Essays on Health Inequality in Low- and Middle-income Countries in Asia

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Doctor of Philosophy

University of York
Economics

April 2020

Abstract

The main objectives of this PhD thesis are to investigate health inequality in low- and middle-income countries in Asia. The thesis consists of four independent chapters. The first two chapters explore the inequality of opportunity in health and the last two chapters evaluate the conditional cash transfer programmes.

Chapter 1 explores the inequality of opportunity in child health in ten developing countries in Asia, i.e. Bangladesh, Nepal, Pakistan, Maldives, India, Cambodia, Myanmar, East Timor, Tajikistan and Kyrgyzstan. We quantify the degree of inequality in child nutritional status between children with advantaged parental socio-economic backgrounds and those with disadvantaged parental socio-economic backgrounds. We then investigate the factors contributing to the observed disparities. The results provide strong evidence that priority should be given to protecting children from marginalised households in order to mitigate the inequality in child health.

Chapter 2 proposes a new approach to measuring the inequality of opportunity with copulas to overcome some of the shortcomings of the existing methods. The proposed approach is applied to the inequality in body mass associated with its intergenerational transmission from parents to children in Indonesia.

Chapter 3 evaluates the conditional cash transfer programme in Indonesia, the *Program Keluarga Harapan* (PKH). We estimate its impacts on the entire distributions of child health and household expenditure. We explore the mechanism by which the PKH affects child nutritional status by decomposing the treatment effect on health into the part that can be explained by the change in household expenditure and the remaining part that is due to behavioural changes. An improvement in height among children aged 24-36 months is observed. Its improvement is explained not by the increase in household expenditure but by the behavioural changes of the beneficiaries.

Chapter 4 re-visits the conditional cash transfer programme in India, the *Janani Suraksha Yojana* (JSY) and examines the existing non-experimental evidence of its impact on maternal healthcare use through the partial identification approach. We find that the average treatment effects estimated in the previous studies are outside of the average treatment effect bounds estimated under weaker but more credible assumptions, thereby suggesting that average treatment effect in previous studies may have been over- or under-estimated due to untenable identification assumptions.

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Part I

Preface

The study into health inequality has played an important role in health economics. In recent decades, many health economics scholars have studied the inequality in health and healthcare service (van Doorslaer et al., 2000, 2004; van Doorslaer and Jones, 2004; van Doorslaer and Koolman, 2004; van Doorslaer et al., 2006; Wagstaff and van Doorslaer, 2000). The majority of this literature focuses on developed countries; research on health inequality in developing countries, where substantial demographic changes and remarkable improvement in living conditions are occurring, has started only recently. This PhD thesis investigates health inequality in low- and middle-income countries in Asia.

Strong economic growth in Asian countries, without doubt, provides wider opportunities for better health. In the case of child health, for example, the number of stunted children in Asia, which was 134.6 million in 2000, declined to 83.6 million in 2017 (UNICEF, 2017). Despite the strong economic growth, its health benefits are not necessarily equally distributed. A large volume of the literature has shown that children whose parents have lower socio-economic backgrounds tend to experience worse health conditions than those with parents who have more advantaged socio-economic backgrounds (Case and Paxson, 2002; Bradley and Corwyn, 2002). Children's health is thus heavily influenced by the characteristics of the families into which they are born, which causes the intergenerational transmission of socio-economic status and health, making it even harder for poor households to find a way to get out of poverty (Case et al., 2002). But for appropriate social policies to ensure reasonable fairness, the economic growth itself brings little benefit to health equity (Marmot et al., 2008). Given that health is one of the most important elements of human capital (Johnson and Schoeni, 2011; Bleakley, 2010), inequality in this regard can be an obstacle for sound and inclusive development. Investigating health inequality and effective policies to mitigate it, should contribute to assisting the sustainable and resilient development of the low- and middle-income countries in the region.

The thesis consists of four independent chapters. The first two chapters explore the inequality of opportunity (IOp) in health. The IOp is an illegitimate form of inequality whereby causes are not attributable to individual choices. In both studies, we regard the environmental circumstances where children grew up as factors beyond children's control and estimate how much of the overall inequality is explained by these circumstances.

Chapter 1 explores the IOp in child malnutrition in ten developing countries in Asia, where a high proportion of children still remain vulnerable to food insecurity. This study takes account of multi-dimensional aspects of household and parental socio-economic status, and partitions children into distinct types through a data-driven clustering method. This is followed by a comparison of the malnutrition rates between types. Next, we decompose the observed between-type disparity in malnutrition rates into the factors that are associated with the observed disparity through a non-linear decomposition method. The results indicate that in all ten countries, significant between-type disparities are found. We find the largest difference in Pakistan as 21.7 percentage points and the smallest difference in Maldives as 5.9 percentage points. In five of the ten countries, the difference in household affluence explains the largest part of the observed between-type disparity. All the results suggest that priority should be given to protecting children from marginalised households in order to mitigate the inequality in child health.

Chapter 2 measures inequality of opportunity using copulas within the framework developed by Roemer (1998). Non-parametric and parametric methods have been widely used to measure IOp, but each of them tends to have a significant technical concern when the sources of inequality are not categorical and multi-dimensional, and when a severe specification error is suspected respectively. Chapter 2 introduces an alternative approach to measuring ex-post IOp using copulas which model various structures of dependence between efforts, factors for which individuals are responsible, and circumstances, factors beyond the individual's control. Semi-parametric estimation of the outcome distribution and flexible modelling of the dependence structure between efforts and circumstances help to overcome the concerns in the existing methods. The method is applied to measuring inequality in the body mass index (BMI), attributable to its intergenerational transmission from parents to their adult offspring in Indonesia. A 21-year gap between the first wave and the last wave of the Indonesian Family Life Survey is exploited to estimate the effect of parental BMI when offspring are below 15 years old on the BMI of grown-up off-

spring. The results show that 16.2 per cent of the overall variance in grown-up children's BMI is associated with intergenerational BMI transmission.

The last two chapters investigate the effectiveness of conditional cash transfer programmes, which are one of the most widely adopted programmes in the world in the last two decades after their success in Latin American countries. *Progresa/Oportunidades* in Mexico and *Bolsa Escola* in Brazil are well known as the most successful programmes. Conditional cash transfer programmes transfer money to low-income households with pregnant mothers and/or children contingent on investments in human capital. The cash transfer combined with multiple conditionalities encourages poor households to invest in long-term human capital development. Conditional cash transfer programmes are justified by social equity concerns as they redistribute resources from richer to poorer households. Recently, conditional cash transfer programmes have been launched in several countries beyond Latin America; Asian countries began to introduce the programmes. We evaluate the impacts of conditional cash transfer programmes conducted in Indonesia and India on health and healthcare use in chapter 3 and chapter 4 respectively.

Chapter 3 investigates the impact of a conditional cash transfer programme in Indonesia, the *Program Keluarga Harapan* (PKH), on the marginal and joint distributions of child nutritional status and household expenditure 26-30 months after its implementation. The cluster-randomised control trial project conducted in Indonesia provides an opportunity to semi-parametrically estimate the causal impacts on the dependence between them. The results show that the PKH increases the higher quantiles of weight-for-age z-score among children aged between 25-36 months. Its improvement is explained not by the rise in the household expenditure due to the PKH but by the change in the association between nutritional status and household expenditure. Furthermore, the PKH strengthens the positive association between nutritional status and household expenditure among them. For the other younger age groups, the improvement in nutritional status is not observed.

Chapter 4 re-estimates the causal impacts of a conditional cash transfer programme in India, the *Janani Suraksha Yojana* (JSY), on maternal and child healthcare use. The main goal is to provide new evidence and assess the validity of the identification assumptions employed in previous studies on the JSY, through the conservative partial identification approach. We find

that the average treatment effects estimated under the conditional independence assumption lie outside the bound of the treatment effects that are estimated under weaker but more credible assumptions, thereby suggesting that the selection bias could not have been fully controlled for by the observable characteristics and that the average treatment effects estimated in the previous studies may have been over- or under-estimated.

Acknowledgements

I wish to thank my supervisors, Professor Andrew M. Jones and Professor Nigel Rice. I am grateful for their close supervision and valuable advice throughout my research life in York. I could not have completed my PhD thesis without their considerable support and assistance. I would like to express my gratitude to my thesis advisory panel (TAP) member, Professor Cheti Nicoletti. My work benefited from her penetrating and thoughtful insights during the TAP meetings.

I gratefully acknowledge helpful comments from participants at seminars, conferences and workshops. In particular, chapter 4 has benefited from constructive discussions made by Dr Laura Anselmi in the 6th European Health Economics Association (EuHEA) PhD Student-Supervisor and Early Career Researcher Conference in 2019. I acknowledge her helpful suggestions on the earlier chapter draft.

I dedicate this thesis to my family and to my friends, who have supported me during my good and bad times. Special thanks go to all of my colleagues in the Health Econometrics and Data Group (HEDG) for their support and suggestions. It has been with my great pleasure to see all of them every Wednesday morning at the Alcuin Bistro for the past four years. I benefited from the open discussions in a friendly atmosphere.

The studies in this thesis use the Demographic Health Surveys (DHS) in chapter 1, the Indonesian Family Life Surveys (IFLS) in chapter 2, the Impact Evaluation of PNPM Generasi Program in chapter 3, the District Level Household and Facility Survey (DLHS) and the National Family Health Survey (NFHS) in chapter 4. I acknowledge the original collectors of the data, the authorised distributors of the data, the survey respondents and the relevant funding agencies. They bear no responsibility for the results and interpretations in each study.

Lastly, my productive life in York was made possible largely through scholarships from the Kikawada foundation (2016-2018) and the Honjo International Scholarship Foundation (2018-2020), and I would like to acknowledge here the generosity of these organisations.

Declaration

I declare that this thesis is a presentation of original work and I am the sole author. This work has not previously been presented for an award at this, or any other, University. All sources are acknowledged as References.

I declare that this thesis has been written by myself as part of my PhD research course in Economics at the University of York. I wish to confirm that there are no known conflicts of interest associated with the studies in this thesis and there has been no financial support for studies that could have influenced their results and interpretations.

Chapter 1 is published in *Economics & Human Biology*, 2019, volume 35 pp.144-161.

A previous version of chapter 2 was presented at the European Health Economics Association (EuHEA) Conference held in Maastricht, June 2018 and the Health Econometrics and Data Group (HEDG) seminar at the University of York, March 2018.

Chapter 3 is published in *Journal of Human Capital*, 2020, volume 14(1) pp.122-164. A previous version of chapter 3 was presented at the 8th Italian Congress of Econometrics and Empirical Econometrics (ICEEE) held in Lecce, January 2019, the 24th Spring Meeting of Young Economists (SMYE) held in Brussels, April 2019, the World Congress of the International Health Economics Association (iHEA) held in Basel, July 2019, the HEDG seminar at the University of York, October 2018, the seminar at the Waseda University, June 2019.

Chapter 4 was presented at the 6th European Health Economics Association (EuHEA) PhD Student-Supervisor and Early Career Researcher Conference held in Porto, July 2019 and the 2nd Asian Workshop of Econometrics and Health Economics (AWEHE) held in Hokkaido, December 2019.

Part II

Chapter 1

Ex-ante Inequality of Opportunity in Child Malnutrition: New Evidence from Ten Developing Countries in Asia

Abstract

This study explores the inequality of opportunity in child malnutrition in ten developing countries in Asia, where a high proportion of children still remain vulnerable to food insecurity. This study takes account of multidimensional aspects of household and parental socio-economic status, and partitions children into distinct types through a data-driven clustering method. This is followed by a comparison of the malnutrition rates between types. Next, we decompose the observed disparity into the factors that are associated with the between-type disparity in malnutrition rates through a non-linear decomposition method. The results indicate that in all ten countries, significant between-type disparities are found. We find the largest difference in Pakistan as 21.7 percentage points and the smallest difference in Maldives as 5.9 percentage points. In five of the ten countries, the difference in household affluence explains the largest part of the observed between-type disparity. All the results suggest that priority should be given to protecting children from marginalised households in order to mitigate the inequality in child health.

JEL code: D63, I12, I14, I15

Keywords: Inequality of opportunity; Clustering; Malnutrition; Decomposition; Asia

1.1 Introduction

The Sustainable Development Goals (SDGs), adopted by all United Nations Member States in 2015, call for action to end all forms of malnutrition among children under five years of age and to protect them from preventable deaths (United Nations, 2015). Malnutrition triggers a potentially lethal cycle of worsening illness and further deterioration in nutritional status. Numerous studies have revealed that malnutrition in the early stages of life increases the risk of poor child development, resulting in a greater likelihood of death from infectious diseases in childhood (Pelletier et al., 1995; Caulfield et al., 2004). According to UNICEF (2017), nearly half of all deaths in children under five are attributable to malnutrition. Moreover, the negative impact of restricted development due to malnutrition in childhood is persistent and its long-term effects are not limited to the health domain.[1] Ill health in childhood has persistent adverse impacts on the development of both cognitive and non-cognitive skills (Sánchez, 2017; Walker et al., 2011; Grantham-McGregor et al., 2007; Victora et al., 2008), motor development, educational outcomes (Glewwe et al., 2001) and future earnings (Smith, 2009; Johnson and Schoeni, 2011). Not taking action at the very early stages of life will have lifelong, irreversible adverse consequences for the future health of children, and even for the subsequent generation and the welfare of societies (Walker et al., 2011).

The stunting rate is gradually declining globally; in 2016, 22.9 per cent of children under the age of five had stunted growth, compared with 32.7 per cent in 2000 (UNICEF, 2017). Despite this enormous progress, more than six million children still die before their fifth birthday every year. Moreover, there is a large regional disparity in the prevalence of malnutrition. In 2016, two out of five stunted children in the world lived in Southern Asia, where the prevalence rate of stunting exceeds 30 per cent (UNICEF, 2017). Certainly, developing countries in Asia are experiencing strong economic growth, which has led to a number of children under five being able to escape the misery caused by poverty and malnutrition. However, the benefits of economic growth have not necessarily been enjoyed equally by all people within a country, and therefore health inequality has not disappeared. The malnutrition rate is persistently higher among poor children in rural areas. Numerous studies have revealed significant relationships between parental socio-economic status and child health. For example, children whose parents come from lower socio-economic

[1] See for example, Strauss and Thomas (2007); Almond and Mazumder (2013); Almond and Currie (2011); Case et al. (2005); Alderman et al. (2006); Smith et al. (2012); Blackwell et al. (2001); Portrait et al. (2017); Victora et al. (2008).

backgrounds tend to have worse health compared with those parents who have higher socio-economic backgrounds (Case et al., 2002; Currie and Moretti, 2003; Currie and Vogl, 2013).

Achieving equity and equality in child health would be an essential public policy objective to ensure sustainable and resilient economic growth. Addressing health inequality among children is as important, or potentially more important, as mitigating it among adults. First, in contrast to adults, health disparity among children cannot generally be attributed to their own lifestyles for which they should be held responsible. Clearly, children cannot choose their parents and they have no control over the environments in which they grow up. In this sense, health disparity among children may well be considered inequitable. Second, the first few years of life are of huge importance because vital development occurs in all domains at this stage, including the structural and functional capacity development of the brain (Grantham-McGregor et al., 2007). Mitigating health inequality by improving the nutritional status of impoverished children would help to bring about long-term benefits for the children themselves, as well as for society in general.

In this paper, we examine the existence of inequality of opportunity in child health in ten developing countries in Asia; namely Bangladesh, Nepal, Pakistan, Maldives, India, Cambodia, Myanmar, East Timor, Tajikistan and Kyrgyzstan. We first partition the children in each country into a few *types*, namely, a set of people with the same circumstances in which children grow up (Roemer, 1998). Then, we compare the nutritional status across different *types*. In particular, we test for the existence of inequality of opportunity by comparing the nutritional status between the least advantaged type and the most advantaged type. If a significant difference is detected, this is a sign of the existence of inequality of opportunity (Peragine and Serlenga, 2007; Lefranc et al., 2008, 2009; Aaberge et al., 2011; Lefranc and Trannoy, 2017; Ramos and Van de gaer, 2016).

Next, in order to better understand the factors that are related to health inequality in child development, we decompose the observed between-type difference in health status into the factors that are associated with the observed difference by the non-linear decomposition approach developed by Fairlie (2005). The decomposition analysis would be helpful for policymakers who seek to design effective policies to mitigate health inequality. The results show that significant between-type disparities are found in all ten countries. The largest difference is observed in Pakistan and the smallest difference is found in Maldives. In five of the ten countries, the dif-

ference in household affluence explains the largest part of the observed between-type disparity. Exploiting the Demographic and Health Survey (DHS) in which data collection procedures and interviewer training are standardised across countries, this paper ensures the comparability of the results across different countries.

1.2 Related literature

The existing literature on health inequality can be roughly categorised into two types (Wagstaff and van Doorslaer, 2004). The first type explores how health differs across various socio-economic dimensions. This variation of the literature typically analyses the difference in health across different segments of the population with regression-based approaches (Kamal, 2011; Pathak and Singh, 2011; Van de Poel and Speybroeck, 2009) or explores income-related or wealth-related health inequality with the concentration index (Wagstaff et al., 1991). Socio-economic health inequality rests on the normative idea that everyone should have an equal opportunity to enjoy better health regardless of their socio-economic status. The majority of the existing literature on child health inequality focuses mainly on a single country (e.g. Zere and McIntyre, 2003; Subramanyam et al., 2010; Hong and Mishra, 2006; Hong et al., 2006; Hong, 2007; Thang and Popkin, 2003). On both a regional and global level, for example, Wagstaff and Watanabe (2000); Houweling et al. (2003) and Van de Poel et al. (2008) analyse the socio-economic inequality in child nutritional status and Van de Poel et al. (2007) and Smith et al. (2005) analyse the urban-rural inequality across various developing countries.

The second type of literature focuses on inequality in health itself, rather than on the correlations between health and socio-economic status. This variation of the literature analyses the distribution of health with the techniques commonly applied to income inequality research (Le Grand, 1987; Gakidou et al., 2000). For example, Pradhan et al. (2003) explore global inequality in child nutritional status and decompose it into within- and between-country inequality. Castillo-Salgado et al. (2001) apply the Gini index to measuring infant mortality rates across South American countries and Fang et al. (2010) analyse the regional differences in inequality in health in China. This type of literature usually does not clearly distinguish between legitimate and illegitimate inequality.

The approach utilised by this paper does not belong to either strand of research in the strictest

sense; rather, it can be regarded as a mixture of both or an extension of the first type. The inequality of opportunity is an illegitimate inequality whereby causes are not attributable to individual choices (Roemer, 1998). Equality is achieved when the same opportunities to secure better health are shared by all children, irrespective of the circumstances in which they grow up. This goal is often referred to as the ex-ante approach, which promotes equality of outcomes among those facing the same circumstances by making their average outcomes as equal as possible (Pignataro, 2012; Ferreira and Gignoux, 2011). Inequality of opportunity is a more general concept of inequality than income- or wealth-related health inequality in the sense that household income or wealth is just a subset of unjustifiable sources of inequality (Fleurbaey and Schokkaert, 2012; Schokkaert, 2015). In fact, in the literature on income-related health inequality, other parental and household factors, such as parental educational backgrounds, tend not to be recognised as illegitimate inequality sources unless they are captured by income.

In contrast to the existing research, this paper takes account of multidimensional circumstances– a set of factors over which individuals have no control. In the real world, unjustifiable sources of health inequality are wide-ranging (Cutler et al., 2008) and income and wealth are just subsets of circumstance factors. From children's perspective, parental educational backgrounds and location of domicile may well be regarded as important factors that can be sources of unfair health inequality. Consideration of the multidimensionality of circumstances allows us to explore the in-depth health inequality which could not be captured by just a single index such as income or wealth. Taking account of various aspects of inequality source is especially important as there is a general consensus that composite factors, including affluence, education and occupation, together represent various aspects of socio-economic status better than any of these elements alone does (White, 1982).

1.3 Data

1.3.1 The Demographic and Health Survey

The Demographic and Health Survey (DHS) project is an ongoing collaboration between the United States Agency for International Development and country-specific agencies. They conduct nationally representative household sample surveys covering a range of population health indicators in low- and middle-income countries (Corsi et al., 2012). The DHS data has been

Table 1.1: Country and year

Country	Region	Year	N	Malnutrition rate
Bangladesh	South	2014	6,988	0.483
Nepal	South	2016	3,759	0.414
Pakistan	South	2012/13	4,807	0.536
Maldives	South	2009	3,521	0.323
India	South	2015/16	40,200	0.548
Cambodia	South East	2014	4,332	0.431
Myanmar	South East	2015/16	5,616	0.361
East Timor	South East	2009/10	10,968	0.718
Tajikistan	Central	2012	8,800	0.355
Kyrgyzstan	Central	2012	5,483	0.212

gathered on the basis of comparable nationally representative household surveys that have been conducted in more than 85 countries worldwide since 1984. The DHS respondents are selected using a two-stage sampling process stratified by urban and rural location. Populations selected in the DHS are representative of the entire country or region of interest. Key advantages of the DHS include the national coverage and high participation rates (response rates typically exceed 90 per cent). In addition, the DHS questionnaire has been standardised and pre-tested to ensure comparability across populations and over time. Standard data collection procedures and interviewer training in the DHS ensure that the data is both reliable and comparable.

This study uses the latest DHS conducted in ten Asian countries listed in Table 1.1.[2] In each country, selected households are visited by a trained interviewer who conducts a brief household interview, completes a household roster and identifies eligible women (aged 15-49) for an individual interview. In this study, we use the data concerning children aged under five.

1.3.2 Variables

Nutritional status

We measure child nutritional status by a height-for-age z-score (HAZ score), a weight-for-age z-score (WAZ score) and a weight-for-height z-score (WHZ score) of children under five. From the actual measurements of the heights and weights of the children, the HAZ, WAZ and WHZ scores were calculated. The HAZ/WAZ scores measure the difference between the height/weight of an individual and the medial value of the "healthy" reference population for the same sex

[2] Afghanistan, Kazakhstan, Indonesia, Philippines, Sri Lanka, Thailand, Uzbekistan and Vietnam are excluded from this study because their DHS data does not contain recent child nutritional information that has been surveyed post-2005.

and age, divided by the standard deviation of the reference population (O'Donnell et al., 2008).[3] The HAZ score measures the long-term cumulative nutritional conditions and a low HAZ score is associated with chronic under-nutrition caused by poor nutrition (Walker et al., 2007). The WAZ score, on the other hand, measures the short-term nutritional conditions. A low WAZ score is associated with the condition of being underweight. Finally, the WHZ score measures current nutritional status and is used to screen children at risk and to measure short-term changes in nutritional status. A low WHZ score is associated with the condition of wasting. Low HAZ, WAZ, and WHZ scores are mainly caused by poor diet and often compounded by recurrent infectious diseases including diarrhoea. The risk of death increases with descending scores (Black et al., 2008). For details of the computations of these scores, see World Health Organization (2006). A HAZ score, WAZ score and WHZ score below -2, i.e. two standard deviations below the international reference median, are often indicators of being stunted, underweight and wasting, respectively. In this study, children with malnutrition are defined as those who have any of these three malnutrition conditions. Of the ten countries analysed in this study, East Timor shows the highest malnutrition rate, followed by India and Pakistan. Kyrgyzstan shows the lowest malnutrition rate.

Circumstance factors

In this study, we consider the nine circumstance factors that are assumed to capture multidimensional circumstances, on the basis of which we partition the observations into a few clusters. They are (I) Maternal health, (II) Maternal education, (III) Paternal education, (IV) Demography, (V) Housing condition, (VI) Affluence, (VII) Sanitation, (VIII) Parental occupation, and (IX) Media exposure (Table 1.2). Certainly, these nine circumstance factors are just a subset of conceivable circumstances, and each country typically may have a country-specific circumstance factor, such as the Caste system in India. However, in this research, we consider the circumstance variables that are surveyed in all ten countries. Considering only the common circumstance variables allows us to compare the results across countries. First, as a measurement of maternal health, following Smith et al. (2005), we use maternal body mass index (BMI).[4] BMI

[3] In the standard that the World Health Organization issued in 2006, the reference population is healthy and well-nourished children in the United States. This standard captures the growth and development process of children from widely diverse ethnic and cultural backgrounds (O'Donnell et al., 2008). Their rationales are discussed in de Onis and Lobstein (2010).

[4] Certainly, paternal BMI could also be an important circumstance factor. In most countries, however, information about heights and weights of fathers is not surveyed in the DHS dataset. In this paper we consider only maternal BMI to achieve international comparability of the results.

Table 1.2: Circumstance factors and their composition elements

	Factor	Composition element
(I)	Maternal health	Maternal body mass index (kg/m^2)
(II)	Maternal education	Maternal education years
(III)	Paternal education	Paternal education years
(IV)	Demography	Maternal age at birth, Family size, Number of children, Living in urban cities
(V)	Housing condition	Natural/rudimentary/finished floors, Natural/rudimentary/finished walls, Natural/rudimentary/finished roofs
(VI)	Affluence	Radio, TV, Refrigerator, Bicycle, Motorcycle, Car, Mobile phone, Electricity, Safe fuel
(VII)	Sanitation	Piped water, Flushing toilet, Water treatment for drinking
(VIII)	Parental occupation	Farmer, Professional worker, Non-manual worker, Manual worker
(IX)	Media exposure	Frequency of TV watching, Frequency of radio listening, Frequency of newspaper reading

is defined as an individual's weight divided by their height squared and then expressed in units of kg/m^2. Information on heights and weights in the DHS was based on actual measurements and therefore was less likely to be subject to measurement errors such as under-reporting of weight and over-reporting of height (Gorber et al., 2007). The effects of maternal health on child health are reported in both developed and developing countries (Bhalotra and Rawlings, 2013; Özaltin et al., 2010; Ahsan and Maharaj, 2018; Ferreira et al., 2009; Li et al., 2009). For example, malnourished women are more likely to deliver smaller babies and less successful at breastfeeding their children (Beard, 2001; Gillespie, 1997).

Second, we use the maternal and paternal educational backgrounds, which are measured by the number of years of education completed by a mother and a father. This is motivated by a number of empirical research papers on the relationship between parental educational backgrounds and child development (Chen and Li, 2009; Currie and Moretti, 2003; Ahsan and Maharaj, 2018; Appoh and Krekling, 2005). For example, it is argued that more educated mothers are capable of better processing information, using effectively healthcare facilities and communicating with healthcare providers for children (Smith et al., 2005; Levin et al., 2000).

Next, for the circumstance factors (IV)-(IX), we create continuous summary indices from continuous and binary variables by the principal component analysis.[5] Individual covariates in each

[5]Certainly the principal component analysis is designed primarily for continuous variables, but it also works with binary variables as well, although the derived score may not be as numerically meaningful as the one derived from continuous variables. In this study, the score is used only in the clustering process and the difficulty of

factor are summarised in Table 1.2. We create a demographic circumstance index from maternal age at birth, family size, number of children in the household, location of living (urban/rural). Family size affects child health not only because there are more mouths to feed for a given income but also because of the likelihood of crowding and the spread of infection (Hatton et al., 2018). Becker (1960), Becker and Lewis (1973) and Becker and Tomes (1986) formulated the link between family size and child outcomes (the quantity and quality trade-offs). The detrimental effects of family size on child health are reported in several countries (Hatton et al., 2018; Liu, 2014; Alderman, 1990; Glick et al., 2007; Blake, 1981). With regard to location of living, urban children generally have a better nutritional status than those in rural areas (Van de Poel et al., 2007).

As a measurement of household affluence, we create its index from the following nine asset-ownership variables (Filmer and Pritchett, 2001; Rutstein and Staveteig, 2014; Montgomery et al., 2000): (i) owning a radio, (ii) owning a television, (iii) having a refrigerator, (iv) having a bicycle, (v) having a motorcycle (vi) having a car, (vii) having a mobile phone, (viii) access to electricity, and (ix) the availability of safe cooking fuel (i.e. electricity, LPG, natural gas). The higher value of affluence index is associated with the better-off living standards. Household affluence is one of the important socio-economic factors that create inequality in health (Chalasani, 2012; Huda et al., 2017; Aizawa and Helble, 2017).

The index for housing characteristics is derived from the materials of floors, walls, roofs, and number of rooms in a household. The housing condition is closely related to child health. Dilapidated, poor and crowded housing has been regarded as one of the important causes of child illness (Marmot, 1999; Bradley et al., 2001). Household sanitary condition is an equally important factor for child health. As a measurement of household sanitary condition levels, we create its index from the following dummy variables: (i) access to drinking water, (ii) having a flushing toilet in a house, and (iii) whether water has a treatment for drinking. It is known that sanitary conditions have a substantial influence on health (Ahsan et al., 2017; Caulfield et al., 2004; Checkley et al., 2004). For example, poor water condition is known as a leading cause of diarrhoea (Konteh, 2009).

numerical interpretation is of less concern herein.

In respect of parental occupation, four occupation types for each mother and father are used in constructing its index: (i) professional/technical/managerial work, (ii) clerical, sales or employment in the service sector, (iii) farming[6], and (iv) manual work.[7] Not currently participating in the labour force is a benchmark category.[8] The association between parental employment status and child health is reported by Ruhm (2004); Blau et al. (1996) and McGuire and Popkin (1990).

The last category, media exposure, includes the mother's frequency of watching TV, listening to the radio and reading newspapers. Frequencies of exposure to the respective media are divided into three levels: (i) not at all/less than once a week, (ii) at least once a week, and (iii) almost every day (benchmark category). As the mass media often disseminate information that could lead to children receiving better nutrition or avoiding an illness or disease, the degree of maternal media exposure is an important factor to take into consideration (Olken, 2009). The descriptive statistics of nutritional status and circumstance variables are shown in Table 1.A.1 in the Appendix.

1.4 Methods

1.4.1 Defining types

When we define *types*, considering multidimensional circumstance factors is important to reflect the various circumstances that children may face. However, defining the *types* is not always easy in cases where we have multidimensional circumstance variables. As we consider more and more circumstance factors, we are likely to suffer the curse of dimensionality (Ferreira and Gignoux, 2011). The precision of the analysis deteriorates as the number of circumstance factors increases, because the exponentially growing number of *types* often leads to very few observations in each *type*. The situation becomes even worse when circumstance variables are continuous; here, discrete *types* can still be defined, but the choice of cut-off point to make a continuous variable discrete tends to be arbitrary. Li Donni et al. (2015) proposed a way of defining endogenously finite types with a latent class approach to deal with the multidimensionality of circumstances, and O'Neill et al. (1999) proposed a method of dealing with the continuity of circumstance variables with kernel estimation. In this paper, we deal with the multidimensionality of circumstances using the clustering approach, which is a data-driven technique of clustering to find distinctive

[6]Either as an employee or as an owner.
[7]Including both skilled and non-skilled manual work.
[8]This includes those not seeking employment, such as homemakers.

subgroups among the observations. Clustering is one of the unsupervised machine learning approaches that partition the observations into a pre-specified number of non-overlapping clusters so that the observations within each group become similar to each other, while at the same time the observations in different groups differ from one another. We use the K-means clustering method, which is one of the most popular clustering methods due to its tractable algorithm and high computational speed.

K-means clustering requires us to firstly specify a desired number of clusters K. Then, the K-means algorithm will assign each observation to exactly one of the K clusters so that the within-cluster variation, summed over all clusters, becomes as small as possible. In the K-means clustering, the within-cluster variation for the Kth cluster is measured by the sum of all of the pairwise squared Euclidean distances between the observations in the Kth cluster, divided by the total number of observations in the Kth cluster. Before applying the K-means clustering, we normalise the circumstance factors so that they will range from 0 to 1, which allows us to calculate the Euclidean distances between the observations.

Determining the optimal number of clusters, K^*, in a data set is a fundamental issue in clustering. If the optimal number of clusters were known a priori, we would be able to use that number, but such information is seldom available in advance. In most cases, the selection of a number of clusters tends to be a later consideration, and so it is important to try several numbers of clusters and compare their performances measured by some objective criteria. One of the methods commonly used in the literature to choose the optimal number of clusters is the elbow method. This method first calculates the total within-cluster sum of square variation (WSS) and plots the total WSS as a function of the number of clusters. In the plot, we should choose a number of clusters so that adding another cluster does not improve the total WSS very much. In the plotted WSS, the location of a bend/knee in the plot is generally considered to be an indicator of the appropriate number of clusters. However, spotting a bend/knee in the plot usually requires our subjective judgement and hence the choice of the optimal number of clusters is often non-unanimous. Different people may choose a different number of clusters from the same plot. Therefore, we consider an alternative approach called the average silhouette method (Kaufman and Rousseeuw, 1990).

The average silhouette method computes the average silhouette of observations for different numbers of K. The average silhouette determines how well each observation lies within its cluster, so a high average silhouette indicates a good clustering. The optimal number of clusters K^* is the one that maximises the average silhouette over a range of the cluster numbers K (Kaufman and Rousseeuw, 1990). When we plot the curve of the average silhouette against the number of clusters K, the location of the maximum is considered to be the optimal number of clusters (Kaufman and Rousseeuw, 1990).

After the K-means algorithm assigns each observation to one of the K^* clusters, we compare the child malnutrition rates between clusters. If we find a significant difference across clusters, it is a sign of the existence of inequality in child nutritional status that is associated with the circumstances. We define the most advantaged type as the better-off class with the lowest malnutrition rate, and the least advantaged type as the worse-off class with the highest malnutrition rate (Roemer, 1998).[9]

1.4.2 Non-linear Oaxaca-Blinder decomposition

Overall decomposition

Next, we decompose the observed between-type differences regarding the malnutrition rates between the least advantaged type and the most advantaged type to better understand what aspects of circumstances are strongly associated with the observed difference in malnutrition rate. We decompose the between-type difference into the part which is attributable to the difference in the distribution of circumstance variables (the explained part) and the part which is not (the unexplained part).

We employ the non-linear decomposition method (Fairlie, 1999, 2005), which is an extension of the linear Oaxaca-Blinder decomposition (Oaxaca, 1973; Blinder, 1973) to the parametric non-linear case. We use the following notations and the explanations of the decomposition method follows Aizawa (2018a). $g = \{L, M\}$ represents the group to which individuals belong. In this study, $g = L$ is the least advantaged type, while $g = M$ is the most advantaged type. N^g denotes the observation size of the group g. Y denotes a binary child malnutrition status

[9]It is worth noting that the most advantaged type does not necessarily have better circumstance variables than the other types because in this paper the distinction between the most advantaged type and the least advantaged type is based on the malnutrition rate of each type, not on the circumstance variables themselves.

variable. Y becomes one if a child has any of the malnutrition conditions, and $X = \{X_1, ..., X_k\}$ is a vector of individual circumstance variables that are used for the clustering. As Y is a binary variable, $\overline{Y}^g = \frac{1}{N^g} \sum_{i=1}^{N^g} Y_i$ denotes the proportion of children in group g whose value of Y equals 1.

The predicted probability of individual i in group g is expressed as

$$\widehat{Y}_i^g = F(X_i^g \widehat{\beta}^g), g = \{L, M\} \tag{1.1}$$

where $F(.)$ denotes a logistic distribution and $\widehat{\beta}^g$ is a vector of coefficients which are estimated separately for group g. In the spirit of the Oaxaca-Blinder decomposition (Oaxaca, 1973; Blinder, 1973), we decompose the between-type difference in the average predicted probabilities as follows:

$$\overline{Y}^L - \overline{Y}^M = \underbrace{\sum_{i=1}^{N^L} \frac{F(X_i^L \widehat{\beta}^L)}{N^L} - \sum_{i=1}^{N^M} \frac{F(X_i^M \widehat{\beta}^M)}{N^M}}_{Observed\ difference} \tag{1.2}$$

$$= \underbrace{\left\{ \sum_{i=1}^{N^L} \frac{F(X_i^L \widehat{\beta}^L)}{N^L} - \sum_{i=1}^{N^M} \frac{F(X_i^M \widehat{\beta}^L)}{N^M} \right\}}_{Explained\ part} + \underbrace{\left\{ \sum_{i=1}^{N^M} \frac{F(X_i^M \widehat{\beta}^L)}{N^M} - \sum_{i=1}^{N^M} \frac{F(X_i^M \widehat{\beta}^M)}{N^M} \right\}}_{Unexplanined\ part} \tag{1.3}$$

where $\sum_{i=1}^{N^M} \frac{F(X_i^M \widehat{\beta}^L)}{N^M}$ is an average of counterfactual predicted probability that would be observed if the children in the most advantaged type had the coefficient vector that the least advantaged type has. The first bracket in equation (1.3) relates to the part of the gap which can be due to the between-type differences in the distribution of X. We call this the explained part. Another interpretation of the explained part is that it shows how much we could make the observed difference smaller, if we were able to nullify the between-type difference in the distributions of circumstance variables. The second bracket represents the term due to the between-type differences in the function determining levels of Y, which we call the unexplained part. The unexplained part is the remaining part of the observed difference that cannot be explained by the between-type difference in the distribution of X. The unexplained part reflects the difference due to not only parental efforts but also circumstance factors that are not considered in this study.

Basically, the explained part is the average differences in the predicted probabilities of the least advantaged type and the most advantaged type samples with the same coefficient vector. However, the decomposition in equation (1.3) is dependent on the choice of reference group (Oaxaca

and Ransom, 1994). Namely, equation (1.3) is based on the reference coefficient vector, $\widehat{\beta}^L$, and the decomposition result varies if we use $\widehat{\beta}^M$ as a reference coefficient vector instead. To deal with this problem, we use the coefficients estimated with the pooled sample ($\widehat{\beta}^P$) as the reference coefficient vector (Fairlie, 2005). So equation (1.2) can be rewritten as

$$\begin{aligned}\overline{Y}^L - \overline{Y}^M &= \underbrace{\Big\{\sum_{i=1}^{N^L}\frac{F(X_i^L\widehat{\beta}^P)}{N^L} - \sum_{i=1}^{N^M}\frac{F(X_i^M\widehat{\beta}^P)}{N^M}\Big\}}_{Explained\,part} + \\ &\underbrace{\Big\{\sum_{i=1}^{N^L}\frac{F(X_i^L\widehat{\beta}^L)}{N^L} - \sum_{i=1}^{N^L}\frac{F(X_i^L\widehat{\beta}^P)}{N^L} + \sum_{i=1}^{N^M}\frac{F(X_i^M\widehat{\beta}^P)}{N^M} - \sum_{i=1}^{N^M}\frac{F(X_i^M\widehat{\beta}^M)}{N^M}\Big\}}_{Unexplanined\,part} \quad (1.4)\end{aligned}$$

The first bracket in equation (1.4) is interpreted as the explained part, while the second bracket is interpreted as the unexplained part.

Detailed decomposition

The next step is to further decompose the explained part into its contributions made by individual covariates. Identifying the contributing factors would help policymakers understand the underlying mechanism that drives the disparity between types. However, in contrast to the linear Oaxaca-Blinder decomposition method, the procedure of the detailed decomposition of the nonlinear model is not simple. The approach of sequentially switching the covariate distributions is proposed by Fairlie (2005).

The fundamental idea of the detailed decomposition is to first find one-to-one matching of the observations of each type and sequentially switch each marginal distribution of X. When the observation sizes of each type are identical ($N^M = N^L$), finding one-to-one matching pairs is straightforward. However, when the sample sizes are different, we cannot find one-to-one matched pairs, and in general K-means clustering generally does not partition observations into equal-sized clusters. To deal with such a common situation, the following algorithm is proposed (Fairlie, 2005).

First, we estimate the coefficients using the pooled samples ($\widehat{\beta}^P$) and calculate predicted probabilities for each population, namely $\widehat{Y}_i^g = F(X_i^g\widehat{\beta}^P)$. Next, we draw the random samples with size N^L from the observations of the most advantaged type with a replacement. Then, each observation in the least and most advantaged types is separately ranked by the predicted probabilities and matched by their respective rankings. This procedure allows us to find a one-to-one pair of observations from each type. The contribution made by each covariate is then calculated as a marginal contribution made by a respective covariate to the explained disparity. The con-

tribution made by each covariate to the disparity is equal to the change in the average predicted probability that is observed when we replace the covariate distribution of the least advantaged type with that of the most advantaged type while holding the distribution of the other variables constant.

For example, the marginal contributions made by X_1 and X_2 to the explained part are expressed by

$$\Delta_{X_1} = \sum_{i=1}^{N^L} \frac{F(\widehat{\beta}_0^P + X_{1i}^L \widehat{\beta}_1^P + X_{2i}^L \widehat{\beta}_2^P +, ..., X_{ki}^L \widehat{\beta}_k^P)}{N^L} - \sum_{i=1}^{N^L} \frac{F(\widehat{\beta}_0^P + \widetilde{X}_{1i}^M \widehat{\beta}_1^P + X_{2i}^L \widehat{\beta}_2^P +, ..., X_{ki}^L \widehat{\beta}_k^P)}{N^L} \quad (1.5)$$

$$\Delta_{X_2} = \sum_{i=1}^{N^L} \frac{F(\widehat{\beta}_0^P + \widetilde{X}_{1i}^M \widehat{\beta}_1^P + X_{2i}^L \widehat{\beta}_2^P +, ..., X_{ki}^L \widehat{\beta}_k^P)}{N^L} - \sum_{i=1}^{N^L} \frac{F(\widehat{\beta}_0^P + \widetilde{X}_{1i}^M \widehat{\beta}_1^P + \widetilde{X}_{2i}^M \widehat{\beta}_2^P +, ..., X_{ki}^L \widehat{\beta}_k^P)}{N^L}, \quad (1.6)$$

where $\widetilde{X}_i^M = \{\widetilde{X}_{1i}^M, ..., \widetilde{X}_{ki}^M\}$ is a vector of covariates of the drawn sample of the most advantaged type. Similarly, we can sequentially estimate the marginal contributions for every covariate. Although the sum of contributions of each covariate equals the explained part, the individual contributions can vary depending on the order of the switching procedure, which is widely known as the path dependency problem. To deal with this problem, we randomise the order of the covariate switching in each replication, which approximates average results over all possible orderings. We implement 5,000 decompositions for each country.

1.5 Results

1.5.1 Clustering

First, we choose the optimal number of clusters, K^*, for each country. Figure 1.1 plots the average silhouette width against the number of clusters. The number of clusters which maximises the average silhouette is the optimal number of clusters. The average silhouette width method chooses two clusters for Bangladesh, Nepal, Pakistan, India, Cambodia, East Timor and Tajikistan. This method chooses three clusters for Maldives, four clusters for Myanmar, and six clusters for Kyrgyzstan.[10] Figure 1.2 illustrates the mean values of nine circumstance factors for each cluster in the respective countries, which reveals that K-means clustering succeeds in partitioning the observations into clusters with a distinctive difference in the characteristic of circumstance factors. For example, the clusters in Bangladesh, Pakistan, India, Cambodia,

[10] As a robustness check, we repeated the implementation of the K-means algorithm with different initial cluster assignments, and obtained very stable results.

Figure 1.1: Average silhouette width plot

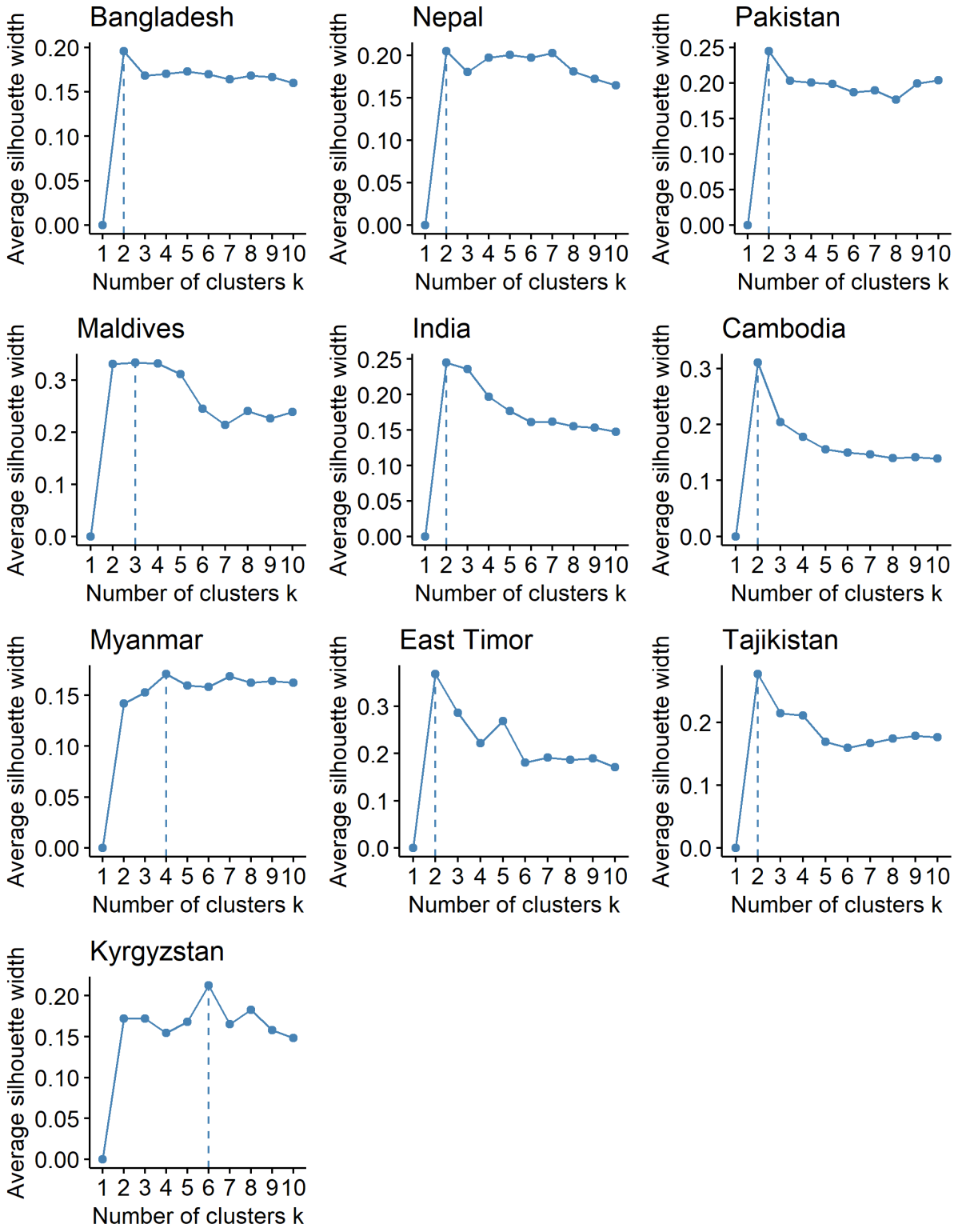


Table 1.3: Malnutrition rates and between-type difference

	Least advantaged type			Most advantaged type			Difference
	Cluster	N	Percentage	Cluster	N	Percentage	Percentage
Bangladesh	2	3665	56.7	1	3323	39.0	17.7
Nepal	1	2172	44.6	2	1587	37.1	7.5
Pakistan	2	2657	63.3	1	2150	41.6	21.7
Maldives	3	960	35.4	1	1361	29.5	5.9
India	2	19782	64.8	1	20418	45.1	19.6
Cambodia	2	2077	48.9	1	2255	37.7	11.2
Myanmar	1	1525	42.6	3	763	22.5	20.1
East Timor	2	6708	75.6	1	4260	65.7	9.9
Tajikistan	1	4663	38.5	2	4137	32.2	6.3
Kyrgyzstan	4	475	26.5	2	737	14.7	11.9

Myanmar and East Timor have large between-cluster differences of affluence. In Cambodia and East Timor, the first cluster has larger mean values for all the circumstance factors than the second cluster. Figure 1.3 illustrates the malnutrition rates with their 95 per cent level confidence intervals across clusters in each country. Comparing the malnutrition rates for each cluster in each country, we define the most advantaged clusters and the least advantaged clusters. Table 1.3 shows the pairs of these two clusters and their malnutrition rates. We find the largest difference in the rate in Pakistan as 21.7 percentage points, and the smallest difference in Maldives as 5.9 percentage points. The descriptive statistics stratified by the types are provided in Tables 1.A.3 and 1.A.4 in the Appendix. In the following decomposition analysis, we explore the factors that are strongly associated with these observed between-type disparities in the malnutrition rates.

1.5.2 Decomposition results

In this subsection, we examine the decomposition results for each country one by one. To facilitate the interpretation of the detailed decomposition results, the results are aggregated across the category which was used in the K-means clustering (Table 1.2). Full results without this aggregation are provided in Table 1.A.2 in the Appendix.

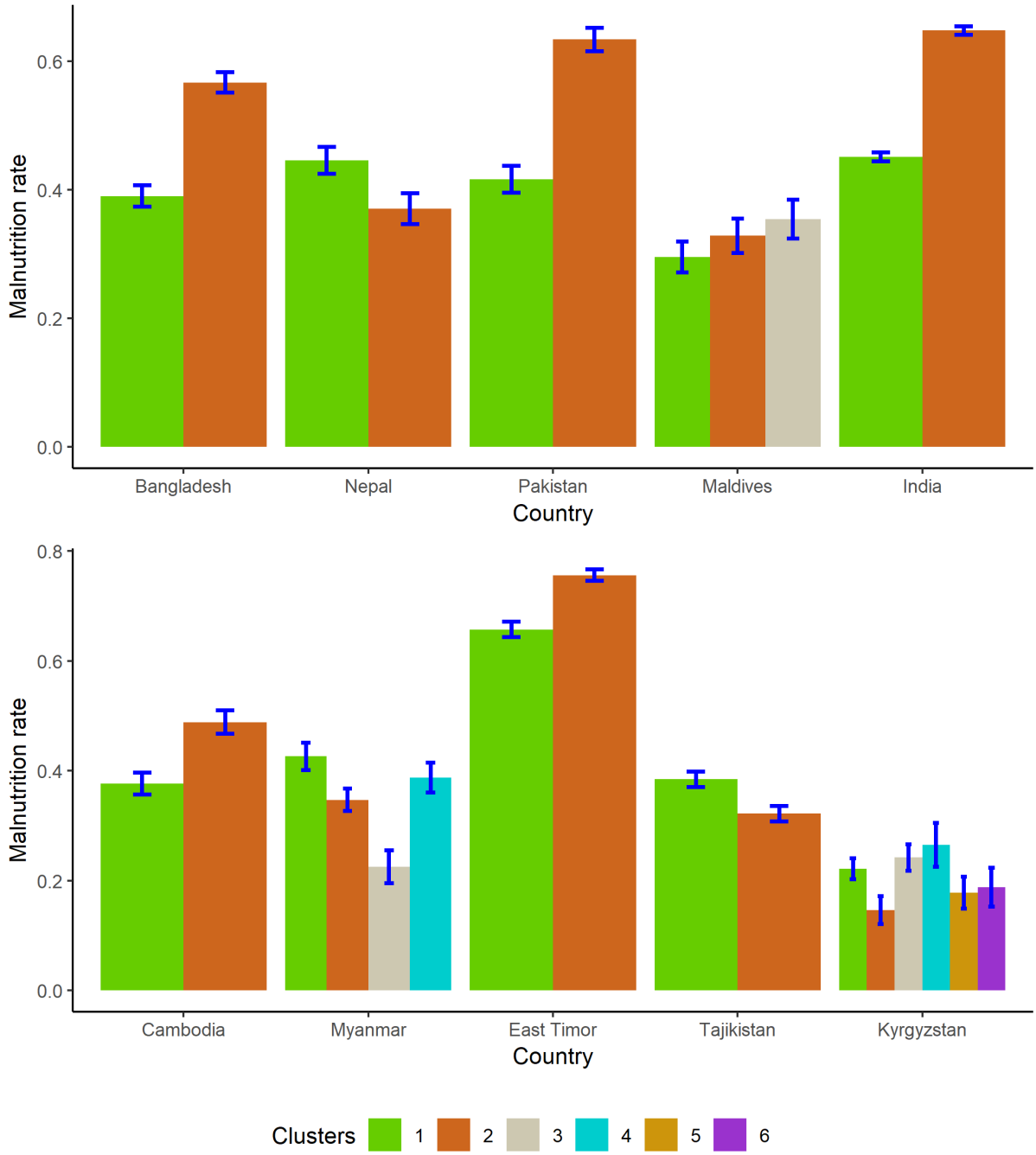
Bangladesh, Nepal, Pakistan and Maldives

Table 1.4 shows the decomposition results for Bangladesh, Nepal, Pakistan and Maldives. First, in Bangladesh, the malnutrition rate of the least advantaged type is 56.7 per cent and that of the most advantaged type is 39.0 per cent. The between-type difference in the malnutrition rate is 17.7 percentage points ($p < 0.01$). When we decompose this observed difference into the explained part and the unexplained part, we find that the explained part explains the 102.1 per

Figure 1.2: Mean values of circumstances across clusters

Note: Each circumstance variable is normalised so that each has a value from 0 to 1.

Figure 1.3: Malnutrition rates across clusters



Note: 95% confidence intervals are illustrated.

cent (=18.0 percentage points) of the observed difference, which is partly offset by the negative contribution made by the unexplained part.

The observation that the relative contribution of the explained part is over 100 per cent suggests that if children among the least advantaged type had the covariate distribution that children in the most advantaged type have, children in the least advantaged type would have a smaller malnutrition rate than children in the most advantaged type. The finding that the relative contribution of the unexplained part is negative suggests that the between-type difference in the functional form in equation (1.1) buffers the observed disparity in malnutrition rate that is associated with the difference in covariates. For example, the unexplained part can become negative, if the negative relationship between affluence and malnutrition is weaker among the most advantaged type than that among the least advantaged group. Also, the negative relative contribution of the unexplained part suggests that there might exist some unobservable factors that substantially contribute to mitigating the gap in malnutrition rate.

Among the explained part, the difference in household affluence shows the largest contribution, explaining the 48.8 per cent (=8.8 percentage points) of the explained difference ($p < 0.01$). The difference in maternal health also shows a large contribution and this explains the 17.8 per cent (=3.2 percentage points) of the explained difference ($p < 0.01$). A significant effect is also found for paternal educational background ($p < 0.01$).

Second, in Nepal, the malnutrition rates for the least advantaged type and the most advantaged type are 44.6 per cent and 37.1 per cent, respectively. The difference, 7.5 percentage points, is significant ($p < 0.01$). The decomposition analysis reveals that the 95.6 per cent of the observed difference, i.e. 7.2 percentage points, is explained by the between-type difference in the observed covariate distributions ($p < 0.01$). Among all the categories, maternal education and sanitary condition ($p < 0.01$) show the large contributions to the observed difference. They explain 28.5 per cent (=2.1 percentage points) and 20.1 per cent (=1.4 percentage points) of the explained difference, respectively. However maternal education shows a higher standard error, making its contribution statistically insignificant. We also find significant contributions made by the differences in maternal health, demography, affluence and maternal media exposure (all $p < 0.01$).

Third, in Pakistan, 63.3 per cent of children in the least advantaged type have a nutritional problem, compared with 41.6 per cent of children in the most advantaged type. The difference is 21.7 percentage points ($p < 0.01$). The difference in the observed covariate distributions accounts for the 92.0 per cent (=20.0 percentage points) of the observed difference in malnutrition rate ($p < 0.01$). Maternal education ($p < 0.01$) is the largest contributor to the observed difference, explaining the 25.5 per cent (=5.1 percentage points) of the explained difference. The differences in maternal health, paternal education, housing and media exposure also show significant contributions to the observed difference (all $p < 0.01$).

Finally, in Maldives, the malnutrition rate of the least advantaged type is 35.4 per cent and that of the most advantaged type is 29.5 per cent. The difference is 5.9 percentage points ($p < 0.05$) and this difference is the smallest among all the countries analysed in this paper. The 86.2 per cent (=5.1 percentage points) of the difference is associated with the difference in the observed covariate distributions between the types ($p < 0.01$). The differences in parental occupation and demography show large contributions to the observed difference, but the contribution of parental occupation is not statistically significant at the 5 per cent level. Demography shows the largest significant contribution to the observed difference, and it explains the 25.8 per cent (=1.3 percentage points) of the explained difference ($p < 0.05$). The differences in housing characteristics and affluence also show significant contributions (both $p < 0.01$).

India, Cambodia, Myanmar and East Timor

Table 1.5 shows the decomposition results for India, Cambodia, Myanmar and East Timor. First, in India, 64.8 per cent of children in the least advantaged type suffer nutritional problems, while 45.1 per cent of children in the most advantaged type do. The difference, 19.6 percentage points, is statistically significant ($p < 0.01$). The 19.0 percentage points are explained by the between-type difference in the covariate distributions. A detailed decomposition result shows that the differences in household affluence ($p < 0.01$), maternal health ($p < 0.01$) and maternal education ($p < 0.01$) exhibit large contributions to the observed difference. They account for the explained part by 28.7 per cent (=5.4 percentage points), 17.8 per cent (=3.4 percentage points) and 14.8 per cent (=2.8 percentage points), respectively. The differences in paternal education, sanitation, parental occupation and media exposure also show significant contributions (all $p < 0.01$).

Table 1.4: Decomposition results: Bangladesh, Nepal, Pakistan and Maldives

	Bangladesh			**Nepal**		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability						
Least advantaged type	0.567***	0.008		0.446***	0.010	
Most advantaged type	0.390***	0.008		0.371***	0.012	
Overall decomposition						
Observed difference	0.177***	0.011		0.075***	0.016	
Explained part	0.180***	0.010	102.063	0.072***	0.015	95.643
Unexplained part	-0.004	0.006	-2.063	0.003	0.006	4.357
Detailed decomposition						
Maternal health	0.032***	0.003	17.787	0.005***	0.001	6.883
Maternal education	0.005	0.005	2.511	0.021	0.016	28.525
Paternal education	0.023***	0.006	12.824	0.002	0.005	2.787
Demography	-0.002	0.005	-1.065	0.012***	0.005	16.283
Housing	0.007	0.009	3.908	0.001	0.002	0.919
Affluence	0.088***	0.014	48.820	0.008***	0.003	11.488
Sanitation	0.009	0.006	5.114	0.014***	0.003	20.145
Parental occupation	0.004	0.008	2.072	0.002	0.002	2.620
Media exposure	0.014	0.011	8.028	0.008***	0.003	10.525
	Pakistan			**Maldives**		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability						
Least advantaged type	0.633***	0.009		0.354***	0.016	
Most advantaged type	0.416***	0.010		0.295***	0.013	
Overall decomposition						
Observed difference	0.217***	0.014		0.059***	0.021	
Explained part	0.200***	0.012	92.013	0.051***	0.019	86.232
Unexplained part	0.017***	0.007	7.987	0.008	0.009	13.768
Detailed decomposition						
Maternal health	0.014***	0.003	6.758	-0.003***	0.001	-5.505
Maternal education	0.051***	0.015	25.490	0.010	0.011	19.688
Paternal education	0.026***	0.007	12.979	-0.002	0.002	-3.743
Demography	0.005	0.007	2.488	0.013**	0.006	25.826
Housing	0.030***	0.012	15.236	0.007***	0.003	13.892
Affluence	0.028*	0.015	13.884	0.012***	0.005	23.851
Sanitation	0.013	0.008	6.520	-0.001	0.003	-2.808
Parental occupation	0.006	0.006	3.112	0.017	0.015	32.927
Media exposure	0.027***	0.011	13.531	-0.002	0.003	-4.380

Contribution (Contr) of the explained and unexplained part is the one to the observed difference.
Contribution (Contr) of the individual category is the one to the explained part.
Standard errors (SEs) are obtained by bootstrap with 5,000 repetitions.
*$p < 0.10$,**$p < 0.05$,***$p < 0.01$.

Next, the malnutrition rate in Cambodia among the least advantaged type is 48.9 per cent, and among the most advantaged type it is 37.7 per cent. The difference, 11.2 percentage points, is statistically significant ($p < 0.01$). We find that the difference in covariates makes a larger contribution to the between-type disparity than is actually observed. It explains the 117.7 per cent (=13.2 percentage points) of the observed difference ($p < 0.01$). This suggests that if the children in the least advantaged type had the covariate distribution that the children in the most advantaged type have, the children belonging to the least advantaged type would have a smaller malnutrition rate than the children in the most advantaged type. The differences in household affluence and sanitation explain larger parts of the observed difference. The difference in household affluence explains 43.5 per cent (=5.7 percentage points) of the explained difference ($p < 0.01$). The difference in sanitation explains the 32.2 per cent (=4.2 percentage points) of the explained difference ($p < 0.01$). We also find a significant contribution made by the difference in maternal health ($p < 0.01$).

In Myanmar, the malnutrition rate of the least advantaged type is 42.6 per cent, whilst that of the most advantaged type is 22.5 per cent. The observed between-type difference is 20.1 percentage points and it is statistically significant ($p < 0.01$). The explained part accounts for the 88.7 per cent (=17.8 percentage points) of the observed difference ($p < 0.01$). A detailed decomposition result shows that the difference in household affluence explains the 46.1 per cent (=8.2 percentage points) ($p < 0.01$) and paternal education explains the 12.9 per cent (=2.3 percentage points) ($p < 0.01$) of the explained difference. We also find significant contributions made by the differences in maternal health ($p < 0.01$) and maternal media exposure ($p < 0.05$).

In East Timor, 75.6 per cent of children belonging to the least advantaged type have some conditions of malnutrition, and 65.7 per cent of children belonging to the most advantaged type suffer from malnutrition. The difference is 9.9 percentage points and this difference is statistically significant ($p < 0.01$). The between-type difference in covariates explains the 93.6 per cent (=9.3 percentage points) of the observed disparity in malnutrition rate ($p < 0.01$). Looking at the result of the detailed decomposition, we observe that the difference in household affluence shows the largest contribution, explaining the 28.6 per cent (=2.6 percentage points) of the explained difference ($p < 0.01$). The second largest significant contributor is the difference

in sanitary condition, explaining the 21.6 per cent (=2.0 percentage points) of the explained difference ($p < 0.01$). We also find that the differences in maternal health ($p < 0.01$), maternal education ($p < 0.05$) and demographic characteristics ($p < 0.05$) show significant contributions.

Tajikistan and Kyrgyzstan

Table 1.6 shows the decomposition results for Tajikistan and Kyrgyzstan. In Tajikistan, the malnutrition rate of the least advantaged type is 38.5 per cent, while the rate of the most advantaged type is 32.2 per cent. The difference is 6.3 percentage points ($p < 0.01$). The observed difference in covariate accounts for the 100.7 per cent (=6.3 percentage points) of the observed disparity ($p < 0.01$), which suggests that if the children in the least advantaged type had the covariate distributions that the children in the most advantaged type have, the disparity in malnutrition rate would disappear. The detailed decomposition reveals that the difference in parental occupation types shows the largest contribution to the disparity, explaining the 75.1 per cent (=4.7 percentage points) of the explained part ($p < 0.01$). The difference in affluence also contributes to the explained difference by 15.3 per cent (=1.0 percentage points) ($p < 0.01$). Although they are small, the differences in maternal health ($p < 0.05$) and maternal educational attainment ($p < 0.01$) show significant contributions.

Last of all, in Kyrgyzstan, among the least advantaged type, the malnutrition rate is 26.5 per cent, and the rate among the most advantaged type is 14.7 per cent. The difference is 11.9 percentage points ($p < 0.01$). We find that the difference in covariates explains the 74.3 per cent (=8.8 percentage points) of the observed difference ($p < 0.01$). The detailed decomposition reveals that the difference in maternal education is the largest contributor, explaining the 46.9 per cent of the explained difference (=4.1 percentage points) ($p < 0.01$).

1.5.3 Heterogeneity between male and female children

Although the difference in the physical developmental process between male and female children is taken account of when calculating the HAZ, WAZ and WHZ scores, the degree of inequality and its determinants can differ between them. Wamani et al. (2007) report that in sub-Saharan Africa male children under five years of age are more likely to become stunted than female children, and that boys have a steeper socio-economic gradient of under-nutrition. In this section, we explore the heterogeneity of the determinants between boys and girls. We split the sam-

Table 1.5: Decomposition results: India, Cambodia, Myanmar and East Timor

	India			Cambodia		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability						
Least advantaged type	0.648***	0.003		0.489***	0.011	
Most advantaged type	0.451***	0.004		0.377***	0.011	
Overall decomposition						
Observed difference	0.196***	0.005		0.112***	0.015	
Explained part	0.190***	0.004	96.688	0.132***	0.013	117.738
Unexplained part	0.007***	0.003	3.312	-0.020***	0.007	-17.738
Detailed decomposition						
Maternal health	0.034***	0.002	17.810	0.013***	0.002	9.852
Maternal education	0.028***	0.004	14.800	-0.001	0.004	-0.993
Paternal education	0.009***	0.003	4.884	0.001	0.003	0.690
Demography	-0.007***	0.002	-3.949	-0.017*	0.009	-12.860
Housing	0.024***	0.004	12.682	0.005	0.010	3.431
Affluence	0.054***	0.006	28.675	0.057***	0.017	43.454
Sanitation	0.023***	0.004	12.363	0.042***	0.012	32.232
Parental occupation	0.012***	0.003	6.292	0.021	0.017	15.857
Media exposure	0.012***	0.005	6.450	0.011	0.010	8.352
	Myanmar			**East Timor**		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability						
Least advantaged type	0.426***	0.013		0.756***	0.005	
Most advantaged type	0.225***	0.015		0.657***	0.007	
Overall decomposition						
Observed difference	0.201***	0.021		0.099***	0.009	
Explained part	0.178***	0.016	88.719	0.093***	0.009	93.628
Unexplained part	0.023*	0.013	11.281	0.006**	0.003	6.372
Detailed decomposition						
Maternal health	0.017***	0.003	9.681	0.009***	0.001	9.699
Maternal education	-0.006	0.005	-3.241	0.008**	0.004	8.756
Paternal education	0.023***	0.007	12.874	-0.004**	0.002	-4.487
Demography	-0.005	0.012	-2.538	0.009**	0.004	9.255
Housing	0.022*	0.012	12.594	0.006	0.005	6.318
Affluence	0.082***	0.018	46.058	0.026***	0.009	28.627
Sanitation	0.016	0.010	8.724	0.020***	0.005	21.644
Parental occupation	0.008	0.007	4.613	0.018*	0.010	18.962
Media exposure	0.020**	0.010	11.219	0.001	0.006	1.233

Contribution (Contr) of the explained and unexplained part is the one to the observed difference.
Contribution (Contr) of the individual category is the one to the explained part.
Standard errors (SEs) are obtained by bootstrap with 5,000 repetitions.
*$p < 0.10$,**$p < 0.05$,***$p < 0.01$.

Table 1.6: Decomposition results: Tajikistan and Kyrgyzstan

	Tajikistan			Kyrgyzstan		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability						
Least advantaged type	0.385***	0.007		0.265***	0.021	
Most advantaged type	0.322***	0.008		0.147***	0.013	
Overall decomposition						
Observed difference	0.063***	0.010		0.119***	0.025	
Explained part	0.063***	0.010	100.776	0.088***	0.021	74.319
Unexplained part	0.000	0.002	-0.776	0.030***	0.012	25.681
Detailed decomposition						
Maternal health	0.002**	0.001	2.416	-0.001	0.001	-1.223
Maternal education	0.001***	0.000	1.204	0.041***	0.008	46.920
Paternal education	-0.001	0.001	-2.224	-0.005	0.005	-5.553
Demography	0.002	0.003	2.480	0.014	0.010	16.165
Housing	0.001	0.003	2.206	0.015	0.014	16.832
Affluence	0.010***	0.003	15.309	-0.004	0.006	-4.078
Sanitation	0.002	0.003	3.454	0.028	0.023	31.799
Parental occupation	0.047***	0.011	75.105	0.002	0.005	2.282
Media exposure	0.000	0.002	0.050	-0.003***	0.001	-3.190

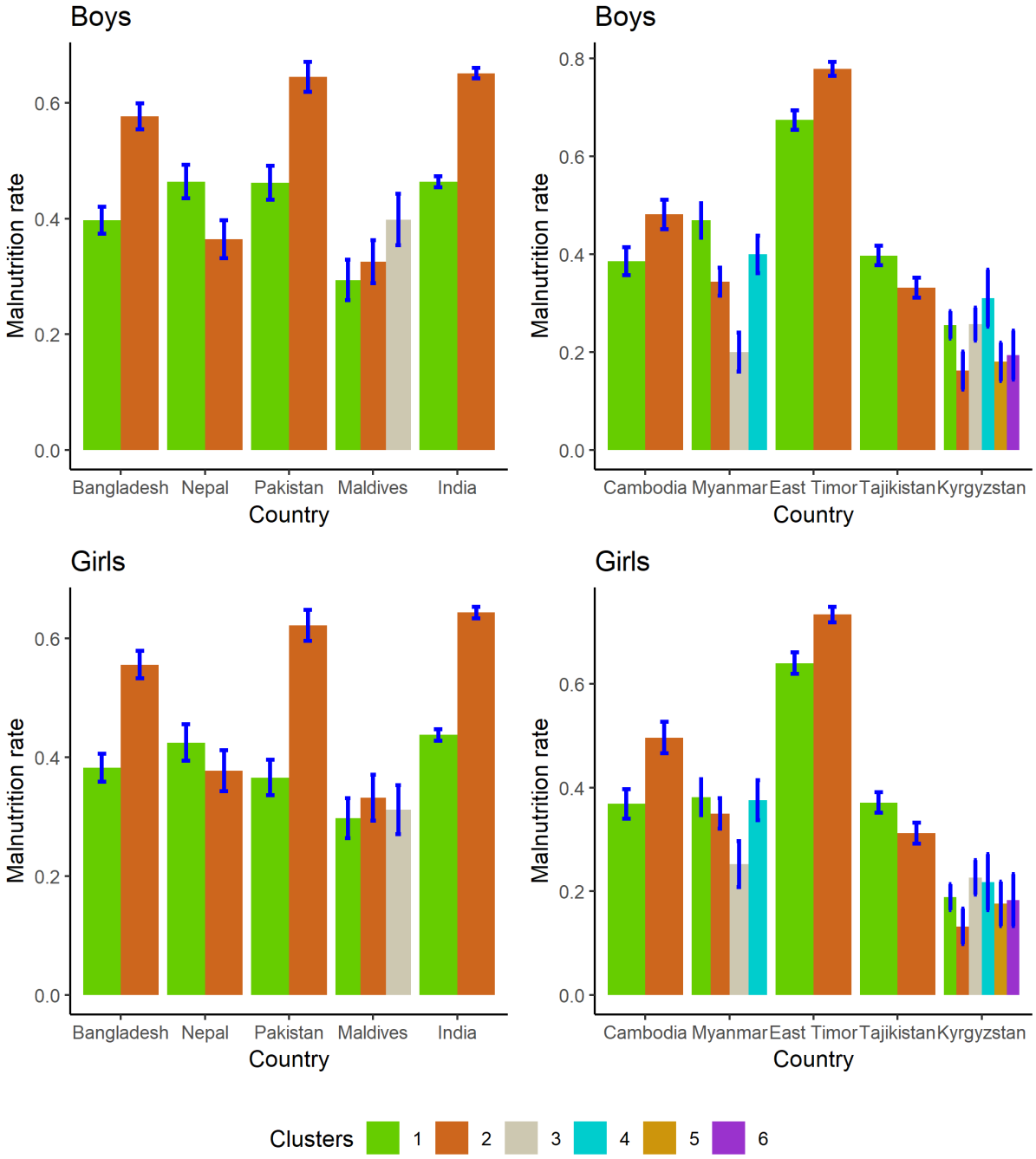
Contribution (Contr) of the explained and unexplained part is the one to the observed difference.
Contribution (Contr) of the individual category is the one to the explained part.
Standard errors (SEs) are obtained by bootstrap with 5,000 repetitions.
$^{*}p < 0.10, ^{**}p < 0.05, ^{***}p < 0.01$.

ple and implement the decomposition analysis for each subsample. Figure 1.4 illustrates the malnutrition rates of each cluster, from which we redefine the most advantaged types and the least advantaged types, and explore the factors that are strongly associated with the observed between-type disparity in the malnutrition rate.

Tables 1.7 and 1.8 present the decomposition results for boys and girls respectively. In Bangladesh, Pakistan, India, Cambodia, Myanmar, East Timor and Tajikistan, we find that the subsample analysis shows very similar results to the main results of the entire sample analysis and we do not find substantial heterogeneity between boys and girls. On the other hand, in Nepal, Maldives and Kyrgyzstan, we find notable differences in terms of the proportion of the explained part and the largest determinants. In this subsection, we discuss the results obtained from these three countries.

First in Nepal, compared with the main result, the result of the boys' sample shows that a much larger proportion of the explained part is contributed by the difference in maternal education ($p < 0.01$). While in the entire sample analysis its relative contribution is 28.5 per cent, here it

Figure 1.4: Malnutrition rates of boys and girls across clusters



Note: 95% confidence intervals are illustrated.

Table 1.7: Decomposition results for boys

	Bangladesh			Nepal			Pakistan			Maldives			India		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability															
Least advantaged type	0.577***	0.011		0.464***	0.016		0.645***	0.014		0.398***	0.022		0.652***	0.005	
Most advantaged type	0.397***	0.012		0.364***	0.017		0.462***	0.015		0.294***	0.018		0.464***	0.005	
Overall decomposition															
Observed difference	0.180***	0.017		0.100***	0.023		0.183***	0.020		0.105***	0.028		0.188***	0.007	
Explained part	0.176***	0.014	97.882	0.099***	0.021	99.210	0.176***	0.018	96.162	0.092***	0.025	87.729	0.185***	0.006	98.418
Unexplained part	0.004	0.009	2.118	0.001	0.009	0.790	0.007	0.009	3.838	0.013	0.011	12.271	0.003	0.004	1.582
Detailed decomposition															
Maternal health	0.032***	0.005	18.019	0.004***	0.001	4.307	0.013***	0.004	7.101	-0.002**	0.001	-2.625	0.036***	0.002	19.246
Maternal education	-0.001	0.007	-0.286	0.057***	0.022	57.245	0.047**	0.021	26.944	0.024	0.016	26.312	0.026***	0.005	13.975
Paternal education	0.018**	0.009	10.248	0.006	0.007	6.520	0.027***	0.010	15.257	0.005	0.005	5.716	0.014***	0.003	7.757
Demography	0.002	0.007	1.346	0.004	0.008	4.154	0.001	0.010	0.442	0.003	0.010	3.794	-0.007***	0.003	-3.892
Housing	0.015	0.013	8.272	-0.006	0.005	-6.229	0.024	0.017	13.601	0.008***	0.003	8.214	0.029***	0.006	15.658
Affluence	0.087***	0.019	49.674	0.009***	0.003	8.854	0.027	0.019	15.429	-0.002	0.009	-2.632	0.053***	0.009	28.613
Sanitation	0.020**	0.009	11.165	0.017***	0.004	16.975	0.016	0.011	8.994	0.013**	0.006	14.462	0.015***	0.005	8.332
Parental occupation	0.009	0.011	5.278	0.003	0.004	3.536	0.004	0.008	2.453	0.046**	0.022	50.609	0.015***	0.004	8.049
Media exposure	-0.007	0.015	-3.730	0.004	0.004	4.495	0.017	0.015	9.719	-0.004	0.004	-3.859	0.004	0.007	2.257

	Cambodia			Myanmar			East Timor			Tajikistan			Kyrgyzstan		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability															
Least advantaged type	0.481***	0.015		0.469***	0.018		0.778***	0.007		0.397***	0.010		0.310***	0.030	
Most advantaged type	0.385***	0.014		0.199***	0.021		0.674***	0.010		0.331***	0.010		0.162***	0.019	
Overall decomposition															
Observed difference	0.096***	0.021		0.269***	0.029		0.105***	0.013		0.066***	0.013		0.148***	0.035	
Explained part	0.118***	0.018	123.487	0.227***	0.022	84.445	0.101***	0.012	96.584	0.065***	0.013	99.544	0.122***	0.031	82.309
Unexplained part	-0.022***	0.009	-23.487	0.042***	0.018	15.555	0.004	0.004	3.416	0.000	0.002	0.456	0.026	0.016	17.691
Detailed decomposition															
Maternal health	0.016***	0.003	13.304	0.014***	0.003	6.089	0.007***	0.001	7.001	0.004***	0.001	6.290	-0.002**	0.001	-1.360
Maternal education	0.005	0.005	3.991	-0.001	0.007	-0.414	0.010*	0.006	10.031	0.000	0.000	0.546	0.042***	0.012	34.426
Paternal education	-0.003	0.004	-2.209	0.027***	0.009	11.749	-0.009***	0.003	-8.568	-0.003	0.002	-4.422	-0.009	0.007	-7.574
Demography	-0.019	0.013	-15.922	0.022	0.017	9.495	0.007	0.006	7.402	-0.002	0.005	-3.639	0.015	0.014	12.263
Housing	0.008	0.015	6.858	0.013	0.016	5.515	0.010	0.007	10.077	0.002	0.004	2.473	0.020	0.021	16.210
Affluence	0.050**	0.023	41.890	0.118***	0.025	51.757	0.031***	0.012	30.336	0.004	0.004	5.532	0.002	0.009	1.539
Sanitation	0.036**	0.016	30.744	0.004	0.014	1.583	0.019***	0.006	19.189	0.007	0.005	11.398	0.059	0.036	48.279
Parental occupation	0.017	0.024	14.512	0.006	0.010	2.479	0.025*	0.014	24.684	0.054***	0.015	83.107	-0.002	0.008	-1.932
Media exposure	0.008	0.014	6.850	0.026*	0.014	11.641	0.000	0.009	-0.195	-0.001	0.002	-1.279	-0.002	0.002	-1.883

Contribution (Contr) of the explained and unexplained part is the one to the observed difference.
Contribution (Contr) of the individual category is the one to the explained part.
Standard errors (SEs) are obtained by bootstrap with 5,000 repetitions.
*$p < 0.10$,**$p < 0.05$,***$p < 0.01$.

is 57.2 per cent. In the result of the girls' sample, on the other hand, we find that the largest contributor is the difference in demographic factors ($p < 0.01$) and the difference in maternal education does not show a significant contribution.

Next, in Maldives, we find interesting heterogeneities between boys and girls. In the boys' sample, the malnutrition rate of the least advantaged type is 39.8 per cent, whilst that of the most advantaged type is 29.4 per cent. The observed between-type difference is 10.5 percentage points and it is statistically significant ($p < 0.01$). The explained part accounts for the 87.7 per cent (=9.2 percentage points) of the observed difference ($p < 0.01$). Looking at the detailed decomposition results, we find a much larger contribution of the difference in parental occupation types ($p < 0.05$) than that observed in the entire sample analysis. On the other hand in the girls' sample, we do not find a statistically significant between-type difference in malnutrition rate, which suggests that the significant difference found in the entire sample is mainly due to the between-type difference of the boys.

In Kyrgyzstan, we find a few notable differences between boys and girls in terms of the proportion of the explained part and its main contributors. For boys, the malnutrition rates for the least advantaged type and the most advantaged type are 31.0 per cent and 16.2 per cent, respectively. The difference is 14.8 percentage points ($p < 0.01$). The difference in the observed covariate distributions accounts for the 82.3 per cent (12.2 percentage points) of the observed difference in malnutrition rate. This relative contribution is larger than the contribution of 74.3 per cent which is estimated in the entire sample analysis. The largest contribution is made by the difference in sanitary condition, but its large standard error makes it statistically insignificant. The difference in maternal education makes the largest significant contribution ($p < 0.01$).

In the girls' sample in Kyrgyzstan, the malnutrition rate of the least advantaged type is 22.7 per cent, whilst that of the most advantaged type is 13.2 per cent. The observed between-type difference is 9.5 percentage points and it is statistically significant ($p < 0.01$). The explained part accounts for only 84.7 per cent (=8.0 percentage points) of the observed difference, which is also larger than the contribution of 74.3 per cent which is estimated in the entire sample analysis. The largest contribution is made by the difference in maternal education ($p < 0.01$), which is consistent with the entire sample analysis.

Table 1.8: Decomposition results for girls

	Bangladesh			Nepal			Pakistan			Maldives			India		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability															
Least advantaged type	0.556***	0.011		0.424***	0.015		0.622***	0.013		0.332***	0.019		0.643***	0.005	
Most advantaged type	0.382***	0.012		0.377***	0.018		0.366***	0.015		0.297***	0.017		0.437***	0.005	
Overall decomposition															
Observed difference	0.173***	0.016		0.047**	0.023		0.256***	0.020		0.035	0.026		0.206***	0.007	
Explained part	0.185***	0.014	107.005	0.048**	0.021	102.623	0.228***	0.017	89.005	0.023	0.023	65.907	0.196***	0.006	94.995
Unexplained part	-0.012	0.009	-7.005	-0.001	0.009	-2.623	0.028***	0.009	10.995	0.012	0.012	34.093	0.010***	0.004	5.005
Detailed decomposition															
Maternal health	0.032***	0.005	17.364	0.006***	0.001	12.035	0.014***	0.005	6.261	-0.001	0.001	-2.934	0.032***	0.002	16.325
Maternal education	0.009	0.006	5.089	-0.024	0.023	-50.156	0.057***	0.022	25.128	0.002	0.007	9.011	0.030***	0.005	15.570
Paternal education	0.028***	0.008	15.055	-0.005	0.007	-10.276	0.023***	0.009	10.216	0.004**	0.002	17.818	0.004	0.004	1.901
Demography	-0.006	0.007	-3.296	0.024***	0.006	50.508	0.008	0.010	3.689	0.023**	0.010	100.445	-0.008***	0.003	-3.992
Housing	0.001	0.012	0.729	0.007***	0.003	13.625	0.040**	0.018	17.515	0.008**	0.004	34.307	0.019***	0.006	9.699
Affluence	0.090***	0.019	48.683	0.010**	0.005	20.473	0.028	0.022	12.214	0.011***	0.004	50.233	0.058***	0.009	29.559
Sanitation	-0.003	0.009	-1.468	0.014***	0.004	28.571	0.012	0.012	5.236	-0.011**	0.005	-46.890	0.031***	0.006	15.956
Parental occupation	-0.003	0.011	-1.823	0.001	0.004	2.828	0.011	0.008	4.786	-0.013	0.022	-55.220	0.009**	0.004	4.489
Media exposure	0.037***	0.015	19.680	0.016***	0.005	32.608	0.034**	0.016	14.905	-0.001	0.004	-5.868	0.021***	0.007	10.491

	Cambodia			Myanmar			East Timor			Tajikistan			Kyrgyzstan		
	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)	Estimates	SEs	Contr (%)
Probability															
Least advantaged type	0.497***	0.016		0.381***	0.017		0.734***	0.008		0.371***	0.010		0.227***	0.016	
Most advantaged type	0.369***	0.014		0.253***	0.022		0.640***	0.011		0.312***	0.011		0.132***	0.017	
Overall decomposition															
Observed difference	0.128***	0.021		0.129***	0.028		0.094***	0.014		0.059***	0.015		0.095***	0.024	
Explained part	0.145***	0.019	113.311	0.127***	0.022	98.919	0.085***	0.013	90.241	0.060***	0.014	101.219	0.080***	0.020	84.735
Unexplained part	-0.017*	0.009	-13.311	0.001	0.018	1.081	0.009**	0.004	9.759	-0.001	0.003	-1.219	0.014	0.015	15.265
Detailed decomposition															
Maternal health	0.011***	0.002	7.625	0.021***	0.005	16.387	0.012***	0.002	13.811	0.000	0.001	-0.054	0.001	0.001	1.421
Maternal education	-0.007	0.005	-4.725	-0.009	0.008	-7.389	0.005	0.006	5.741	0.001	0.001	1.262	0.060***	0.016	74.939
Paternal education	0.007	0.006	5.009	0.014	0.011	10.652	0.000	0.003	-0.526	0.000	0.001	0.728	0.000	0.001	-0.178
Demography	-0.015	0.013	-10.099	-0.035*	0.018	-27.525	0.011**	0.005	12.646	0.007	0.005	10.942	0.005	0.004	6.128
Housing	-0.001	0.014	-0.791	0.033**	0.016	26.104	-0.001	0.008	-1.003	0.000	0.003	0.248	0.003	0.003	3.895
Affluence	0.060***	0.024	41.624	0.043*	0.025	33.756	0.024*	0.013	28.877	0.015***	0.004	24.115	0.003	0.003	3.362
Sanitation	0.052***	0.017	36.103	0.031**	0.014	24.383	0.022***	0.007	26.189	-0.001	0.005	-1.520	-0.003	0.003	-4.281
Parental occupation	0.023	0.024	15.582	0.007	0.009	5.429	0.009	0.015	10.895	0.038***	0.015	62.874	0.003	0.009	3.621
Media exposure	0.014	0.015	9.665	0.023	0.015	18.171	0.003	0.009	3.335	0.001	0.002	1.262	0.009***	0.003	11.090

Contribution (Contr) of the explained and unexplained part is the one to the observed difference.
Contribution (Contr) of the individual category is the one to the explained part.
Standard errors (SEs) are obtained by bootstrap with 5,000 repetitions.
*$p < 0.10$,**$p < 0.05$,***$p < 0.01$.

1.6 Discussion and conclusion

Health inequality among children is not merely an infringement of various social equalities. Given that health is one of the most important elements of human capital (Johnson and Schoeni, 2011), it can be a substantial future obstacle for the sound, inclusive social and economic development of a country. This study explores the inequality of opportunity in child nutritional status in ten developing countries in Asia, where a high proportion of children still remain vulnerable to food insecurity and suffer malnutrition. As determinants of child health inequality are often multidimensional and mutually related, this study considers various aspects of circumstances that are totally beyond the control of children. We partition child observations into distinct types defined by a data-driven clustering method. This study clarifies the sources of unfair inequality in child malnutrition which could not be captured by an unidimensional indicator of socioeconomic status. Then, in order to better understand what aspects of circumstances are strongly associated with the between-type disparity in malnutrition rate, we decompose the observed disparity into its contributing factors with a non-linear decomposition method. The latest DHS data allows us to compare the results across countries. Although the decomposition results in this study do not necessarily reflect the causal relationships, the descriptive association can be equally important from a policymaking perspective.

Our decomposition analysis elucidates the important factors that are associated with the inequality in child nutritional status across the country. First, we find strong evidence of the existence of inequality of opportunity in child nutritional status in all ten countries. The largest between-type disparity in malnutrition rate is observed in Pakistan as 21.7 percentage points. The decomposition results show that in eight countries, the difference in household affluence shows the significant contribution and that in five of the countries, the difference in household affluence exhibits the largest contribution to the observed disparity in malnutrition rate. This is consistent with a variety of literature which reports that children from poorer households tend to be vulnerable to food insecurity and to have higher mortality rates (Chalasani, 2012; Black et al., 2013; Huda et al., 2017).

The difference in maternal health shows a significant contribution in Bangladesh, Nepal, Pakistan, India, Cambodia, Myanmar, East Timor and Tajikistan, where we find the significantly larger maternal BMI among the most advantaged type. The difference in demographic factors

show significant contributions in Nepal, Maldives and East Timor. In Nepal and East Timor, the difference in the maternal age at birth made the largest contribution, while in Maldives the difference in the proportion of households living in urban areas made the largest contribution (Table 1.A.2).

The difference in housing characteristics shows a significant contribution in Pakistan, Maldives and India. In Maldives, the difference in number of rooms is the main contributor. In Pakistan and India, where children in the least advantaged type tend to live in poorer houses, the difference in the materials of housing make large contributions (Table 1.A.2). The difference in sanitary condition, which is known as a leading cause of diarrhoea and intestinal parasites (Konteh, 2009), exhibits a significant contribution in Nepal, India, Cambodia and East Timor, where a much lower proportion of households among the least advantaged type have access to piped water, own a flushing toilet, and treat water for drinking. In these four countries, the between-type difference in the proportion of households with a flushing toilet makes the largest contribution (Table 1.A.2).

In Nepal, Pakistan, India and Myanmar, the difference in maternal exposure to the media significantly explains the observed difference. In these countries, the differences in frequency of watching TV and reading a newspaper made the largest contributions (Table 1.A.2). The difference in the frequency of listening to radio did not show a significant contribution at the five per cent level in these countries. These findings suggest the existence of a gap in knowledge about childrearing that could be obtained by being exposed to the TV and newspaper.

In addition, the difference in maternal educational backgrounds significantly explains the observed disparity in Pakistan, India, East Timor, Tajikistan and Kyrgyzstan. On the other hand, the difference in paternal educational backgrounds shows a significant contribution to the observed disparity in Bangladesh, Pakistan, India and Myanmar. In all the countries except Bangladesh and Myanmar, the contributions made by paternal education are significantly smaller than those made by maternal education ($p < 0.05$). This can be attributed to the observations that, in most Asian countries, mothers play a major role in raising children. Protecting children from mothers with a less advantaged educational background is significant and enhancing the "health-related literacy" among mothers should be considered. Moreover, enhancing educational

opportunities for children could lessen the inequality in nutritional status for future generations.

The difference in parental occupation types show a significant contribution in India and Tajikistan. In these countries, among the least advantaged type, the proportion of parents working as manual workers or farmers is significantly higher and the proportion of parents working as professional workers is significantly lower than among the most advantaged type. In India, the between-type difference in the proportion of fathers working as farmers shows the largest significant contribution. In Tajikistan, the larger proportion of non-manual workers among fathers of the most advantaged type makes the largest significant contribution. These findings imply that the inequality in child nutritional status is significantly and strongly associated with paternal occupation-related socio-economic status.

Overall, all the evidence from this study suggests that priority should be given to protecting children from marginalised households to mitigate the inequality in child health. To promote child health among poor households, conditional cash transfer programmes, which transfer money to households contingent on investments in human capital, have been implemented globally (Handa and Davis, 2006).[11] In Asia, some countries have launched conditional cash transfer programmes (Rawlings and Rubio, 2005), but the number of countries which introduced these programmes is still limited in the region.[12] Given the reported improvement in nutritional status of children from poor households in a number of Latin American countries (Behrman and Hoddinott, 2005; Gertler, 2004; Maluccio and Flores, 2005; Attanasio et al., 2005), governments in Asian countries might also wish to assess the possibility of introducing the conditional cash transfer programme.

Moreover, as food insecurity is a key risk factor for child malnutrition, encouraging social movement to promote affordable food could also be beneficial (Capone et al., 2014; Baig-Ansari et al., 2006). The food assistance programmes undertaken across the world show higher returns for very young children in poor households (Lentz and Barrett, 2013). The in-kind food assistance programme targeting poor households could be an alternative option to the conditional cash transfer programmes. While the conditional cash transfer programmes, which allow households to choose more diverse diets, tend to result in the increased consumption of nutritionally rich

[11]The most famous examples are *Progresa/Oportunidades* in Mexico and *Bolsa Escola* in Brazil.

[12]In Asia, Indonesia introduced a conditional cash transfer programme called the PKH (*Program Keluarga Harapan*) in 2007. In India and Nepal, similar programmes were introduced for pregnant women in order to promote institutional delivery, both in 2005.

foods such as animal-source foods, vegetables and fruits, in-kind food and community vouchers allow agencies to target specific nutrition interventions such as vitamin-fortified oils (Meyer and TANGO International Inc., 2012).

Providing education about nutrition to mothers with limited educational backgrounds appears to be equally important (Bryce et al., 2008). The success of nutrition education in terms of reducing malnutrition has been reported in Pakistan (Khan et al., 2013). Policymakers should ensure that children can equally enjoy better health and affordable healthcare access, irrespective of parental occupations. Once we take into account the intergenerational benefits, the potential benefit of early intervention would be exponential (Black and Hurley, 2014).

Finally, it is worth discussing a limitation and some potential extensions to this study. First, thanks to the detailed DHS information, we are able to consider multidimensional aspects of household and parental socio-economic status, but the circumstance factors beyond the control of children are without doubt much more complex (Currie and Vogl, 2013). For example, health inequality starts even at the prenatal stage (Dipietro et al., 1999; Kramer, 1987; Almond, 2006) and genetic factors are one such circumstance factor. However, such information is rarely available in large-scale general-purpose surveys. Moreover, in some countries, institutional inequalities, such as the Caste system in India, are also likely to play a considerable role in creating health inequality (Van de Poel and Speybroeck, 2009). In this study, in order to make the results comparable across countries, we did not consider country-specific factors, but including more detailed country-specific circumstance factors could possibly allow us to define finer types in a single-country study.

Second, if there were some large-scale natural disasters and epidemic shocks around the time of data collection, the between-type gap may have reflected their heterogeneous effects. However, the main goal of this study is to describe in detail the existing inequality of opportunity in child malnutrition. Hence, the heterogeneous effects of these shocks, if any, are also incorporated as a part of the inequality associated with the circumstance factors. Third, as this study involves cross-sectional analysis, we did not discuss the trends of health inequality over time. Exploring the trend of health inequality and changes in the contributing factors for each country with the multiple waves of the DHS data would be a promising research topic and complement the

findings of this study.

1.A Appendix

Table 1.A.1: Descriptive statistics

	BGD	NPL	PAK	MDV	IND	KHM	MMR	TLS	TJK	KGZ
	mean	mean	mean	mean	mean	mean	mean	mean	mean	mean
Child malnutrition	0.48	0.41	0.54	0.32	0.55	0.43	0.36	0.72	0.36	0.21
Mother BMI	21.62	21.36	23.70	24.10	21.32	22.08	22.69	20.51	23.16	23.93
Maternal education years	2.72	1.99	1.80	3.81	2.88	2.95	2.89	3.57	5.05	5.32
Paternal education years	2.64	2.66	2.80	3.60	3.40	3.23	3.05	2.52	5.39	5.54
Demography	0.03	-0.02	-0.05	0.01	0.00	0.04	0.02	0.04	0.02	0.04
Housing	0.02	-0.09	-0.01	-0.03	-0.06	-0.06	-0.22	0.01	-0.02	0.16
Affluence	-0.02	-0.08	-0.01	0.18	-0.05	-0.05	-0.23	-0.01	0.03	0.03
Sanitation	-0.07	-0.23	-0.15	-0.00	-0.15	-0.21	0.03	-0.02	-0.16	-0.24
Occupation	0.00	0.02	-0.01	-0.00	1.50	0.01	-0.00	-0.02	-0.00	0.01
Media exposure	-0.02	-0.12	-0.01	0.03	-0.17	-0.11	-0.11	-0.06	-0.07	-0.04
Mother age at birth	25.58	25.77	28.86	28.22	27.31	28.57	30.67	31.33	27.51	28.45
Family size	6.05	7.45	11.56	10.21	6.45	5.85	6.74	8.14	10.16	6.81
Number of children	1.34	1.63	2.79	2.06	1.74	1.53	1.58	2.22	2.64	1.85
Urban	0.32	0.58	0.45	0.14	0.25	0.27	0.24	0.21	0.26	0.25
Natural floors	0.00	0.11	0.01	0.00	0.10	0.00	0.00	0.01	0.00	0.00
Rudimentary floors	0.00	0.00	0.00	0.00	0.07	0.66	0.63	0.03	0.21	0.26
Finished floors	0.31	0.29	0.42	0.99	0.49	0.26	0.30	0.34	0.49	0.69
Natural walls	0.11	0.06	0.08	0.00	0.21	0.16	0.07	0.65	0.11	0.19
Rudimentary walls	0.09	0.52	0.20	0.00	0.12	0.14	0.65	0.03	0.32	0.37
Finished walls	0.79	0.41	0.66	0.93	0.67	0.70	0.27	0.31	0.57	0.40
Natural roofs	0.02	0.11	0.04	0.00	0.08	0.07	0.30	0.23	0.04	0.00
Rudimentary roofs	0.00	0.00	0.26	0.00	0.05	0.00	0.01	0.01	0.00	0.01
Finished roofs	0.98	0.88	0.42	1.00	0.63	0.93	0.68	0.75	0.90	0.99
Number of rooms	2.32	2.92	2.78	3.67	2.01	1.64	1.83	2.76	3.33	2.45
Radio	0.03	0.28	0.19	0.90	0.09	0.34	0.29	0.36	0.24	0.36
TV	0.43	0.47	0.64	0.99	0.59	0.62	0.54	0.20	0.97	0.99
Refrigerator	0.20	0.11	0.48	0.88	0.26	0.11	0.12	0.05	0.47	0.73
Bicycle	0.25	0.44	0.29	0.55	0.47	0.53	0.35	0.12	0.26	0.27
Electricity	0.62	0.89	0.95	1.00	0.86	0.56	0.56	0.36	0.99	1.00
Car	0.01	0.05	0.10	0.04	0.06	0.18	0.06	0.02	0.45	0.55
Mobile phone	0.90	0.97	0.90	0.99	0.92	0.88	0.68	0.44	0.95	0.99
Safe fuel	0.13	0.18	0.40	0.92	0.31	0.18	0.16	0.01	0.65	0.64
Motorcycle	0.08	0.18	0.40	0.38	0.37	0.72	0.54	0.12	0.02	0.02
Piped water	0.06	0.26	0.32	0.09	0.26	0.15	0.12	0.18	0.39	0.45
Flushing toilet	0.18	0.73	0.73	0.97	0.45	0.53	0.40	0.35	0.13	0.08
Water treatment	0.10	0.17	0.08	0.51	0.38	0.65	0.74	0.87	0.81	0.52
Mother professional worker	0.02	0.03	0.02	0.11	0.02	0.04	0.05	0.02	0.06	0.13
Mother manual worker	0.06	0.04	0.05	0.14	0.05	0.14	0.24	0.02	0.11	0.03
Mother non-manual worker	0.02	0.06	0.07	0.13	0.04	0.22	0.16	0.08	0.04	0.05
Mother farmer	0.15	0.48	0.07	0.01	0.13	0.34	0.15	0.30	0.02	0.03
Father professional worker	0.06	0.05	0.11	0.14	0.07	0.09	0.08	0.16	0.14	0.16
Father manual worker	0.34	0.41	0.47	0.32	0.33	0.30	0.56	0.06	0.52	0.40
Father non-manual worker	0.23	0.33	0.27	0.30	0.23	0.14	0.08	0.16	0.28	0.11
Father farmer	0.24	0.17	0.12	0.22	0.32	0.46	0.27	0.61	0.03	0.31
Watch TV less than once a week	0.09	0.24	0.21	0.01	0.08	0.16	0.19	0.09	0.13	0.07
Watch TV once a week	0.50	0.40	0.46	0.97	0.61	0.53	0.52	0.30	0.81	0.92
Read newspaper less than once a week	0.09	0.20	0.20	0.35	0.15	0.17	0.25	0.17	0.19	0.27
Read newspaper once a week	0.06	0.04	0.04	0.26	0.18	0.06	0.12	0.17	0.26	0.43
Listen to radio less than once a week	0.02	0.31	0.15	0.08	0.06	0.25	0.20	0.14	0.15	0.19
Listen to radio once a week	0.02	0.26	0.04	0.81	0.09	0.24	0.20	0.31	0.23	0.30
Observations	6988	3759	4807	3521	40200	4332	5616	10968	8800	5483

BGD: Bangladesh; NPL: Nepal; PAK: Pakistan; MDV: Maldides; IND: India
KHM: Cambodia; MMR: Myanmar; TLS: East Timor; TJK: Tajikistan; KGZ: Kyrgyzstan

Table 1.A.2: Full results of detailed decomposition without aggregation

	BGD	NPL	PAK	MDV	IND	KHM	MMR	TLS	TJK	KGZ
Mother BMI	0.0320***	0.00476***	0.0135***	-0.00279***	0.0338***	0.0126***	0.0170***	0.00896***	0.00151**	-0.00108*
Maternal education years	0.00453	0.0187	0.0508***	0.00992	0.0281***	-0.00121	-0.00574	0.00807*	0.000746*	0.0415***
Paternal education years	0.0231***	0.00181	0.0258***	-0.00188	0.00927***	0.000852	0.0227***	-0.00414*	-0.00139	-0.00484
Mother age at birth	0.00168*	0.00865**	0.00320**	0.000871	-0.000403	0.000488*	0.00236***	0.00307***	0.0000765	0.00394**
Family size	-0.00140	0.00000867	0.00152	-0.0000402	0.000104	-0.00334	-0.000575	0.000435	0.00366**	0.00599*
Number of children	0.000410	-0.000343	0.00331*	-0.000135	0.00373***	0.000148	0.0104***	-0.0000243	-0.000481	-0.00222**
Urban	-0.00266	0.00209	-0.00308	0.0124**	-0.0110***	-0.0134	-0.0166	0.00507	-0.00173	0.00652
Natural floors	0	0.000829	0.0000433	0	0.00312***	0	0.0000192	0.000233***	0	0
Rudimentary floors	0.00000834	-0.0000420	0.0000280	0	0.000223**	-0.000872	-0.0232***	0.000206	-0.000141	0.0228***
Finished floors	-0.000201	0.00121	-0.0144*	0.000101	0.0107***	0.00323	0.0158	0.00856*	-0.00426***	-0.0162***
Natural walls	-0.000450	-0.000956	-0.00589	0	0.00513	-0.00240	-0.00556	-0.0110	0.000645	-0.00314
Rudimentary walls	0.000565	0.000533	0.00201	0.0000588	0.000881	0.000236	-0.0111	-0.0000380	0.0166	0.0139
Finished walls	0.00322	0.000951	0.0323**	0.000526	-0.00838	0.000773	0.0150	0.00696	-0.0127	0.00180
Natural roofs	-0.00371	-0.00470	0.00919***	0	-0.00355***	-0.0518***	-0.0521	0.00409	0.000698	0
Rudimentary roofs	-0.000229	0.00162***	0.00928	0	0.000509	-0.000674	-0.00298*	-0.000276	-0.000169	-0.00973
Finished roofs	0.00634**	0.00959***	-0.000610	-0.000727	0.00893***	0.0605***	0.0710	-0.00576	0.00114**	0.0105
Number of rooms	0.00165	-0.00207	-0.00147	0.00698**	0.00665***	0.000317	0.0183***	0.00285	-0.000126	-0.00517**
Radio	0.00130	0.00455***	0.00468***	-0.00124	0.00287***	0.00113	-0.000368	-0.000359	-0.000157	0.00162
TV	0.0251**	0.0000446	-0.00308	0.0000857	0.00847*	0.00924	-0.000426	-0.0130**	-0.000278	-0.000506
Refrigerator	0.0341***	0.000797	0.0201**	-0.00326	0.0187***	0.0160**	0.00404	0.00366	0.00518**	0.00505**
Bicycle	0.000551**	-0.000935	0.0000417	0.00213*	0.000467	0.000890	0.0121***	0.00884***	0.00262***	-0.00136
Electricity	0.0106	0.00282***	0.00888***	0	0.00731***	0.00334	0.000619	0.0117*	0.000634	0.000736
Car	0.000725	0.000477	-0.00539*	-0.000612	0.00268**	0.000259	0.0217***	0.00176	-0.000210	-0.00116
Mobile phone	0.00921***	0.000772	-0.00629**	-0.00166*	0.00356***	-0.00140	0.0120	0.0110**	0.00299***	0.000170
Safe fuel	0.000894	-0.00122	0.0198**	0.00643***	0.00587*	0.0154**	0.0251**	0.000521	-0.00141	-0.00851
Motorcycle	0.00546*	0.000187	-0.0108**	0.0102**	0.00431*	0.00986**	0.00568	0.00239	0.000225	0.000381
Piped water	-0.00768**	0.00178*	-0.00430	-0.00307	-0.00387**	0.00522	0.00316**	0.00110	0.00106	-0.0215**
Flushing toilet	0.0117**	0.0101***	0.0177**	0.00202	0.0207***	0.0337***	0.0116*	0.0174***	0.00100	0.0499**
Water treatment	0.00530*	0.000712*	-0.000444	-0.000414	0.00669***	0.00154	0.000439	0.00146**	0.0000976	-0.000280
Mother professional worker	-0.000798	0.00344**	0.00527***	0.000527	0.000675	0.00561*	-0.00289	0.00181	0.00481***	-0.0000289
Mother manual worker	0.00106	-0.00129	-0.0000352	0.00912**	0.000229	-0.00386	0.00681**	-0.000316*	0.00168**	-0.000677
Mother non-manual worker	-0.000371	0.000493	0.000296	-0.00180	-0.000659***	0.00377	-0.00316	-0.00408***	0.000158	-0.00274**
Mother farmer	0.00530*	-0.000753	-0.00743***	0.00190	0.00131	-0.000987	0.00439**	0.0161***	-0.000294	-0.00127
Father professional worker	0.00212	-0.00268**	-0.00139	0.0118	-0.0000585	0.00961	0.00467	-0.0116	0.0191**	-0.00368
Father manual worker	-0.00152	0.00247	0.00171	-0.0294	0.000927**	0.0303	-0.00164	-0.00439	-0.0322	0.000267
Father non-manual worker	0.000356	0.00372*	0.00614	0.0227	-0.00244	0.0142	0.00315	-0.0159	0.0582***	0.000856
Father farmer	-0.00241	-0.00299*	0.00161	0.00214	0.0121***	-0.0381	-0.00316	0.0361	-0.00432	0.00926
Watch TV less than once a week	0.00321	0.00123*	-0.000612	-0.000241	-0.00160*	-0.00161	-0.00200	-0.000192	-0.000355	-0.0000443
Watch TV once a week	0.00692	0.00287**	0.00880	0.000206	0.00782*	0.0135	0.0122	0.00186	-0.00201	0.0000480
Read newspaper less than once a week	0.00192	-0.000786	0.0188***	-0.000313	0.00125	-0.00150	0.00208	0.00299**	0.000230	0.0000579
Read newspaper once a week	0.000438	-0.0000597	0.00727**	-0.00279	0.00368	-0.00450	0.00638	0.000166	0.00202	0.000521
Listen to radio less than once a week	0.000559	0.00203	-0.00480***	-0.00258***	0.000211*	-0.0000319	0.0000596	-0.000660*	0.000613*	0.0000453
Listen to radio once a week	0.00148	0.00145	-0.00227**	0.00364**	0.000944	0.00474*	0.000918	-0.00301	-0.000495	-0.00337***
Observations	6988	3759	4807	3519	40200	4332	5616	10968	8800	5476

Standard errors are suppressed.
BGD: Bangladesh; NPL: Nepal; PAK: Pakistan; MDV: Malvides; IND: India
KHM: Cambodia; MMR: Myammar; TLS: East Timor; TJK: Tajikistan; KGZ: Kyrgyzstan.
Zero indicates that the distribution of correspoing variables does not have between-type difference.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 1.A.3: Descriptive statistics stratified by types

	BGD L adv	BGD M adv	NPL L adv	NPL M adv	PAK L adv	PAK M adv	MDV L adv	MDV M adv	IND L adv	IND M adv
	mean	mean	mean	mean	mean	mean	mean	mean	mean	mean
Child malnutrition	0.57	0.39	0.45	0.37	0.63	0.42	0.35	0.30	0.65	0.45
Mother BMI	20.61	22.74	21.27	21.50	22.85	24.75	24.36	23.89	20.18	22.43
Maternal education years	2.06	3.44	0.53	3.98	0.21	3.77	6.67	3.42	1.45	4.27
Paternal education years	1.75	3.62	2.22	3.26	2.00	3.80	4.19	3.49	2.37	4.40
Demography	0.08	-0.02	0.02	-0.07	0.00	-0.12	0.01	0.07	0.15	-0.15
Housing	-0.50	0.59	-0.12	-0.05	0.72	-0.91	-0.04	-0.01	-0.91	0.76
Affluence	-0.97	1.02	-0.13	-0.01	-0.78	0.93	-0.06	0.34	-1.18	1.05
Sanitation	-0.57	0.49	-0.38	-0.02	-0.64	0.46	-0.84	1.35	-1.10	0.78
Occupation	0.59	-0.64	0.04	-0.01	-0.21	0.23	-0.84	1.35	2.44	0.59
Media exposure	-0.64	0.66	-0.11	-0.12	-0.51	0.61	-0.02	0.03	-0.79	0.43
Mother age at birth	25.98	25.14	26.99	24.09	29.43	28.17	28.70	27.34	27.58	27.05
Family size	5.88	6.22	7.44	7.45	11.79	11.26	10.24	10.25	6.48	6.42
Number of children	1.38	1.30	1.69	1.55	2.94	2.61	2.10	2.04	1.88	1.61
Urban	0.17	0.48	0.54	0.62	0.30	0.64	0.07	0.25	0.10	0.39
Natural floors	0.00	0.00	0.12	0.09	0.01	0.01	0.00	0.00	0.17	0.04
Rudimentary floors	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.06	0.07
Finished floors	0.09	0.55	0.28	0.30	0.22	0.67	0.99	0.99	0.22	0.75
Natural walls	0.17	0.05	0.06	0.07	0.13	0.02	0.00	0.00	0.34	0.08
Rudimentary walls	0.13	0.04	0.52	0.52	0.31	0.06	0.00	0.00	0.15	0.08
Finished walls	0.68	0.91	0.42	0.41	0.46	0.90	0.93	0.92	0.50	0.83
Natural roofs	0.03	0.00	0.12	0.10	0.07	0.01	0.00	0.00	0.13	0.03
Rudimentary roofs	0.00	0.00	0.00	0.01	0.39	0.10	0.00	0.00	0.06	0.04
Finished roofs	0.96	1.00	0.88	0.89	0.40	0.44	1.00	0.99	0.49	0.76
Number of rooms	2.03	2.64	2.82	3.05	2.57	3.04	3.42	3.84	1.73	2.27
Radio	0.02	0.05	0.25	0.33	0.16	0.23	0.92	0.89	0.05	0.13
TV	0.17	0.72	0.47	0.48	0.48	0.84	0.98	0.99	0.30	0.87
Refrigerator	0.02	0.40	0.10	0.12	0.27	0.74	0.82	0.93	0.04	0.48
Bicycle	0.24	0.25	0.45	0.43	0.28	0.29	0.56	0.49	0.51	0.44
Electricity	0.39	0.87	0.88	0.91	0.92	0.99	1.00	1.00	0.73	0.98
Car	0.00	0.02	0.04	0.06	0.04	0.17	0.03	0.06	0.01	0.12
Mobile phone	0.83	0.98	0.96	0.98	0.84	0.96	0.98	0.99	0.86	0.98
Safe fuel	0.02	0.26	0.17	0.20	0.19	0.66	0.89	0.95	0.07	0.54
Motorcycle	0.03	0.15	0.19	0.16	0.28	0.55	0.25	0.50	0.17	0.56
Piped water	0.01	0.11	0.23	0.29	0.24	0.41	0.06	0.13	0.11	0.42
Flushing toilet	0.05	0.32	0.68	0.81	0.58	0.93	0.95	0.99	0.19	0.71
Water treatment	0.03	0.17	0.17	0.17	0.03	0.13	0.49	0.48	0.21	0.55
Mother professional worker	0.01	0.04	0.04	0.01	0.01	0.04	0.05	0.19	0.00	0.03
Mother manual worker	0.03	0.09	0.03	0.05	0.07	0.03	0.21	0.04	0.06	0.04
Mother non-manual worker	0.03	0.01	0.07	0.06	0.07	0.07	0.09	0.19	0.03	0.04
Mother farmer	0.22	0.06	0.47	0.49	0.12	0.02	0.02	0.01	0.23	0.04
Father professional worker	0.02	0.12	0.06	0.04	0.05	0.18	0.09	0.27	0.02	0.12
Father manual worker	0.16	0.53	0.40	0.42	0.54	0.38	0.49	0.00	0.34	0.31
Father non-manual worker	0.21	0.26	0.31	0.35	0.21	0.35	0.04	0.71	0.12	0.33
Father farmer	0.42	0.04	0.19	0.15	0.17	0.07	0.37	0.00	0.48	0.17
Watch TV less than once a week	0.14	0.03	0.23	0.25	0.25	0.16	0.02	0.01	0.13	0.04
Watch TV once a week	0.22	0.81	0.38	0.42	0.28	0.69	0.97	0.97	0.33	0.88
Read newspaper less than once a week	0.04	0.15	0.18	0.23	0.05	0.38	0.38	0.31	0.08	0.22
Read newspaper once a week	0.01	0.12	0.05	0.03	0.00	0.09	0.20	0.34	0.03	0.32
Listen to radio less than once a week	0.01	0.03	0.28	0.36	0.11	0.19	0.08	0.10	0.05	0.06
Listen to radio once a week	0.01	0.04	0.23	0.29	0.03	0.05	0.84	0.77	0.05	0.12
Observations	3665	3323	2172	1587	2657	2150	960	1361	19782	20418

L adv: Least advantaged type; M adv: Most advantaged type
BGD: Bangladesh; NPL: Nepal; PAK: Pakistan; MDV: Maldives; IND: India
KHM: Cambodia; MMR: Myanmar; TLS: East Timor; TJK: Tajikistan; KGZ: Kyrgyzstan

Table 1.A.4: Descriptive statistics stratified by types

	KHM L adv	KHM M adv	MMR L adv	MMR M adv	TLS L adv	TLS M adv	TJK L adv	TJK M adv	KGZ L adv	KGZ M adv
	mean	mean	mean	mean	mean	mean	mean	mean	mean	mean
Child malnutrition	0.49	0.38	0.43	0.23	0.76	0.66	0.38	0.32	0.27	0.15
Mother BMI	21.67	22.46	22.24	24.10	20.16	21.06	22.91	23.43	23.94	23.59
Maternal education years	2.47	3.39	1.09	2.63	2.55	5.18	5.00	5.11	4.78	2.47
Paternal education years	2.78	3.64	1.24	3.35	2.16	3.10	5.56	5.21	4.84	6.14
Demography	-0.17	0.24	0.00	0.20	0.01	0.08	0.18	-0.17	-0.90	-0.01
Housing	-0.87	0.68	-1.04	1.61	-0.53	0.85	-0.23	0.20	-1.02	0.24
Affluence	-1.08	0.89	-1.10	2.00	-0.76	1.17	-0.16	0.24	0.28	-0.02
Sanitation	-0.98	0.51	0.24	-2.83	-1.03	1.57	-0.38	0.09	3.56	-0.59
Occupation	-1.34	1.26	-0.39	-0.21	-1.03	1.57	-1.19	1.34	-0.27	0.02
Media exposure	-0.52	0.27	-0.40	0.56	-0.40	0.49	-0.21	0.09	0.32	0.24
Mother age at birth	28.61	28.53	31.35	30.81	32.08	30.15	27.28	27.77	29.93	28.29
Family size	5.51	6.17	6.64	7.15	8.06	8.26	10.50	9.77	5.66	6.62
Number of children	1.55	1.51	1.68	1.41	2.20	2.24	2.73	2.53	1.67	1.77
Urban	0.06	0.47	0.11	0.75	0.10	0.39	0.17	0.37	0.81	0.19
Natural floors	0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.00	0.00	0.00
Rudimentary floors	0.85	0.49	0.71	0.32	0.04	0.02	0.21	0.22	0.06	0.25
Finished floors	0.06	0.45	0.17	0.64	0.20	0.57	0.43	0.56	0.93	0.71
Natural walls	0.27	0.06	0.12	0.00	0.77	0.47	0.12	0.10	0.00	0.21
Rudimentary walls	0.13	0.14	0.74	0.32	0.04	0.03	0.38	0.25	0.06	0.44
Finished walls	0.58	0.80	0.13	0.67	0.19	0.50	0.50	0.65	0.53	0.33
Natural roofs	0.12	0.01	0.50	0.02	0.31	0.11	0.04	0.03	0.00	0.00
Rudimentary roofs	0.00	0.00	0.02	0.00	0.01	0.00	0.00	0.00	0.06	0.00
Finished roofs	0.87	0.98	0.47	0.98	0.67	0.88	0.91	0.89	0.93	1.00
Number of rooms	1.33	1.93	1.63	2.45	2.61	2.98	3.35	3.30	2.09	2.41
Radio	0.26	0.42	0.23	0.33	0.29	0.47	0.23	0.24	0.25	0.36
TV	0.37	0.85	0.33	0.95	0.06	0.43	0.97	0.98	1.00	0.99
Refrigerator	0.00	0.21	0.03	0.56	0.01	0.13	0.37	0.59	0.90	0.74
Bicycle	0.46	0.59	0.27	0.58	0.05	0.22	0.24	0.29	0.15	0.25
Electricity	0.26	0.83	0.39	0.96	0.18	0.64	0.99	1.00	1.00	0.99
Car	0.14	0.22	0.02	0.26	0.00	0.06	0.41	0.50	0.65	0.57
Mobile phone	0.80	0.95	0.49	0.98	0.27	0.72	0.94	0.97	1.00	0.99
Safe fuel	0.02	0.33	0.05	0.60	0.00	0.03	0.60	0.70	0.99	0.63
Motorcycle	0.58	0.84	0.38	0.68	0.03	0.27	0.01	0.03	0.02	0.02
Piped water	0.03	0.26	0.09	0.01	0.12	0.27	0.30	0.49	0.99	0.37
Flushing toilet	0.25	0.78	0.25	0.71	0.21	0.58	0.07	0.20	0.85	0.00
Water treatment	0.61	0.69	0.74	0.18	0.85	0.90	0.81	0.82	0.55	0.50
Mother professional worker	0.00	0.08	0.04	0.12	0.00	0.05	0.04	0.08	0.16	0.16
Mother manual worker	0.06	0.22	0.31	0.13	0.02	0.03	0.13	0.09	0.02	0.03
Mother non-manual worker	0.05	0.38	0.09	0.29	0.05	0.14	0.03	0.06	0.13	0.07
Mother farmer	0.67	0.05	0.10	0.02	0.41	0.12	0.02	0.02	0.01	0.03
Father professional worker	0.01	0.16	0.05	0.20	0.01	0.39	0.00	0.29	0.40	0.15
Father manual worker	0.09	0.49	0.69	0.50	0.01	0.15	0.98	0.00	0.37	0.39
Father non-manual worker	0.02	0.26	0.05	0.22	0.00	0.41	0.00	0.59	0.20	0.13
Father farmer	0.88	0.07	0.18	0.06	0.97	0.03	0.00	0.07	0.01	0.31
Watch TV less than once a week	0.20	0.12	0.21	0.12	0.09	0.09	0.15	0.11	0.00	0.00
Watch TV once a week	0.30	0.75	0.32	0.84	0.14	0.55	0.78	0.85	0.98	0.99
Read newspaper less than once a week	0.09	0.23	0.14	0.32	0.13	0.23	0.19	0.19	0.28	0.29
Read newspaper once a week	0.01	0.10	0.05	0.33	0.10	0.28	0.22	0.31	0.50	0.51
Listen to radio less than once a week	0.25	0.25	0.17	0.18	0.13	0.15	0.16	0.14	0.19	0.22
Listen to radio once a week	0.18	0.30	0.14	0.21	0.20	0.48	0.20	0.27	0.40	0.32
Observations	2077	2255	1525	763	6708	4260	4663	4137	475	737

L adv: Least advantaged type; M adv: Most advantaged type
BGD: Bangladesh; NPL: Nepal; PAK: Pakistan; MDV: Maldives; IND: India
KHM: Cambodia; MMR: Myanmar; TLS: East Timor; TJK: Tajikistan; KGZ: Kyrgyzstan

Chapter 2

A Copula-based Measure of Inequality of Opportunity: With an Application to Health Inequality in Body Mass in Indonesia

Abstract

Inequality of opportunity (IOP) is an illegitimate form of inequality whereby causes are not attributable to individual choices. Non-parametric and parametric methods have been widely used to measure IOP, but each of them tends to have a significant technical concern when the sources of inequality are not categorical and multi-dimensional, and when a severe specification error is suspected respectively. This paper introduces an alternative approach to measuring ex-post IOP using copulas which model various structures of dependence between efforts, factors for which individuals are responsible, and circumstances, factors beyond individual's control. Semi-parametric estimation of the outcome distribution and flexible modelling of the dependence structure between efforts and circumstances help to overcome the concerns in the existing methods. The method is applied to measuring inequality in the body mass index (BMI), attributable to its intergenerational transmission from parents to their adult offspring in Indonesia. A 21-year gap between the first wave and the last wave of the Indonesian Family Life Survey is exploited to estimate the relationship between parental BMI when offspring are below 15 years old and the BMI of grown-up offspring. The results show that 15.1 per cent of the overall variance in grown-up children's BMI is associated with intergenerational BMI transmission.

JEL codes: I14, I18

Keywords: inequality of opportunity, copulas, intergenerational mobilities of BMI, Indonesia

2.1 Introduction

There is a large volume of literature on the measurement of inequality of opportunity (IOP). From the viewpoint of responsibility sensitive egalitarianism (Dworkin, 1981a,b; Cohen, 2011; Arneson, 1989; Roemer, 1996), not all kinds of inequalities are ethically objectionable, and two types of inequality can be defined. One is legitimate inequality, in which differences in outcomes are attributable to individual responsibility, and the other is illegitimate inequality, in which outcome differences cannot be attributed to individual responsibility. A typical example of the former is differences in body mass across individuals with different lifestyles, caused, for instance, by dietary choices and daily exercise habits. Such inequality is considered morally 'fair', because the difference in the consequential health outcome is the result of an individual's freedom of choice. On the other hand, illegitimate inequality is typically exemplified by different mortality rates across different backgrounds, such as genetics, parental social class and race, and inequality attributable to factors beyond the scope of individual control is viewed as 'unfair'. This distinction suggests that equality in outcomes is not always the most equitable or justifiable situation, because it could fail to hold people accountable for things for which they should be held partially responsible. In other words, the unobjectionable situation for responsibility-sensitive egalitarians is not a perfectly equal distribution of the outcome per se but a distribution in which differences in the outcomes, due to individuals' voluntary choices, remain, and any disparity owing to circumstances beyond their control is appropriately compensated. The question of 'inequality of what' supports the equality of opportunity (EOP) concept, which is achieved when all people, regardless of their circumstances, have the same chances of achieving an outcome. In this sense, EOP is popularly formulated in the 'level-the-playing-field' metaphor (Roemer, 1996; Rosa Dias and Jones, 2007).

In the literature on EOP, Roemer (1996, 1998, 2002) established a seminal framework in which all the factors influencing individual attainment can be sorted into effort factors and circumstance factors. Effort factors are those for which individuals are held partially responsible, while circumstance factors are those that are beyond an individual's control and are sources of unjustifiable inequality. In the context of health, effort factors typically include lifestyle choices such as diet, smoking, alcohol consumption, exercise and so forth, and disparities that arise unavoidably are typically considered neutral. Circumstances, on the other hand, are typified by race, family socioeconomic and biological background and so on. As Roemer (1996) argues, effort factors are

not necessarily independent of circumstance factors, as some of the former can be more or less influenced by some of the latter. A typical example is that smoking behaviours are more or less affected by parents' smoking behaviours and socio-economic status. Such effects are assumed to be a part of circumstances in the Roemer framework.[1]

As described above, we can define conceptually both legitimate and illegitimate inequality. The question, however, is how we should measure overall illegitimate inequality. This paper proposes a new method for measuring IOP. The key features of the proposed method are twofold: first, copula functions model a structure of dependence between efforts and circumstances, making it possible to express simply the hypothetical outcome distribution in a situation where EOP is achieved. Second, the proposed method generates a hypothetical outcome from the semi-parametrically estimated outcome distribution function, without relying on a linear functional form of the outcome.

This paper illustrates the application of the proposed method to the measurement of IOP in relation to individuals' body mass in Indonesia. This application measures the inequality in body mass index (BMI) of early adults aged between 20 and 35, with respect to the intergenerational transmission of BMI from their parents. The results show that 15.1 per cent of the overall variance is associated with the IOP. Last of all, the estimated results are compared with the ones estimated by the traditional parametric method.

2.2 Related literature

The idea of EOP embodies two fundamental principles. The reward principle demands that inequalities, due to different levels of effort, should be considered acceptable, and the compensation principle claims that inequalities, due to circumstances, should be eliminated. The compensation principle has two approaches, namely the ex-ante approach and the ex-post approach (Fleurbaey and Peragine, 2013; Pignataro, 2012; Li Donni et al., 2014). The ex-ante approach promotes equality of outcomes among those facing the same circumstances, by making their average outcomes as equal as possible. Ex-ante EOP is achieved when the set of opportunities is the same for all people, irrespective of their circumstances. The ex-post approach, on the other hand, en-

[1] On the other hand, from the viewpoint of Swift (2005), circumstances only include past variables that are independent of efforts (Jusot et al., 2013). Jusot et al. (2013) discuss whether these differences in normative principles matter empirically.

courages the compensation of inequality caused by different circumstantial factors among those exerting the same level of effort. When the outcome does not vary across people with different circumstances at any given effort level, ex-post EOP is achieved.[2]

At the stage of testing the existence of the IOP, the ex-ante and ex-post compensation approaches can be divided further into two approaches, namely a non-parametric method and a parametric method. The non-parametric method defines *types*, i.e. a set of people with the same circumstances, and *tranches*, i.e. a set of people exerting the same efforts. An ex-ante non-parametric approach typically tests the existence of IOP by applying the stochastic dominance test for outcome distributions across different *types* defined by parental socio-economic status, etc. (e.g. Peragine and Serlenga, 2007; Lefranc et al., 2008, 2009; Aaberge et al., 2011). An ex-post non-parametric approach measures between-*type* inequality among those who belong to the same *tranche*. Checchi and Peragine (2010) proposed non-parametric ways to measure IOP from the ex-ante and ex-post perspectives.

The advantage of the non-parametric method is that it does not make any assumptions about the functional form of the outcome variable. However, the precision of the estimation deteriorates as the number of circumstance factors becomes large, because the exponentially growing number of *types* often leads to very few observations in each *type* and unreliably imprecise estimates. Besides, as the number of effort variables increases, the number of possible *tranches* that needs to be considered is also exponentially growing. The situation becomes even worse when circumstance and effort variables are continuous. When circumstance variables are continuous, discrete *types* can be still defined, but continuous information of circumstance variables tend to be lost and full information of circumstances are not exploited.[3]

On the other hand, the parametric method allows us to define finer categories of *types*, relying on a functional form assumption of the outcome variable. The parametric method mostly

[2]However, it is known that the ex-ante and the ex-post approaches are logically incompatible in a general setting in which circumstances and efforts are not additively separable in the outcome function (Fleurbaey and Peragine, 2013). This stems from incompatibility between the reward principle and the compensation principle in a general setting. For further discussion, see, for example, Ferreira and Peragine (2015).

[3]Li Donni et al. (2015) proposed a way to define endogenously finite types with a latent class approach and deal with the multidimensionality of circumstances. O'Neill et al. (1999) proposed a method to deal with the continuity of circumstance variables, using kernel estimation. Aizawa (2019) used the clustering approach to taking into account the multidimensionality of circumstances. These studies implicitly take into account unobservable circumstances.

employs a regression-based approach, and the measurement of IOP is implemented through two steps. The first step estimates a conditional mean function of the outcome variable and obtains estimates of the effects of circumstances and efforts, following which the parametric approach constructs a counterfactual outcome distribution by suppressing the valuations of circumstances. The second stage indirectly measures the IOP by comparing the inequality level between the observed distribution and the derived hypothetical distribution. This indirect approach is based on the spirit of fairness gap proposed by Fleurbaey and Schokkaert (2009).[4]

In contrast to the non-parametric model, the parametric method can handle a large number of continuous circumstance and effort variables in a multivariate regression model. Bourguignon et al. (2007), for example, first construct a linear structural model, as shown in equation (2.1).

$$\begin{cases} Y_i = \alpha + \beta_C C_i + \beta_E E_i + \epsilon_i^Y \\ E_i = \gamma + \delta C_i + \epsilon_i^E, \end{cases} \tag{2.1}$$

where Y_i is an outcome variable. E_i and C_i are efforts and circumstances, respectively. ϵ_i^Y and ϵ_i^E are error terms. When we compute the hypothetical outcome in which the variation of circumstances is suppressed, one needs to take into account the indirect effect of C on E. To address this matter, Bourguignon et al. (2007) estimate the reduced form of the structural equation, as shown in equation (2.2).

$$Y_i = \underbrace{(\alpha + \beta_E \gamma)}_{\zeta} + \underbrace{(\beta_C + \beta_E \delta)}_{\kappa} C_i + \underbrace{(\beta_E \epsilon_i^E + \epsilon_i^Y)}_{\xi_i}. \tag{2.2}$$

In equation (2.2), $\kappa = (\beta_C + \beta_E \delta)$, which is the coefficient of C, reflects the direct and indirect partial effects of C on Y. Conceptually the direct effect means the partial effect of C on Y after controlling for E. The indirect effect is the partial impact of C on Y through E.

Finally, in order to quantify the IOP, Bourguignon et al. (2007) predict an individual outcome under the hypothetical situation in which C is constant, and compares the Gini coefficient of the original distribution with that in a hypothetical situation in which everybody faces the same level circumstances. For example, when C is fixed at its sample mean level, $C_i = \overline{C}$ for all i, the hypothetical individual outcome is estimated by $Y_i^{EOP} = \widehat{\zeta} + \widehat{\kappa}\overline{C} + \widehat{\xi}_i$. By subtracting the inequality

[4]For a more detailed review of the direct approach, see, for example, Ramos and Van de gaer (2016).

level of the hypothetical distribution Y^{EOP} from the that of the original outcome distribution Y, the parametric method quantifies the degree of IOP. This type of indirect quantification is based on the fairness gap approach (Fleurbaey and Schokkaert, 2009), which is widely used in empirical studies (Pistolesi, 2008; Salehi-Isfahani et al., 2014; Belhaj Hassine, 2012).[5]

However, contrary to the non-parametric method, the parametric approach may suffer specification errors. In the context of health, normally there is little consensus about the functional form of the health outcome variable. Furthermore, not all of the continuous health outcome variables can be best specified in a linear model; for example, medical care expenditure often shows a heavily skewed distribution.[6] In the presence of a significant specification error, the estimated IOP can be biased downwards because the model cannot model satisfactorily an association between circumstances and an outcome.[7] Pistolesi (2008) proposes a semi-parametric method which does not rely on the linearity assumption. Pistolesi (2008) estimates the conditional distributions of outcome and efforts by employing the duration model (Foresi and Peracchi, 1995), and from the estimated conditional distribution, the author predicts the counterfactual individual hypothetical outcome level by assuming that the individual effort and the outcome in the hypothetical situation, where variation in C is suppressed, are predicted at the same quantiles as those in the actual distributions. This rank preservation assumption implies that while the circumstances can change the absolute values of efforts and outcomes of individuals, circumstances do not influence the relative ranks/positions of individuals in their respective distributions.

Comprehensive reviews of empirical studies on EOP are found in Pignataro (2012); Ramos and Van de gaer (2012, 2016); Ferreira and Peragine (2015); and Roemer and Trannoy (2016). Amongst them, Roemer and Trannoy (2016) present a detailed discussion on the broad concept of EOP, while Ramos and Van de gaer (2016) provide a concise, recent review of the methodologies from the viewpoint of both ex-ante and ex-post approaches. For the health context, Fleurbaey

[5]The fairness gap approach is normatively consistent with the ex-post approach. The other direct quantification method, which calculates the inequality after removing the effect of all legitimate variables, is called the direct unfairness approach, and it is consistent with the ex-ante approach. For more details, see Fleurbaey and Schokkaert (2009).

[6]When the nonlinear specification is appropriate, the reduced form of a structural model is rarely used; instead, a structural recursive nonlinear model with a multivariate normality assumption of error terms is often employed (Rosa Dias, 2010; García-Gómez et al., 2015).

[7]When there is a significant specification error, the outcome variation that should have been attributable to circumstances would be captured by the error term. As the error term is not regarded as the sources of illegitimate inequality in the Roemer framework and its contribution to the outcome is differenced-out when we indirectly quantify the IOP using the fairness gap approach, the estimated IOP can be smaller than the IOP estimated with the correctly-specified model.

and Schokkaert (2012) provide extensive discussions.

This paper addresses the aforementioned shortcomings of the parametric and the non-parametric methods by introducing a new alternative method for measuring ex-post IOP. Instead of making a parametric assumption on the conditional expectation function of the outcome variable – as in equation (2.1) – it imposes an assumption on its conditional distribution function as is done in Pistolesi (2008). We use copula functions to model the dependence structure between circumstances and efforts. Copulas capture a wide range of types of dependence structures by a function. Then hypothetical outcomes when the variation of circumstances are suppressed will be simulated, taking account of the estimated original dependence structure. The method rests on the semi-parametric ex-post compensation approach.

Specifically, the proposed method has a number of advantages over the traditional approaches. The proposed method is in particular of use when circumstance and effort variables are continuous. The advantage over the non-parametric method is that circumstances and efforts do not have to be discrete in order to define *types* and *tranches*. Therefore information from continuous variables is fully exploited herein. Contrary to the traditional parametric method, the proposed method does not impose a linearity assumption for the conditional mean of the outcome variable, which allows for heterogeneous impacts of circumstances on the distribution of the outcome.

2.3 Model setting

This paper uses the following notation: $Y \in \mathbb{R}_+$ is a non-negative, measurable, interpersonally comparable and one-dimensional outcome variable that has a distribution $F_Y(y)$. $C = (C_1, ..., C_p)' \in \mathbb{R}^p$ denotes the vector of circumstances, and $E = (E_1, ..., E_q)' \in \mathbb{R}^q$ stands for the vector of efforts. $X = (C', E')' \in \mathbb{R}^{p+q}$ is a vector of potential determinants of Y. We consider a general model of the form

$$Y = m(X, \eta) = m(C, E, \eta), \tag{2.3}$$

where η is an unobservable factor. Following the Roemer framework (Roemer, 1998), we make the following assumption on the distinction between E and C.

Assumption 1 *Unambiguous responsibility-cut and measurability of efforts and circumstances:*

X can be divided unambiguously into E and C and both E and C are measurable.

This assumption states that the determinants of Y can be sorted into either E or C, but it does not require the independence between E and C.[8]

$F_X(x)$, $F_E(e)$, $F_C(c)$ are joint distributions of X, efforts and circumstances, respectively. $f_X(x)$, $f_E(e)$ and $f_C(c)$ stand for their respective joint densities. $F_{E_m}(e_m)(m = 1, ..., q)$ and $F_{C_n}(c_n)(n = 1, ..., p)$ are marginal distributions of E_m and C_n, and $f_{E_m}(e_m)$ $(m = 1, ..., q)$ and $f_{C_n}(c_n)$ $(n = 1, ..., p)$ are their marginal densities. Finally, $v(F_Y(y))$, where $v : F_Y \mapsto \mathbb{R}$, is an inequality statistic.

Applying the law of iterated expectation, the association between the outcome and its determinants is expressed as follows (Pistolesi, 2008):

$$F_Y(y) = \int_X F_{Y|X}(y|x)dF_X(x) \tag{2.4}$$

$$= \int_C \int_E F_{Y|E,C}(y|e,c)dF_{E|C}(e|c)dF_C(c), \tag{2.5}$$

where $F_{Y|X}(y|x)$ and $F_{Y|E,C}(y|e,c)$ are conditional distributions of the outcome, given X, and given E and C, respectively, while $F_{E|C}(e|c)$ is a conditional distribution of E given C. Equation (2.5) reflects the argument that E is influenced by C (Roemer, 1996).

2.3.1 Copulas

In general, multivariate joint distributions can be expressed by their marginal distributions and a copula. Hence, copulas allow us to model dependence structures of variables from their marginal distributions, and they have the property that a functional association between underlying variables is not influenced by their marginal behaviours. Sklar's theorem states that for an n-variate distribution function $F(y_1, ..., y_n)$, the copula is a distribution function $\mathbb{C} : [0, 1]^n \mapsto [0, 1]$ that satisfies, for all real values of $y_1, .., y_n$,

$$F(y_1, ..., y_n) = \mathbb{C}(F_1(y_1), ..., F_n(y_n); \theta), \tag{2.6}$$

[8]Other than efforts and circumstances, Lefranc et al. (2009) build up the model where outcomes are assumed to be dependent not only on efforts and circumstances, but also on luck, which is a set of random factors that affect all individuals' decision makings. However, in this study, following the original Roemer framework, we assume that X can be divided into E and C only. This implies that in the proposed model the random factors are included in efforts.

where $\theta \in \Theta$ is a parameter vector of the copula. If $F_1, .., F_n$ are continuous, the copula can be uniquely expressed.[9] Alternatively, equation (2.6) can be expressed as

$$\mathbb{C}(u_1, ..., u_n; \theta) = F(F_1^{-1}(u_1), ..., F_n^{-1}(u_n)). \tag{2.7}$$

Every copula has its derivative (Nelsen, 2006). For example, let $\mathbb{C}(u_1, u_2)$ be a bivariate copula. For any u_2, the partial derivative $\frac{\partial \mathbb{C}(u_1,u_2)}{\partial u_1}$ exists for almost all u_1. For such u_1 and u_2, the range of its derivative is

$$0 \leq \frac{\partial \mathbb{C}(u_1, u_2)}{\partial u_1} \leq 1. \tag{2.8}$$

Similarly, the partial derivative $\frac{\partial \mathbb{C}(u_1,u_2)}{\partial u_2}$ exists for almost all u_2.

Although a long list of copulas has been proposed in the statistical literature, we focus only on the several examples that are frequently used in applied econometrics. Table 2.1 lists the functions of copulas typically employed in empirical studies, their parameter ranges and the number of parameters. First, the Product copula, which takes the simplest form, does not allow dependences among marginal distributions and consequently does not have a dependence parameter. Second, the Frank copula is capable of modelling both positive and negative dependence and is suitable for data that exhibit weak tail dependence (Trivedi and Zimmer, 2007). Third, the Clayton copula can better capture strong left tail dependence and relatively weak right tail dependence, but it is not suitable for negative dependence. As its dependence parameter goes to 0, the marginal distributions become independent. Fourth, the Gumbel copula, similar to the Clayton copula, is not able to model negative dependence, but it can better capture strong right tail dependence and relatively weak left tail dependence. Fifth, the Joe copula also allows only positive dependence and is appropriate to model the very strong right tail dependence and relatively weak left tail dependence between marginals. Finally, the Gaussian copula is flexible in the sense that it allows for equal degrees of positive and negative dependence. It can accommodate at most $n(n-1)/2$ parameters, which reflect pairwise correlations. The multiple parameters of the Gaussian copula help model various dependence structures between marginals. For more details about copulas in general, see Nelsen (2006). For their econometric applications,

[9] $y_1, .., y_n$ need to be continuous so that copula expression is always uniquely expressed. It is agreed that while the lack of uniqueness of copula presentation for discrete distributions is a theoretical issue, it does not inhibit empirical application for the estimation of joint distribution functions (Trivedi and Zimmer, 2007).

Table 2.1: Copulas

Copula	$C(u_1, ..., u_n)$	Parameter range	Number of parameters
Product	$\prod_{i=1}^{n} u_i$	NA	0
Frank	$-\frac{1}{\theta} ln(1 + \frac{\prod_{i=1}^{n} exp(-\theta u_i)-1}{(exp(-\theta)-1)^{n-1}})$	$(-\infty, \infty)$	1
Clayton	$(\sum_{i=1}^{n} u_i^{-\theta} - n + 1)^{-\frac{1}{\theta}}$	$(0, \infty)$	1
Gumbel	$exp[-[\sum_{i=1}^{n}(-ln(u_i))^{\theta}]^{\frac{1}{\theta}}]$	$[1, \infty]$	1
Joe	$1 - [1 - \prod_{i=1}^{n}(1 - (1 - u_i)^{\theta})]^{\frac{1}{\theta}}$	$[1, \infty]$	1
Gaussian	$\Phi_n(\Phi^{-1}(u_1), ..., \Phi^{-1}(u_n); \theta)$	$[-1, 1]$	$n(n-1)/2$

Note: $\Phi_n(.;\theta)$ is a multivariate normal distribution function with correlation parameters θ. $\Phi(.)$ is a univariate normal distribution function.

see Trivedi and Zimmer (2007).

2.4 Methodology

2.4.1 Hypothetical outcome distribution

In order to measure the illegitimate inequality attributable to circumstances, we obtain a hypothetical outcome distribution whereby variations of circumstances are suppressed. Let $F_Y^{EOP}(y)$ be the hypothetical outcome distribution in which people are faced with the same circumstances and consequently, by definition, there exists no IOP. Therefore, $v(F_Y^{EOP}(y))$ is viewed as legitimate inequality according to the opportunity-egalitarian view. Next, following the fairness gap approach (Fleurbaey and Schokkaert, 2009), we indirectly quantify the absolute level of IOP by subtracting it from the inequality of the observed outcome distribution, $\Delta = v(F_Y(y)) - v(F_Y^{EOP}(y))$. We also measure the relative level of IOP by $1 - \frac{v(F_Y^{EOP}(y))}{v(F_Y(y))}$.

In the hypothetical situation, everybody has the same constant circumstance, denoted as $\widetilde{C}$, which are called the reference values of C, such as their sample means. We make the following assumption about the range of the reference levels.

Assumption 2 *Restricted range of the reference values of C:*
$\widetilde{C}$ needs to be contained in the observed support of C.

This assumption states that the reference values need to be set between the maximum and the minimum C, which guarantees that marginal distributions $F_{C_n}(\widetilde{c_n})$ $(n = 1, ..., p)$ lie between 0 and 1. This assumption is essential in modelling the joint distribution of E and C, when C is evaluated at $\widetilde{C}$. This assumption corresponds to the common support assumption used in the

parametric method that requires that $\widetilde{C}$ should be in the support of C.

Next, exploiting the property of copulas that a functional association between underlying variables is not influenced by their marginal behaviours, we assume that the dependence structure between efforts and circumstances does not change when we manipulate the marginal distribution of C.

Assumption 3 *Stability of dependence structure:*
The original copula functional form and its parameter vector θ are stable when the marginal distributions of circumstances are manipulated.

This assumption states that original functional associations between underlying variables are not influenced by the manipulations of their marginal distributions. The important point to note is that it does not require that the distribution of E is unaffected when we manipulate the distribution of C. In contrast, this assumption ensures that the effect of C on E will be considered through the estimated copula function when we construct the hypothetical distribution of Y. In the parametric case, this assumption corresponds to the exogeneity assumption requiring that C is independent of ϵ^E in equation (2.1).

Under the assumptions above, the following proposition holds.

Proposition 1 *The actual outcome distribution and the hypothetical outcome distribution can be expressed using copulas as follows:*

$$F_Y(y) = \int_C \int_E F_{Y|E,C}(y|e,c)\Psi(e,c)dF_E(e)dF_C(c) \tag{2.9}$$

$$F_Y^{EOP}(y) = \int_E F_{Y|E,C}(y|e,c=\widetilde{c})\Psi(e,c=\widetilde{c})dF_E(e), \tag{2.10}$$

where $\Psi(e,c) = \frac{dF_{E|C}}{dF_E} = \frac{c(F_{E_1}(e_1),...,F_{E_q}(e_q),F_{C_1}(c_1),...,F_{C_p}(c_p);\theta)}{c_e(F_{E_1}(e_1),...,F_{E_q}(e_q);\theta_e)c_c(F_{C_1}(c_1),...,F_{C_p}(c_p);\theta_c)}$ *is a weighting factor.* $c(F_{E_1}(e_1),...,F_{E_q}(e_q),\ F_{C_1}(c_1),...,F_{C_p}(c_p);\theta)$, $c_e(F_{E_1}(e_1),...,F_{E_q}(e_q);\theta_e)$ *and* $c_c(F_{C_1}(c_1),...,F_{C_p}(c_p);\theta_c)$ *are copula density functions.*

Proof of the proposition is provided in the Appendix.

The weighting factor, $\Psi(e,c) = \frac{dF_{E|C}}{dF_E}$, in equation (2.9) is basically a ratio between the conditional effort distribution given circumstances and the unconditional effort distribution. The

use of weighting factor to estimate a counterfactual distribution was firstly proposed by DiNardo et al. (1996) in the context of the income inequality analysis. Intuitively, $\Psi(e, c = \widetilde{c})$ in equation (2.10) re-weights the observations so that the weighted observations will have the distribution of E that would be realised when the variation of C is suppressed. In other words, the indirect impact of levelling the circumstances on efforts are taken into consideration through this weighting factor. Therefore, $\Psi(e, c = \widetilde{c})$ makes sure that $F_Y^{EOP}(y)$ is the hypothetical outcome distribution after taking account of the original dependence structure between E and C.

Based on the spirits of fairness-gap measurement approach (Fleurbaey and Schokkaert, 2009), inequality due to circumstances is then indirectly measured by calculating the difference between inequality of the original outcome distribution and that of the hypothetical outcome distribution, i.e. $\Delta = v(F_Y(y)) - v(F_Y^{EOP}(y))$. As discussed above, $v(F_Y^{EOP}(y))$ can be seen as legitimate inequality attributable to the different levels of efforts, and Δ can be regarded as the contribution of specific circumstances to overall inequality (Ferreira and Gignoux, 2011; Trannoy et al., 2010).

This method does not make a functional form assumption about the conditional mean of outcome, which implies that we are not assuming that efforts and circumstances are additively separable in the outcome function. It is known that the size of estimated IOP can vary according to the reference values of circumstances when outcomes are not additively separable into efforts and circumstances (Fleurbaey and Schokkaert, 2009, 2012).[10] Certainly, the set of the mean values of circumstances, i.e. $\widetilde{C} = \overline{C}$, is a common choice in the empirical literature, but there is some arbitrariness left. It is recommended to calculate IOP by setting various reference values and then taking their average as part of a sensitivity analysis (Ramos and Van de gaer, 2012).

2.4.2 Estimation

Replacing the functions in equation (2.10) with their respective estimators and taking the average across E,

$$\begin{aligned} \widehat{F}_Y^{EOP}(y) &= \int_E \widehat{F}_{Y|E,C}(y|e, c = \widetilde{c})\widehat{\Psi}(e, c = \widetilde{c})d\widehat{F}_E(e). \\ &= \frac{1}{N}\sum_{i=1}^{N} \widehat{F}_{Y|E,C}(y|e = e_i, c = \widetilde{c})\widehat{\Psi}(e = e_i, c = \widetilde{c}). \end{aligned} \tag{2.11}$$

[10] This means that when the additive separability of E and C in the outcome is imposed, the choice of reference values does not make a difference.

For the derivation of the hypothetical distribution of Y, we first need to estimate $F_{Y|E,C}(y|e,c)$, the conditional distribution function and the weighting factor.

Estimation of the conditional distribution function

First, the conditional distribution function, $F_{Y|E,C}(y|e,c)$, is estimated by employing the distributional regression method (Foresi and Peracchi, 1995; Chernozhukov et al., 2013).[11] The basic idea behind the distributional regression is to model directly the conditional distribution of Y, given E and C, through a family of binary response models in the event that the dependent variable y_i exceeds each threshold of Y, which we denote as q_Y^j $(j = 1, ..., J)$.[12] In essence, the distributional regression estimates the consistent conditional distribution by approximating it by a bunch of step functions.

The conditional distribution of Y at the j_{th} threshold is given by

$$F_{Y|E,C}(q_Y^j|e,c) = P(y \leq q_Y^j|e,c) = \Lambda(\alpha^j + e'\beta_E^j + c'\beta_C^j + I(e,c)'\beta_{EC}^j), \tag{2.12}$$

where $\Lambda(.)$ is a strictly increasing link function[13], and $I(e,c)_i$ is a vector of two-way interaction terms between E and C. Equation (2.12) is estimated by making an assumption on $\Lambda(.)$. The coefficients α^j and $\beta^j = (\beta_E^{j\prime}, \beta_C^{j\prime}, \beta_{EC}^{j\prime})'$ are allowed to vary with the threshold of Y. For example, if we assume that the link function is a standard normal distribution function, α^j, β_E^j, β_C^j and β_{EC}^j are estimated by running a probit regression of a dummy variable $1\{y_i \leq q_Y^j\}$ on a constant term, E_i, C_i, and interaction terms between E and C. For the probit version, the maximum likelihood estimators are given by

$$\begin{pmatrix} \widehat{\alpha}^j \\ \widehat{\beta}_E^j \\ \widehat{\beta}_C^j \\ \widehat{\beta}_{EC}^j \end{pmatrix} = \underset{\substack{a \in \mathbb{R} \\ b_E \in \mathbb{R}^q \\ b_C \in \mathbb{R}^p \\ b_{EC} \in \mathbb{R}^{pq}}}{\arg\max} \sum_{i=1}^{N} [1\{y_i \leq q_Y^j\} ln(\Phi(a + e_i' b_E + c_i' b_C + I(e,c)_i' b_{EC})) + 1\{y_i > q_Y^j\} ln(1 - \Phi(a + e_i' b_E + c_i' b_C + I(e,c)_i' b_{EC}))], \tag{2.13}$$

[11] Alternatively, the quantile regression method can be employed (Machado and Mata, 2005; Melly, 2005).

[12] For example, the empirical application of this study uses the 100 thresholds which are uniformly chosen from the 0.001th and 0.999th quantiles of Y.

[13] In practice, a logistic, probit or identity distribution function is often used.

where $1(.)$ is an indicator function and $\Phi(.)$ is a normal distribution function. Estimating the coefficients for every $j = 1, ..., J$ and interpolating these estimated points, we can construct an approximately continuous conditional distribution function of Y (Chernozhukov et al., 2013).[14]

Estimation of the weighting factor

Next, our estimation of the weighting factor, $\Psi(e, c) = \frac{c(F_{E_1}(e_1),...,F_{E_q}(e_q),F_{C_1}(c_1),...,F_{C_p}(c_p);\theta)}{c_e(F_{E_1}(e_1),...,F_{E_q}(e_q);\theta_e)c_c(F_{C_1}(c_1),...,F_{C_p}(c_p);\theta_c)}$, requires estimations of (i) the copula function of C and E, (ii) the copula function of E and (iii) the copula function of C.

Two-step sequential likelihood estimation is employed to estimate a copula. This separates the estimation of the marginals from that of the dependence parameter(s), and produces consistent estimates of the dependence parameter(s) (Trivedi and Zimmer, 2007). For example, in order to model the joint distribution of E and C, the first step estimates univariate marginal distributions of each element of E and C by these empirical cumulative distribution functions, i.e. $\widehat{F}_{E_m}(e_m) = \frac{1}{N}\sum_{i=1}^{N} 1(e_{mi} \leq e_m)$ for $m = 1, ..., q$ and $\widehat{F}_{C_n}(c_n) = \frac{1}{N}\sum_{i=1}^{N} 1(c_{ni} \leq c_n)$ for $n = 1, ..., p$.

The second step then estimates the dependence parameter(s), using the estimated marginal distributions from the first step. It is essential to select an appropriate copula for modelling a joint distribution. If dependence structures were known a priori, it would be easier to choose an appropriate copula, but such information is seldom available in advance. The selection of the copula tends to be a posterior consideration, and so it is important to estimate several copulas and compare their performances.

The dependence parameters are estimated by employing the maximum likelihood method from the marginal distributions with parametric assumptions of each copula function (Trivedi and Zimmer, 2007). An appropriate copula is the one which can best capture the dependence structures of the data. Maximised likelihood values, Akaike information criteria (AIC) and Bayesian information criteria (BIC) provide guidance on the selection.[15] The AIC and BIC select the best

[14] It is known that the estimated conditional function may be non-monotonic in the sense that $q_Y^j > q_Y^k$ does not necessarily lead to $\widehat{F}_{Y|E,C}(q_Y^j) > \widehat{F}_{Y|E,C}(q_Y^k)$. In such a case, the estimated distribution function cannot be directly inverted. To deal with this potential non-monotonicity, following Chernozhukov et al. (2010), we re-arrange the estimated conditional distribution so that the re-arranged distribution function satisfies monotonicity.

[15] $AIC = -2 * ln(likelihood) + 2 * K$ and $BIC = -2 * ln(likelihood) + K * ln(K)$, where K is the number of parameters of a copula function.

model, taking account of the trade-off between the bias due to misspecification and the sample variation resulting from over-fitting (Stone, 1977). The parameter estimator is given by

$$\widehat{\theta} = \arg\max_{\theta \in \Theta} \sum_{i=1}^{N} ln\, c(\widehat{F}_{E_1}(e_{1i}), ..., \widehat{F}_{E_q}(e_{qi}), \widehat{F}_{C_1}(c_{1i}), ..., \widehat{F}_{C_p}(c_{pi}); \theta), \tag{2.14}$$

where $c(.)$ is the likelihood function of a specific copula. Likewise, the copula functions of E and C are estimated respectively by

$$\widehat{\theta_e} = \arg\max_{\theta_e \in \Theta} \sum_{i=1}^{N} ln\, c_e(\widehat{F}_{E_1}(e_{1i}), ..., \widehat{F}_{E_q}(e_{qi}); \theta_e) \tag{2.15}$$

$$\widehat{\theta_c} = \arg\max_{\theta_c \in \Theta} \sum_{i=1}^{N} ln\, c_c(\widehat{F}_{C_1}(c_{1i}), ..., \widehat{F}_{C_p}(c_{pi}); \theta_c). \tag{2.16}$$

Then we obtain an estimate of $\Psi(e, c)$.

Once we estimate $F_{Y|E,C}$ and Ψ, we can generate the hypothetical distribution of Y from $\widehat{F}_Y^{EOP}$.[16]

2.4.3 Direct and indirect effects

As discussed in the introduction, some observed efforts may depend on circumstances, it is therefore insightful to decompose $\Delta = v(F_Y(y)) - v(F_Y^{EOP}(y))$ further into two parts: a direct effect and an indirect effect, following Bourguignon et al. (2007), Ferreira and Gignoux (2011) and Pistolesi (2008). The direct effect is the illegitimate inequality attributable solely to the distribution of circumstances in the absence of any interplay between efforts and circumstances, while the indirect effect is the counterpart which is caused by the dependence of efforts on circumstances.

We prepare for the counterfactual distribution of Y, denoted as $F_Y^{EOP,direct}(y)$, which would be realised when the variation of circumstances are suppressed while the variation of efforts are unaffected. When manipulating the marginal distributions of each element of circumstances does not affect the joint or marginal distributions of efforts, the hypothetical outcome distribution

[16] Noting that the statistical property that as the random variable $U = F_Y^{EOP}(y)$, is uniformly distributed on $(0, 1)$, the random variable $Y = \widehat{F}_Y^{EOP,-1}(U)$ has distribution $\widehat{F}_Y^{EOP}$.

under the EOP is expressed by

$$\begin{aligned}\widehat{F}_Y^{EOP,direct}(y) &= \int_E \widehat{F}_{Y|E,C}(y|e, c = \widetilde{c})\widehat{\Psi}(e, c)d\widehat{F}_E(e) \\ &= \frac{1}{N}\sum_{i=1}^{N} \widehat{F}_{Y|E,C}(y|e = e_i, c = \widetilde{c})\widehat{\Psi}(e = e_i, c = c_i). \end{aligned} \tag{2.17}$$

Note that the weighting factor in equation (2.17) is a function of both E and C. c_i in the weighting factor is the observed circumstances before its manipulation, not the fixed reference value. Intuitively, different from the weighting factor in equation (2.10), i.e. $\widehat{\Psi}(e = e_i, c = \widetilde{c})$, the weighting factor in equation (2.17), i.e. $\widehat{\Psi}(e = e_i, c = c_i)$, does not take into consideration the indirect pathway by which C influences E when we suppress the variation of C. Hence, $\widehat{F}_Y^{EOP,direct}(y)$ is the hypothetical outcome distribution with circumstances fixed at the reference level and with original efforts unaffected by our levelling circumstances.

Finally, in the fairness-gap spirits (Fleurbaey and Schokkaert, 2009), $\widehat{\Delta}^{direct} = v(\widehat{F}_Y(y)) - v(\widehat{F}_Y^{EOP,direct}(y))$ yields the direct effect. Subtracting this from the total IOP measurement gives the indirect effect, i.e. $\widehat{\Delta}^{indirect} = \widehat{\Delta} - \widehat{\Delta}^{direct}$.

2.5 Empirical application: Ex-post IOP in intergenerational transmission of body mass in Indonesia

2.5.1 Background and motivation

This section illustrates the proposed copula-based measurement approach through its application to the intergenerational transmission of the body mass index (BMI) in Indonesia. The growing number of overweight and obese people is a major public health issue in both developed and developing countries (Kelly et al., 2008). According to World Health Organization (2015), 39 per cent of the adult population in the world were overweight and around 13 per cent of them were obese in 2014. A higher BMI is a leading risk factor in chronic diseases such as cardiovascular disease, stroke, diabetes and musculoskeletal disorders (Ng et al., 2014). As well as over-nutrition, low- and middle-income countries are faced with the persistence of under-nutrition and the consequences of being underweight, which is also associated with elevated morbidity and mortality (Strauss and Thomas, 2007; Waaler, 1984). These two forms of malnutrition are one of the major public health issues in low- and middle-income countries.

The existence of the intergenerational transmission of BMI is increasingly recognised internationally and parental over- and under-nutritions are strong significant predictors of those of their offspring (Dolton and Xiao, 2015; Freeman et al., 2012; Yan, 2015; Brown and Roberts, 2013; Costa-Font and Gil, 2013; Li et al., 2009; Whitaker et al., 1997). Dolton and Xiao (2017) estimate intergenerational elasticity across six countries[17] with substantially different economic development and finds that intergenerational elasticity is very similar across these nations and relatively constant. Intergenerational BMI transmission is a result of both genetic inheritance and family environment (Frayling et al., 2007; Bouchard, 2009; Burke et al., 2001; Brown and Roberts, 2013; Martin, 2008; Salsberry and Reagan, 2007), and a significant correlation is found to persist even after children have grown up (Abrevaya and Tang, 2011; Reynolds et al., 2007; Laitinen et al., 2001). Given the medical evidence that extreme BMI can have detrimental effects on health (Forouzanfar et al., 2015; Ng et al., 2014; Jung, 1997; Waaler, 1984), the large intergenerational transmission of BMI would be against the ideal of equal opportunities in relation to good health. This analysis focuses on the BMI of grown-up children aged between 20 and 35 and the BMI of their parents in their childhood and adolescence.

While there is epidemiological evidence that parental BMI is important determinants of BMI in their offspring, it seems also quite true that the current BMI of offspring is largely due to their own current lifestyles, revolving around matters such as dietary choices and daily exercise. The responsibility-cut demarcation in this application is based on the following normative criteria. First, from the viewpoint of children, they obviously cannot choose their parents, and therefore the adiposity of their parents in their childhood and adolescence is beyond their responsibility or control. Second, it seems reasonable to think that, after they reach adulthood, they should be held responsible for the lifestyle they choose.

The goal of this application is to measure the ex-post IOP in BMI associated with its intergenerational transmission among grown children aged between 20 and 35 in Indonesia. As 15 is the age of majority in Indonesia, i.e. the recognised threshold of adulthood,[18] this application assumes that the BMI of their parents when they were children or adolescents aged below 15 are illegitimate causes of inequality. Hence, we treat only parental BMI as circumstances herein

[17] The United Kingdom, the United States, China, Indonesia, Spain and Mexico

[18] *http://www.youthpolicy.org/factsheets/country/indonesia/* (accessed 18/07/2017)

and aim to quantify the inequality attributable solely to parental BMI, which implies that all the other factors are treated as efforts in this study. However, it is noteworthy that treating the other factors as efforts does not decrease the usefulness of the method at all. In general, the demarcation between efforts and circumstance varies, depending on the context of research, and the flexibility of normative judgement regarding the demarcation is indeed an advantage of the EOP research framework (Schokkaert, 2015). We admit that the inherent determinants of BMI can be enormous, and parental BMI are just a subset of conceivable circumstances for their offspring. Furthermore, it is true that the BMI of offspring can also be influenced by socioeconomic characteristics, family environments, peer pressure from school mates and so forth, which can also be potentially regarded as circumstances in the sense that they are not controllable by respondents. In this sense, the inequality attributable to the intergenerational transmission can also be interpreted as the fraction of the entire IOP.

The study of Indonesia is of interest not only from the viewpoints of public health but also economic perspectives. Indonesia has the largest population in Southeast Asia and the fourth largest population in the World (United Nations, 2016). As in other developing countries in Southeast Asia, Indonesia has been showing high economic growth and a strong increase in overweight and obese people. It has been reported that obesity has increased rapidly across all Indonesian population groups including rural and low-income people (Aizawa and Helble, 2017; Aizawa, 2018b; Roemling and Qaim, 2012). Although the prevalence rate of being underweight has been exhibiting a decline, under-nutrition has not yet entirely disappeared (Witoelar et al., 2009; Oddo et al., 2012; Vaezghasemi et al., 2014, 2016; Hanandita and Tampubolon, 2015; Doak et al., 2005; Roemling and Qaim, 2013). Studying Indonesia provides a good case study in Southeast Asia, which is currently experiencing strong economic growth, a rapid increase in obesity and persistent under-nutrition, especially among the poor.

2.5.2 Data

The Indonesian Family Life Survey (IFLS)

The Indonesian Family Life Survey (IFLS) is a large-scale, ongoing longitudinal survey and was designed and implemented by the RAND Corporation, launched first in 1993/94. It currently has five waves (as of 2018), the latest of which was completed in 2014. The survey questions are very extensive, covering a household's economy, education, employment and a wide range of

health conditions. The sample is representative of people living in 13 of the 27 provinces in the country, where about 83 per cent of the total population resides.[19] The high re-contact rates of each wave are one of the strengths of the IFLS[20], which helps to lessen the potential risk of bias owing to non-random attrition. In fact, its re-contact rates are as high as, or higher than, most longitudinal surveys available in the United States and Europe (Strauss et al., 2016).

Outcome variable

The health outcome of interest is the BMI of grown-up children aged between 20 and 35. From measurements of the heights and weights of the respondents, BMI was calculated, which is defined as an individual's weight divided by their height squared and expressed internationally in units of kg/m^2. Hence, BMI conveys cardinal information about body mass. Information on heights and weights in the IFLS was based on actual measurements, and therefore it was less likely to be subject to measurement errors such as under-reporting of weights and over-reporting of heights (Gorber et al., 2007), which substantially enhances the credibility of the result.[21]

Circumstance variables

Circumstance variables in this application are parental BMI when respondents were below 15 years old. This study exploits the panel structure of the IFLS to obtain them, i.e. the 21-year gap between the first wave (completed in 1993/94) and the last wave (completed in 2014) of the IFLS allows us to obtain that information (Figure 2.1). The high re-contact rates in the IFLS make this possible. Parental BMI in 1993/94 may well be recognised as circumstances affecting the BMI of their adult offspring in 2014.

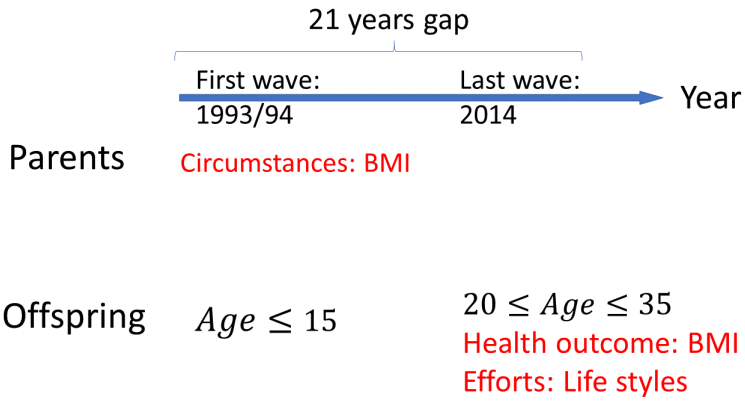
The fundamental reason not to use contemporaneous parents' BMI as circumstances is that they are not necessarily uncontrollable by grown-up offspring. For instance, it is quite likely that grown-up sons/daughters encourage their overweight/obese parents to try to participate in physical activities to lose weight. It is also likely that grown-up children play important roles

[19]Four provinces on Sumatra (North Sumatra, West Sumatra, South Sumatra and Lampung), all five of the Javanese provinces (DKI Jakarta, West Java, Central Java, DI Yogyakarta and East Java) and four provinces covering the remaining major island groups (Bali, West Nusa Tenggara, South Kalimantan and South Sulawesi) are included (Strauss et al., 2016).

[20]For example, the re-contact rate in the latest wave among the households interviewed in the first wave was reported at 92.0 per cent. Among the households interviewed in the first wave, 86.9 per cent were involved in all five waves.

[21]In the survey, heights were measured using a Seca plastic height board and weight measurements were taken using Seca floor-model scales developed in collaboration with UNICEF. The floor-model scales have a digital readout and are accurate to the nearest 0.1 kg.

Figure 2.1: BMI of grown-up children and parental BMI in the IFLS



Note: The outcome variable (Y) in this application is the BMI of grown-up children aged between 20 and 35 years. The circumstance variables (C) are parental BMI when offspring were below 15 years old, which is obtained from the data in the first wave of the IFLS. The effort variables (E) are lifestyles of grown-up children, of which information is obtained from the last wave of the IFLS.

in the choice of daily meals for their parents. In these cases, parental BMI in 2014 may not be undoubtedly viewed as circumstances for grown-up children. On the other hand, before offspring become mature, they are by far less likely to influence parents' BMI and it is plausible to regard them as exogenous for children.

In the Roemer framework, a part of E is dependent on C. It is highly probable that family environments in childhood shape children's weight-related behaviours and life-long beliefs throughout their development. Especially before children grow up, their parents play an important role in choosing foods, preparing meals and monitoring children's use of time. These parental controls could influence weight-related behaviours, values and preferences, even after children reach adulthood (Wardle et al., 2001; Fisher et al., 2002; Fogelholm et al., 1999; Birch and Davison, 2001; Davison and Birch, 2002). In this sense, parts of the lifestyles in adulthood are dependent on parental behaviours during development. Following the Roemer framework, such influences are treated as parts of circumstances, and their contributions to the IOP are captured as the indirect effect.

Effort variables

The set of effort variables used in this analysis is composed of the following five categories: 1) education, 2) long-term household affluence, 3) food expenditure and choices, 4) exercise and 5) smoking. First, years of education completed by the respondents are included in the model. As

variables reflecting the households' affluence, logarithmic family-size adjusted household wealth[22] is used. The advantage of using wealth over income is that the former, as a stock of income, is suitable as an indicator reflecting the long-term living standards of households. In addition, wealth is less susceptible to temporary economic shocks and seasonal events such as drought, which is important for the analysis of developing countries in which agriculture plays an important role.

Next, as measurements of food expenditure and choices, the logarithmic amount of family-size adjusted total expenditure on food is used.[23] The proportion of expenditure spent on prepared foods outside the home to total food expenditure is used in this study, in order to capture the different food choices made by the household. Increasing the consumption of foods prepared outside the home, including meals at restaurants, is considered one of the main factors contributing to excess body fat. In the case of the United States, the rise in the share of food budget spent on foods prepared away from the home versus total food expenditure is reported as being part of the reason for an increase in obesity (McCrory et al., 1999). Moreover, the analysis also includes the share of food expenditure spent on staple foods,[24] which is important, because developing Asian countries are experiencing a rapid change in diet, known as the 'nutrition transition' (Popkin, 2001; Popkin and Du, 2003; Popkin and Gordon-Larsen, 2004; Popkin et al., 2006). As in traditional Indonesian cultures, unprocessed staples have played a major role (Roemling and Qaim, 2012), a lower share of staple food could be considered to indicate the lesser importance of traditional cuisine in diet choice.

Furthermore, this study uses information about daily vigorous and moderate physical activities. In the IFLS, vigorous activities are defined as those that make one breathe much harder than normal and may include heavy lifting, digging, ploughing, aerobics, fast cycling and cycling with a load. Moderate activities make one breathe somewhat harder than normal and may include carrying light loads, cycling at a regular pace or mopping the floor. For both vigorous

[22]Wealth is defined as the aggregated total value of the following various assets, which are commonly found in typical Indonesian households: House and land occupied by a household; Other house/building (including land); Land (not used for farming); Poultry; Livestock/fish pond; Hard stem plant not used for farming, or a non-farm business; Vehicles (cars, boats, bicycles, motorbikes); Household appliances (radio, tape recorder, television, fridge, sewing or washing machine, video and CD player, cell phone, etc.); Savings/certificates of deposits/stocks; Receivables; Jewellery; Household furniture and utensils.

[23]In the survey, the reporting of food expenditure information was done according to the household unit. This study assumes that household members share expenditure on an equal basis.

[24]Hulled, uncooked rice, sago/flour, cassava, tapioca and other staple foods.

Table 2.2: Descriptive statistics

	count	mean	sd	p50	min	max
BMI	4564	23.21	4.38	22.40	13.95	43.39
Father's BMI in 1993	4564	21.17	2.74	20.71	13.93	33.71
Mother's BMI in 1993	4564	21.97	3.41	21.44	12.07	33.91
Education years	4564	11.16	3.45	12.00	0.00	16.00
ln(Wealth)	4564	16.51	1.84	16.66	0.00	20.91
ln(Food expenditure)	4564	12.04	0.60	12.03	9.82	14.49
Prepared food ratio	4564	0.15	0.19	0.07	0.00	1.00
Staple food ratio	4564	0.18	0.15	0.16	0.00	0.94
Vigorous activity	4564	2.43	5.86	0.00	0.00	28.00
Moderate activity	4564	5.79	7.41	3.00	0.00	28.00
Eating frequency	4564	0.17	1.28	-0.11	-1.45	9.11

sd=Standard deviation; p50=Median

and moderate physical activities, work that takes fewer than ten minutes is not counted as an exercise. The intensity of these exercises is calculated by the time spent and the frequency of the respective activity.[25] Finally, the index of frequency of eating unhealthy foods in a week[26], which is obtained by the principal component analysis using the first principal component, is included.

Sample selection

After dropping the outliers[27] and deleting observations with missing values, the sample size becomes 4,564.[28] The Kolmogorov–Smirnov test fails to reject the equality of the offspring's BMI distribution of the complete observations and that of the incomplete observations which are deleted due to missing values. The descriptive statistics are shown in Table 2.2. Figure 2.2 shows the estimated density and cumulative distribution functions. The parametric Shapiro-Francia test and the non-parametric Kolmogorov-Smirnov test reject the normality of BMI ($p < 0.01$).

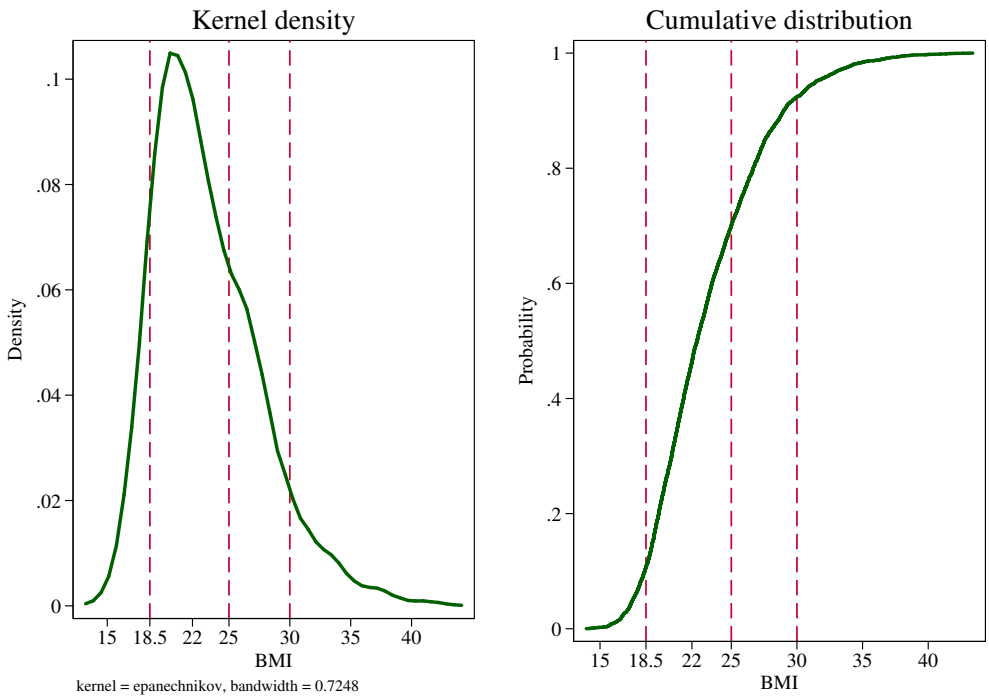
[25]The variable is the product of the time they usually spend in a day (1 is given, if they did no exercise; 2 is given to less than 30 minutes' exercise; 3 is given to exercises between 30 minutes and 4 hours; 4 is given, if it is more than 4 hours) and the days on which they do this in a week. The lowest value is zero (no exercise in a week) and the highest is 28 (more than 4 hours and 7 days a week).

[26]Unhealthy foods considered herein are instant noodles, fast foods, soft drinks, fried snacks and sweet snacks.

[27]Some observations show extremely high or low BMI, which seems biologically impossible. Observations from the top 0.1 per cent or bottom 0.1 per cent of BMI of respondents and parental BMI are dropped from the sample as possible outliers.

[28]The original sample size is 14,774 of which 12,129 observations have information of respondent's BMI and 7,575 observations had an interview in the first wave. 23 observations are deleted as possible outliers and 2,297 observations with missing values of either efforts or circumstances are deleted.

Figure 2.2: Kernel density and cumulative distribution functions of BMI



2.5.3 Results

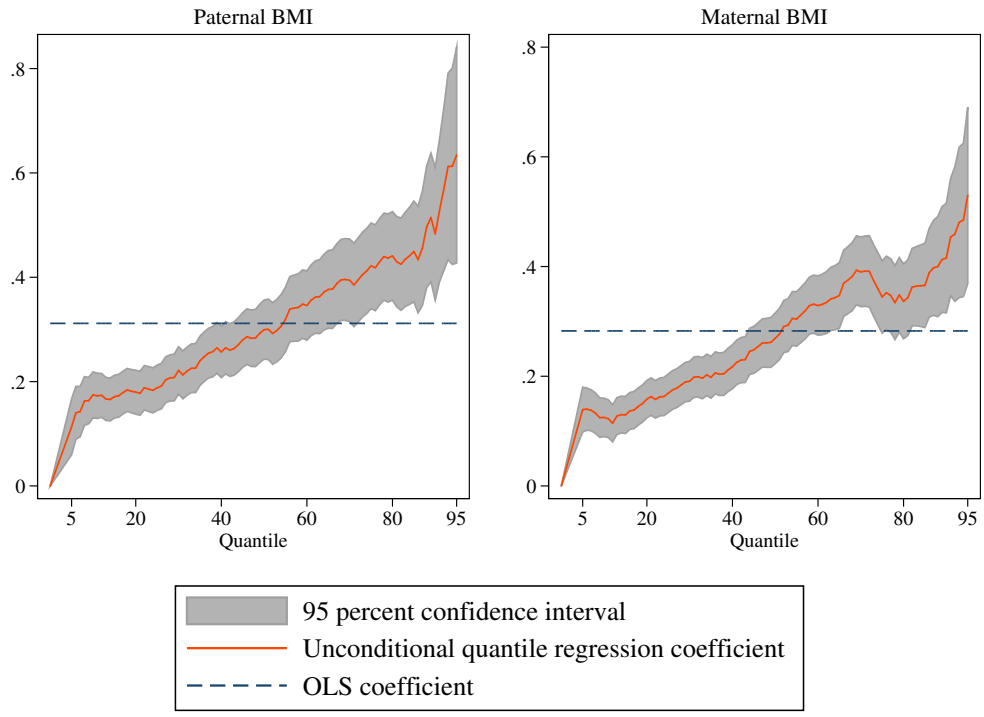
Regression analysis

First, we implement a regression analysis to understand better the association between the BMI of children and that of their parents. We focus predominantly on the effects of parental BMI here. Table 2.3 shows the results of the ordinary least squares (OLS) regression and unconditional quantile regressions.[29] The OLS estimation result in the first column illustrates that both maternal and paternal BMI in 1993 had significant associations with the BMI of their child/children in 2014 ($p < 0.01$). We cannot find a significant difference in their coefficient sizes at the 5 per cent level.

Figure 2.3 illustrates their heterogeneous effects at the different points of distribution. The unconditional quantile regression results reveal that the impacts of parental BMI on their child/children's BMI vary across the distribution. That is to say, the parental BMI are more relevant around the middle and the right tail of the offspring's BMI distribution, which is suggestive of the nonlinear relations between them. The existence of heterogeneous effects across the distribution is in line

[29]The unconditional quantile regression was proposed by Firpo et al. (2009), and a brief explanation is available in the Appendix.

Figure 2.3: Unconditional quantile regression coefficients of parental BMI



Note: We estimate the marginal effect of parental BMI when offspring were children on the quantiles of BMI of grown-up children. Estimated coefficients of respective circumstance variables by the unconditional quantile regression are plotted. Efforts variables are controlled for (Not shown here).

with previous literature (Dolton and Xiao, 2015, 2017; Classen, 2010). Again, the differences in the paternal and maternal BMI' coefficient sizes are not significant at the 5 per cent level for every estimated quantile point.

Measurement of inequality of opportunity

Table 2.4 presents the maximised log-likelihood values, AIC and BIC of the copula estimations. For the estimation of the copula function for E and C and that of E, we fail to obtain results for the Frank copula, because its log-likelihood values did not converge during the estimation. For the copula function estimation of C, the Clayton copula did not converge. Failure of convergence could be an indication of inconsistency between the data properties and the copula restriction on the dependence parameter (Trivedi and Zimmer, 2007). Among the others, the Gaussian copula, which can have multiple dependence parameters, shows the highest likelihoods and the lowest AIC and BIC in the estimations of the joint distribution of E and C and that of E, dominating the other copulas in terms of fitting to the data. The Gumbel copula shows the highest likelihood and the lowest AIC and BIC for the estimation of the joint distribution of C, which implies that the parental BMI have strong right tail dependence. Hence, in this application, we use the

Table 2.3: Regression analysis

	(1) OLS	(2) 0.05 quantile	(3) 0.20 quantile	(4) 0.35 quantile	(5) 0.50 quantile	(6) 0.65 quantile	(7) 0.80 quantile	(8) 0.95 quantile
Father's BMI in 1993	0.312***	0.114***	0.180***	0.241***	0.300***	0.377***	0.441***	0.612***
	(0.0250)	(0.0274)	(0.0215)	(0.0241)	(0.0289)	(0.0380)	(0.0432)	(0.104)
Mother's BMI in 1993	0.283***	0.135***	0.158***	0.199***	0.269***	0.347***	0.337***	0.531***
	(0.0210)	(0.0212)	(0.0174)	(0.0194)	(0.0236)	(0.0306)	(0.0348)	(0.0821)
Education years	-0.0978***	-0.0543**	-0.0566***	-0.0995***	-0.0782***	-0.107***	-0.131***	-0.180***
	(0.0195)	(0.0230)	(0.0186)	(0.0205)	(0.0244)	(0.0303)	(0.0328)	(0.0688)
ln(Wealth)	0.143***	0.104**	0.137***	0.181***	0.210***	0.206***	0.146***	0.0754
	(0.0305)	(0.0465)	(0.0358)	(0.0369)	(0.0413)	(0.0489)	(0.0493)	(0.104)
ln(Food expenditure)	0.127	0.159	-0.0413	0.247**	0.266*	0.129	0.171	-0.442
	(0.110)	(0.131)	(0.108)	(0.119)	(0.142)	(0.179)	(0.188)	(0.396)
Prepared food ratio	-0.640*	0.104	-0.214	-0.557	-0.746	-0.951*	-1.026*	-0.00474
	(0.348)	(0.404)	(0.348)	(0.387)	(0.457)	(0.563)	(0.576)	(1.254)
Staple food ratio	-1.605***	-0.166	-1.041**	-1.494***	-1.654***	-2.135***	-1.200	-2.111
	(0.469)	(0.608)	(0.471)	(0.514)	(0.600)	(0.732)	(0.767)	(1.664)
Vigorous activity	-0.0695***	-0.00327	-0.0354***	-0.0604***	-0.0874***	-0.0998***	-0.0903***	-0.0966***
	(0.00904)	(0.0124)	(0.0109)	(0.0116)	(0.0129)	(0.0150)	(0.0160)	(0.0308)
Moderate activity	0.0229***	0.00961	0.0216***	0.0225**	0.0265**	0.0176	0.0300**	0.0206
	(0.00802)	(0.00936)	(0.00798)	(0.00899)	(0.0106)	(0.0131)	(0.0141)	(0.0280)
Eating frequency	-0.0848*	-0.131**	-0.111**	-0.112**	-0.105*	-0.0938	-0.106	0.236
	(0.0498)	(0.0622)	(0.0491)	(0.0525)	(0.0614)	(0.0756)	(0.0797)	(0.190)
Observations	4564	4564	4564	4564	4564	4564	4564	4564
R-squared	0.127	0.0201	0.0506	0.0732	0.0860	0.0821	0.0742	0.0369

Standard errors in parentheses.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.4: Maximised log-likelihood, AIC and BIC of the copula estimations

Copula	Frank	Clayton	Gumbel	Joe	Gaussian
Joint distribution of E and C					
Log likelihood	NA	204.569	59.098	7.666	1970.634
AIC	NA	−407.139	−116.197	−13.332	−3851.268
BIC	NA	−409.139	−118.197	−15.332	−3769.969
Joint distribution of E					
Log likelihood	NA	145.601	14.721	0.011	1664.229
AIC	NA	−289.202	−27.442	1.977	−3238.458
BIC	NA	−291.202	−29.442	−0.023	−3157.158
Joint distribution of C					
Log likelihood	143.909	NA	158.932	131.828	154.536
AIC	−285.818	NA	−315.864	−261.657	−307.073
BIC	−287.818	NA	−317.864	−263.657	−309.073

Note: NA means that a likelihood function did not converge. AIC: Akaike information criteria. BIC: Bayesian information criteria.

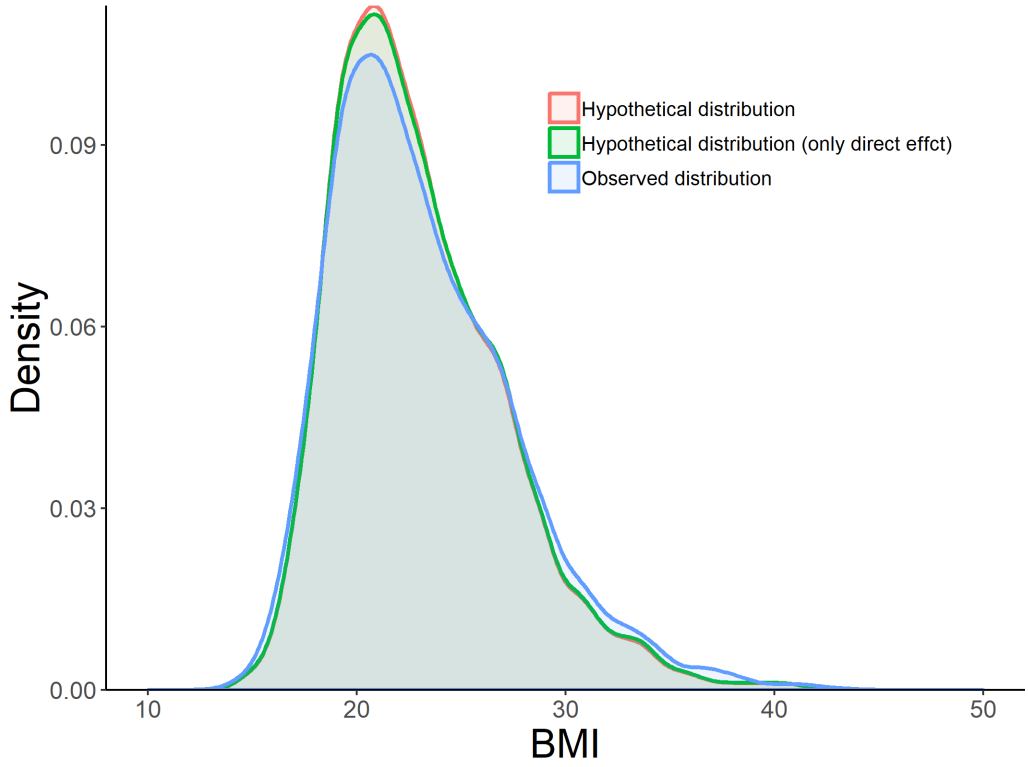
Gaussian copula and the Gumbel copula to model the respective joint distributions.

In the distributional regression, we implement 100 probit estimations and 5,000 random samples are drawn from the hypothetical health distribution. Figure 2.4 illustrates the density functions of the offspring's BMI under the original covariate distributions and under the hypothetical distribution where circumstance values are set to their sample means. We find that the hypothetical BMI distributions exhibit less variation, compared with the observed BMI distribution. This perfectly makes sense because the hypothetical distributions are those distributions when we suppress the variation of the circumstance variables. Hence, the reduction in the variation of BMI can be interpreted as a part of the total BMI variation associated with its intergenerational transmission (Fleurbaey and Schokkaert, 2009).

Table 2.5 summarises the main estimation results of this application. Standard errors are obtained by bootstrap with 500 repetitions. Table 2.5 is composed of two sections, indicated in the rows 1) Inequality measurement and 2) IOP percentage. The first section reports the mean, variance (Var), coefficient of variation (CV), Gini coefficient (Gini) and Theil index (Theil) of the originally observed health distribution and two hypothetical distributions.[30] The first hypothetical distribution is the one in which we take into account both direct and indirect effects of circumstances on the outcome. The second hypothetical distribution is the one in which we only consider their direct effects. These two hypothetical distributions reflect the inequality under

[30]Inequality statistics are defined as follows: $Var(Y) = \frac{1}{N}\sum_{i=1}^{N}(y_i - \overline{y})^2$; $CV(Y) = \frac{Std.Dev.}{\overline{y}} = \sqrt{\frac{1}{N}\sum_{i=1}^{N}(y_i - \overline{y})^2}/\overline{y}$; $Gini(Y) = \frac{2\sum_{i=1}^{N} iy_i}{n\sum_{i=1}^{N} y_i} - \frac{N+1}{N}$; and $Theil(Y) = \frac{1}{2N\overline{y}^2}\sum_{i=1}^{N}(y_i - \overline{y})^2$.

Figure 2.4: Density functions of the hypothetical and observed BMI



EOP, where the variations in circumstances are suppressed. These inequality statistics are based on the hypothetical situation when the reference values are set to their sample means.

The second section reports the percentage share of the IOP in relation to overall inequality. The first row in the second section reports the total effect, i.e. $100 * \{1 - \frac{v(F_Y^{EOP}(y))}{v(F_Y(y))}\}$. The second and third rows show the percentage share of the direct effect and the indirect effect in relation to the IOP respectively. The result suggests that the shares of IOP in relation to overall inequality vary, depending on the inequality statistics. With respect to the variance, for example, the IOP accounts for around 15.1 per cent when the sample means are used as reference values. Compared to the case of variance, we observe relatively smaller variations in the case where the coefficient of variation, the Gini coefficient, and the Theil index are used as inequality statistics.

2.5.4 Comparison with the parametric approach

Next, we measure the IOP with the parametric approach (Bourguignon et al., 2007). Assuming the linearity of effort factors and BMI[31], we estimate the counterfactual distribution of effort

[31] As we include the two-way interaction terms between C and E, the parametric model does not impose the additivity of individual factors.

Table 2.5: Estimation results

	Mean		**Var**		**CV**		**Gini**		**Theil**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs
Inequality measurement										
Original distribution: $v(F_Y)$	23.208***	0.065	19.177***	0.470	0.189***	0.002	0.104***	0.001	0.017***	0.000
Hypothetical distribution: $v(F_Y^{EOP})$	22.995***	0.096	16.283***	0.562	0.175***	0.003	0.096***	0.001	0.015***	0.000
Hypothetical distribution (direct): $v(F_Y^{EOP,direct})$	23.030***	0.099	16.550***	0.545	0.177***	0.003	0.097***	0.001	0.015***	0.000
IOP percentage										
Total effect			15.090***	2.483	6.998***	1.164	7.020***	1.178	13.327***	2.113
Direct effect			13.699***	2.458	6.383***	1.134	6.367***	1.057	12.171***	2.028
Indirect effect			1.392	1.164	0.615	0.494	0.653	0.534	1.155	0.922

Note: Standard errors (SEs) are obtained by bootstrap with 500 repetitions. Number of observations is 4,564.
IOP percentage is the proportion of inequality of opportunity to the observed inequality.
Total effect $(\Delta) = 1 - \frac{v(F_Y^{EOP})}{v(F_Y)}$
Direct effect $(\Delta^{direct}) = v(F_Y) - v(F_Y^{EOP,direct})$
Indirect effect $(\Delta^{indirect}) = \Delta - \Delta^{direct}$.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

factors and BMI in a situation where everybody faces a fixed parental BMI. The associated OLS coefficient estimates are provided in Appendix 2.A.3. We do not consider the non-parametric approach in this application study because continuous circumstance and effort variables require a large number of observations. Possible numbers of types and *tranches* in this application study are by far larger than the number of available samples, which makes it impossible to conduct the non-parametric analysis.

Results with the parametric approach are shown in Table 2.6, where associated standard errors are obtained by bootstrap with 500 repetitions. In the parametric method, we find that 11.4 per cent of the total variance is associated with the parental BMI, which is lower than the estimates in Table 2.5. The smaller effects of parental BMI are also observed for coefficient of variation, Gini coefficient and Theil index. The observation that estimated IOP in the parametric model is smaller than that estimated by our semi-parametric estimation may suggest that, due to the possible specification error in the parametric model, the association between outcome and circumstances may not have been satisfactorily modelled, thereby resulting in the downwardly biased IOP estimates. The second row in the second section in Table 2.6 indicates that a large part of the IOP is due to the direct effect of parental BMI on BMI of offspring. The very small indirect effect in the parametric method suggests that the relationships between C and E may not have been satisfactorily modelled by a linear regression model.

2.5.5 Sensitivity analysis

We repeat estimations, using several reference values of circumstance variables, which is suggested by Ramos and Van de gaer (2016). Table 2.7 reports the average of the inequality statistics when the reference values are set from the 0.05_{th} to 0.95_{th} quantiles. Specifically, this is calculated by averaging inequalities which are estimated when the reference values are set to the 0.05_{th}, 0.20_{th}, 0.35_{th}, 0.50_{th}, 0.65_{th}, 0.80_{th} and 0.95_{th} quantiles of circumstance variables. The result suggests that the shares of IOP in relation to overall inequality vary, depending on the reference values of circumstances. For example, intergenerational transmission of BMI accounts for around 15.2 per cent on average when the reference values are set to various quantiles of circumstances.

Table 2.6: Estimation results of the parametric model

	Mean		**Var**		**CV**		**Gini**		**Theil**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs
Inequality measurement										
Original distribution: $v(F_Y)$	23.208***	0.063	19.177***	0.483	0.189***	0.002	0.104***	0.001	0.017***	0.000
Hypothetical distribution: $v(F_Y^{EOP})$	23.202***	0.068	16.999***	0.421	0.178***	0.002	0.098***	0.001	0.015***	0.000
Hypothetical distribution (direct): $v(F_Y^{EOP,direct})$	23.201***	0.068	16.999***	0.421	0.178***	0.002	0.098***	0.001	0.015***	0.000
IOP percentage										
Total effect			11.356***	0.971	5.823***	0.517	5.584***	0.530	10.661***	0.931
Direct effect			11.355***	0.972	5.820***	0.517	5.580***	0.531	10.648***	0.933
Indirect effect			0.001	0.003	0.003	0.010	0.004	0.009	0.014	0.023

Note: Standard errors (SEs) are obtained by bootstrap with 500 repetitions. Number of observations is 4,564.
IOP percentage is the proportion of inequality of opportunity to the observed inequality.
Total effect $(\Delta) = 1 - \frac{v(F_Y^{EOP})}{v(F_Y)}$
Direct effect $(\Delta^{direct}) = v(F_Y) - v(F_Y^{EOP,direct})$
Indirect effect $(\Delta^{indirect}) = \Delta - \Delta^{direct}$.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 2.7: IOP estimation results under various reference values

Copula-based method

	Mean		Var		CV		Gini		Theil	
	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs
Inequality measurement										
Original distribution: $v(F_Y)$	23.208***	0.065	19.177***	0.470	0.189***	0.002	0.104***	0.001	0.017***	0.000
Hypothetical distribution: $v(F_Y^{EOP})$	23.057***	0.102	16.455***	0.575	0.174***	0.003	0.096***	0.002	0.015***	0.000
Hypothetical distribution (direct): $v(F_Y^{EOP,direct})$	23.110***	0.110	16.905***	0.623	0.176***	0.003	0.097***	0.002	0.015***	0.000
IOP percentage										
Total effect			14.063***	2.794	7.748***	1.305	7.474***	1.327	14.075***	2.379
Direct effect			11.582***	3.149	6.795***	1.341	6.370***	1.356	12.164***	2.518
Indirect effect			2.481*	1.506	0.953*	0.574	1.104*	0.591	1.911*	1.121

Parametric method

	Mean		Var		CV		Gini		Theil	
	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs	Estimates	SEs
Inequality measurement										
Original distribution: $v(F_Y)$	23.208***	0.063	19.177***	0.483	0.189***	0.002	0.104***	0.001	0.017***	0.000
Hypothetical distribution: $v(F_Y^{EOP})$	23.213***	0.072	17.060***	0.432	0.179***	0.002	0.098***	0.001	0.016***	0.000
Hypothetical distribution (direct): $v(F_Y^{EOP,direct})$	23.205***	0.080	17.060***	0.432	0.179***	0.002	0.098***	0.001	0.016***	0.000
IOP percentage										
Total effect			10.992***	0.963	5.132***	0.529	4.870***	0.536	8.816***	0.930
Direct effect			10.991***	0.963	5.090***	0.551	4.826***	0.557	8.721***	0.977
Indirect effect			0.001	0.006	0.043	0.109	0.044	0.111	0.095	0.214

Note: Standard errors (SEs) are obtained by bootstrap with 500 repetitions. Number of observations is 4,564.
The average of the distributional statistics are reported when C is set from 0.05th quantile to 0.95th quantile.
IOP percentage is the proportion of inequality of opportunity to the observed inequality.
Total effect $(\Delta) = 1 - \frac{v(F_Y^{EOP})}{v(F_Y)}$
Direct effect $(\Delta^{direct}) = v(F_Y) - v(F_Y^{EOP,direct})$
Indirect effect $(\Delta^{indirect}) = \Delta - \Delta^{direct}$.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

2.6 Discussion and conclusion

In welfare economics, two types of inequality can be defined: legitimate inequality and illegitimate inequality. This distinction is based on the normative judgements as to whether the causes of inequality are things for which an individual can be held responsible. The IOP is the illegitimate inequality attributable to circumstances beyond the individual's control. This paper proposes a new methodology to measure the ex-post IOP with copulas. The copula function models dependence structures between effort and circumstance variables, following which the hypothetical outcome distribution in a situation in which the variations of circumstances are suppressed is derived. The proposed semi-parametric model is particularly useful when the non-parametric method is not applicable because effort and circumstance factors are continuous variables.

It is worth noting that, as discussed by Ferreira and Gignoux (2011), the measurement of IOP yields only the *lower limit* of illegitimate inequality.[32] In most cases, some of the circumstance variables are unobservable or non-quantifiable. The real cause of illegitimate inequality can consist of a far more complex set of circumstances, but only its subset is available to researchers. In this sense, as Wagstaff and Kanbur (2015) and Kanbur and Wagstaff (2015, 2016) argue, the importance of showing the lower limit for policymakers deserves consideration. The result of low IOP should not be received optimistically, either, and needs to be carefully interpreted. It seems important in implementing an empirical analysis to ask oneself how meaningful it is in the research context to report the lower limit of illegitimate inequality. Furthermore, the actual demarcation between efforts and circumstances is not obvious to everyone and often requires personal normative judgement regarding distributional justice. Of course, there might not always be consensual agreement on the distinction. Judgement about which situations are unfair can vary from place to place and from time to time (Whitehead, 1992); however, one of the benefits of the EOP framework is that it can handle our various normative values in the model, making it possible to discuss the upper and lower limits of illegitimate inequality (Schokkaert, 2015; García-Gómez et al., 2015). To make the most of the versatility of the framework, it is important to state explicitly the standpoint of one's own normative judgement on the demarcation in empirical analyses.

[32] A notable exception is Niehues and Peichl (2014), in which the authors propose measuring the upper limit of illegitimate inequality with longitudinal data.

This paper applies the copula-based method in order to study the ex-post IOP in BMI distribution among Indonesian people aged between 20 and 35. The application exploits the longitudinal nature of IFLS data and measures the inequality associated with the intergenerational transmission of BMI from parents to offspring. The circumstance variables are paternal and maternal BMI when respondents were in their childhood and adolescence. As effort variables, various lifestyle choices are included. The results suggest that the proportions of IOP vary, depending on the choice of inequality statistics and the choice of the reference values of circumstance variables. Although these estimates do not necessarily reflect the causal relationship between them, this study shows that intergenerational transmission of BMI is forming the non-negligible part of entire BMI distribution. When the reference values are set to the sample means of the circumstance variables, 15.1 per cent of the overall variance in the respondents' BMI is found to be associated with their parents' BMI.

Last of all, it is worth discussing the limitations of this study. Most of the copulas can deal with multiple marginal distributions, but not all of the popular copula functions can have multiple dependence parameters. If a certain copula can have only a single parameter, the parameter is supposed to capture dependences of all the possible combinations of marginal distributions. In this case, these copulas cannot always capture the dependence structures satisfactorily. When the X dimension is large, the practical choice of copula functions is likely to be more constrained. In such a case, we might not be able to enjoy the full benefit of employing copulas in modelling relations between C and E. One practical solution to deal with this issue would be to reduce dimensions by the principal component analysis before estimating copula functions. Another solution would be to consider a much wider array of copula functions which are capable of flexibly modelling more complex multi-dimensional dependency. A more thorough exploration of further possibilities would be our future research topic and this would expand the potential of copulas in the study into the IOP.

2.A Appendix

2.A.1 Proof of proposition 1

In this proof, C and E denote the sets of circumstance and effort variables, i.e. $C = (C_1, ..., C_p)$ and $E = (E_1, ..., E_q)$.

From equation (2.5),

$$\begin{aligned} F_Y(y) &= \int_C \int_E F_{Y|E,C}(y|e,c) \frac{dF_{E|C}(e|c)}{dF_E(e)} dF_E(e) dF_C(c) \\ &= \int_C \int_E F_{Y|E,C}(y|e,c) \Psi(e,c) dF_E(e) dF_C(c), \end{aligned} \tag{2.A.1}$$

where $\Psi(e,c)$ is a weighting factor and expressed as

$$\Psi(e,c) = \frac{dF_{E|C}(e|c)}{dF_E(e)} = \frac{f_{E|C}(e|c)}{f_E(e)} = \frac{f_{E,C}(e,c)}{f_E(e) f_C(c)} \tag{2.A.2}$$

Next, the joint density functions, $f_{E,C}(e,c)$, $f_E(e)$ and $f_C(c)$ are expressed by their copula functions as follows;

$$\begin{aligned} f_{E,C}(e,c) &= \frac{\partial^{p+q} F_{E,C}(e,c)}{\partial E_1 ... \partial E_q \partial C_1 ... \partial C_p} \\ &= \frac{\partial^{p+q} \mathbb{C}(F_{E_1}, ..., F_{E_p}, F_{C_1}, ..., F_{C_p}; \theta)}{\partial E_1 ... \partial E_q \partial C_1 ... \partial C_p} \\ &= \frac{\partial^{p+q} \mathbb{C}(F_{E_1}, ..., F_{E_p}, F_{C_1}, ..., F_{C_p}; \theta)}{\partial F_{E_1}(e_1) ... \partial F_{E_q}(e_q) \partial F_{C_1}(c_1) ... \partial F_{C_p}(c_p)} \frac{\partial F_{E_1}(e_1) ... \partial F_{E_q}(e_q) \partial F_{C_1}(c_1) ... \partial F_{C_p}(c_p)}{\partial E_1 ... \partial E_q \partial C_1 ... \partial C_p} \\ &= c(F_{E_1}, ..., F_{E_p}, F_{C_1}, ..., F_{C_p}; \theta) \prod_{m=1}^{q} f_{E_m}(e_m) \prod_{n=1}^{p} f_{C_n}(c_n) \end{aligned} \tag{2.A.3}$$

$$\begin{aligned} f_E(e) &= \frac{\partial^q F_E(e)}{\partial E_1 ... \partial E_q} \\ &= \frac{\mathbb{C}_e(F_{E_1}, ..., F_{E_p}; \theta_e)}{\partial F_{E_1}(e_1) ... \partial F_{E_q}(e_q)} \frac{\partial F_{E_1}(e_1) ... \partial F_{E_q}(e_q)}{\partial E_1 ... \partial E_q} \\ &= c_e(F_{E_1}, ..., F_{E_p}; \theta_e) \prod_{m=1}^{q} f_{E_m}(e_m) \end{aligned} \tag{2.A.4}$$

$$\begin{aligned} f_C(c) &= \frac{\partial^p F_C(c)}{\partial C_1 ... \partial C_p} \\ &= \frac{\mathbb{C}_c(F_{C_1}, ..., F_{C_p}; \theta_c)}{\partial F_{C_1}(c_1) ... \partial F_{C_p}(c_p)} \frac{\partial F_{C_1}(c_1) ... \partial F_{C_p}(c_p)}{\partial C_1 ... \partial C_p} \\ &= c_c(F_{C_1}, ..., F_{C_p}; \theta_c) \prod_{n=1}^{p} f_{C_n}(c_n), \end{aligned} \tag{2.A.5}$$

where $F_{E_m}(e_m)$ and $F_{C_n}(c_n)$ are marginal distribution functions of E_m and C_n. $f_{E_m}(e_m)$ and $f_{C_n}(c_n)$ are their respective marginal density functions.

Substituting equation (2.A.3)-(2.A.5) into equation (2.A.2),

$$\begin{aligned}\Psi(e,c) &= \frac{c(F_{E_1},...,F_{E_p},F_{C_1},...,F_{C_p};\theta)\prod_{m=1}^{q} f_{E_m}(e_m)\prod_{n=1}^{p} f_{C_n}(c_n)}{c_e(F_{E_1},...,F_{E_p};\theta_e)\prod_{m=1}^{q} f_{E_m}(e_m)c_c(F_{C_1},...,F_{C_p};\theta_c)\prod_{n=1}^{p} f_{C_n}(c_n)} \\ &= \frac{c(F_{E_1}(e_1),...,F_{E_p}(e_p),F_{C_1}(c_1),...,F_{C_p}(c_p);\theta)}{c_e(F_{E_1}(e_1),...,F_{E_q}(e_q);\theta_e)c_c(F_{C_1}(c_1),...,F_{C_p}(c_p);\theta_c)} \end{aligned} \tag{2.A.6}$$

This gives equation (2.9).

Secondly, from equation (2.5) the hypothetical outcome distribution can be defined by,

$$F_Y^{EOP}(y) = \int_C \int_E F_{Y|E,C}(y|e,c)dF_{E|C}(e|c)dF_C(c=\widetilde{c}) \tag{2.A.7}$$

Under the assumption of *Stability of the conditional distribution function*, equation (2.A.7) becomes

$$\begin{aligned} F_Y^{EOP}(y) &= \int_E F_{Y|E,C}(y|e,c=\widetilde{c})dF_{E|C}(e|c=\widetilde{c}) \\ &= \int_E F_{Y|E,C}(y|e,c=\widetilde{c})\frac{dF_{E|C}(e|c=\widetilde{c})}{dF_E(e)}dF_E(e) \\ &= \int_E F_{Y|E,C}(y|e,c=\widetilde{c})\Psi(e,c=\widetilde{c})dF_E(e), \end{aligned} \tag{2.A.8}$$

where

$$\Psi(e,c=\widetilde{c}) = \frac{dF_{E|C}(e|c=\widetilde{c})}{dF_E(e)} = \frac{f_{E|C}(e|c=\widetilde{c})}{f_E(e)} = \frac{f_{E,C}(e,c=\widetilde{c})}{f_E(e)f_C(c=\widetilde{c})} \tag{2.A.9}$$

is a weighting factor. Under the assumption of *Stability of dependence structure*, substituting equation (2.A.3)-(2.A.5) to equation (2.A.9),

$$\Psi(e,c=\widetilde{c}) = \frac{c(F_{E_1}(e_1),...,F_{E_p}(e_p),F_{C_1}(\widetilde{c_1}),...,F_{C_p}(\widetilde{c_p});\theta)}{c_e(F_{E_1}(e_1),...,F_{E_q}(e_q);\theta_e)c_c(F_{C_1}(\widetilde{c_1}),...,F_{C_p}(\widetilde{c_p});\theta_c)} \tag{2.A.10}$$

The assumption of *Restricted range of the reference values of C* guarantees $0 \leq F_{C_n}(\widetilde{c_n}) \leq 1$, for all $n = 1,...,p$.

This gives equation (2.10).

■

2.A.2 Unconditional quantile regression

The unconditional quantile regression developed by Firpo et al. (2009) uses the recentred influence function (RIF) of quantiles of the outcome variable, y. Coefficients of explanatory variables at each quantile is estimated by regressing the vector of RIF values on a set of covariates. The difference from OLS is that the RIF regression uses the estimated RIF of quantiles as a dependent variable. The RIF vector is obtained by the following equation:

$$RIF(y; q_\tau) = q_\tau + \frac{\tau - I(y \leq q_\tau)}{f(q_\tau)}, \tag{2.A.11}$$

where q_τ is the τ_{th} quantile of the outcome variable of interest and $f(q_\tau)$ is the unconditional kernel density of y at the τ_{th} quantile. $I(y \leq q_\tau)$ is an indicator function and it becomes 1 if y is smaller or equal to the τ_{th} quantile of the outcome distribution.

2.A.3 OLS regression results

Tables 2.A.1 and 2.A.2 show the OLS regression results for effort variables and for child BMI respectively.

Table 2.A.1: OLS regression results for efforts

	(1) Education years	(2) ln(Wealth)	(3) ln(Food exp)	(4) Prepared food ratio	(5) Staple food ratio	(6) Vigorous activity	(7) Moderate actvity	(8) Eating frequency
Father's BMI in 1993	0.265***	0.0868	-0.0310	0.0260***	-0.0120***	-0.271	0.0828	0.0167
	(0.0988)	(0.0578)	(0.0203)	(0.00621)	(0.00437)	(0.178)	(0.245)	(0.0430)
Mother's BMI in 1993	0.144	0.0240	-0.0414**	0.0203***	-0.00898**	-0.272*	0.181	0.00999
	(0.0923)	(0.0542)	(0.0190)	(0.00563)	(0.00413)	(0.163)	(0.234)	(0.0400)
Father's BMI*Mother's BMI	-0.00222	-0.000606	0.00247***	-0.000830***	0.000300	0.00923	-0.00813	0.000244
	(0.00417)	(0.00243)	(0.000878)	(0.000262)	(0.000187)	(0.00741)	(0.0107)	(0.00185)
Constant	3.412	14.43***	12.45***	-0.461***	0.491***	9.829**	3.862	-0.515
	(2.151)	(1.267)	(0.433)	(0.131)	(0.0951)	(3.865)	(5.302)	(0.919)
Observations	4564	4564	4564	4564	4564	4564	4564	4564
R-squared	0.0470	0.0134	0.0242	0.0162	0.0165	0.00352	0.00155	0.00496

Standard errors in parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 2.A.2: OLS regression results for BMI

	(1) BMI	
Father's BMI in 1993	-0.583	(0.500)
Mother's BMI in 1993	0.883**	(0.415)
Education years	0.0252	(0.187)
ln(Wealth)	0.203	(0.315)
ln(Food expenditure)	-0.676	(0.972)
Prepared food ratio	-3.193	(3.282)
Staple food ratio	-2.685	(4.533)
Vigorous activity	-0.153	(0.0984)
Moderate activity	0.0431	(0.0766)
Eating frequency	-1.360***	(0.430)
Father's BMI*Mother's BMI	-0.00272	(0.00617)
Father's BMI*Education years	0.00195	(0.00776)
Father's BMI*ln(Wealth)	-0.0142	(0.0133)
Father's BMI*ln(Food expenditure)	0.0928**	(0.0410)
Father's BMI*Prepared food ratio	0.147	(0.130)
Father's BMI*Staple food ratio	0.133	(0.194)
Father's BMI*Vigorous activity	0.00529	(0.00426)
Father's BMI*Moderate activity	-0.00324	(0.00324)
Father's BMI*Eating frequency	0.0283	(0.0183)
Mother's BMI*Education years	-0.00748	(0.00609)
Mother's BMI*ln(Wealth)	0.0112	(0.0111)
Mother's BMI*ln(Food expenditure)	-0.0531	(0.0341)
Mother's BMI*Prepared food ratio	-0.0299	(0.107)
Mother's BMI*Staple food ratio	-0.0789	(0.149)
Mother's BMI*Vigorous activity	-0.00123	(0.00354)
Mother's BMI*Moderate activity	0.00216	(0.00255)
Mother's BMI*Eating frequency	0.0305**	(0.0143)
Constant	15.03	(11.67)
Observations	4564	
R-squared	0.132	

Standard errors in parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Chapter 3

Joint Impact of the Conditional Cash Transfer on Child Nutritional Status and Household Expenditure in Indonesia

Abstract

Child malnutrition, which hampers sound human capital development, is often associated with the low socio-economic status of poor households in developing countries. This study investigates the impact of a conditional cash transfer programme in Indonesia, the *Program Keluarga Harapan* (PKH), on the marginal and joint distributions of child nutritional status and household expenditure 26-30 months after its implementation. The cluster-randomised control trial project conducted in Indonesia provides an opportunity to semi-parametrically estimate the causal impacts on the dependence between them. The results show that the PKH increases the higher quantiles of weight-for-age z-score among children aged between 25 and 36 months. Its improvement is explained not by the rise in household expenditure due to the PKH but by the change in the association between nutritional status and household expenditure. Furthermore, the PKH strengthens the positive association between nutritional status and household expenditure. For the other younger age groups, a significant improvement in nutritional status is not observed.

JEL code: I14, I15, I18, I38

Keywords: Conditional cash transfer; Malnutrition; Change-in-changes estimator; Copulas; Indonesia

3.1 Introduction

In developing countries, malnutrition is a substantial factor in the causes of death among children aged under five. In fact, child malnutrition contributes to nearly half of all deaths in children under five years of age, translating into the loss of approximately 3 million young lives every year (UNICEF, 2017). Malnutrition accounts for 35 per cent of the burden of disease in children under five (Black et al., 2008). It also impedes their sound human capital development beyond the health domain (Hoddinott et al., 2013). The first few years of a child's life are of enormous importance for growth because vital development occurs in all areas in these very early stages, including the development of the brain's structural and functional capacity (Grantham-McGregor et al., 2007). Mounting evidence has shown that health conditions in childhood have persistent and continued effects on adult health, cognitive development, educational achievement and even future income.[1] Malnourished children may be at high risk of impaired health and function throughout their lives (Martorell, 1995). As these conditions extend into adulthood and hence the reproductive years, restricted development in childhood affects subsequent generations, perpetuating an intergenerational vicious cycle of economic and health inequality (Black and Hurley, 2014; World Bank, 2006).

Numerous studies have revealed significant relationships between parental socio-economic status (SES) and child health. Children in poorer households are often at a higher risk of experiencing malnutrition (Case et al., 2002; Currie and Moretti, 2003; Currie and Vogl, 2013; Aizawa, 2019), partly because they tend to live in dilapidated, poor and crowded houses with poor sanitary conditions (Ahsan et al., 2017; Caulfield et al., 2004; Marmot, 1999) and with parents who often have limited educational backgrounds (Chen and Li, 2009; Currie and Moretti, 2003; Ahsan and Maharaj, 2018; Appoh and Krekling, 2005). In contrast to adults, any health disparity among children generally cannot be attributed to their own lifestyles for which they can be held responsible. Clearly, children cannot choose their parents and they have no control over the environment in which they grow up. In this sense, such health disparities among children may well be considered inequitable.

Addressing child malnutrition is hugely important for both the resilient economic growth of countries and for individuals because the economic costs attributable to child malnutrition are

[1] See for example Case and Paxson (2010); Strauss and Thomas (2007); Hoddinott et al. (2013) for a review.

substantial. According to World Bank (2006), productivity loss due to malnutrition is estimated to be more than 10 per cent of lifetime incomes. The Sustainable Development Goals, adopted in 2015 by the United Nations, call for worldwide action to end all forms of malnutrition among children under five years of age and to protect them from avoidable death (UNICEF, 2017).

Conditional cash transfer (CCT) programmes, which transfer money to households contingent on investments in human capital, have been some of the most adopted initiatives in the last two decades after the success of the CCT programmes in Mexico (*Progresa*, renamed *Oportunidades* in 2001) and Brazil (*Bolsa Escola*) (Handa and Davis, 2006). To date, these CCT programmes have been launched in several countries in Latin America, Africa and Asia.[2] The primary objectives of the CCTs are to mitigate short-term poverty, assist long-term well-being through investments in human capital development and break the vicious cycle of intergenerational poverty.

CCTs are expected to improve the welfare of beneficiaries through two main mechanisms: cash transfer and conditionality. Conditionality is considered to be the main driver of behavioural change in beneficiaries. An unconditional cash transfer (UCT), which transfers cash without any strings attached, may be theoretically preferable to CCTs under the assumption that individual beneficiaries are rational and fully informed and that the market is functioning perfectly (Bassett, 2008). Nevertheless, imposing conditionality is justified in situations where myopic individuals with incomplete information undervalue the long-term benefits of human capital development and do not fully recognise the risk of child malnutrition. CCTs are designed to help poor households with high discount rates to make better decisions. Furthermore, in the presence of market failure such as positive externalities, individually optimal levels of human capital investment may be lower than socially optimal investment levels; child immunisation and household investment in sanitary conditions are typical examples. In such cases, imposing conditionality can help them attain a socially desirable and optimal level of human capital investment (Fiszbein et al., 2009; Adato and Hoddinott, 2007).

The main objective of this study is to estimate the impacts of the CCT pilot programme in Indonesia on the marginal and joint distributions of child nutritional status and household expenditure. We take advantage of the randomised experimental design that was implemented

[2]For reviews of the CCT programmes, see Rawlings and Rubio (2005).

for the purpose of evaluating the programme. We measure the impacts on their association by simulating the hypothetical marginal and joint distribution of health and expenditure for the treatment group in the absence of the treatment, which is achieved by the change-in-changes (CIC) method with an additional identification assumption regarding a copula structure controlling the dependence between health and expenditure (Athey and Imbens, 2006; Cañón Salazar, 2016). Estimating the hypothetical joint distribution not only makes it possible to measure the impact on the association between child health and household expenditure but also allows us to explore the mechanism by which the CCT affects nutritional status. We estimate how much of the treatment effect on health is due to the change in household expenditure. We decompose the impact on the marginal distribution of health into the part that is attributable to the rise in expenditure (expenditure effect) and the remaining part that cannot be explained by the change in expenditure (behavioural effect) through a re-weighting decomposition approach (DiNardo et al., 1996). This decomposition analysis helps us disentangle the pathways by which CCTs influence a child's nutritional status.

3.2 The conditional cash transfer programme in Indonesia

Despite rapid and strong economic growth in Indonesia, 87 million people still remain vulnerable to food insecurity, and the country has the fifth highest number of stunted children in the world (FAO, IFAD and WFP, 2015). In 2007, Indonesia introduced a pilot conditional cash transfer programme called the PKH (*Program Keluarga Harapan*). This initiative is aimed at very poor households in seven provinces that were chosen based on their willingness to participate and the extent to which they represented the country's diversity.[3] Like other CCT programmes in Latin America, the PKH is intended to improve the welfare of poor households by providing them with a quarterly cash transfer in order to improve the health and educational outcomes of the participating children (Table 3.1-A).

The PKH aims to mitigate household poverty in the short term while encouraging human capital investment by enhancing school enrolment and improving the health conditions of children and pregnant women in the long term. The provision of subsidies, in principle, is conditional on the fulfilment of the following conditions (Table 3.1-B): in respect of pregnant women, they must

[3]The provinces selected were DKI Jakarta, West Java, East Java, West Sumatra, North Sulawesi, East Nusa Tenggara and Gorontalo.

take iron supplements and visit an office for postnatal care; with regard to children aged under 6 years, they must complete immunisation programmes and receive regular health check-ups; and for children aged 6-15, they must achieve 85 per cent school attendance. Field facilitators advise the beneficiary households to comply with the conditionalities, informing them of their rights and obligations and monitoring eligibility. If these conditionalities are not complied with, subsequent transfer payments are reduced and, after a series of warnings, the transfers cease altogether.[4]

The PKH cash benefit is transferred directly to mothers only and the amount of the cash transfer is around 12 per cent of the total expenditure of the beneficiary household (World Bank, 2012). Moreover, the amount of subsidy depends on the number of children in the household as well as their age (Table 3.1-C), and it ranges from IDR 600,000 (approximately USD 65) to IDR 2,200,000 (approximately USD 240) per year including the fixed base cash transfer.[5] IDR 1,000,000 per year is provided to households with children aged under 6 or to those with pregnant or lactating mothers. Households with a primary school student (aged 7-12) receive IDR 800,000 per year while IDR 1,000,000 per year is awarded to households with a secondary school student (aged 13-15). The government limited the maximum transfer to IDR 2,200,000 regardless of the number of children in the household. There are no specific rules as to how the transferred cash must be used. As well as the cash transfer, the PKH beneficiaries can receive in-kind benefits. They can use maternal and infant healthcare services at district hospitals, community health centres (*puskesmas*), community health posts (*posyandu*) and village delivery posts (*polindes*) without out-of-pocket expenditure in accordance with the applicable regulations to comply with the conditionality.

3.3 Experimental evidence of impacts of CCTs on anthropometric and nutritional outcomes

Evidence of the causal impacts of CCTs on child anthropometric measures is extremely mixed across countries (Manley et al., 2013; Hoddinott and Bassett, 2008). In contrast to healthcare utilisation where the majority of CCTs show positive impacts, impacts on nutrition-related measures are more sensitive to the timing of the impact measurement and the extent of the

[4]However, in practice, the verification system did not begin until at least 2010 (Cahyadi et al., 2018).
[5]The exchange rate as of 1 August 2007 is used for the currency conversion.

Table 3.1: PKH target, cash transfer amount and conditionality

Panel A: PKH target indicators

	Health indicators
1	Four prenatal care visits
2	Taking iron tablets during pregnancy
3	Delivery assisted by a trained professional
4	Two postnatal care visits
5	Complete childhood immunisations
6	Adequate monthly weight increases for infants
7	Monthly weighing for children under three and biannually for children under five
8	Vitamin A twice a year for children under five
	Education indicators
9	Primary school enrolment of children 6-to-12 years old
10	Minimum attendance rate of 85 per cent for primary school-aged children
11	Junior secondary school enrolment of children 13-to-15 years old
12	Minimum attendance rate of 85 per cent for junior secondary school-aged children

Panel C: PKH cash transfer amount per year

PKH assistance	Amount (Rp)
Fixed cash transfer	200,000
Cash transfer per household with	
a. Child aged less than 6 years	800,000
b. Pregnant or lactating mother	800,000
c. Children of primary school age	400,000
d. Children of secondary school age	800,000
Minimum transfer per household	600,000
Maximum transfer per household	2,200,000

Panel B: PKH conditionality

Households with pregnant or lactating women	(i)	Complete four antenatal care visits and take iron tablets during pregnancy
	(ii)	Be assisted by a trained professional during the birth
	(iii)	Lactating mothers must complete two post-natal care visits
Households with children aged 0-6 years	(iv)	Ensure that the children have complete childhood immunization and take Vitamin A capsules a minimum of twice a year.
	(v)	Take children for growth monitoring check-ups (monthly for infants 0-11 months, and quarterly for children 1-6 years).
Households with children aged 6-15 years	(vi)	Enrol their children in primary school and ensure attendance for a minimum of 85 per cent of school days.
	(vii)	Enrol junior secondary school children and ensure attendance for a minimum of 85 per cent of school days.
Households with children aged 16-18 years but have not completed nine years of primary and secondary school	(viii)	Enrol their children in an education programme to complete 9 years equivalent.

Source: Ministry of Social Affairs of Indonesia (2007)

treatment (Fiszbein et al., 2009; Leroy et al., 2009; Glassman et al., 2007). Moreover, significant results tend to be observed only in some beneficiary subgroups (Lagarde et al., 2007). In this section, we firstly review experimental evidence of the impacts of CCTs on child nutritional outcomes in Latin America, where the various types of CCTs were designed, and we then review the existing evidence in relation to the CCT in Indonesia. Programme details, major studies and their impacts are summarised in Table 3.2.

3.3.1 Limited improvements in Honduras and Brazil

The CCTs in Honduras (*Programa de Asignación Familiar*) and Brazil (*Bolsa Alimentação*) reveal no significant effect on child anthropometric outcomes (Morris et al., 2004; Hoddinott and Bassett, 2008). The lack of impact on nutritional status in Honduras is in all likelihood due to the small size of the transfer, which is less than 4 per cent of average beneficiary expenditure and around 10 per cent of household consumption, and the infrequent distribution of the transfer (IFPRI, 2003; Hoddinott and Bassett, 2008). In Brazil, where around 8 per cent of household expenditure is transferred, a negative impact on the height-for-age z-score (HAZ score) for children younger than 7 is even observed (Morris et al., 2004). Morris et al. (2004) attribute this counterintuitive negative effect to beneficiaries perhaps misunderstanding the programme's eligibility criteria. Morris et al. (2004) argue that some beneficiaries may have had a misconception that benefits would be discontinued if their child's growth began to improve.

3.3.2 Significant improvements in Colombia, Mexico and Nicaragua

On the other hand, significant improvements in child nutritional outcomes have been found in Colombia, Mexico and Nicaragua. The CCT programme in Colombia (*Familias en Acción*), which transfers around 24 per cent of household expenditure, improved the HAZ score by 0.16 in respect of newborns and infants aged under 24 months ($p < 0.10$), translating into a 6.9 percentage point decrease in the probability of stunting (Attanasio et al., 2005). However, studies do not show any impact on the nutritional status of children aged 24 months and older.

Positive impacts of the programme in Mexico (*Progresa/Oportunidades*) on child nutritional status are reported by several studies (Behrman and Hoddinott, 2001; Leroy et al., 2008; Rivera et al., 2004; Gertler, 2004; Behrman and Hoddinott, 2005; Hoddinott and Bassett, 2008). For example, Gertler (2004) reports that children aged 12-36 months who were exposed to the pro-

Table 3.2: Evidence in Latin America

Country and CCT programme name	Transfer size (USD $)	Major studies	Exposure duration	Impacts on anthropometrics and nutritional outcomes
Honduras *Programa de Asignación Familiar*	$17 on average ≤4% of HH expenditure ≈10% of HH consumption	IFPRI (2003)	2 years	No significant impact among all children
Brazil *Bolsa Alimentação*	$6.25–18.70/beneficiary ≈8% of HH expenditure ≈10% of HH consumption	Morris et al. (2004)†	6 months	Decrease in HAZ score by 0.13 (0-84m)**
Colombia *Familias en Acción*	$50 on average ≈24% of HH expenditure ≈30% of HH consumption	Attanasio et al. (2005)	1 year	Increase in HAZ score by 0.16 (0-24m)* Decrease in probability of stunting by 6.9 p.p. (0-24m)**
Mexico *Progresa/ Oportunidades*	$20 on average ≈20% of HH expenditure ≈25% of HH consumption	Behrman and Hoddinott (2001)	1 year	Increase in height by 1.02 cm (12-36m)** Increase in height by 0.84 cm (6-36m)** Increase in height by 0.98 cm (12-30m)** Increase in height by 0.70 cm (12-42m)** Increase in height by 0.62 cm (12-48m)**
		Leroy et al. (2008)†	2 years	Increase in HAZ score by 0.41 (0-6m)** Increase in WAZ score by 0.47 (0-6m)** Increase in height by 1.5 cm (0-6m)** Increase in weight by 0.76 kg (0-6m)***
		Rivera et al. (2004)	2 years	Increase in height by 1.1 cm (0-6m, poorest 50th percentile)**
		Gertler (2004)	1 year	Increase in height by 0.96 cm (12-36m‡)***
		Behrman and Hoddinott (2005)	1 year	Increase in height by 1.02 cm (12-36m)** Increase in height by 1.22 cm (24-36m)**
		Hoddinott and Bassett (2008)	1 year	Decrease in probability of stunting by 7.3 p.p. (0-36m)* Decrease in probability of stunting by 18.0 p.p. (12-36m)***
Nicaragua *Red de Protección Social*	$25 on average ≈18% of HH expenditure ≈20% of HH consumption	Maluccio and Flores (2005)	2 years	Decrease in probability of stunting by 5.5 p.p. (0-36m)* Decrease in probability of being underweight by 6.2 p.p. (0-36m)**

Source: Bassett (2008); Lagarde et al. (2007); Gaarder et al. (2010); Ranganathan and Lagarde (2012); and individual studies.

Note: HH= Household; HAZ= Height for age; WAZ= Weight for age; Stunting is defined by HAZ<-2; and being underweight is defined by WAZ<-2.

Parenthesis indicates the age of children measured by month.

Impacts are intent-to-treat effects among eligible households located in the treatment cluster, unless otherwise indicated.

† Treatment is defined as the actual enrolment. ‡ At the time of follow-up survey. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

gramme for one year are taller than children from controlled areas by 0.96 cm ($p < 0.01$), but a reduction in the likelihood of being stunted was not observed. Rivera et al. (2004) report an increase in height by 1.1 cm ($p < 0.05$), after two years of exposure to the treatment, among infants younger than 6 months at baseline living in the poorest households. Behrman and Hoddinott (2005) also find similar results, reporting the increase in height by 1.02 cm ($p < 0.10$) among children aged 12-36 months at baseline after a one-year exposure to the *Progresa*. The authors argue that the main positive impact comes from the children aged 24-36 months who are given nutritional supplements when they are underweight.[6] The large transfer size, approximately one-fifth of per capita household expenditure, and the provision of a nutritional supplement contribute to the significant positive effect of *Progresa/Oportunidades* (World Bank, 2008; Handa and Davis, 2006).

In Nicaragua, the CCT (*Red de Protección Social*) reduces the prevalence of being stunted and underweight by 5.5 ($p < 0.10$) and 6.2 ($p < 0.05$) percentage points respectively (Maluccio and Flores, 2005). This impressive improvement is attributed to the relatively large transfer, approximately 18 per cent of average monthly household expenditure, and the programme's wide range of nutrition-related conditionality (Maluccio and Flores, 2005; Hoddinott and Bassett, 2008). On the other hand, it does not lower the probability of being wasted, which is due to the very low level of wasting at baseline (Maluccio and Flores, 2005).

Bassett (2008) argues that the size of the transfer appears to be critical in terms of nutritional improvement. The successful CCT programmes in Mexico, Nicaragua and Colombia transfer approximately 20 per cent of the average beneficiary expenditure, while in Honduras and Brazil, where no significant improvement was found, the transfer is below 8 per cent of the average beneficiary expenditure. Fernald et al. (2008) report that the larger transfer yields greater health improvement in the case of Mexico. Bassett (2008) also points out that the successful CCT programmes have multiple nutrition-related conditionalities, including a micronutrient supplement in Nicaragua and a nutritional supplement in Mexico, which could have brought about the even larger impact on nutritional status.

[6] *Progresa* provided a nutritional supplement (called a *papilla*) to pregnant and lactating women, children aged between 4 and 24 months, and children aged between 2 and 5 with any signs of malnutrition. The supplement for children constituted 20 per cent of their daily caloric requirements and 100 per cent of their micronutrient requirements (Rosado et al., 2000).

3.3.3 Evidence in Indonesia

In Indonesia, while the PKH significantly improved the use of maternal and child healthcare services (Alatas, 2011; Kusuma et al., 2017, 2016), the impacts of the PKH on nutritional status are mixed, although they are not statistically significant; a positive impact of the PKH on the mean weight-for-age z-score (WAZ) and a negative impact on the mean HAZ score have been reported two years after the PKH was implemented (Alatas, 2011). Kusuma et al. (2017) estimate the impact on children's food consumption and nutritional outcomes. Regarding food consumption, the authors find significant positive impacts of the PKH on the consumption of milk ($p < 0.10$) and fish ($p < 0.10$). In respect of the nutritional outcome, they report that, for children aged 24-36 months at baseline in 2007, the PKH reduces the prevalence of wasting and severe wasting by 6.3 percentage points ($p < 0.05$) and 3.7 percentage points ($p < 0.10$) respectively. However, a reduction in the likelihood of being stunted is not observed (Kusuma et al., 2017).

Cahyadi et al. (2018) recently reported the continued effect on various outcomes by re-surveying the households to measure the medium-run performance of the PKH. They show cumulative effects on the prevalence of stunting six years after the PKH was implemented. In their six-year follow-up survey, the prevalence of being stunted declined by approximately 9-11 percentage points ($p < 0.05$) among children aged 0-60 months. On the other hand, no significant impact was observed regarding the likelihood of being underweight. Cahyadi et al. (2018) highlight the possible cumulative effect of the PKH on children's height. This follow-up research suggests that the period for which children are exposed to the treatment is also a significant factor of nutritional improvement.

Our study's main contributions to the literature are threefold. First, this study estimates the treatment effect on the entire distribution of health outcomes and household expenditure, which tends to be unexplored in the majority of empirical studies on CCTs. Certainly, estimating the average treatment effect provides valuable information to policymakers, but a sole focus on the average treatment effect could potentially miss important distributional effects that do not show up as a change in means. A more thorough evaluation becomes possible by exploring the impacts on the entire outcome distributions beyond their means. Second, this paper estimates the impact of the PKH on the joint distribution of health outcomes and household expenditure, which has not specifically been examined by the existing literature. CCT programmes are likely to,

directly and indirectly, affect both nutritional status and households' purchasing power through the cash transfer and behavioural changes. Hence, they are likely to change the association between health outcomes and household expenditure, as well as individual outcomes. Examining the impact on the joint distribution allows us to analyse whether the PKH strengthens or weakens the association between household expenditure and health. Third, this paper aims to disentangle the mechanism by which the PKH affects health outcomes by estimating how much of the changes in health outcomes is driven solely by the change in the distribution of household expenditure. This is achieved by decomposing the estimated effect on health into the part that can be attributed to the change in household expenditure and the remaining part that cannot be explained by it.

3.4 Data

3.4.1 Randomisation design of the PKH pilot programme

The pilot PKH programme was conducted in 2007 in six provinces[7] within which the richest 20 per cent of districts were excluded. The selection of eligible sub-districts was based on certain characteristics, including the prevalence of malnutrition, the poverty rate, school drop-outs and the availability of health and education facilities. A total of 1,085 sub-districts that were considered "supply-side ready" were randomly selected to participate in the programme. As a result, 769 sub-districts were included in the treatment group and the remaining 316 sub-districts were retained as control groups. Targeted households were those classified as extremely poor by Statistics Indonesia using both economic and asset-based poverty measurements.[8]

3.4.2 Sample construction

A baseline household survey in the treatment and control sub-districts was conducted between June and August 2007 by the National Planning Agency and the World Bank. It was designed as a cluster-randomised control trial at sub-district level. Randomising over sub-districts (not households or villages) captures the intra-sub-district spill-over effects, while eliminating any bias from inter-sub-district spill-over effects. Ethical approval was not necessary because this study

[7] They are Jakarta, West Java, East Java, North Sulawesi, East Nusa Tenggara and Gorontalo.

[8] Under the definition supplied by Statistics Indonesia, a very poor household is one that has less-than-poverty-line expenditure overall, spends a large proportion of its available income on basic staple foods, cannot afford medical treatment (except at community health clinics or other subsidised or free public health facilities) and cannot afford sufficient new or replacement clothing.

utilises public data without identifiable private information.[9]

First, 180 treatment sub-districts and 180 control sub-districts were randomly selected. Both treatment and control samples were stratified by urban and rural classification. Within each selected sub-district, eight villages were then randomly selected. Per village, one ward was randomly selected. Next, five households per ward were randomly selected based on PKH beneficiaries criteria (two households with pregnant/lactating women and three households with children aged 6 to 15) (Figure 3.1). Eventually, 14,326 households consisting of 73,563 individuals were interviewed in the baseline survey. The distributions of household characteristics are balanced in the baseline survey (Sparrow et al., 2008). About 75 per cent of the survey households were located in urban areas. The same households were interviewed again in the follow-up survey conducted between October and December 2009, allowing us to construct fully balanced panel data to evaluate the impact of the PKH approximately 26-30 months after its implementation.

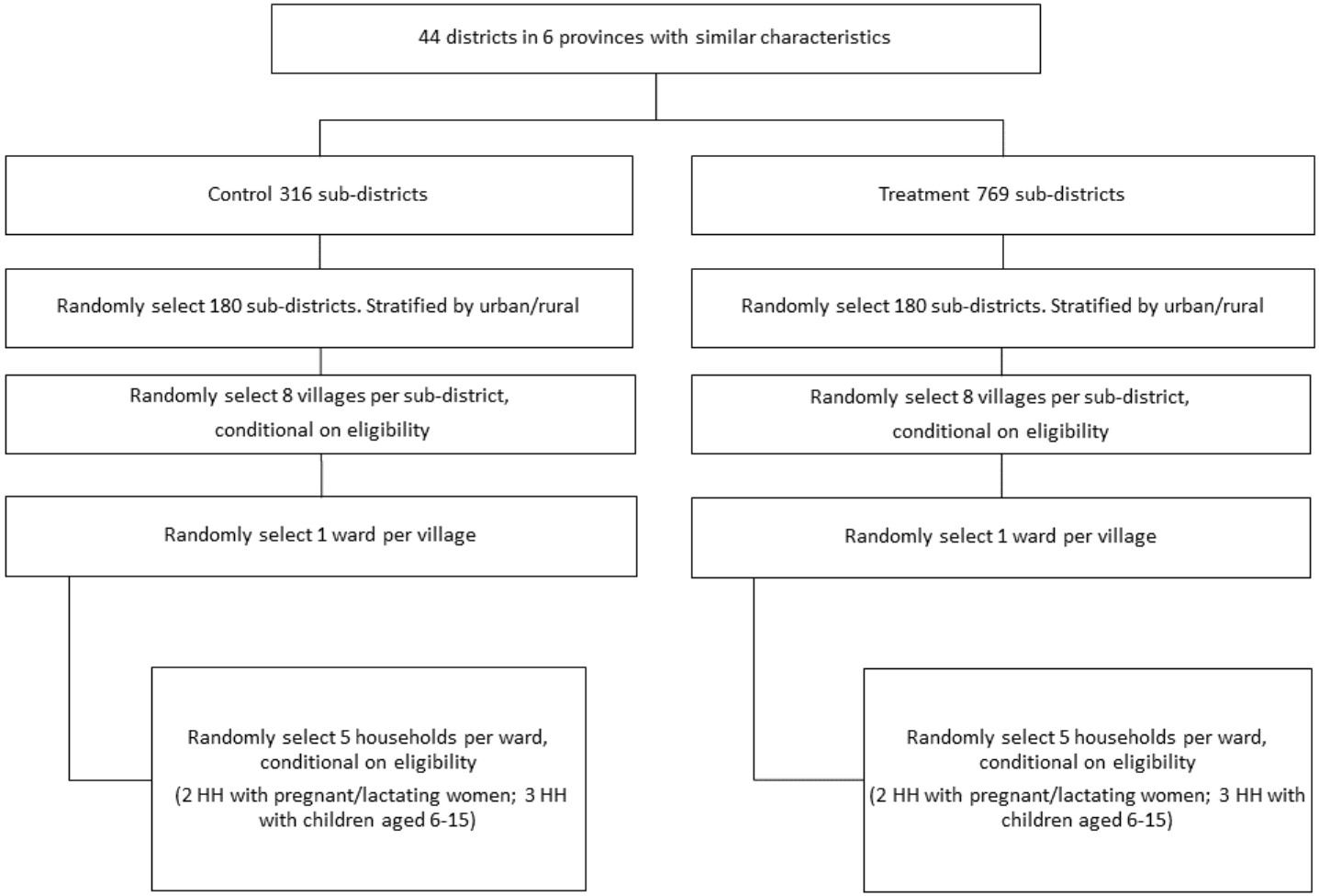
3.4.3 Nutritional status

We measure child development by a weight-for-age z-score (WAZ score) and a height-for-age z-score (HAZ score). These scores were calculated on the basis of actual measurements of the heights and weights of the respondents. The WAZ/HAZ scores measure the difference between the weight/height of an individual and the median value of the "healthy" reference population for the same sex and age, divided by the standard deviation of the reference population.[10] The WAZ and HAZ scores are commonly used as measurements of the nutritional status of children (Glewwe et al., 2001; Case and Paxson, 2010; Caulfield et al., 2004; Pelletier et al., 1993). The WAZ score measures short-term nutritional conditions. A low WAZ score is associated with the condition of being underweight. Meanwhile, the HAZ score measures long-term cumulative nutritional conditions and a low HAZ score is associated with chronic under-nutrition caused by poor food consumption (Walker et al., 2007). Low WAZ and HAZ scores are both caused by poor diet and often compounded by recurrent infectious diseases. The risk of death increases with descending WAZ and HAZ scores (Black et al., 2008). A WAZ score and a HAZ score below -2 (i.e., two standard deviations below the international reference median) are defined as the

[9] We use the Impact Evaluation of PNPM Generasi Program data set provided by the World Bank.

[10] According to World Health Organization (2006), the reference population refers to healthy and well-nourished children in the United States. This standard captures the growth and development process of children from widely diverse, ethnic and cultural backgrounds (O'Donnell et al., 2008). Their rationales are discussed in de Onis and Lobstein (2010).

Figure 3.1: PKH sampling design



Source: Sparrow et al. (2008). Note: HH=Households.

indicators of being underweight and stunted respectively, while scores below -3 are defined as the indicators of being severely underweight and severely stunted respectively.

3.4.4 Household expenditure

As a measurement of household welfare or socio-economic status, we use the logarithm of household expenditure on non-food purchases and food consumption during the last 12 months of the interview process.[11] To reflect the difference in household compositions, the equivalent household expenditure is calculated using the Oxford scale[12], which assigns a weight of 1.0 to the first household member, 0.7 to each additional adult and 0.5 to each child, then calculating the weighted average expenditure per household member.[13] Certainly, a household's food consumption is directly related to a child's nutritional status, but non-food purchases are equally important to their health. For example, a household's spending on child healthcare and investment in its sanitary conditions are essential elements of better child health. The spending on non-food purchases and food consumption can reflect both the long-term and short-term living standards of the household. Compared with income, household expenditure is less susceptible to seasonal events such as drought, which is significant in the analysis of developing countries where agriculture plays an important role.

3.5 Method

3.5.1 Notation

First, $g \in \{A, B\}$ denotes the control and treatment groups and $t \in \{1, 2\}$ denotes the time period. The group $g = B$ in period $t = 2$ is the only group that is exposed to the treatment. In the first period ($t = 1$), both groups had no treatment. H and Y denote continuous health outcomes and expenditure. Conditional health and expenditure in group g in period t are denoted by H_{gt}

[11] Specifically, non-food includes (a) housing and household facilities, (b) miscellaneous goods and services, (c) educational costs, (d) health costs, (e) clothing, shoes and headwear, (f) durable goods and household equipment, (g) taxes, (h) health insurances, and (i) feasts and rituals. Food consumption measures the monetary values of the food consumed, and this includes not only food purchased but also that which the participants produce themselves or receive from other sources. As the survey information on food consumption relates to just one week, the total values are multiplied by 52 (weeks) to ensure consistency with the measurement of non-food purchases.

[12] Also known as the OECD equivalence scale.

[13] As robustness checks, we calculate the equivalent expenditure by the square root scale equivalisation methods, which divide the total household expenditure by the squared family size, and the equivalent expenditure which divides the total expenditure by family size. However, we did not find systematic differences in the final results across the adjustment methods. This is mainly thanks to the successful stratified data collection across household compositions in both the control and treatment groups. The t-test and the Kolmogorov-Smirnov test did not indicate a significant difference in the household compositions between the control and treatment groups.

and Y_{gt} respectively. H_{gt} and Y_{gt} have marginal distributions, $F_{H,gt}(h)$ and $F_{Y,gt}(y)$, and their supports are denoted by $\mathbb{H}_{gt}$ and $\mathbb{Y}_{gt}$.

Following Rubin (1974), we assume that each individual has two possible outcomes, namely $H^I_{gt} \in \mathbb{H}^I_{gt}$ and $Y^I_{gt} \in \mathbb{Y}^I_{gt}$ in the presence of the treatment and $H^N_{gt} \in \mathbb{H}^N_{gt}$ and $Y^N_{gt} \in \mathbb{Y}^N_{gt}$ in the absence of the treatment. We can only observe one of them for each person, but never both. That is H^I_{gti} and Y^I_{gti} are observed only if $g = B$ and $t = 2$, and H^N_{gti} and Y^N_{gti} are observed unless $g = B$ and $t = 2$.

Let $F_{H,Y,gt}$ be the bivariate *joint* distributions of (H, Y) and let $v(F_{H,Y,gt})$, $v(F_{H,gt})$ and $v(F_{Y,gt})$ denote the distributional summary statistics of interest. We define the treatment effects on the joint distribution as $\tau_{HY} = v(F^I_{H,Y,B2}) - v(F^N_{H,Y,B2})$ and on the marginal distributions as $\tau_H = v(F^I_{H,B2}) - v(F^N_{H,B2})$ and $\tau_Y = v(F^I_{Y,B2}) - v(F^N_{Y,B2})$. As we condition on whether the household resides in a treatment sub-district, not on the actual participation in the PKH programme, in essence, the estimates reflect the intention-to-treat effect.

The fundamental identification problem with regard to the treatment impacts is that for any individual in group $g = B$ in time period $t = 2$, we cannot observe their potential outcomes in the absence of the treatment, i.e. the outcome that a person in group $g = B$ and time period $t = 2$ would have experienced had he/she not been exposed to the treatment. The impossibility of observing (H^N_{B2}, Y^N_{B2}) prevents us from estimating $v(F^N_{H,Y,B2})$, $v(F^N_{H,B2})$ or $v(F^N_{Y,B2})$, and hence we cannot identify the treatment effects without further assumptions.

3.5.2 Estimation of the counterfactual distributions by change-in-changes estimators

The experimental study design gives us a reliable source of identification. Athey and Imbens (2006) propose a non-linear difference-in-differences model, which is also known as the change-in-changes model (CIC). The CIC does not rely on functional form assumptions and can non-parametrically estimate the entire counterfactual distribution of the outcome for the treatment group in the post-treatment period in the absence of the treatment, i.e. $F^N_{H,B2}$ and $F^N_{Y,B2}$. Specifically, the CIC non-parametrically estimates the change across time that happened in the control group, and estimates the counterfactual distribution for the treatment group by assuming that

the distribution in the treatment group would have experienced the same change without the treatment. The CIC allows the effects of time and treatment to differ across individuals. Estimation of the counterfactual distribution makes it possible to identify, in essence, the treatment effect on any univariate functional of the outcome variable beyond its mean.

Suppose H and Y have the following general structures in the absence of the treatment:

$$H^N = m(u^H, t) \tag{3.1}$$

$$Y^N = n(u^Y, t), \tag{3.2}$$

where $m(.)$ and $n(.)$ are unknown functions, and u^H and u^Y denote the unobservable characteristics of individuals for H and Y respectively. We assume u^H and u^Y are not necessarily independent. Equations (3.1) and (3.2) imply that the outcomes do not directly depend on the group indicator and that all relevant unobservable characteristics in each outcome can be captured in a single index. We do not impose specific functional form assumptions.

With the CIC model, the full marginal distributions of H^N_{B2} and Y^N_{B2} are identified through

$$F^N_{H,B2}(h) = F^N_{H,B1}(F^{-1,N}_{H,A1}(F^N_{H,A2}(h))) \tag{3.3}$$

$$F^N_{Y,B2}(y) = F^N_{Y,B1}(F^{-1,N}_{Y,A1}(F^N_{Y,A2}(y))). \tag{3.4}$$

Equations (3.3) and (3.4) imply that the counterfactual distribution in the absence of the treatment can be identified from the observable distributions. Assumptions used to estimate them by the CIC are described in section 3.A.1 in the Appendix. Equations (3.3) and (3.4) can be more intuitively interpreted, if we take their inverse as follows:

$$F^{-1,N}_{H,B2}(\alpha) = F^{-1,N}_{H,A2}(F^N_{H,A1}(F^{-1,N}_{H,B1}(\alpha))) \tag{3.5}$$

$$F^{-1,N}_{Y,B2}(\alpha) = F^{-1,N}_{Y,A2}(F^N_{Y,A1}(F^{-1,N}_{Y,B1}(\alpha))). \tag{3.6}$$

For example, equation (3.6) states that an individual at the αth quantile of the counterfactual expenditure distribution in the treatment group ($g = B$) in the post-treatment period ($t = 2$) without the treatment would be at the $F^N_{Y,A1}(F^{-1,N}_{Y,B1}(\alpha))$th quantile point of the observable distribution in the control group ($g = A$) in the post-treatment period ($t = 2$).

3.5.3 Extension of the CIC for the bivariate joint distribution

An additional assumption regarding the joint distribution of unobservable characteristics enables us to measure distributional effects with multiple treatment outcomes (Cañón Salazar, 2016). We assume the invariance of dependence structure. Specifically, this assumes that within each group, the dependence structure of (u^H, u^Y) is invariant across time, i.e. $(u^H, u^Y) \perp t|g$. This assumption states that the dependence structure between u^H and u^Y can systematically vary across the groups, but not over time within groups. This, however, does not necessarily require that individual i should have the same values (u_i^H, u_i^Y) in each period, but rather this assumption only requires that the dependence structure of (u^H, u^Y) is stable across time given the group. This assumption, though not testable, is plausible when group composition does not change over time. The immediate implication is that the dependence of (u_{B1}^H, u_{B1}^Y) is assumed to be identical to that of (u_{B2}^H, u_{B2}^Y). More detailed explanations are provided in section 3.A.2 in the Appendix.

The counterfactual joint distribution in group B in period $t = 2$ in the absence of the treatment is given by:

$$F_{H,Y,B2}^N(h, y) = F_{H,Y,B1}^N(F_{H,A1}^{-1,N}(F_{H,A2}^N(h)), F_{Y,A1}^{-1,N}(F_{Y,A2}^N(y))). \tag{3.7}$$

The identification of the counterfactual joint distribution leads to the identification of the treatment impact on the joint distribution. In addition, this allows us to identify the counterfactual conditional distribution of H given Y (Cañón Salazar, 2016). We subsequently make the most of this property in disentangling the mechanism by which the PKH affects child health by decomposing the impact on child health into two components (see section 3.5.5).

If the invariance of dependence structure assumption fails, we cannot identify the counterfactual joint distribution in group B in period $t = 2$ in the absence of the treatment. However, it does not prevent us from estimating the impacts on the marginal distributions of health outcome and expenditure. Certainly, as the invariance of dependence structure assumption is the one relating to the hypothetical distribution of unobservable factors in the absence of the treatment, we cannot test it from the available data. However we did not find strong evidence against its plausibility. First, we did not find any large-scale natural disaster or epidemic shock between the time of data collection which could threaten the assumption. Second, in this study, as we use fully

balanced panel data, both groups did not experience structural changes in their within-group compositions.

3.5.4 Copulas

Joint distribution and copulas

In general, joint distributions can be expressed by their marginal distributions and a copula function. Copulas allow us to model dependence structures of variables from their marginal distributions, and they have the property that a functional association between underlying variables is not influenced by their marginal behaviours (Nelsen, 2006; Trivedi and Zimmer, 2007). In general, applying Sklar's theorem, there exists a copula function, $C(.,.)$, such that

$$F_{H,Y}(h,y) = C(F_H(h), F_Y(y); \theta), \tag{3.8}$$

where $\theta \in \Theta$ is a parameter vector. Various copulas are proposed in the statistical literature (Nelsen, 2006; Gudendorf and Segers, 2010) and major parametric copulas are introduced in section 3.A.3 in the Appendix. We employ a two-step maximum likelihood estimation to estimate a copula function for the joint outcome distribution. This separates the estimation of the marginals from that of the dependence parameter(s) and produces consistent estimates of the dependence parameter(s) (Trivedi and Zimmer, 2007). The estimation procedure is explained in section 3.A.4 in the Appendix.

Simulation from the estimated copula

We make pseudo-random draws with the conditional sampling method, using the estimated copula function. In essence, the conditional sampling method generates the random samples from the multivariate joint distribution using the following three steps. Let $C(v_1, v_2)$ be a bivariate copula. First, we draw v_1 from the standard uniform distribution. Second, we set $v_2 = \frac{\partial C(v_1,v_2)}{\partial v_1}$.[14] The pair (v_1, v_2) are uniformly distributed variables from the copula function

[14]Every copula has its derivative (Nelsen, 2006). For any v_2, the partial derivative $\frac{\partial C(v_1,v_2)}{\partial v_1}$ exists for almost all v_1. For such v_1 and v_2, the range of its derivative is $0 \leq \frac{\partial C(v_1,v_2)}{\partial v_1} \leq 1$. Similarly, the partial derivative $\frac{\partial C(v_1,v_2)}{\partial v_2}$ exists for almost all v_2.

$C(.)$ because the second step exploits the property of the derivative of copulas,

$$\frac{\partial C(v_1, v_2)}{\partial v_1} = \lim_{\Delta v_1 \to 0} \frac{C(v_1 + \Delta v_1, v_2) - C(v_1, v_2)}{\Delta v_1} \tag{3.9}$$

$$= Pr[V_2 \leq v_2 | V_1 \leq v_1]. \tag{3.10}$$

Third, we generate the random variables $H = \hat{F}_H^{-1}(v_1)$ and $Y = \hat{F}_Y^{-1}(v_2)$. The outcomes (H, Y) can be considered as a set of pseudo-random sampling from the joint distribution of (H, Y). We generate the set of (H_{B2}^N, Y_{B2}^N) of which size is as large as the sample size of the treated groups $(g = B, t = 2)$, which allows us to calculate the distributional statistics of the counterfactual marginal and joint distributions, i.e. $v(F_{H,B2}^N), v(F_{Y,B2}^N)$ and $v(F_{H,Y,B2}^N)$.

3.5.5 Decomposition of the treatment effect on health

Estimating the counterfactual joint distribution allows us to quantify how much of the treatment effect on the health distribution is contributed by the change in the distribution of household expenditure. We decompose the treatment effect on the health distribution into two effects: the expenditure effect and the behavioural effect. The expenditure effect is the part of the total effect attributable to the change in the expenditure distribution due to the treatment, and the behavioural effect is the remaining part which is not attributable to the expenditure effect. Hence, the behavioural effect captures the effect attributable to the changes in the association between health and household expenditure, which is induced by the behavioural changes due to the treatment. This decomposition exercise does not necessarily produce causal effects, but the decomposition would help us explore the pathways by which CCTs affect child nutrition.

We decompose the total effect into the expenditure effect and the behavioural effect by adding and subtracting counterfactual distributional statistics $v(F_{H,B2}^{IN})$, as follows:

$$\underbrace{v(F_{H,B2}^I) - v(F_{H,B2}^N)}_{Total\ effect} = \underbrace{\{v(F_{H,B2}^I) - v(F_{H,B2}^{IN})\}}_{Expenditure\ effect} + \underbrace{\{v(F_{H,B2}^{IN}) - v(F_{H,B2}^N)\}}_{Behavioural\ effect}. \tag{3.11}$$

The distribution $F_{H,B2}^{IN}$ is a counterfactual health distribution of group B in the post-treatment period, which is composed of the conditional distribution of H with the treatment and the marginal distribution of Y without the treatment. In other words, $F_{H,B2}^{IN}$ is the health distribution with the treatment in a situation where no one changed their expenditure patterns even in the

presence of the treatment. Formally, applying the law of iterated expectation, $F^{I}_{H,B2}$, $F^{N}_{H,B2}$, and $F^{IN}_{H,B2}$ are expressed by

$$F^{I}_{H,B2} = \int_{\mathcal{Y}} F^{I}_{H|Y,B2}(h)dF^{I}_{Y,B2}(y) \tag{3.12}$$

$$F^{N}_{H,B2} = \int_{\mathcal{Y}} F^{N}_{H|Y,B2}(h)dF^{N}_{Y,B2}(y) \tag{3.13}$$

$$F^{IN}_{H,B2} = \int_{\mathcal{Y}} F^{I}_{H|Y,B2}(h)dF^{N}_{Y,B2}(y). \tag{3.14}$$

The expenditure effect in equation (3.11) captures the effect on the health distribution due to the difference in the marginal expenditure distribution, i.e. the difference between $F^{I}_{Y,B2}$ and $F^{N}_{Y,B2}$. The behavioural effect, which reflects the difference in the conditional distribution of health given expenditure, i.e. the difference between $F^{I}_{H|Y,B2}$ and $F^{N}_{H|Y,B2}$, measures the effects on health that are attributable to the change in the association between H and Y due to the treatment.

To estimate the expenditure and behavioural effects, we need to estimate the counterfactual health distribution, $F^{IN}_{H,B2}$. This counterfactual health distribution can be estimated by employing the re-weighting decomposition approach (DiNardo, Fortin, and Lemieux, 1996) (hereafter denoted as "DFL weighting"). In essence, we replace the marginal distribution of Y^{I}_{B2} with the marginal distribution of Y^{N}_{B2}, using the DFL weighting factor to produce the counterfactual distribution as follows:

$$\begin{aligned} F^{IN}_{H,B2} &= \int_{\mathcal{Y}} F^{I}_{H|Y,B2}(h)dF^{N}_{Y,B2}(y) \\ &= \int_{\mathcal{Y}} F^{I}_{H|Y,B2}(h)\Psi(Y)dF^{I}_{Y,B2}(y), \end{aligned} \tag{3.15}$$

where $\Psi(Y) = \frac{dF^{N}_{Y,B2}}{dF^{I}_{Y,B2}}$ in equation (3.15) is the DFL weighting factor. Intuitively, the weighting factor re-weights the entire treated observations so that they would have the health outcome distribution that would be realised under the marginal expenditure distribution without the treatment.

For the implementation of the decomposition, we need to estimate the DFL weighting factor,

which can be done from the observed and simulated data:

$$\begin{aligned}
\Psi(Y) &= \frac{dF^{N}_{Y,B2}(y)}{dF^{I}_{Y,B2}(y)} = \frac{f(Y|g=B,t=2,N)}{f(Y|g=B,t=2,I)} = \frac{Pr(Y|g=B,t=2,N)}{Pr(Y|g=B,t=2,I)} \\
&= \frac{Pr(Y,g=B,t=2,N)/Pr(g=B,t=2,N)}{Pr(Y,g=B,t=2,I)/Pr(g=B,t=2,I)} \\
&= \frac{Pr(N|g=B,t=2,Y)Pr(g=B,t=2,Y)/Pr(g=B,t=2,N)}{Pr(I|g=B,t=2,Y)Pr(g=B,t=2,Y)/Pr(g=B,t=2,I)} \\
&= \frac{Pr(N|g=B,t=2,Y)Pr(g=B,t=2,I)}{Pr(I|g=B,t=2,Y)Pr(g=B,t=2,N)} \\
&= \frac{Pr(N|g=B,t=2,Y)Pr(I|g=B,t=2)Pr(g=B,t=2)}{Pr(I|g=B,t=2,Y)Pr(N|g=B,t=2)Pr(g=B,t=2)} \\
&= \frac{\{1-Pr(I|g=B,t=2,Y)\}Pr(I|g=B,t=2)}{Pr(I|g=B,t=2,Y)Pr(N|g=B,t=2)},
\end{aligned} \tag{3.16}$$

where $Pr(I|g=B,t=2,Y)$ is the probability of getting the treatment in group B in period 2, given the covariate Y, and it is estimated by the logit model. The ratio $\frac{Pr(I|g=B,t=2)}{Pr(N|g=B,t=2)}$ becomes one because the identical numbers of untreated samples and treated samples are simulated with the conditional sampling method (see section 3.5.4).

The DFL weighting is based on the assumption that the support of Y^{N}_{B2} is in support of Y^{I}_{B2}, i.e. $\mathbb{Y}^{N}_{B2} \in \mathbb{Y}^{I}_{B2}$. This common support assumption requires the overlap in the expenditure distributions in group B, in the sense that there is no such value of Y that is observed only in the absence of the treatment. Although the validity of this common support assumption cannot be tested from the observed distributions, given that the PKH is a cash transfer programme, it is unlikely that the upper support of Y^{N}_{B2} exceeds that of Y^{I}_{B2}. The failure of the common support assumption prevents us from estimating the counterfactual health distribution with the re-weighting factor, because such a situation makes the propensity score zero or one. Intuitively, if the propensity score is close to zero or one, observations will get close to zero or an extremely high weight in estimates of the contractual distribution with the DFL re-weighting approach, potentially leading to an imprecise estimation. We deal with the potential lack of common support by trimming the samples outside the top 0.1 per cent and bottom 0.1 per cent of the propensity score (Crump et al., 2009).

3.5.6 Entropy balance weighting

Despite randomisation, pre-treatment differences across groups are expected to be present, and controlling for the pre-treatment characteristics can increase statistical precision and help the

identification assumptions to be satisfied (Abadie and Imbens, 2011; Abadie, 2005). We balance the distributions of these pre-treatment variables with entropy balance weighting proposed by Hainmueller (2012), which non-parametrically assigns weights such that the covariate distributions in the weighted data satisfy a set of moment conditions set by a researcher. Specifically, weights are assigned to the control group so that the moments of the weighted covariate distributions in the control group match those in the treatment group. Balancing the higher moments of the covariate distributions can further enhance the credibility of the treatment effect estimation. The algorithm of the entropy balance weighting is provided in section 3.A.5 in the Appendix.

3.6 Results

3.6.1 Sample selection

The original sample size for children aged below 36 months who construct fully balanced panel data is 7,090 observations.[15] Then, 56 observations are dropped because they do not have complete information about pre-treatment individual-, household- and village-level characteristics in either the baseline or follow-up surveys. The Kolmogorov-Smirnov test does not indicate systematic differences in the distribution of HAZ score, WAZ score and household expenditure between the dropped samples and the remaining samples, implying that dropping the observations with missing information about pre-treatment characteristics is not likely to produce severe selection bias. Following the previous studies in Mexico (Behrman and Hoddinott, 2005), we implement an analysis across three age groups in the baseline: (1) 0-12 months; (2) 13-24 months; and (3) 25-36 months. Since the constructed data is fully balanced panel data, the structures of each age group are consistent across periods, which helps identification assumptions to be satisfied. The sample sizes of each age group are 3,020, 2,738 and 1,276 respectively.[16]

3.6.2 Entropy balance weighting

First, we balance the distribution of pre-treatment individual-, household- and village-level characteristics for each age group by the entropy balance weighting. Specifically, as individual-

[15] From the initial 11,938 observations, 4,848 observations are dropped because they do not have complete nutritional status and/or household expenditure information in either the baseline or follow-up surveys. However, the Kolmogorov-Smirnov test fails to reject the equality in the distribution of the household- and village-level characteristics between the dropped samples and the included samples, implying that the missing information is random.

[16] As the height and weight were measured among children aged under five, children aged 25-36 months in the baseline have fewer samples in the follow-up survey, compared with children aged 0-12 months and 13-24 months.

and household-level characteristics, we balance the distribution of sex, residential location (urban/rural) and family size. As village-level characteristics, we balance the distribution of population size (divided by 1,000), ratio of farmers, Islamic population ratio, and the existence of community health centres (*puskesmas*), community health facilities, birth attendants, private clinics, government hospitals, private hospitals and drug stores. Their distributions are balanced up to their second moments. The covariate distributions before and after the balancing are provided in Table 3.3.

3.6.3 Copula selection of the joint distribution of health and expenditure of the treatment group

Copula selection results are shown in Table 3.4, which presents the maximised log-likelihood values, Akaike information criteria (AIC) and Bayesian information criteria (BIC) of the maximum-likelihood estimations. We observe the failure of convergence for some copulas, which could be an indication of inconsistency between the data properties and the copula restriction on the dependence parameter (Trivedi and Zimmer, 2007). For the joint distribution of the WAZ score and expenditure, the Gaussian copula, the Student-t copula and the FGM copula show the highest likelihoods and the lowest AIC and BIC for children aged 0-12 months, children aged 13-24 months and children 25-36 months respectively. In the estimations of the joint distribution of HAZ score and expenditure, the Student t copula shows the highest likelihoods and the lowest AIC and BIC among children aged 0-12 months. The Placket copula shows the highest likelihoods and the lowest AIC and BIC for children aged 13-24 months. For children aged 25-36 months, the FGM copula shows the highest likelihoods and the lowest AIC and BIC. These selected copulas are used to simulate the counterfactual distributions of health and expenditure.

3.6.4 Impacts on the marginal distributions of WAZ score, HAZ score and expenditure

Figure 3.2 shows the estimated cumulative distribution functions of WAZ score, HAZ score and the household expenditure of the control group ($g = A$) and the treatment group ($g = B$) in the post-treatment period ($t = 2$). In addition, Figure 3.2 illustrates the estimated counterfactual cumulative distribution functions of the treatment group if they had not been treated. The horizontal difference between the distribution of the treatment group and the counterfactual dis-

Table 3.3: Entropy balance weighting results

Age 0-12 months

Pre-balancing	Treatment group			Control group			Post-balancing	Treatment group			Control group		
	Mean	Variance	Skewness	Mean	Variance	Skewness		Mean	Variance	Skewness	Mean	Variance	Skewness
Male	0.499	0.250	0.005	0.505	0.250	−0.021	Male	0.499	0.250	0.005	0.499	0.250	0.005
Urban	0.490	0.250	0.042	0.479	0.250	0.085	Urban	0.490	0.250	0.042	0.489	0.250	0.042
Family size	6.386	4.671	1.090	6.094	3.718	1.115	Family size	6.386	4.671	1.090	6.386	4.671	1.153
Population	4.446	22.945	6.045	4.269	17.864	6.236	Population	4.446	22.945	6.045	4.446	22.942	6.692
Farmer ratio	69.341	718.165	−1.056	69.307	772.110	−1.103	Farmer ratio	69.341	718.165	−1.056	69.332	718.074	−1.105
Islam ratio	79.080	1428.361	−1.480	78.003	1489.190	−1.407	Islam ratio	79.080	1428.361	−1.480	79.069	1428.180	−1.488
Puskesmas	0.168	0.140	1.772	0.167	0.139	1.787	Puskesmas	0.168	0.140	1.772	0.168	0.140	1.772
Community health facility	0.356	0.230	0.600	0.342	0.225	0.666	Community health facility	0.356	0.230	0.600	0.356	0.229	0.600
Birth attendant	0.849	0.129	−1.945	0.786	0.168	−1.396	Birth attendant	0.849	0.129	−1.945	0.848	0.129	−1.943
Private clinic	0.213	0.168	1.403	0.215	0.169	1.386	Private clinic	0.213	0.168	1.403	0.213	0.168	1.403
Governmental hospital	0.016	0.015	7.801	0.014	0.014	8.213	Governmental hospital	0.016	0.015	7.801	0.016	0.015	7.799
Private hospital	0.033	0.032	5.261	0.023	0.022	6.420	Private hospital	0.033	0.032	5.261	0.033	0.032	5.260
Drug store	0.102	0.092	2.633	0.106	0.095	2.552	Drug store	0.102	0.092	2.633	0.102	0.092	2.632

Age 13-24 months

Pre-balancing	Treatment group			Control group			Post-balancing	Treatment group			Control group		
	Mean	Variance	Skewness	Mean	Variance	Skewness		Mean	Variance	Skewness	Mean	Variance	Skewness
Male	0.512	0.250	−0.046	0.487	0.250	0.051	Male	0.512	0.250	−0.046	0.511	0.250	−0.046
Urban	0.493	0.250	0.029	0.504	0.250	−0.016	Urban	0.493	0.250	0.029	0.493	0.250	0.029
Family size	6.247	4.939	1.392	6.039	4.557	1.398	Family size	6.247	4.939	1.392	6.246	4.938	1.317
Population	4.330	21.710	6.081	4.551	23.370	6.372	Population	4.330	21.710	6.081	4.329	21.707	6.994
Farmer ratio	67.117	817.539	−1.007	66.859	889.911	−0.929	Farmer ratio	67.117	817.539	−1.007	67.107	817.419	−0.941
Islam ratio	74.913	1629.793	−1.174	76.657	1545.496	−1.312	Islam ratio	74.913	1629.793	−1.174	74.902	1629.550	−1.193
Puskesmas	0.140	0.121	2.073	0.161	0.135	1.842	Puskesmas	0.140	0.121	2.073	0.140	0.121	2.073
Community health facility	0.353	0.229	0.617	0.338	0.224	0.686	Community health facility	0.353	0.229	0.617	0.353	0.228	0.617
Birth attendant	0.838	0.136	−1.836	0.799	0.161	−1.489	Birth attendant	0.838	0.136	−1.836	0.838	0.136	−1.835
Private clinic	0.228	0.176	1.294	0.215	0.169	1.387	Private clinic	0.228	0.176	1.294	0.228	0.176	1.294
Governmental hospital	0.017	0.017	7.395	0.015	0.014	8.076	Governmental hospital	0.017	0.017	7.395	0.017	0.017	7.394
Private hospital	0.033	0.032	5.208	0.035	0.034	5.085	Private hospital	0.033	0.032	5.208	0.033	0.032	5.207
Drug store	0.129	0.112	2.219	0.113	0.101	2.439	Drug store	0.129	0.112	2.219	0.129	0.112	2.218

Age 25-36 months

Pre-balancing	Treatment group			Control group			Post-balancing	Treatment group			Control group		
	Mean	Variance	Skewness	Mean	Variance	Skewness		Mean	Variance	Skewness	Mean	Variance	Skewness
Male	0.498	0.251	0.006	0.528	0.249	−0.112	Male	0.498	0.251	0.006	0.498	0.250	0.007
Urban	0.508	0.251	−0.031	0.472	0.249	0.112	Urban	0.508	0.251	−0.031	0.507	0.250	−0.030
Family size	6.235	5.371	0.894	5.941	4.397	1.053	Family size	6.235	5.371	0.894	6.233	5.369	1.017
Population	4.264	11.995	1.613	4.391	28.073	6.977	Population	4.264	11.995	1.613	4.264	12.043	1.973
Farmer ratio	66.018	895.619	−0.900	66.893	888.611	−0.953	Farmer ratio	66.018	895.619	−0.900	65.992	895.477	−0.892
Islam ratio	71.006	1772.190	−0.939	68.648	1864.203	−0.830	Islam ratio	71.006	1772.190	−0.939	70.982	1771.488	−0.955
Puskesmas	0.165	0.138	1.804	0.160	0.135	1.853	Puskesmas	0.165	0.138	1.804	0.165	0.138	1.803
Community health facility	0.404	0.241	0.393	0.352	0.228	0.620	Community health facility	0.404	0.241	0.393	0.404	0.241	0.393
Birth attendant	0.798	0.162	−1.486	0.756	0.185	−1.189	Birth attendant	0.798	0.162	−1.486	0.798	0.161	−1.483
Private clinic	0.214	0.169	1.394	0.248	0.187	1.169	Private clinic	0.214	0.169	1.394	0.214	0.168	1.393
Governmental hospital	0.006	0.006	12.669	0.014	0.014	8.367	Governmental hospital	0.006	0.006	12.669	0.006	0.006	12.665
Private hospital	0.037	0.035	4.928	0.037	0.036	4.915	Private hospital	0.037	0.035	4.928	0.037	0.035	4.926
Drug store	0.107	0.096	2.542	0.118	0.104	2.368	Drug store	0.107	0.096	2.542	0.107	0.096	2.539

Table 3.4: Copula estimation

Copula	WAZ score and expenditure			HAZ score and expenditure		
	Log likelihood	AIC	BIC	Log likelihood	AIC	BIC
Age 0-12 months						
Clayton	NA	NA	NA	NA	NA	NA
Frank	0.828	0.344	−1.656	0.004	1.992	−0.008
Gumbel	0.127	1.747	−0.253	0.086	1.827	−0.173
AMH	0.854	0.291	−1.709	0.004	1.992	−0.008
Joe	0.000	2.000	0.000	0.130	1.740	−0.260
Gaussian	1.025	−0.051	−2.051	0.013	1.974	−0.026
Student-t	0.911	0.178	−1.822	0.503	0.994	−1.006
FGM	0.872	0.256	−1.744	0.004	1.992	−0.008
Plackett	0.807	0.387	−1.613	0.004	1.992	−0.008
Galambos	0.338	1.324	−0.676	0.309	1.382	−0.618
Husler Reiss	0.383	1.234	−0.766	0.328	1.343	−0.657
Tawn	0.018	1.963	−0.037	NA	NA	NA
Age 13-24 months						
Clayton	3.009	−4.018	−6.018	NA	NA	NA
Frank	1.257	−0.513	−2.513	2.326	−2.651	−4.651
Gumbel	1.549	−1.098	−3.098	0.764	0.472	−1.528
AMH	1.338	−0.677	−2.677	2.315	−2.630	−4.630
Joe	0.972	0.057	−1.943	0.142	1.716	−0.284
Gaussian	1.507	−1.015	−3.015	2.242	−2.484	−4.484
Student-t	3.899	−5.799	−7.799	2.091	−2.183	−4.183
FGM	1.165	−0.330	−2.330	2.312	−2.625	−4.625
Plackett	1.306	−0.613	−2.613	2.331	−2.663	−4.663
Galambos	0.934	0.132	−1.868	0.666	0.668	−1.332
Husler Reiss	0.802	0.396	−1.604	0.659	0.682	−1.318
Tawn	1.926	−1.852	−3.852	0.830	0.341	−1.659
Age 25-36 months						
Clayton	0.540	0.921	−1.079	NA	NA	NA
Frank	0.764	0.473	−1.527	0.109	1.782	−0.218
Gumbel	0.000	2.000	0.000	0.000	2.000	0.000
AMH	0.742	0.517	−1.483	0.108	1.783	−0.217
Joe	0.000	2.000	0.000	0.000	2.000	0.000
Gaussian	0.760	0.481	−1.519	0.090	1.821	−0.179
Student-t	0.708	0.584	−1.416	0.050	1.900	−0.100
FGM	0.819	0.362	−1.638	0.113	1.775	−0.225
Plackett	0.739	0.523	−1.477	0.108	1.785	−0.215
Galambos	NA	NA	NA	0.012	1.975	−0.025
Husler Reiss	NA	NA	NA	0.019	1.963	−0.037
Tawn	NA	NA	NA	NA	NA	NA

Note: NA means that a likelihood function did not converge. AIC: Akaike information criteria. BIC: Bayesian information criteria.

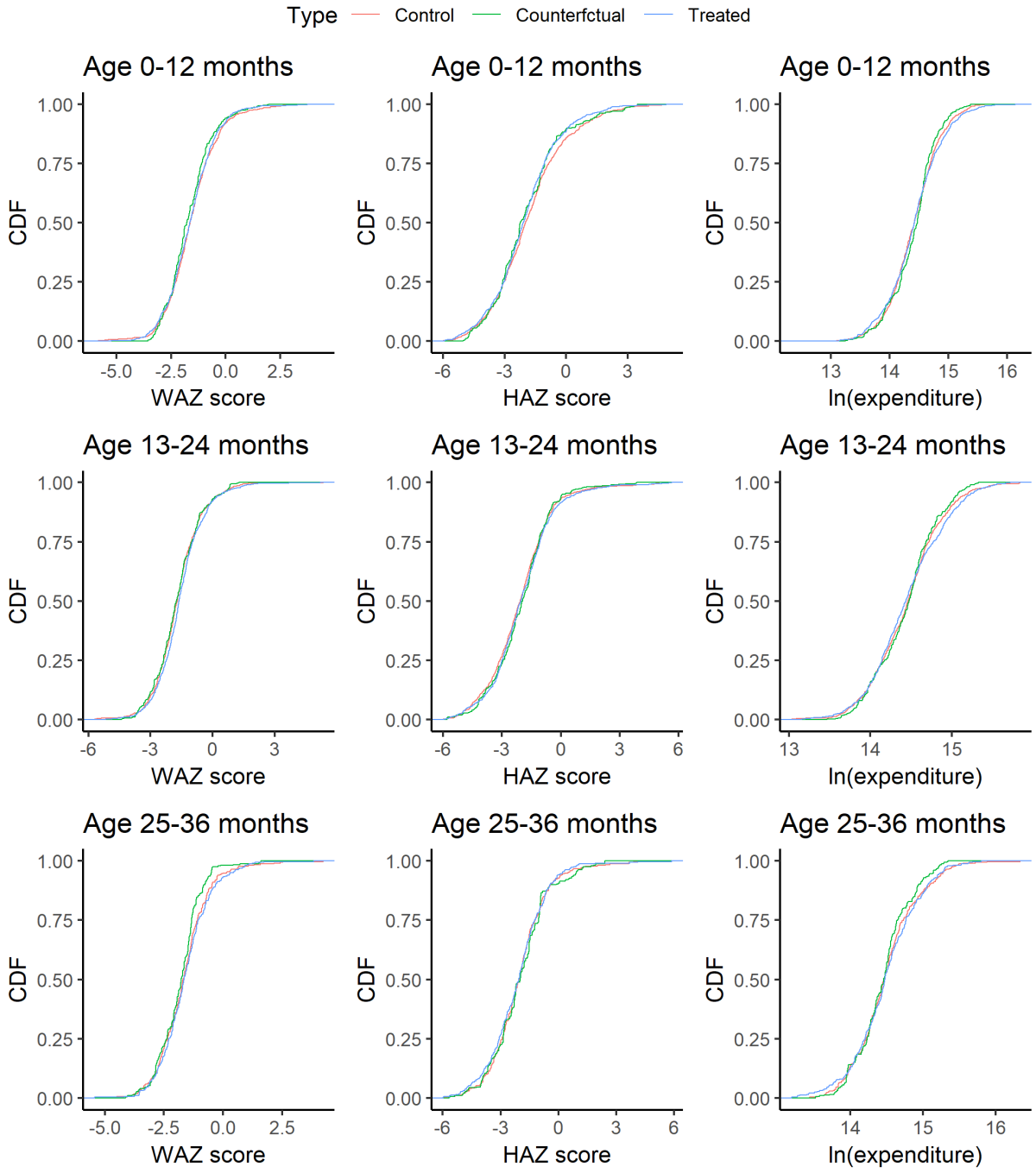
tribution corresponds to the quantile treatment effect. The non-parametric Kolmogorov-Smirnov test rejects the equality of the distributions ($p < 0.01$), but stochastic dominance of the distributions of the treatment group over the counterfactual distributions is not observed in all cases. Table 3.5 reports the impacts of the PKH programme on the mean and deciles of each outcome distribution. Associated standard errors are obtained by bootstrap with 500 repetitions. First, on the distribution of the WAZ score, the impact of the PKH is mixed across the age groups. For children aged 0-12 months, increases in the WAZ score are observed in the middle and higher percentiles of the distribution, but we did not find significant increases. For children aged 13-24 months, the impact on the WAZ score distribution shows positive signs, but it is not statistically significant except on the 40th percentile ($p < 0.10$). For children aged 25-36 months, we find a significant large positive impact on the mean ($p < 0.10$), the 80th ($p < 0.01$) and the 90th ($p < 0.05$) percentiles of the distribution.

The result for children aged 25-36 months shows that the significant improvement is not observed uniformly across the distribution. At the lower percentile of the distribution, the significant increase is not found, thereby suggesting that the PKH did not work effectively among those who most need the intervention. The null of the improvement in the lower percentile of the distribution would indicate the importance of re-designing the CCT programme with appropriate conditionality so that the benefits would effectively reach the neediest.

Another interesting finding is that children aged 0-12 months and children aged 13-24 months show little improvement in health. The most plausible explanation for this is that these children are too young to get the benefit of the programme. In Indonesia, breastfeeding was almost universal with over 96 per cent of children in the country being breastfed (Beatty et al., 2017; World Health Organization, 2010). Continued breastfeeding at least until age two is recommended in Indonesia and the exclusive breastfeeding rate is higher among poorer mothers (Beatty et al., 2017). Children aged 0-24 months who grew mainly through breastfeeding may have enjoyed little benefit from the PKH.

Next, on the distribution of the HAZ score, the treatment effect on the mean and deciles of the distribution is insignificant among all three age groups. This could be attributable to the relatively short evaluation period, as argued by Cahyadi et al. (2018). In contrast to the WAZ

Figure 3.2: HAZ score, WAZ score and expenditure distributions in the post-treatment period



score, the improvement in the HAZ score requires exposure to the programme over a sustained period of time (UNICEF, 2013; Strauss and Thomas, 1998; Waterlow et al., 1977). The distributional impacts in the medium and long term with the follow-up surveys would be worth exploring and this would complement the findings made by Cahyadi et al. (2018).[17] However, note that treatment exposure duration in this study, i.e. for 26-30 months, is not necessarily shorter than other experimental studies in Latin America (see Table 3.2).

Lastly, on the distribution of household expenditure, the significant positive impact is observed on the 90th percentile of the distribution among children aged below 12 months ($p < 0.10$) and on the 80th ($p < 0.05$) and 90th ($p < 0.10$) percentiles among children aged 13-24 months. Among children aged 25-36 months, we do not observe the significant increase in household expenditure.

Table 3.5 also shows the treatment impact on the prevalence of malnutrition. A decline in the prevalence of being underweight ($WAZ < -2$) is observed across all three age groups, but it is statistically insignificant. For the prevalence of being stunted ($HAZ < -2$), we observe an increase among children aged 13-24 months and children aged 25-36 months, and a decrease among children aged below 12 months, although these changes are not statistically significant.

3.6.5 Decomposition of the treatment effect on health

Then, we decompose the treatment effect on the distribution of nutritional status in order to estimate how much of the treatment effect is attributable to the change in the household expenditure due to the PKH (equation (3.11)). Table 3.6 and Table 3.7 report the expenditure and behavioural effects of the PKH on the marginal distributions of WAZ and HAZ scores. We find that much larger proportions of the total effects are contributed by the behavioural effect, which implies that the PKH impacts the marginal health distribution by directly affecting health rather than influencing it through the change in household expenditure.

One likely explanation for the behavioural change is the knowledge transfer from healthcare providers to beneficiary mothers through the conditionality. Previous studies in Indonesia showed that the PKH increased healthcare use among mothers and children; for example, prenatal care, institutional delivery, postnatal care, immunisations of children and growth-monitoring check-ups

[17] As of August 2019, the six-year follow-up survey data was not yet publicly available. Once the data becomes available to the public, estimating the long-term effects would complement the findings of this study.

Table 3.5: Treatment effect on the WAZ score, HAZ score and expenditure

	WAZ score		**HAZ score**		**Expenditure**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs
Age 0-12 months						
Mean	0.063	0.111	-0.072	0.188	0.011	0.035
10th percentile	-0.092	0.135	-0.212	0.219	-0.074	0.065
20th percentile	-0.030	0.113	-0.078	0.160	-0.046	0.045
30th percentile	0.067	0.107	0.091	0.190	-0.032	0.045
40th percentile	0.096	0.099	0.041	0.166	-0.030	0.042
50th percentile	0.136	0.106	0.153	0.182	-0.049	0.037
60th percentile	0.063	0.101	0.018	0.207	-0.030	0.042
70th percentile	0.104	0.140	-0.015	0.244	0.037	0.051
80th percentile	0.160	0.199	0.048	0.310	0.083	0.058
90th percentile	0.072	0.219	-0.066	0.527	0.124*	0.065
Undernutrition	-0.029	0.045	-0.021	0.048		
Severe undernutrition	0.009	0.024	-0.017	0.041		
Age 13-24 months						
Mean	0.144	0.105	0.047	0.164	0.014	0.038
10th percentile	0.200	0.138	0.089	0.305	-0.031	0.058
20th percentile	0.129	0.150	0.039	0.163	-0.003	0.051
30th percentile	0.188	0.119	-0.087	0.154	-0.055	0.058
40th percentile	0.180*	0.107	-0.059	0.159	-0.054	0.050
50th percentile	0.131	0.098	-0.073	0.175	-0.039	0.036
60th percentile	0.119	0.104	-0.039	0.153	-0.003	0.043
70th percentile	0.037	0.149	0.065	0.168	0.052	0.056
80th percentile	0.071	0.161	0.070	0.191	0.123**	0.056
90th percentile	0.084	0.236	0.277	0.335	0.125*	0.068
Undernutrition	-0.071	0.044	0.020	0.051		
Severe undernutrition	-0.036	0.024	0.012	0.035		
Age 25-36 months						
Mean	0.232*	0.128	-0.137	0.222	0.033	0.054
10th percentile	0.048	0.216	-0.140	0.327	-0.004	0.092
20th percentile	0.152	0.161	-0.214	0.231	-0.036	0.092
30th percentile	0.074	0.169	-0.050	0.209	0.011	0.061
40th percentile	0.119	0.135	-0.108	0.200	0.041	0.066
50th percentile	0.127	0.132	-0.073	0.176	0.013	0.061
60th percentile	0.121	0.134	-0.171	0.172	0.047	0.064
70th percentile	0.222	0.137	-0.095	0.213	0.103	0.072
80th percentile	0.466***	0.193	0.010	0.229	0.136	0.087
90th percentile	0.483**	0.236	-0.121	0.774	0.131	0.094
Undernutrition	-0.034	0.055	0.028	0.056		
Severe undernutrition	0.000	0.032	0.043	0.055		

Note: Standard errors (SEs) are obtained by bootstrap with 500 repetitions.
Sample sizes of each age group are 3,020, 2,738 and 1,276 respectively.
Pre-treatment characteristics are balanced with entropy balance weighting.
Undernutrition is the condition of z-score being below -2.
Severe undernutrition is the condition of z-score being below -3.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

all saw significant increases (Alatas, 2011; Kusuma et al., 2017). The improvement of health-related knowledge as a result of more frequent health clinic visits and interactions with health professionals due to conditionality contributes to the improved WAZ score. Furthermore, previous studies also showed that the PKH changed the composition of household expenditure. Beneficiary households increased the proportion of health expenditure to total expenditure by two percentage points by lowering the proportions of other expenditure (Alatas, 2011). The increased healthcare utilisation and the re-orientation towards higher healthcare use suggest that the PKH enhanced the "health literacy" among beneficiaries.

3.6.6 Treatment effect on the dependence measurements

Next, we look at the treatment effect on the dependence between nutritional status and household expenditure. The most well-known dependence concept is the Pearson correlation coefficient, which measures linear dependence.[18] We also use rank correlations as alternative measurements of dependence; in this paper we use Spearman's rho and Kendall's tau.[19] Both rank correlation measurements do not depend on the functional forms of the marginal distributions (Trivedi and Zimmer, 2007). The degree of dependence, measured by correlation coefficients, allows us to quantify the change in dependence and the change in copula functions made by the treatment. The difference in the dependence measurements in the presence of the treatment and those in the absence of the treatment gives the effect of the PKH on the dependence measurements.

Table 3.8 reports the effects on the dependence measurements between expenditure and the WAZ score and those between expenditure and the HAZ score. Without the treatment, the dependence measurement indicates either statistically null dependence or positive dependences. A positive dependence implies concordance between expenditure and HAZ/WAZ score; that is, large values of household expenditure are associated with the large value of WAZ/HAZ score. We observe that the PKH significantly increases the degrees of dependence, measured by the three measurements, between expenditure and WAZ score among children aged 25-36 months ($p < 0.05$). On the dependence between household expenditure and HAZ score, the PKH does not show significant effects for all three age groups.

[18] Formerly known as the Pearson product-moment correlation coefficient.

[19] They are defined by $\rho_s(H,Y) = \rho(F_H(h), F_Y(y))$ and $\rho_\tau(H,Y) = Pr\{(h_1 - h_2)(y_1 - y_2) > 0\} - Pr\{(h_1 - h_2)(y_1 - y_2) < 0\}$, where $\rho(.,.)$ is a linear correlation coefficient and (h_1, y_1) and (h_2, y_2) are two independent pairs of random variables from $F_{H,Y}$.

Table 3.6: Decomposition of the treatment effect on the WAZ score

WAZ score	**Total effect**		**Expenditure effect**		**Behavioural effect**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs
Age 0-12 months						
Mean	0.063	0.111	0.000	0.006	0.063	0.111
10th percentile	-0.092	0.135	0.000	0.008	-0.092	0.136
20th percentile	-0.030	0.113	0.000	0.013	-0.030	0.114
30th percentile	0.067	0.107	0.000	0.010	0.067	0.109
40th percentile	0.096	0.099	0.000	0.007	0.096	0.100
50th percentile	0.136	0.106	0.000	0.008	0.136	0.106
60th percentile	0.063	0.101	0.000	0.010	0.063	0.100
70th percentile	0.104	0.140	-0.005	0.010	0.109	0.141
80th percentile	0.160	0.199	-0.010	0.010	0.170	0.202
90th percentile	0.072	0.219	0.000	0.014	0.072	0.219
Age 13-24 months						
Mean	0.144	0.105	0.000	0.008	0.144	0.105
10th percentile	0.200	0.138	0.000	0.018	0.200	0.138
20th percentile	0.129	0.150	0.000	0.021	0.129	0.150
30th percentile	0.188	0.119	0.000	0.012	0.188	0.121
40th percentile	0.180*	0.107	0.000	0.008	0.180*	0.108
50th percentile	0.131	0.098	0.000	0.007	0.131	0.098
60th percentile	0.119	0.104	0.000	0.010	0.120	0.104
70th percentile	0.037	0.149	0.000	0.009	0.037	0.150
80th percentile	0.071	0.161	0.000	0.015	0.071	0.161
90th percentile	0.084	0.236	-0.010	0.015	0.094	0.235
Age 25-36 months						
Mean	0.232*	0.128	0.011	0.030	0.222*	0.129
10th percentile	0.048	0.216	0.000	0.037	0.048	0.220
20th percentile	0.152	0.161	0.004	0.044	0.148	0.160
30th percentile	0.074	0.169	0.001	0.036	0.072	0.163
40th percentile	0.119	0.135	0.015	0.029	0.104	0.134
50th percentile	0.127	0.132	0.020	0.028	0.107	0.127
60th percentile	0.121	0.134	0.010	0.030	0.111	0.135
70th percentile	0.222	0.137	0.015	0.031	0.207	0.136
80th percentile	0.466***	0.193	0.001	0.044	0.465***	0.195
90th percentile	0.483**	0.236	-0.002	0.062	0.484**	0.233

Note: Standard errors (SEs) are obtained by bootstrap with 500 repetitions.
Sample sizes of each age group are 3,020, 2,738 and 1,276 respectively.
Pre-treatment characteristics are balanced with entropy balance weighting.

$$\underbrace{v(F^{I}_{H,B2}) - v(F^{N}_{H,B2})}_{Total\ effect} = \underbrace{\{v(F^{I}_{H,B2}) - v(F^{IN}_{H,B2})\}}_{Expenditure\ effect} + \underbrace{\{v(F^{IN}_{H,B2}) - v(F^{N}_{H,B2})\}}_{Behavioural\ effect}.$$

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 3.7: Decomposition of the treatment effect on the HAZ score

HAZ score	**Total effect**		**Expenditure effect**		**Behavioural effect**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs
Age 0-12 months						
Mean	-0.072	0.188	0.001	0.011	-0.072	0.190
10th percentile	-0.212	0.219	0.000	0.012	-0.212	0.221
20th percentile	-0.078	0.160	0.000	0.021	-0.078	0.160
30th percentile	0.091	0.190	0.000	0.010	0.091	0.192
40th percentile	0.041	0.166	0.000	0.012	0.041	0.169
50th percentile	0.153	0.182	0.000	0.011	0.153	0.183
60th percentile	0.018	0.207	0.000	0.010	0.018	0.207
70th percentile	-0.015	0.244	0.000	0.018	-0.015	0.246
80th percentile	0.048	0.310	-0.010	0.019	0.058	0.312
90th percentile	-0.066	0.527	-0.001	0.029	-0.065	0.530
Age 13-24 months						
Mean	0.047	0.164	0.001	0.010	0.046	0.164
10th percentile	0.089	0.305	0.000	0.008	0.089	0.305
20th percentile	0.039	0.163	0.000	0.013	0.039	0.162
30th percentile	-0.087	0.154	0.000	0.010	-0.086	0.153
40th percentile	-0.059	0.159	0.000	0.008	-0.059	0.160
50th percentile	-0.073	0.175	0.000	0.015	-0.073	0.176
60th percentile	-0.039	0.153	0.000	0.015	-0.039	0.156
70th percentile	0.065	0.168	0.000	0.012	0.065	0.170
80th percentile	0.070	0.191	0.000	0.009	0.070	0.192
90th percentile	0.277	0.335	-0.001	0.009	0.278	0.337
Age 25-36 months						
Mean	-0.137	0.222	0.013	0.033	-0.150	0.218
10th percentile	-0.140	0.327	-0.001	0.037	-0.139	0.319
20th percentile	-0.214	0.231	0.000	0.048	-0.213	0.226
30th percentile	-0.050	0.209	0.009	0.033	-0.060	0.204
40th percentile	-0.108	0.200	0.008	0.036	-0.116	0.201
50th percentile	-0.073	0.176	0.009	0.038	-0.082	0.174
60th percentile	-0.171	0.172	0.000	0.031	-0.171	0.170
70th percentile	-0.095	0.213	0.002	0.038	-0.097	0.214
80th percentile	0.010	0.229	0.016	0.050	-0.006	0.226
90th percentile	-0.121	0.774	-0.003	0.053	-0.118	0.773

Note: Standard errors (SEs) are obtained by bootstrap with 500 repetitions.
Sample sizes of each age group are 3,020, 2,738 and 1,276 respectively.
Pre-treatment characteristics are balanced with entropy balance weighting.

$$\underbrace{v(F^{I}_{H,B2}) - v(F^{N}_{H,B2})}_{Total\ effect} = \underbrace{\{v(F^{I}_{H,B2}) - v(F^{IN}_{H,B2})\}}_{Expenditure\ effect} + \underbrace{\{v(F^{IN}_{H,B2}) - v(F^{N}_{H,B2})\}}_{Behavioural\ effect}.$$

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 3.8: Treatment effect on the dependence measurements between expenditure and nutritional status

WAZ score	**With treatment**		**Without treatment**		**Treatment effect**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs
Age 0-12 months						
Pearson's correlation	-0.002	0.057	0.071	0.047	-0.073	0.075
Kendall's tau	-0.004	0.042	0.047	0.037	-0.051	0.057
Spearman's rho	-0.007	0.063	0.071	0.054	-0.077	0.085
Age 13-24 months						
Pearson's correlation	0.008	0.053	0.060	0.064	-0.052	0.094
Kendall's tau	0.022	0.034	0.043	0.039	-0.021	0.057
Spearman's rho	0.032	0.051	0.063	0.057	-0.031	0.086
Age 25-36 months						
Pearson's correlation	0.114	0.071	-0.100	0.069	0.214**	0.107
Kendall's tau	0.098**	0.045	-0.065	0.047	0.163**	0.074
Spearman's rho	0.146**	0.068	-0.098	0.071	0.243**	0.112
HAZ score	**With treatment**		**Without treatment**		**Treatment effect**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs
Age 0-12 months						
Pearson's correlation	0.017	0.064	0.011	0.061	0.006	0.087
Kendall's tau	0.007	0.037	0.012	0.042	-0.005	0.056
Spearman's rho	0.010	0.056	0.018	0.062	-0.008	0.082
Age 13-24 months						
Pearson's correlation	0.016	0.048	0.127***	0.050	-0.110	0.082
Kendall's tau	0.012	0.030	0.086***	0.036	-0.074	0.052
Spearman's rho	0.017	0.044	0.131***	0.053	-0.114	0.077
Age 25-36 months						
Pearson's correlation	0.101	0.065	-0.009	0.062	0.109	0.086
Kendall's tau	0.062	0.039	0.001	0.042	0.061	0.055
Spearman's rho	0.097*	0.058	0.002	0.064	0.096	0.082

Note: Standard errors (SEs) are obtained by bootstrap with 500 repetitions.
Sample sizes of each age group are 3,020, 2,738 and 1,276 respectively.
Pre-treatment characteristics are balanced with entropy balance weighting.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

The increase in the dependence among children aged 25-36 months is consistent with the increase in their WAZ score. Given that the change in the marginal distribution of expenditure is not statistically significant among this age group, the increase in the dependence between WAZ score and expenditure suggests that the PKH improves the WAZ score of children in relatively better-off households among the beneficiaries. Again, this implies the PKH does not necessarily reach the children who most need the intervention. Improving the nutritional outcomes of children in the poorest households is essential to achieve the ultimate goal of the CCT programme.

3.7 Discussion and conclusion

Numerous research papers have suggested that malnutrition during the early stages of life has irreversible adverse impacts on the development of both cognitive and non-cognitive skills (Sánchez, 2017; Walker et al., 2011; Grantham-McGregor et al., 2007), motor development, educational outcome (Glewwe et al., 2001) and future earnings (Smith, 2009; Johnson and Schoeni, 2011).[20] Despite the country's strong economic growth, the child malnutrition rate in Indonesia remains high. The PKH was introduced in 2007 with the intention of improving the welfare of poor households. Like other CCT programmes across the world, the PKH aims to change health-related behaviours through cash transfers and multiple conditionalities.

The PKH is expected to improve child nutritional status by cash transfer and through the associated conditionalities. As a lack of household purchasing power is one of the fundamental causes of child malnutrition, the cash transfer would contribute to improved nutritional status through increased quantity and quality of food consumption by overcoming budget constraints on appropriate nutritious diets. In addition, imposing conditions related to children's health and monitoring compliance help poor households increase their healthcare utilisation to the desired level. This study evaluates the treatment effects on the entire distribution of child health outcomes and household expenditure and then explores the pathway through which the PKH influences child nutritional status by estimating its impacts on the joint distribution of them.

The results show that the impact on the total household expenditure distribution is very modest and statistically indistinguishable from zero at most parts of the distribution. The statistically significant increase is observed only at the higher percentile of the expenditure distribution of the households with children aged 0-12 months and 13-24 months. The PKH programme has improved the WAZ score among children aged 25-36 months. Decomposition analysis shows that the improvement of the WAZ score is not explained by the change in the household expenditure distribution, which is not surprising given that the increase in the total expenditure is not observed among the households with children aged 25-36 months. The decomposition analysis suggests that their improvement in WAZ score is driven by behavioural changes in beneficiary households.

[20] For more evidence, see for example Strauss and Thomas (2007); Almond and Currie (2011); Case et al. (2005); Alderman et al. (2006); Blackwell et al. (2001); Portrait et al. (2017) and Victora et al. (2008).

Overall, this study shows that the PKH did not improve the nutritional condition of those at high risk of under-nutrition. One possible explanation is that the amount of cash transfer is not sufficiently large. Compared with the successful CCTs in Latin America, where the transferred cash is typically above 20 per cent of average beneficiary household expenditure, the cash transfer of the PKH is around 12 per cent of the total expenditure of beneficiary households (World Bank, 2012). Furthermore, in contrast to the successful CCTs in Latin America, the PKH does not have a nutrition-related conditionality such as participants taking a nutritional supplement (Maluccio and Flores, 2005; Skoufias, 2005). In Mexico, for example, mothers must visit a clinic at least once a month to pick up packets of supplements per eligible child if signs of malnutrition are detected by the clinic's personnel. Although the effectiveness of the nutrition supplement itself is not yet fully confirmed, the Indonesian government may wish to assess the option to introduce a nutrition-related conditionality.

Some additional complementary conditionalities related to feeding practice would potentially enhance the effectiveness of the PKH on child health outcomes. For example, while attending a nutritional education workshop or utilising a counselling service are included in CCT programmes in Colombia[21], Brazil, El Salvador, Mexico, Nicaragua and Peru (Bassett, 2008), the conditionality of the PKH does not include mothers participating in nutrition counselling. Providing educational opportunities in which beneficiaries frequently get advice about child diets from healthcare professionals could assist the nutritional improvement.

Adding the conditionality regarding hygiene and sanitation promotions would also be an option to enhance the effectiveness of the CCT programme (Hoddinott and Bassett, 2008). For example, significant improvements in child health due to the promotion of hand-washing using soap have been reported in India (Bhutta et al., 2008). Combining education counselling sessions on diets, hygiene and other topics relating to child health and growth with regular growth monitoring would also further help to promote child growth by maximising the potential effectiveness of the CCT (Hoddinott and Bassett, 2008).

In addition, this study provides evidence that the PKH strengthens the positive dependence between child nutritional status and household expenditure among children aged 25-36 months.

[21] In Colombia, attending workshops is encouraged but not mandatory.

In the existing literature, the impact of CCTs on the joint distribution has not been afforded any examination, but its impact should not be completely ignored in the process of economic policy evaluations because the rationale of the CCTs rests on social equity which requires that everyone, regardless of their socio-economic background, should have the equal opportunity to achieve sound human capital development (Fiszbein et al., 2009). We would like to emphasise that the CCTs should be designed with appropriate conditionalities to ensure that their benefits will effectively reach the neediest.

By way of conclusion, it is worth discussing a limitation and some potential extensions of this study. First, since the respondents are not representative of the sub-district populations as they were instead selected by means of purposive sampling, the implications of this study are not necessarily applicable to the entire population (Deaton and Cartwright, 2018). In other words, although we find that the PKH changes the association between nutritional status and expenditure among the eligible households, it is still uncertain whether it is strong enough to change the socio-economic gradient in health in the entire population, including the non-poor ineligible households.

Second, the effectiveness of each conditionality of the PKH is worth exploring. The PKH has several education- and health-related conditionalities (Table 3.1-B), and careful examination of the effectiveness of each one would contribute to a more thorough evaluation of the policy. However, the randomisation design of the data used in this study does not provide us with the opportunity to conduct a detailed investigation of what conditions mainly trigger the effect. The ideal experimental design to identify the marginal contributions of each conditionality would be a randomised controlled trial with multiple treatment groups, each of which complies with each conditionality of the programme and a control group receiving no transfers. Exploitation of structural modelling with multiple outcomes and a more detailed randomisation design would promote a better understanding of the detailed causal pathways.

3.A Appendix – Notes on methodology

3.A.1 Assumptions for the CIC

First, we assume the independence between treatment assignment and latent outcomes:

Assumption 1 *Random treatment assignment*
u^H and u^Y are independent of the treatment assignment.

This is a core identification assumption which allows identification of the treatment effect parameter. In this study, as eligibility for the PKH is randomly assigned, this assumption is usually satisfied. Next, the CIC imposes an assumption on the unobserved characteristics.

Assumption 2 *Strict monotonicity (Athey and Imbens, 2006)*
$m(u^H, t)$ and $n(u^Y, t)$ are strictly increasing in $u^H \in \mathbb{U}^H$ and $u^Y \in \mathbb{U}^Y$ for $t \in \{1, 2\}$.

The strict monotonicity assumption requires that the higher level of u corresponds to the strictly higher outcomes. Equations (3.1) and (3.2) with strict monotonicity imply that the outcomes of an individual i with $u_i^j = u^j, j \in \{H, Y\}$ will be the same in a given period of time, regardless of the group status. In other words, in the absence of the treatment, all differences between the two groups in a given time period are attributable to the differences in the distribution of u conditional on g.

In addition, the CIC makes an assumption about the distribution of $u^j, j \in \{H, Y\}$.

Assumption 3 *Time invariance within groups (Athey and Imbens, 2006)*
The distribution of u^j can systematically vary across the groups, but not over time within groups.

$$u^j \perp t|g,\ j \in \{H, Y\}. \tag{3.A.1}$$

Time invariance within groups is a core assumption for the CIC. Conceptually, the time invariance assumption in the CIC is a generalisation of the common trend assumption used in the standard difference-in-differences method. In essence, the time invariance within groups assumption requires that the population in a given group does not change over time. In our case, as we use fully balanced panel data, group composition is preserved over time. This assumption is required to extrapolate the distribution of u^j in the second period from its distribution in the first period. Conditional on the group, the marginal distribution of $u^j_{|g,t=1}$ is identical to the

marginal distribution of $u^j_{|g,t=2}$ for $j \in \{H, Y\}$. Time invariance of the distribution of u^j, however, does not require that a particular individual i has the same realisation u^j_i in each period. This assumption only requires that the distribution of u^j is the same across time given the group.

Lastly, the CIC makes an assumption on the support of u^j.[22]

Assumption 4 *Common support (Athey and Imbens, 2006)*
The support of u^j in the group B is in the support of u^j in the group A.

$$\mathbb{U}^j_{|g=B} \in \mathbb{U}^j_{|g=A},\ j \in \{H, Y\}. \tag{3.A.2}$$

The common support assumption implies the following four support conditions of the outcome:

$$\mathbb{H}^N_{B1} \in \mathbb{H}^N_{A1} \tag{3.A.3}$$

$$\mathbb{H}^N_{B2} \in \mathbb{H}^N_{A2} \tag{3.A.4}$$

$$\mathbb{Y}^N_{B1} \in \mathbb{Y}^N_{A1} \tag{3.A.5}$$

$$\mathbb{Y}^N_{B2} \in \mathbb{Y}^N_{A2}. \tag{3.A.6}$$

The support assumption ensures that the counterfactual distributions of H^N_{B2} and Y^N_{B2} are identified at all points in the support of H^N_{A2} and Y^N_{A2}.

3.A.2 Identifying the joint distribution with the extended CIC

The invariance of dependence assumption, combined with the assumptions for the CIC model, allows us to relate the joint cumulative distribution of (H^N_{gt}, Y^N_{gt}) to the joint cumulative distributions of (u^H_{gt}, u^Y_{gt}) as follows (Cañón Salazar, 2016):

$$\begin{aligned} F^N_{H,Y,gt}(h, y) &= Pr\{m(u^H_{gt}, t) \leq h, n(u^Y_{gt}, t) \leq y\} \\ &= Pr\{u^H_{gt} \leq m^{-1}(h; t), u^Y_{gt} \leq n^{-1}(y; t)\} \\ &= F_{U^H_{gt}, U^Y_{gt}}(m^{-1}(h; t), n^{-1}(y; t)), \end{aligned} \tag{3.A.7}$$

where $F_{U^H_{gt}, U^Y_{gt}}(.,.)$ is a joint distribution function of (u^H_{gt}, u^Y_{gt}).

[22] Technically, this assumption is not the vital assumption for estimating the counterfactual distribution. This assumption ensures that the counterfactual distribution is all identified in the support of Y^j_{A2}. Even without this assumption, the distribution of $Y^{N,j}_{B2}$ in the support of Y^j_{A2} is identified, although the distribution of $Y^{N,j}_{B2}$ outside the support of Y^j_{A2} is not identified. For details, see Corollary 3.1 of Athey and Imbens (2006).

3.A.3 Major parametric copulas

In the statistical literature, a long list of copulas has been proposed for modelling the dependence between the variables, but we focus only on the several examples that are frequently used empirically in econometrics. A particular group of copulas which has proven useful is the Archimedean family copulas, which are easily derived and are capable of capturing various types of dependence structures. The Archimedean family copulas take the general form $C(v_1, v_2) = \psi^{-1}(\psi(v_1) + \psi(v_2))$, where $\psi(.)$ is called a generator function and its functional form is unique to each Archimedean family copula. Product copula, Frank copula, Clayton copula, Gumbel copula, Joe copula and Ali-Mikhail-Haq (AMH) copula are major Archimedean family copulas.

First, the Product copula, which takes the simplest form, does not allow dependence between marginal distributions and consequently does not have a dependence parameter. Second, the Frank copula is capable of modelling both positive and negative dependence, and is suitable for data that exhibit weak tail dependence (Trivedi and Zimmer, 2007). The Clayton copula can better capture strong left tail dependence and relatively weak right tail dependence, but it is not suitable for negative dependence. As its dependence parameter goes to 0, the marginal distributions become independent. The Gumbel copula, similar to the Clayton copula, is not able to model negative dependence, but it can better capture strong right tail dependence and relatively weak left tail dependence. The Joe copula also allows only positive dependence and is appropriate for modelling the very strong right tail dependence and relatively weak left tail dependence between marginals. The AMH copula is suitable only for modelling the weak dependence.

The elliptical family copulas are also frequently employed in the empirical literature. The elliptical copulas are simply the copulas with elliptically contoured distributions. The Gaussian copula and the Student t copula are major copulas in the elliptical family. The elliptical family copulas are flexible in the sense that they allow for positive and negative dependence. They are suitable for the marginals with a symmetric dependence structure. A disadvantage of the elliptical family copulas is that they do not have a closed functional form. The Student t copula has two dependence parameters, (θ_1, θ_2). The first parameter governs the heaviness of the tails. As $\theta_1 \to \infty$, the Student t copula becomes the Gaussian copula with parameter θ_2.

In addition, we consider the Plackett copula and the Farlie-Gumbel-Morgenstern (FGM) copula. The Plackett copula can model both positive and negative dependences well. If the marginals have relatively strong tail dependences, the Plackett copula is better at modelling them than the Gaussian copula. The FGM copula is a perturbation of the product copula. Similar to the AMH copula, the FGM copula is only suitable for modelling the two marginals with modest dependence. If the dependence parameter θ goes to zero, the FGM copula becomes the product copula.

Finally, we consider the extreme-value copulas. A copula is called an extreme-value copula if there exists a copula function C_F such that

$$C_F(v_1^{\frac{1}{n}}, v_2^{\frac{1}{n}})^n \to C(v_1, v_2) \, as \, n \to \infty. \tag{3.A.8}$$

The common extreme-value copulas are the Galambos copula, Hüsler Reiss copula and Tawn copula.[23] The extreme-value copulas are used mainly in the field of finance for modelling the dependence structure between rare events, but they can also be used to model data with positive dependence. Compared with the class of Archimedean family copulas, one advantage is that they can better model the marginals with asymmetric dependence and strong tail dependence. For more details about the extreme-value copulas, see Gudendorf and Segers (2010). Table 3.A.1 lists the functions of copulas employed in this paper, their parameter ranges and the number of parameters.

3.A.4 Copula selection and parameter estimation

We employ maximum likelihood estimation to parametrically estimate a copula function for the joint outcome distribution. This separates the estimation of the marginals from that of the dependence parameter(s) and produces consistent estimates of the dependence parameter(s) (Trivedi and Zimmer, 2007). The first step non-parametrically estimates univariate marginal distributions of health and expenditure. The second step then estimates the dependence parameter(s), using the estimated marginal distributions from the first step. It is essential to select an appropriate copula for modelling a joint distribution. If dependence structures were known a priori, it would be easier to choose an appropriate copula, but such information is seldom available in advance.

[23]The Gumbel copula, which is also a family of Archimedean family copulas, also belongs to the extreme-value copulas.

Table 3.A.1: Copulas

Copula	$C(v_1, v_2)$	Parameter range	Number of parameters
Product	$v_1 v_2$	NA	0
Clayton	$(v_1^{-\theta} + v_2^{-\theta} - 1)^{-\frac{1}{\theta}}$	$(0, \infty)$	1
Frank	$-\frac{1}{\theta} ln(1 + \frac{exp(-\theta v_1) exp(-\theta v_2) - 1}{exp(-\theta) - 1})$	$(-\infty, \infty)$	1
Gumbel	$exp\{-[(-ln(v_1))^{\theta} + (-ln(v_2))^{\theta}]^{\frac{1}{\theta}}\}$	$[1, \infty]$	1
AMH	$\frac{v_1 v_2}{1 - \theta(1 - v_1)(1 - v_2)}$	$[-1, 1]$	1
Joe	$1 - [1 - (1 - (1 - v_1)^{\theta})(1 - (1 - v_2)^{\theta})]^{\frac{1}{\theta}}$	$[1, \infty]$	1
Gaussian	$\Phi_2(\Phi^{-1}(v_1), \Phi^{-1}(v_2); \theta)$	$[-1, 1]$	1
Student t	$\int_{-\infty}^{t_{\theta_1}^{-1}(v_1)} \int_{-\infty}^{t_{\theta_2}^{-1}(v_2)} \frac{1}{2\pi(1-\theta_2^2)^{1/2}} \{1 + \frac{(s^2 - 2\theta_2 st + t^2)^{-(\theta_1+2)/2}}{q(1-\theta_2^2)}\} dsdt$	$\theta_1 \in [-1, 1]$, $\theta_2 \in [0, \infty]$	2
FGM	$v_1 v_2 (1 + \theta(1 - v_1)(1 - v_2))$	$[-1, 1]$	1
Plackett	$\frac{1 + (\theta - 1)(v_1 + v_2) - \sqrt{[1 + (\theta - 1)(v_1 + v_2)]^2 - 4\theta(\theta - 1) v_1 v_2}}{2(\theta - 1)}$	$\theta > 0$	1
Galambos	$v_1 v_2 exp\{[(1 - v_1)^{-\theta} + (1 - v_2)^{-\theta}]^{-1/\theta}\}$	$(0, \infty)$	1
Hüsler Reiss	$exp[ln(v_1 v_2)\{\frac{ln(v_2)}{ln(v_1 v_2)} \Phi(V) + (1 - \frac{ln(v_2)}{ln(v_1 v_2)}) \Phi(V)\}]$	$(0, \infty)$	1
Tawn	$v_1 v_2 exp\{-\theta \frac{ln(v_1) ln(v_2)}{ln(v_1 v_2)}\}$	$[-1, 1]$	1

Note: $\Phi_2(.)$ is a bivariate normal distribution function with a correlation coefficient θ.
$\Phi(.)$ is a univariate standard normal distribution function.
$V = \frac{1}{\theta} + \frac{\theta}{2} ln(\frac{ln(v_2)/ln(v_1 v_2)}{1 - ln(v_2)/ln(v_1 v_2)})$.
AMH stands for the Ali-Mikhail-Haq copula.
FGM stands for the Farlie-Gumbel-Morgenstern copula.
q denotes the degreess of freedom.

The selection of the copula tends to be a posterior consideration, and so it is important to estimate several copulas and compare their performances. The dependence parameters are estimated by employing the maximum likelihood method from the marginal distributions with parametric assumptions of each copula function (Trivedi and Zimmer, 2007). An appropriate copula is the one which can best capture the dependence structures of the data. Maximised likelihood values, Akaike information criteria (AIC) and Bayesian information criteria (BIC) provide guidance on the selection.[24] The parameter estimator is given by

$$\hat{\theta} = \arg\max_{\theta \in \Theta} \sum_{i \in \{g=B, t=1\}}^{N} ln\, c(\hat{F}_{H,B1}(h_i), \hat{F}_{Y,B1}(y_i); \theta), \tag{3.A.9}$$

where $c(.)$ is the likelihood function of a specific copula.

3.A.5 Entropy balance weighting

The entropy balancing method has a few advantages over the propensity score inverse probability weighting (PS-IPW). First, the entropy balancing method can "perfectly" balance a set of moments of the covariate distributions between two groups, while the parametric PS-IPW methods often fail to jointly balance all the covariate distributions in practice, partly because of the misspecification of the propensity score. To improve the balance of some covariates, the PS-IPW method usually needs to sacrifice the balance of the other covariates (Iacus et al., 2012). Thanks to the perfect balancing with the entropy balancing method, manual balance checking is no longer necessary. Hence, we do not have to go back and forth between estimating the propensity score and manually checking the balance of the covariates (Hainmueller, 2012).

In the entropy balancing method, each observation in the control group gets a weight satisfying a set of balance constraints set by a researcher a priori (Hainmueller, 2012). Entropy balancing estimates the weights directly from a potentially large set of balance constraints and the normalisation and non-negativity constraints. The optimal weights for the control group are chosen so that they minimise the following entropy distance metric:

$$\widehat{w_i} = \arg\min_{w_i} \sum_{\{i|control\}} w_i ln(w_i/q_i), \tag{3.A.10}$$

[24] $AIC = -2 * ln(likelihood) + 2 * K$ and $BIC = -2 * ln(likelihood) + K * ln(K)$, where K is the number of parameters of a copula function.

subject to the following balance and normalisation constraints:

$$\sum_{\{i|control\}} w_i c_{ri}(X_i) = m_r \; with \; r \in \{1, ..., R\}, \tag{3.A.11}$$

$$\sum_{\{i|control\}} w_i = 1, \; and \tag{3.A.12}$$

$$w_i \geq 0, \; \forall i \in (Control). \tag{3.A.13}$$

Equation (3.A.11) is a set of balance constraints. Equations (3.A.12) and (3.A.13) are the normalisation constraint and the non-negativity constraint respectively. $w_i c_{ri}(X_i) = m_r$ in equation (3.A.11) describes a set of R balance constraints imposed on the moments of the weighted distributions of the covariates in the control group. Each balance constraint equates a certain order moment of the weighted covariate distributions in the control group to the corresponding moment of the covariate distributions in the treatment group. As the entropy balancing can balance the higher order moments of the distributions, the moment constraints can include not only the mean (first moment), but also the variance (second moment), the skewness (third moment) and beyond. For example, if we are interested in estimating the weights for balancing the r-th order moment of a specific covariate, say X_p, we set $c_{ri}(X_{ip}) = (X_{ip})^r$ or $c_{ri}(X_{ip}) = (X_{ip} - \mu_p)^r$ with mean μ_p. The choice of the moment order can vary across the covariates and largely depends on the researcher's a priori knowledge about their distribution types. For example, if the covariates are binary variables, the first order moment balancing is sufficient, while for the case of the variables that are normally distributed, balancing the first and second order moments is sufficient to balance their entire distributions.

Chapter 4

Reviewing the Existing Evidence of the Conditional Cash Transfer in India on Maternal Healthcare through the Partial Identification Approach

Abstract

This study re-estimates the causal impacts of a conditional cash transfer programme in India, namely the *Janani Suraksha Yojana* (JSY), on maternal and child healthcare use. The main goal is to provide new evidence and to assess the validity of the identification assumptions employed in previous studies on the JSY, through the conservative partial identification approach. We find that the average treatment effects estimated under the conditional independence assumption lie outside the bound of the treatment effect estimated under weaker but more credible assumptions, thereby suggesting that selection bias could not have been fully controlled for by the observable characteristics, and that the average treatment effects estimated in the previous studies may have been over- or under-estimated.

JEL codes: I12, I15, I18

Keywords: Conditional cash transfer; Partial identification; Conditional independence; India

4.1 Introduction

4.1.1 Background

The adequate use of healthcare services is an essential factor in successful maternal and child health outcomes (Campbell and Graham, 2006; Chou et al., 2015; Darmstadt et al., 2009; Scott and Ronsmans, 2009). However, women in developing countries are often faced with multiple barriers to accessing health services, and improvements in maternal healthcare access, especially by poor women, remain inadequate. In India, only 51.2 per cent of women access antenatal care, and in 2014, only 38.7 per cent gave birth in a health institution (UNICEF, 2018a). In addition, India has the highest neonatal mortality rate in the world – as of 2015, around 20 per cent (=1.2 million) of global under-five deaths occurred in the country (UNICEF, 2015, 2018b). Promoting institutional delivery and adequate use of antenatal care is a key method in improving infant and neonatal mortalities (Lahariya, 2009; Pathak and Mohanty, 2010; Freedman et al., 2007). However, women's uptake of maternal care in India has been associated strongly with wealth, and facilitating the adequate use of maternal care among poor and marginalised women is still a major challenge (Pathak et al., 2010; Kesterton et al., 2010).

The success of conditional cash transfer (CCT) programmes in Latin America has led to an enthusiastically mirrored response in Africa and Asia, and CCT has become one of the most adopted demand-side programmes to enhance healthcare use (Handa and Davis, 2006; Rawlings and Rubio, 2005). CCTs influence health-seeking behaviours through financial incentives by transferring money to households, contingent on investments in human capital such as health and education. Interest in using financial incentives to promote maternal and child healthcare utilisation has also spread to Southern Asia, which has low maternal healthcare use and high neonatal mortality rates. CCT programmes intending to encourage maternity and child healthcare use have been introduced in India, Nepal and Bangladesh (Devadasan et al., 2008).[1]

On 12 April 2005, the Indian government launched a nationwide CCT programme called *Janani Suraksha Yojana* (JSY), or the 'Safe Motherhood Programme', to promote institutional delivery and the receipt of timely antenatal and postnatal care, in order to reduce infant and neonatal mortalities (Horton, 2010; Bhutta et al., 2010). The JSY has become one of the largest cash

[1] Pakistan and Cambodia also introduced a voucher-type programme with the same aims as the CCTs in South Asia (Jehan et al., 2012; Van de Poel et al., 2014).

transfer programmes in the world in terms of the number of beneficiaries. It provides a cash incentive to mothers that give birth in government-approved health facilities[2], and in this sense, it has a much narrower aim than most of the CCTs in Latin America, which have multiple goals beyond maternal and child healthcare. Funded by central government, the JSY is administered through the National Rural Health Mission. In 2014-2015, 10.4 million women benefited from the scheme, representing a third of all women giving birth in the country annually (Ministry of Health and Family Welfare, 2015).

Evidence in most previous studies has largely relied on inappropriate data or potentially untenable identification assumptions, in order to yield a definitive causal impact. Until now, no study has ever tried to explore the validity of the identification assumptions employed in previous studies on the JSY. The main goal of this study is to re-estimate the causal impact of JSY participation on maternal and child healthcare use, with weaker but more credible identification assumptions than used in previous studies. Specifically, we non-parametrically identify the average treatment effect (ATE) by bounds. The partial identification approach abandons point identification with strong assumptions and instead seeks causal effects with much more credible assumptions (Manski, 1990, 1997; Manski and Pepper, 2000). In this sense, the partial identification approach yields more conservative results.

In this study, we perform inference under a spectrum of assumptions of varying identification power, following the approach taken by Gundersen et al. (2012, 2017); Gonzalez (2005); Gerfin and Schellhorn (2006) and Kreider et al. (2012). Any point-estimate obtained under the conventionally imposed strong assumptions should lie within the bounds, if these assumptions are valid. In this study, we show that the causal impacts based on the assumptions employed in previous studies are larger/smaller than the upper/lower limits of the ATE bounds, thus suggesting that the existing evidence could have been over- or under-estimated.

4.1.2 Janani Suraksha Yojana (JSY)

In the JSY scheme, women of 19 years of age and above are eligible for a cash benefit if they have a below-the-poverty-line card issued by the government, or if they belong to a scheduled caste or tribe. They can receive 600 Indian rupees in urban areas and 700 rupees in rural areas

[2]This includes public hospitals and accredited private institutions.

Table 4.1: Differences in eligibility and cash transfer size between HPSs and LPSs

High performing states (HPSs)	Low performing states (LPSs)
Eligibility	
Marginalised women who gives birth in a public facility for their first two live births. Women need to have a below-the-poverty-line card or belong to a scheduled caste/tribe.	All women who gives birth in a public facility.
Cash transfer size	
600 rupees in urban areas 700 rupees in rural areas	1,000 rupees in urban areas 1,400 rupees in rural areas

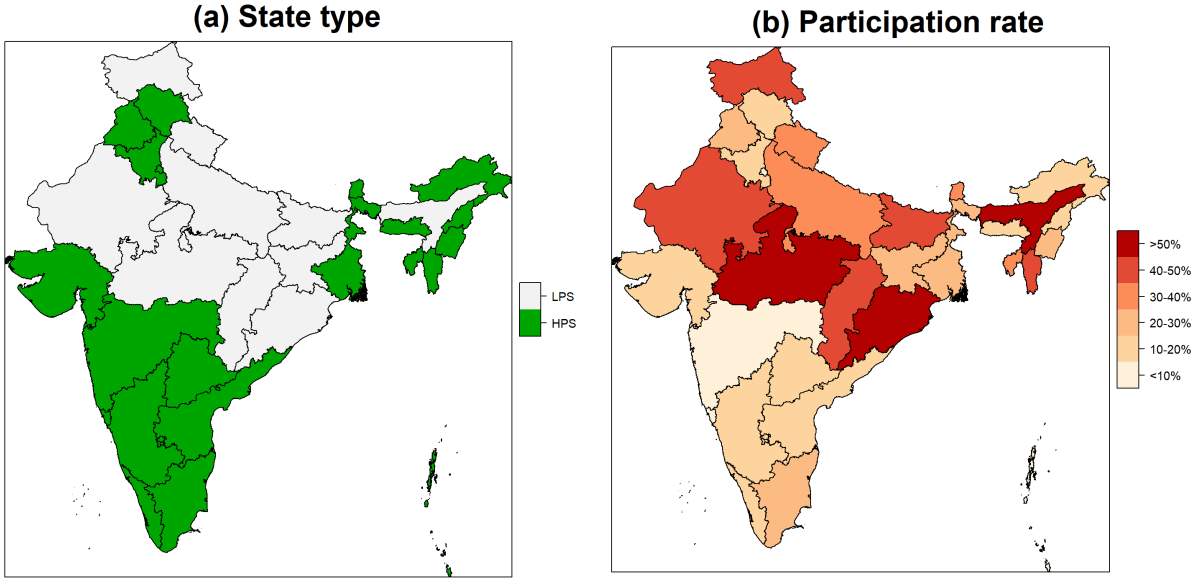
Note: This eligibility criteria and cash transfer amounts began to be applied after November 2006.

after the delivery of their first two live births in a public health facility. The JSY does not cover the actual cost of institutional delivery and maternal healthcare. The basic JSY scheme used to be the same across the country, but after November 2006, different eligibility criteria and cash transfer amounts began to be applied in ten states with high levels of maternal mortality and low levels of institutional delivery (i.e. Uttar Pradesh, Uttaranchal, Bihar, Jharkhand, Madhya Pradesh, Chhattisgarh, Assam, Rajasthan, Odisha and Jammu and Kashmir) (Bredenkamp, 2009). In these ten low-performing states (LPSs), the JSY provides a cash incentive to all women regardless of age, numbers of children or socio-economic status. In other words, every woman who gives birth in a public facility is eligible to receive a cash benefit. Moreover, in the LPSs, a higher cash incentive is provided, namely 1,000 rupees in urban areas and 1,400 rupees in rural areas.[3] In other states that are classified as high-performing states (HPSs), the original eligibility criteria, as well as the same cash incentives for pregnant mothers, continue to be applied. The differences between HPSs and LPSs are summarised in Table 4.1. The left panel of Figure 4.1 illustrates the HPSs and LPSs in the country, and the right panel of Figure 4.1 shows JSY participation rates among women who gave birth between 2010 and 2016 across the states.

The JSY addresses both the demand and the supply constraints of maternal healthcare services, i.e. it also has a supply-side component, in which community-level health workers are given incentive payments for encouraging pregnant women to give birth in official health facilities. Community-level health workers, known as 'accredited social health activists', are the first and

[3]This is around 8-12 paid days off from minimum wage manual labour (Joshi and Sivaram, 2014).

Figure 4.1: State types and JSY participation rates in 2010-16



Source: Author's calculation from the National Family Health Survey.
Note: LPS=Low-performing state; and HPS=High-performing state. The left panel illustrates the HPSs and LPSs in the country, and the right panel shows the participation rates of the JSY across the states.

most important point of contact for pregnant women – they identify and register all pregnant women, assist them in developing their birth plans and arrange the JSY for them. Accredited social health activists are given additional cash incentives to encourage mothers to complete postnatal care. Cash incentives given to health workers are intended to reduce absenteeism and enhance their overall performance.

4.2 Related literature

4.2.1 Evidence of the impacts of the JSY on maternal healthcare use

The positive impacts of CCTs in Latin America on maternal healthcare utilisation are reported by numerous studies (Ranganathan and Lagarde, 2012; Barber and Gertler, 2010; Morris et al., 2004). For systematic literature reviews of the CCT programme, see Glassman et al. (2013); Lagarde et al. (2007); Bassett (2008) and Gaarder et al. (2010). In Mexico and El Salvador, for example, birth attendance by skilled personnel increased by 11.4 percentage points and 12.3 percentage points, respectively, (Urquieta et al., 2009; de Brauw and Peterman, 2011). However, different from CCTs with much broader aims beyond maternal healthcare, studies on those with narrower aims, such as the JSY, are relatively sparse. Most of the existing literature has explored the descriptive associations between JSY receipt and healthcare use and health (Gupta et al., 2012, 2011; Thongkong et al., 2017; Randive et al., 2014, 2013; Mukherjee and Singh, 2018; Puro-

hit et al., 2014; Ng et al., 2014; Gopalan and Varatharajan, 2012), while studies estimating the causal impacts of the JSY are rather limited, mainly because the rigorous randomised controlled trial (RCT) designed for the evaluation of the programme was not conducted in India (Joshi and Sivaram, 2014).

Lim et al. (2010) is the first seminal study to estimate the causal impact of JSY from the observational data. Specifically, Lim et al. (2010) estimate the causal impacts on institutional delivery, use of antenatal care, using the individual-level and district-level data. According to their estimates with matching, the JSY increases institutional deliveries by 43.5 percentage points and skilled birth attendance by 36.6 percentage points. In addition, they report a significant increase in the use of antenatal care by 10.7 percentage points.

After the study by Lim et al. (2010), a number of studies extended the analysis by exploring heterogeneity in the average treatment effect (ATE) across the population and estimating the impacts on the use of various healthcare services (Carvalho et al., 2014). Major research into the causal effects of the JSY are listed in Table 4.2. As well as the increase in maternal healthcare utilisation, reductions in neonatal mortality and increases in the use of child healthcare are reported by Sengupta and Sinha (2018), who estimate causal impacts by employing the inverse probability weighting regression approach. In addition, improvements in the mental health of beneficiary mothers (Powell-Jackson et al., 2016), breastfeeding and further pregnancies (Powell-Jackson et al., 2015; Nandi and Laxminarayan, 2016) are reported as additional – unintended – benefits.

Most studies exploit the second and the third waves of the District Level Household and Facility Survey (DLHS-2 and DLHS-3), data for which were collected in 2002-2004 and 2007-2008. The DLHS-2 was conducted before the JSY was launched, and DLHS-3 was conducted in the initial stage of JSY's implementation. Despite the introduction of JSY in 2005, its proper implementation actually started in 2007 (Das et al., 2011). Das et al. (2011) argues that in DLHS-3, many women who gave birth before 2007 were misclassified as JSY beneficiaries, and only a smaller proportion of women knew about the programme itself at that time. Also, during the first few years of the implementation, many institutions were not adequately prepared for maternal and child healthcare (Gopichandran and Chetlapalli, 2012). As such, it turned out that

the DLHS-3 was not appropriate for causal analysis, and we had to wait for new data to become available in order to estimate the causal effect of the JSY programme.

Recently, Rahman and Pallikadavath (2017) and Rahman and Pallikadavath (2018) have re-estimated the impact of the JSY with the latest wave of the DLHS (DLHS-4), which was implemented in 2013-2014 when JSY had matured enough to be known by almost all pregnant women. Rahman and Pallikadavath (2017) estimated the impacts of the JSY on various healthcare utilisations with propensity score matching (PSM). Rahman and Pallikadavath (2018) additionally estimated the impacts with the fuzzy regression discontinuity design (RDD), which exploits changes of eligibility for the JSY programme with birth orders in HPSs.

4.2.2 Identification assumptions in previous studies

Most of the existing literature that uses individual-level data relies on strong assumptions in order to point-identify the ATE. Previous studies on the JSY imposed the conditional independence assumption (CIA), requiring that, conditional on specific observable characteristics, the selection of the treatment is random (Imbens and Wooldridge, 2009).[4] Methods based on the CIA do not assume the possibility that unobserved differences between participants and non-participants are associated with difference in outcomes. However, participation in the JSY programme is likely to be dependent on individual unobservable characteristics that could also affect healthcare use, such as the degree of being risk-averse. Also, participants in the JSY may be more aware of the importance of maternal healthcare use and the potential risk of delivery at home. If the receipt of the JSY is related to such unobservable characteristics, the approaches based on the CIA still suffer selection bias and fail to estimate the causal impact.

Joshi and Sivaram (2014) estimated the intention-to-treat effect to deal with selection problems that had not been properly addressed, using the eligibility criteria, but we should note that the intention-to-treat effect (ITT) is conceptually different from the treatment effects explored in other literature (Table 4.2). The district-level difference-in-differences (DID) is employed in Lim et al. (2010) and Powell-Jackson et al. (2015), but the effect identified with the district-level DID is also conceptually different from the ATEs identified in other studies that rely on individual-level data (Table 4.2). As the district-level DID exploits variations in the roll-out of the JSY, it

[4] A notable exception is Powell-Jackson et al. (2016), who collected their own data and exploited the fact that some women did not receive the cash due to administrative problems in its disbursement.

Table 4.2: Major studies estimating the causal effects of the JSY

Literature	Data	Methods/Identification source	Treatment indicator	Identified effect	Main outcomes
Study on targeted outcomes					
Lim et al. (2010)	DLHS 2-3	Matching, Regression	JSY participation	ATE	Delivery, birth attendance, mortality
		District-level difference-in-differences	JSY coverage rate	ATE	
Carvalho et al. (2014)	DLHS 3	Propensity score matching	JSY participation	ATE	Immunisation, PNC, breastfeeding
Sengupta and Sinha (2018)	DLHS 3	OLS adjusted with the Inverse probability weighting	JSY participation	ATE	Mortality, stillbirth, PNC, immunisation
Rahman and Pallikadavath (2017)	DLHS 4	OLS, Propensity score matching	JSY participation	ATT	Delivery, ANC, PNC, immunisation
Rahman and Pallikadavath (2018)	DLHS 4	Propensity score matching,	JSY participation	ATT	Delivery, ANC, PNC
		Fuzzy regression discontinuity design	JSY participation	LATE	
Joshi and Sivaram (2014)	DLHS 2-3	Individual-level difference-in-differences	JSY eligibility	ITT	ANC, delivery, PNC, birth attendance
Study on additional unintended benefits					
Powell-Jackson et al. (2015)	DLHS 2-3	District-level difference-in-differences	JSY coverage rate	ATE	Mortality, delivery, ANC, pregnancy, breastfeeding
Powell-Jackson et al. (2016)	Own data, census	Quasi-experimental design, exploiting the administrative problems in the disbursement	JSY participation	ATE	Maternal mental health
Nandi and Laxminarayan (2016)	DLHS 2-3	Propensity score matching + difference-in-differences, exploiting the difference in the incentive structure	JSY participation	ATT	Fertility

Note: ATE=Average treatment effect; ATT=Average treatment effect on the treated; ITT=Intention-to-treat effect; LATE=Local average treatment effect; ANC=Antenatal care; and PNC=Postnatal care.

estimates the effect of percentage point changes in the JSY coverage rate on the probability of healthcare use, not the effect of JSY participation itself. Hence, in this study, we do not consider the ITT or the district-level DID.

The fuzzy RDD can deal with individual unobservable characteristics, but the estimate is local in the sense that it is the impact among those who have a value for the running variable near the cut-off value (Imbens and Angrist, 1994). Hence, it is often difficult to extrapolate the impact for the entire population from the estimated results for the the sub-population. Rahman and Pallikadavath (2018) use the birth order (parity) as a running variable and exploit the gap in the probability of participating in the programme between mothers having one and two children and mothers having three to seven children. However, the parity itself is highly likely to be endogenous, so the observed discontinuity in the probability of programme participation could be invalid as an exogenous source of identification.

In contrast to the existing literature, this study takes a totally different approach. Rather than relying on strong and yet questionable assumptions, to achieve point-identification of the causal impact, we impose just weak but credible identification assumptions to find the bound estimation of the causal effect (Manski, 1990). The partial identification approach highlights what may be learned from the data without invoking potentially untenable assumptions (Manski, 2003, 2013). By gradually adding assumptions, we explore how much we can narrow the estimated bounds. The partial identification approach is very useful in a situation in which a rigorous RCT has not been implemented – and hence valid instrumental variables to deal with possible endogeneity are not available to researchers. The partial identification approach has been applied to various topics in microeconomics, such as labour (Lee, 2009; Gonzalez, 2005; Blundell et al., 2007), crime (Manski, 2013; Manski and Nagin, 1998), education (Ginther, 2000; Huber et al., 2017) and health (Gerfin and Schellhorn, 2006; Gundersen et al., 2017, 2012; Kreider et al., 2012).

The identification assumptions employed in the existing literature have not been sufficiently justified or assessed. In this study, we first re-estimate the causal impacts of the JSY with the DLHS-4, closely following the approaches taken by Rahman and Pallikadavath (2018), Lim et al. (2010) and Sengupta and Sinha (2018), which all rely on the CIA. We then estimate the bounds of ATE through the partial identification approach and compare the point-identified impacts

with the bound-identified impacts, in order to assess the validity of the assumptions used by their respective studies.

4.3 Data

4.3.1 District Level Household and Facility Survey (DLHS)

We use the latest wave of the District Level Household and Facility Survey (DLHS-4), which was conducted in 2013-2014 when the JSY had been rolled out across the country and matured somewhat (Rahman and Pallikadavath, 2017, 2018). As such, the DLHS-4 does not suffer the data contamination problem that is found in DLHS-3 (Das et al., 2011). The DLHS-4 covers the 18 HPSs[5] and three high-performing union states.[6] The DLHS-4 is a representative survey only at the district level, and it collects data only in HPSs, so this study only focuses on the HPSs. We complement our findings in HPSs by proving additional evidence in LPSs using another latest set of survey data, namely the National Family Health Survey (NFHS-4), in which data were collected also from LPSs, though the sample sizes are much smaller. Results for LPSs are discussed in section 4.A.1 in Appendix 4.A.

4.3.2 Outcomes

This study estimates the impacts on the following eight maternal and child healthcare utilisations. First, we estimate the impacts on (1) giving birth at health institutions[7], which is the primary target outcome of the JSY programme and (2) skilled birth attendance. We also estimate the impacts on using (3) antenatal care (ANC) at least once, (4) ANC three times or more, (5) postnatal care (PNC) for mothers, (6) PNC for babies, (7) iron and folic acid (IFA) tablets/syrup during pregnancy and (8) tetanus toxoid (TT) injections to prevent babies from getting tetanus after birth.

4.3.3 Covariates for the point-identification

In order to make our results comparable as much as possible with those in the existing literature, we control for the individual- and household-level confounding factors, closely following Rahman and Pallikadavath (2018). As factors reflecting demographic and socio-economic characteristics,

[5] Andhra Pradesh, Arunachal Pradesh, Goa, Haryana, Himachal, Pradesh, Karnataka, Kerala, Maharashtra, Manipur, Meghalaya, Mizoram, Nagaland, Punjab, Sikkim, Tamil Nadu, Telangana, Tripura and West Bengal.

[6] Andaman and Nicobar Islands, Chandigarh and Puducherry.

[7] Health institution herein includes both the public and the private health sector.

we control for below-the-poverty-line card ownership, maternal age, a residential location (urban/rural) and a birth order (parity). A Hindu dummy variable is used to reflect individual socio-cultural backgrounds. Other religions (e.g. Islam, Christianity, Sikhism, Buddhism) are benchmark groups. In addition, we use dummy variables for the scheduled castes and tribes. Scheduled castes/tribes are the most socially disadvantaged groups, members of which have suffered the greatest burden of social and economic segregation and deprivation (Chitnis, 1997). We control for parental educational levels, measured by the number of education years. Household wealth is captured through a composite index of relative standards of living.[8]

4.3.4 Sample selection

In this study, we focus only on women aged between 15 and 49 who gave birth in the five years prior to the survey, so that all of those analysed in this study gave birth after the proper implementation of the JSY. We restrict our attention to the most recent birth of each woman and exclude women who participated in programmes for childbirth other than the JSY. The DLHS-4 has 84,266 observations. After dropping observations with missing information[9], our sample size becomes 67,595. Descriptive statistics are shown in Table 4.3.

4.4 Methods

4.4.1 Notations

Let $Y = \{0, 1\}$ be an indicator for observed healthcare utilisation. Y becomes 1 if a woman uses a healthcare service. $D = \{0, 1\}$ is an indicator for participation in the JSY, and it equals 1 for those who participated in the JSY programme, and 0 otherwise. Following Rubin (1974), we assume that each individual has two potential outcomes, namely Y_0 in the absence of the treatment and Y_1 in the presence of the treatment. They are latent, in the sense that we can only observe one of them for each person, but never both. What is actually observed for

[8]Following Rahman and Pallikadavath (2018), we derive the wealth index by applying the principal component analysis over various household characteristics. They are cooking fuel, house type, number of dwelling rooms, electricity, house ownership, landholding, radio, television, computer, internet, telephone, mobile phone, washing machine, refrigerator, sewing machine, watch, bicycle, motorcycle, car, tractor, tube well, cart and air cooler.

[9]We dropped 27 observations because of the missing information about below-the-poverty-line card ownership. We dropped 82 and two observations that did not have information about caste and religion, respectively. In total, 2,916 observations were then dropped because of missing information about parity. We dropped 79 observations and 13,171 observations because they did not have information about maternal and parental educational backgrounds, respectively. We dropped eight observations with missing information about household wealth. Lastly, we dropped 386 observations with missing information about outcomes.

Table 4.3: Descriptive statistics for the DLHS-4

	Non-participants mean	Participants mean
Institutional delivery	0.82	0.95
Skilled birth attendance	0.89	0.97
Antenatal care at least once	0.87	0.96
Antenatal care ($\geq$ 3 times)	0.70	0.81
Postnatal care for mother	0.64	0.72
Postnatal care for baby	0.78	0.83
Iron and folic acid (IFA) supplement	0.68	0.82
Tetanus toxoid (TT) injection	0.83	0.93
Below the poverty line card	0.30	0.46
Scheduled caste	0.77	0.75
Scheduled tribe	0.16	0.19
Rural	0.56	0.67
Birth order (parity)	2.01	1.78
Hindu	0.67	0.69
Maternal age	27.23	26.05
Maternal education years	9.81	9.23
Paternal education years	10.92	9.98
Wealth	0.54	-0.26
Observations	52732	14863

Source: DLHS-4.

researchers is

$$Y = DY_1 + (1 - D)Y_0. \tag{4.1}$$

The causal impact of the JSY for individual $j \in J$ is $Y_{1j} - Y_{0j}$, and we are interested in the average treatment effect (ATE), which is defined by $ATE \equiv P(Y_1 = 1) - P(Y_0 = 1)$. Theoretically, the ATE ranges from -1 to +1 and has a width of 2. Let X be observed individual characteristics, which determine Y and D.

Using the law of total probability, we can express $P(Y_1 = 1)$ and $P(Y_0 = 1)$ as

$$P(Y_1 = 1) = P(Y_1 = 1|D = 1)P(D = 1) + \underbrace{P(Y_1 = 1|D = 0)}_{Unobservable} P(D = 0) \tag{4.2}$$

$$P(Y_0 = 1) = \underbrace{P(Y_0 = 1|D = 1)}_{Unobservable} P(D = 1) + P(Y_0 = 1|D = 0)P(D = 0), \tag{4.3}$$

where $P(Y_1 = 1|D = 0)$ is the counterfactual probability that untreated women would use a healthcare service under the treatment, and $P(Y_0 = 1|D = 1)$ is the counterfactual probability that treated women would use a healthcare service if they had not been treated. $P(Y_1 = 1|D = 1)$,

$P(Y_0 = 1|D = 0)$, $P(D = 1)$ and $P(D = 0)$ are immediately identifiable from the distribution of the observed data, but $P(Y_1 = 1|D = 0)$ and $P(Y_0 = 1|D = 1)$ are unobservable, which makes the ATE unidentifiable without further assumptions.

4.4.2 Point-identification with the independence assumption

Researchers must impose assumptions regarding $P(Y_1 = 1|D = 0)$ and $P(Y_0 = 1|D = 1)$, in order to identify the ATE. For example, if we assume that participation in the treatment is random, we can point-identify the ATE from the observed distribution. This assumption is called the 'independence assumption' and can be expressed as

$$P(Y_1 = 1|D = 0) = P(Y_1 = 1|D = 1) \tag{4.4}$$

$$P(Y_0 = 1|D = 0) = P(Y_0 = 1|D = 1). \tag{4.5}$$

The ATE is point-identified by $ATE = P(Y_1 = 1|D = 1) - P(Y_0 = 1|D = 0)$. This independence assumption is valid under the rigorous RCTs, without which it is usually too stringent. Another identification assumption that is conventionally imposed in the policy evaluation literature is the conditional independence assumption (CIA) in which participation in the treatment is assumed to be random, conditional on the observable characteristics X. The CIA is formalised by

$$P(Y_1 = 1|D = 0, X) = P(Y_1 = 1|D = 1, X) \tag{4.6}$$

$$P(Y_0 = 1|D = 1, X) = P(Y_0 = 1|D = 1, X). \tag{4.7}$$

For example, evaluations with the multivariate regression approach and the propensity score matching approach are based on this assumption (Imbens and Wooldridge, 2009; Abadie and Cattaneo, 2018). Although the CIA itself cannot be tested by the data, this assumption can be untenable in many cases. If the decision to participate in the programme is dependent on unobservable characteristics that can affect outcomes, the CIA fails, resulting in a biased causal effect estimate.

In this study, we point-estimate the ATEs by (1) mean comparison between the participants and non-participants, which we call 'Independence', (2) multivariate ordinary least squares (OLS) regression, (3) propensity score matching (PSM), (4) OLS regression with inverse probability weighting adjustment (IPW+OLS), (5) nearest neighbourhood matching (NNM), (6) entropy

Table 4.4: Point-identification approaches and assumptions

Method	**Short name**	**Assumption**
Mean comparison	Independence	Exogenous/independent treatment assignment
Multivariate linear regression	OLS	Conditional independence; Outcome linear functional-from; Additive linearity of the treatment status and the unobservable; Same treatment effect for all individuals
Propensity score matching	PSM	Conditional independence; Propensity score functional-form
Nearest neighbour matching	NNM	Conditional independence
OLS adjusted with the inverse probability weighting	IPW+OLS	Conditional independence
Entropy balance weighting	EBW	Conditional independence
Regression discontinuity design	RDD	Exogenous discontinuity in the probability of the JSY participation at birth order two

balance weighting (EBW) and (7) fuzzy regression discontinuity design (RDD). Their key assumptions are summarised in Table 4.4.

The mean comparison between participants and non-participants rests on the independence assumption, and the OLS, PSM, IPW+OLS, NNM and EBW approaches rest on the CIA. Some also rely on functional form assumptions; for example, the OLS assumes that the treatment variable and the error term are additively separated and the treatment effect is identical for everyone. The PSM and IPW+OLS rely on the functional form assumption of the propensity score. The EBW non-parametrically balances the moments of the covariate distributions, the algorithm of which is explained in the Appendix 4.B.1.

For fuzzy RDD, we also closely follow the approach taken by Rahman and Pallikadavath (2018), which exploits JSY eligibility, i.e. mothers who are eligible for the JSY up to their second live birth. The eligibility change of JSY at birth order two was exploited as a source of identification. Following Rahman and Pallikadavath (2018), we first run an OLS regression of the JSY dummy variable on $z_1 = 1\{parity \leq 2\}$, $z_2 = z_1 * (parity - 2)$, $z_3 = (parity - 2)$ and the other covariates as the first-stage OLS regression, where z_1 and z_2 are excluded instruments. Using the predicted value of the JSY dummy variable, we estimate the causal impact of JSY in the second-stage OLS

regression.

The DLHS is repeated cross-sectional data. If longitudinal data were available, we could possibly employ the fixed effect (FE) model, which deals with the situation where individual unobservable and time-invariant characteristics influence treatment selection. However, the FE model is less appealing for the study on maternal healthcare, because it is not realistic to assume that the degree of risk-aversion is time-invariable; it can change as mothers experience more childbearing. For example, the motivation to join the JSY programme for primiparity can be different from the one for the second or third childbirth. In this study, we do not consider the individual-level DID approach either, mainly for two reasons. First, the JSY is a nationwide programme, which makes it difficult to set up the control group, and second, we have gaps of more than 10 years between data collection before and after JSY implementation; the DLHS-2 was conducted in 2002-2004. The long periods between the waves could substantially deteriorate the credibility of common-trend assumption that is essential for the DID.

4.4.3 Partial identification assumptions

In essence, partial identification first estimates sharp bounds for $P(Y_1 = 1)$ and $P(Y_0 = 1)$ and then constructs a sharp bound of ATE. A sharp bound is defined as the narrowest bound that can be obtained under the maintained assumptions regarding the unobservable distribution. When the bound of $P(Y_t = 1)$ is (LB_t, UB_t), the ATE bound is defined as

$$(LB_1 - UB_0) \leq ATE \leq (UB_1 - LB_0). \tag{4.8}$$

Note that first, the ATE bound is conceptually different from the confidence interval of the point-estimated ATE. The width of the ATE bound reflects the identification power of the imposed assumptions. In addition, bound width indicates tension between the strength of assumptions and their credibility (Manski, 2007), and it does not change when sample sizes change. On the other hand, the width of confidence interval for the point-identified ATE reflects the uncertainty of sampling variability, which does vary when sample sizes change. We discuss the uncertainty of sampling variability for the ATE bound in section 4.4.5. In this study, unless otherwise indicated, we use the term 'bound' to denote an ATE bound of partial identification, and use the term 'interval' to indicate a confidence interval.

Second, as the partial identification approach does not assume that participation in the treatment is random and conditional on the covariates, there is no specific need to condition on a long list of covariates. Hence, the partial identification approach is not susceptible to criticisms of omitted variable bias (Manski and Nagin, 1998). Splitting the sample by a covariate to explore heterogeneity across the population is also possible for partial identification as well, but in this study we do not implement sample splitting.

No assumptions (Worst-case)

First, as a benchmark, we specify a range of $P(Y_1 = 1|D = 0)$ and $P(Y_0 = 1|D = 1)$ to construct a bound for $P(Y_1 = 1)$ and $P(Y_0 = 1)$, without imposing any assumption regarding the counterfactual probabilities. Since $P(Y_1 = 1|D = 0)$ and $P(Y_0 = 1|D = 1)$ are probabilities, they necessarily belong to [0,1], so the bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ are given from equations (4.2) and (4.3) by

$$\underbrace{P(Y_1 = 1|D = 1)P(D = 1)}_{LB_1} \leq \quad \boldsymbol{P(Y_1 = 1)} \quad \leq \underbrace{P(Y_1 = 1|D = 1)P(D = 1) + P(D = 0)}_{UB_1} \tag{4.9}$$

$$\underbrace{P(Y_0 = 1|D = 0)P(D = 0)}_{LB_0} \leq \quad \boldsymbol{P(Y_0 = 1)} \quad \leq \underbrace{P(Y_0 = 1|D = 0)P(D = 0) + P(D = 1)}_{UB_0}. \tag{4.10}$$

In equation (4.9), the lower bound of $P(Y_1 = 1)$ is attained if all non-participants would have used the healthcare had they participated in the JSY. The upper bound of $P(Y_1 = 1)$ is attained if all non-participants would have had no healthcare had they participated in the JSY. The bound width of $P(Y_1 = 1)$ is $P(D = 0)$. The bounds of $P(Y_0 = 1)$ in equation (4.10) can be interpreted in the same fashion, and bound width is $P(D = 1)$. The sharp bound of ATE can be obtained via equation (4.8) (Manski, 1990):

$$\begin{aligned} & \underbrace{P(Y_1 = 1|D = 1)P(D = 1)}_{LB_1} - \underbrace{\{P(Y_0 = 1|D = 0)P(D = 0) + P(D = 1)\}}_{UB_0} \\ \leq \quad & ATE \\ \leq \quad & \underbrace{\{P(Y_1 = 1|D = 1)P(D = 1) + P(D = 0)\}}_{UB_1} - \underbrace{P(Y_0 = 1|D = 0)P(D = 0)}_{LB_0}. \end{aligned} \tag{4.11}$$

This bound is the narrowest sharp band that can be inferred from the data alone, which is often called a 'worst-case' bound. Note that even without any assumption about unobserved probabilities, the data alone tighten the theoretical width of ATE to half, if the support of the outcome variable is bounded (Manski, 1989). It is known that without any identification assumption,

Table 4.5: Partial-identification assumptions

Assumption	**Implication**
Monotone treatment response (MTR)	Individuals do not participate in the programme that makes them worse-off with respect to the outcome.
Positive/negative monotone treatment selection (MTS)	The treated are more/less likely to have the healthcare than the non-treated both in the presence and absence of the treatment.
Monotone instrumental variable (MIV)	Eligible people are less likely to have healthcare conditional on the treatment than ineligible women.

the bound on the ATE in equation (4.11) always has a width of 1 and includes 0. Hence, without additional assumptions, we cannot evaluate the sign of the treatment effect on healthcare use. Next, we consider three assumptions: (i) the monotone treatment response (MTR), (ii) the monotone treatment selection (MTS) and (iii) the monotone instrumental variable (MIV). The key implications of each assumption are summarised in Table 4.5.

Monotone treatment response

Second, we assume that individuals do not select a treatment that would make them worse off with respect to maternal and child healthcare use. This assumption is called the 'monotone treatment response' (MTR) assumption and it implies that, *ceteris paribus*, response varies monotonically with treatment (Manski, 1997).

Formally, the MTR assumes that for all individual $j \in J$, $Y_{0j} \leq Y_{1j}$. We obtain the new bounds for $P(Y_1 = 1|D = 0)$ and $P(Y_0 = 1|D = 1)$ as follows:

$$P(Y_0 = 1|D = 0) \leq \boldsymbol{P(Y_1 = 1|D = 0)} \leq 1 \tag{4.12}$$

$$0 \leq \boldsymbol{P(Y_0 = 1|D = 1)} \leq P(Y_1 = 1|D = 1). \tag{4.13}$$

We observe shrinkage in the counterfactual probability bounds. For $P(Y_1 = 1|D = 0)$, while the bound of this counterfactual probability in the worst-case ranges from 0 to 1, its new range under the MTR assumption ranges from $P(Y_0 = 1|D = 0)$ to 1. Also, the new range of $P(Y_0 = 1|D = 1)$ under the MTR assumption runs from 0 to $P(Y_1 = 1|D = 1)$. The following bounds of $P(Y_1 = 1)$

and $P(Y_0 = 1)$ can be obtained from equations (4.1) and (4.3):

$$\begin{aligned} & P(Y_1 = 1|D = 1)P(D = 1) + P(Y_0 = 1|D = 0)P(D = 0) \\ = \; & \underbrace{P(Y = 1)}_{Updated\, LB_1} \\ \leq \; & \boldsymbol{P(Y_1 = 1)} \\ \leq \; & P(Y_1 = 1|D = 1)P(D = 1) + P(D = 0) \end{aligned} \tag{4.14}$$

and

$$\begin{aligned} & P(Y_0 = 1|D = 0)P(D = 0) \\ \leq \; & \boldsymbol{P(Y_0 = 1)} \\ \leq \; & P(Y_1 = 1|D = 1)P(D = 1) + P(Y_0 = 1|D = 0)P(D = 0) \\ = \; & \underbrace{P(Y = 1)}_{Updated\, UB_0}, \end{aligned} \tag{4.15}$$

where the lower bound of $P(Y_1 = 1)$ and the upper bound of $P(Y_0 = 1)$ in the worst-case scenario are both replaced by $P(Y = 1)$. Via equation (4.8), it follows

$$0 \leq ATE \leq \{P(Y_1 = 1|D = 1)P(D = 1) + P(D = 0)\} - P(Y_0 = 1|D = 0)P(D = 0). \tag{4.16}$$

Under the MTR assumption, the lower bound of ATE becomes 0. Therefore, the MTR assumption precludes a non-negative ATE and assumes away the possibility of the detrimental impact of the JSY. In other words, the MTR assumes that the JSY programme would never decrease the likelihood of receiving healthcare. The MTR assumption, however, leaves open the question of whether the programme has a strong beneficial effect, a mild beneficial effect or no effect at all (Gundersen et al., 2017).

In general, the credibility of the MTR assumption depends on the type of policy we are analysing. As the MTR assumes the sign of the treatment effect, the MTR assumption can be admittedly too stringent in the case where we have no clue at all regarding the sign of the ATE a priori. However, in the context of the JSY programme, it is unlikely that a mother does not use the healthcare service when she is treated and does use the service when she is untreated, because the accredited social health activists, who support pregnant mothers in the JSY programme,

encourage women to receive timely maternal and child healthcare. Furthermore, they encourage women to undergo three antenatal cares and to give birth in a health institution. They also assist pregnant women to obtain tetanus toxoid injections and iron/folic acid supplements. The Ministry of Health and Family Welfare also states, in their guidelines for the implementation of JSY, that all pregnant mothers should receive at least three ANCs. Therefore, it is reasonable to presume that the JSY programme will not decrease healthcare use.

Monotone treatment selection

Third, we make an assumption on the selection mechanism through which women participate in the JSY. Specifically, we make an assumption as to whether or not individuals participating in the JSY programme are more likely to use maternal and child healthcare services on average, conditional on the treatment assignment. This assumption is called the 'monotone treatment selection' (MTS) assumption (Manski and Pepper, 2000) and sounds similar to the MTR assumption, but they are different, although not mutually exclusive. In contrast to the MTR assumption, which is on individual-level potential behaviours, the MTS assumption is on expected probability. The MTS can be regarded as the weaker version of the independence assumption, whereby equalities in equations (4.4) and (4.5) are weakened to the point of becoming inequalities. There are two types of MTS assumption, namely positive MTS and negative MTS, and they make different assumptions regarding the direction of the selection bias.

(i) Positive MTS: When we assume that mothers participating in the JSY are likely to use no fewer healthcare services on average than non-participants, conditional on treatment assignment, this assumption is called the 'positive MTS assumption'. It is plausible in situations where risk-averse women are more likely to participate in the JSY and use healthcare services than women with lower degrees thereof, and/or where women in socially and economically segregated ethnic groups know less about the JSY. The positive MTS assumption is formalised as follows:

$$0 \leq \quad \boldsymbol{P(Y_1 = 1 | D = 0)} \quad \leq P(Y_1 = 1 | D = 1) \tag{4.17}$$

$$P(Y_0 = 1 | D = 0) \leq \quad \boldsymbol{P(Y_0 = 1 | D = 1)} \quad \leq 1. \tag{4.18}$$

Note that in equations (4.17) and (4.18), the MTS assumes only the direction of the selection bias, and it does not assume its strength. The positive MTS assumption lowers the upper bound of $P(Y_1 = 1|D = 0)$ in the worst-case, from 1 to $P(Y_1 = 1|D = 1)$, and raises the lower bound of

$P(Y_0 = 1|D = 1)$ in the worst-case, from 0 to $P(Y_0 = 1|D = 0)$.

The new sharp bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ can then be obtained from equations (4.2) and (4.3) as follows (Manski and Pepper, 2000):

$$P(Y_1 = 1|D = 1)P(D = 1) \leq \quad \boldsymbol{P(Y_1 = 1)} \quad \leq \underbrace{P(Y_1 = 1|D = 1)}_{Updated\, UB_1} \tag{4.19}$$

$$\underbrace{P(Y_0 = 1|D = 0)}_{Updated\, LB_0} \leq \quad \boldsymbol{P(Y_0 = 1)} \quad \leq P(Y_0 = 1|D = 0)P(D = 0) + P(D = 1). \tag{4.20}$$

Compared with their bounds in the worst-case scenario, the upper bound of $P(Y_1 = 1)$ has become smaller and the lower bound of $P(Y_0 = 1)$ has become larger. The sharp bound of ATE is given via the equation (4.8) and it is

$$\begin{aligned} & P(Y_1 = 1|D = 1)P(D = 1) - \{P(Y_0 = 1|D = 0)P(D = 0) + P(D = 1)\} \\ \leq \quad & ATE \\ \leq \quad & P(Y_1 = 1|D = 1) - P(Y_0 = 1|D = 0). \end{aligned} \tag{4.21}$$

We find that, compared with the worst-case ATE bound in equation (4.11), the upper bound in equation (4.21) has become smaller. It is easy to show that the upper bound of ATE under the MTS assumption corresponds to the ATE estimated under the independence assumption (see the note in the Appendix 4.B.2), which implies that the upper bound of ATE is achieved when there exists no selection bias regarding participation in the JSY.

(ii) Negative MTS: If we anticipate that mothers who had the greater difficulty in using healthcare services had higher motivation to participate in the JSY, or they were more strongly encouraged by the accredited social health activists to participate in the JSY, it may be more appropriate to assume that mothers participating in the JSY are likely to use no more health-care services on average than non-participants. This assumption is called the 'negative MTS assumption', which is formalised as follows:

$$P(Y_1 = 1|D = 1) \leq \quad \boldsymbol{P(Y_1 = 1|D = 0)} \quad \leq 1 \tag{4.22}$$

$$0 \leq \quad \boldsymbol{P(Y_0 = 1|D = 1)} \quad \leq P(Y_0 = 1|D = 0). \tag{4.23}$$

The negative MTS assumption raises the worst-case upper bound of $P(Y_1 = 1|D = 0)$ from 0 to $P(Y_1 = 1|D = 1)$ and lowers the worst-case lower bound of $P(Y_0 = 1|D = 1)$ from 1 to

$P(Y_0 = 1|D = 0)$. The bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ become

$$\underbrace{P(Y_1 = 1|D = 1)}_{Updated\, LB_1} \leq \quad \boldsymbol{P(Y_1 = 1)} \quad \leq P(Y_1 = 1|D = 1)P(D = 1) + P(D = 0) \tag{4.24}$$

$$P(Y_0 = 1|D = 0)P(D = 0) \leq \quad \boldsymbol{P(Y_0 = 1)} \quad \leq \underbrace{P(Y_0 = 1|D = 0)}_{Updated\, UB_0}. \tag{4.25}$$

The sharp bound of ATE is given via equation (4.8) and is

$$\begin{aligned} & P(Y_1 = 1|D = 1) - P(Y_0 = 1|D = 0) \\ \leq \quad & ATE \\ \leq \quad & \{P(Y_1 = 1|D = 1)P(D = 1) + P(D = 0)\} - P(Y_0 = 1|D = 0)P(D = 0). \end{aligned} \tag{4.26}$$

We find that, compared with the worst-case bound in equation (4.11), the lower bound of the ATE in equation (4.26) has become larger. Also, this bound shows that the lower bound of ATE under the negative MTS assumption corresponds to the ATE estimated under the independence assumption, which implies that the lower bound of ATE is achieved only when there exists no selection bias regarding the participation in the JSY (see the note in the Appendix 4.B.2).

In general, sources of selection bias are multiple. The ultimate direction of the selection mechanism is expected to vary across the outcomes, but moreover, in essence, there is no definitive way to examine the sources of selection bias and the direction of this bias. However, we can find a clue by comparing the size of the ATE estimated under the independence assumption and the one estimated under the CIA. When the ATE under the independence assumption is larger than that under the CIA, it implies the existence of positive selection bias, part of which is removed by controlling for covariates. On the other hand, when the difference between the ATEs is negative, it suggests the existence of negative selection bias. We infer the sign of selection bias for each outcome, and then we impose either a positive or a negative MTS assumption accordingly.

Monotone instrumental variable

The last assumption we consider is the monotone instrumental variable (MIV) assumption (Manski and Pepper, 2000), which states that the latent probability of having healthcare varies weakly and monotonically with an observed instrument, v. Different from the standard instrumental variable approach (Imbens and Angrist, 1994), the MIV assumption does not impose the mean independence assumption requiring that the latent outcomes are mean-independent of the in-

strument, namely $P(Y_t = 1|v = 1) = P(Y_t = 1|v = 0)$ for $t \in \{0, 1\}$. The instrument v is allowed to be dependent on the mean of the potential outcome, as long as the direction of its effect is monotone. Hence, in this sense, the MIV is weaker than the mean independence assumption. Also, the MIV assumption itself does not impose any assumption regarding the association between the instrument and the treatment status. Thus, the MIV assumption is not susceptible to criticisms of weak instruments.

Following Gundersen et al. (2017, 2012)[10], we use the non-eligibility status of the JSY as an instrument. We assume that ineligible women, who tend to be richer, are more likely to use healthcare services conditional on the treatment than eligible women. This assumption is supported by the observations in developing countries that poorer women are less likely to use maternal healthcare services (Pathak et al., 2010; Pathak and Mohanty, 2010; Kesterton et al., 2010; Balarajan et al., 2011). We discuss the plausibility of our MIV assumption further in section 4.6.2.

Formally, let v be a binary instrument, and $v = 1$ indicates that a woman is not eligible for the JSY. The MIV assumption imposed herein is expressed as

$$P(Y_1 = 1|v = 0) \leq P(Y_1 = 1|v = 1) \tag{4.27}$$

$$\underbrace{P(Y_0 = 1|v = 0)}_{Eligible\ women} \leq \underbrace{P(Y_0 = 1|v = 1)}_{Ineligible\ women}. \tag{4.28}$$

Note that equations (4.27) and (4.28) are the assumptions on the latent outcomes. The MTS assumption is a special case of the MIV assumption, in which the participation status itself is used as an instrument, i.e. $v = D$.

The MIV assumption gives the following sharp bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ in equations

[10]Gundersen et al. (2017, 2012) estimate the ATE bounds of food assistance programmes targeting poor households in the US on nutritional status.

(4.29) and (4.30). Their derivations are provided in the Appendix 4.B.3.

$$
\begin{aligned}
& P(v=0)P(Y_1=1|D=1,v=0)P(D=1|v=0) \\
& +P(v=1)\max\{P(Y_1=1|D=1,v=0)P(D=1|v=0), P(Y_1=1|D=1,v=1)P(D=1|v=1)\} \\
\leq\ & \boldsymbol{P(Y_1=1)} \qquad (4.29) \\
\leq\ & P(v=0)\min\{P(Y_1=1|D=1,v=0)P(D=1|v=0)+P(D=0|v=0), \\
& P(Y_1=1|D=1,v=1)P(D=1|v=1)+P(D=0|v=1)\} \\
& +P(v=1)[P(Y_1=1|D=1,v=1)P(D=1|v=1)+P(D=0|v=1)]
\end{aligned}
$$

and

$$
\begin{aligned}
& P(v=0)P(Y_0=1|D=0,v=0)P(D=0|v=0) \\
& +P(v=1)\max\{P(Y_0=1|D=0,v=0)P(D=0|v=0), P(Y_0=1|D=0,v=1)P(D=0|v=1)\} \\
\leq\ & \boldsymbol{P(Y_0=1)} \qquad (4.30) \\
\leq\ & P(v=0)\min\{P(Y_0=1|D=0,v=0)P(D=0|v=0)+P(D=1|v=0), \\
& P(Y_0=1|D=0,v=1)P(D=0|v=1)+P(D=1|v=1)\} \\
& +P(v=1)[P(Y_0=1|D=0,v=1)P(D=0|v=1)+P(D=1|v=1)].
\end{aligned}
$$

The sharp bound of the ATE can be obtained by equation (4.8).[11] ATE bounds under the MIV assumption, and those in the worst-case scenario, coincide if the worst-case lower and upper bounds of $P(Y_t=1|v=u)$ weakly increase with u; in such cases, the MIV assumption has no identifying power, and the MIV assumption does not make the ATE bounds narrower (Manski and Pepper, 2000; Richey, 2016).

4.4.4 Joint imposition of partial identification assumptions

We consider the case where we jointly impose the assumptions introduced so far. We add mild assumptions one by one and see how much each additional assumption can make the ATE bound

[11] It is known that when we estimate the ATE bound under the MIV assumption with the sample analogue, the estimate can suffer finite-sample bias, which in turn could potentially make the bound narrower than the true bound (Manski and Pepper, 2009). Following Jensen's inequality, the estimated lower bounds are potentially biased upwards because of their maxima operators, and the estimated upper bounds are potentially biased downwards because of their minima operators. Details are provided in the Appendix 4.B.4. To address this bias, we implement a bootstrap-based correction proposed by Kreider and Pepper (2007). This method estimates bias by using the bootstrap distribution and adjusting the sample analogue estimate in accordance with the estimated bias. For example, when we have a random sample of size N, let LB_N be the sample analogue estimate of the lower bound in question. We denote $E^b(LB_N)$ as the mean of the estimate from the bootstrap distribution of size b. Bias is estimated as $E^b(LB_N) - LB_N$, and the bias-corrected estimate is given by $LB_N - \{E^b(LB_N) - LB_N\} = 2LB_N - E^b(LB_N)$. We correct the bias of the upper bound in the same way. In this study, we estimate bias with $b = 100$ bootstrap repetitions. The performance of this method is confirmed by Monte Carlo simulations in Manski and Pepper (2009).

narrower. In the context of the JSY programme, the MTR assumption is the most plausible, followed by the MTS assumption. Lastly, we add the MIV assumption, which seems the strongest of all three assumptions. Hence, in this study we mainly consider the following two cases: MTR+MTS and MTR+MTS+MIV.

When we jointly impose different assumptions, we derive the bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ under the jointly imposed assumptions, in order to get the ATE bound. ATE bounds' estimators and their full derivations are in the Appendix 4.C. For the other combinations, i.e. MTR+MIV and MTS+MIV, their estimators are also provided in the Appendix 4.D.

4.4.5 Inference of partial identification

Estimating the ATE bounds requires us to consider sampling variabilities. The ATE bounds introduced so far concern the conclusions that could be drawn under different assumptions if we were to observe the JSY participation status and outcomes experienced by everyone in the population. Sampling variability, however, arises when these data are available for only a sample of the population. We consider sample variability by constructing the confidence intervals for the bound estimates.

A confidence interval for the bound estimate contains the parameter of interest; however, there is no consensus on its definition, and related research is still ongoing. When we consider a confidence interval in the partial identification, the question arises as to whether to construct an interval over the region of identification (Chernozhukov et al., 2007) or over the actual parameter of interest (Imbens and Manski, 2004).[12]

We obtain the confidence intervals for the estimated bounds via the method developed by Imbens and Manski (2004). The confidence intervals defined by Imbens and Manski (2004) asymptotically cover the true parameter, namely ATE, with a fixed probability, rather than covering the entire identification region with fixed probability.

Formally, the confidence intervals for parameter θ at the $1 - \alpha$ level are defined as sets of the parameters where we cannot reject the null hypothesis that θ is an element of the iden-

[12]Instead, some studies provide confidence intervals for the upper and lower ATE bounds, respectively (Ginther, 2000).

tification set, Θ, at the α level. When the estimated lower and upper bounds are $[\widehat{LB}, \widehat{UB}]$ and their standard errors are $\widehat{\sigma_{LB}}$ and $\widehat{\sigma_{UB}}$, $1-\alpha$ level confidence intervals of Imbens and Manski (2004) are constructed as $CI_{1-\alpha} \in [\widehat{LB} - c\widehat{\sigma_{LB}}, \widehat{UB} + c\widehat{\sigma_{UB}}]$, where parameter c solves $\Phi(c + \frac{(\widehat{\sigma_{UB}} - \widehat{\sigma_{LB}})}{\max\{\widehat{\sigma_{LB}}, \widehat{\sigma_{UB}}\}}) - \Phi(-c) = 1-\alpha$ with the Newton-Raphson method. For more details, see Imbens and Manski (2004). Confidence intervals are obtained by bootstrap with 200 repetitions.

4.5 Results

4.5.1 Point-identification

We point-estimate the ATEs by: (1) Independence, (2) OLS, (3) PSM, (4) IPW+OLS, (5) NNM, (6) EBW and (7) RDD. For these approaches (2)-(7), we use the set of covariates used in Rahman and Pallikadavath (2018). Logistic regression is used to estimate the propensity score.[13] Table 4.6 shows the estimation results, and we observe that the JSY has positive impacts on all outcomes ($p < 0.01$). When we relax the independence assumption and instead impose the CIA, we find larger effects on institutional delivery, skilled birth attendance, PNC for mothers and PNC for babies, and smaller effects on ANC at least once, ANC at least three times, iron and folic acid supplement intakes and tetanus toxoid injections.

The results of the fuzzy RDD approach are shown in Table 4.6. They show the largest ATEs on all outcomes except PNC for mothers. As is well known, the RDD estimates the ATE among a sub-population that has been induced to participate in the treatment with the change in instrumental variables, and these people are often called 'compliers'. Hence, an ATE estimated by RDD can be different from ATEs estimated by other approaches, if compliers are very much different from the entire sample population (Imbens and Angrist, 1994). The jumps and kinks in the probability of participating in the programme, and the probability of using healthcare, are shown in Figure 4.2.

4.5.2 Partial identification

Table 4.7 presents the main result of this study, and it reports changes in the bound estimates for each outcome as we incrementally tighten the ATE bounds by adding stronger assumptions one by one. Herein, we consider the following four cases: (1) No assumption, (2) MTR, (3)

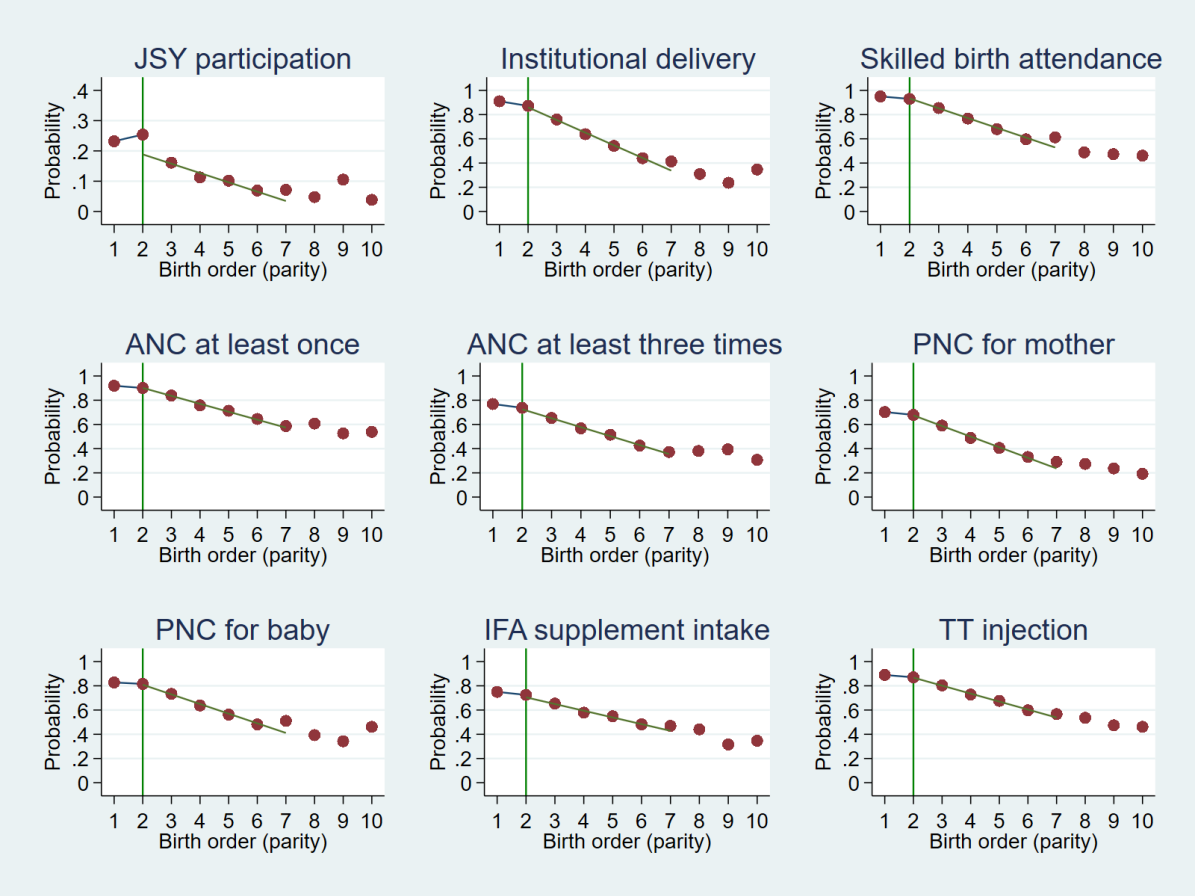
[13] The result for which is available from the author upon request.

Table 4.6: Point-estimated ATE

	Estimates	SEs	Confidence intervals
Institutional delivery			
Independence	0.126***	0.003	(0.12, 0.132)
OLS	0.129***	0.003	(0.123, 0.135)
PSM	0.129***	0.003	(0.123, 0.135)
IPW+OLS	0.132***	0.003	(0.126, 0.138)
NNM	0.122***	0.003	(0.116, 0.128)
EBW	0.131***	0.003	(0.125, 0.137)
RDD	0.19***	0.037	(0.117, 0.263)
Skilled delivery attendance			
Independence	0.081***	0.002	(0.077, 0.085)
OLS	0.088***	0.002	(0.084, 0.092)
PSM	0.088***	0.002	(0.084, 0.092)
IPW+OLS	0.09***	0.002	(0.086, 0.094)
NNM	0.082***	0.002	(0.078, 0.086)
EBW	0.094***	0.002	(0.09, 0.098)
RDD	0.134***	0.030	(0.075, 0.193)
Antenatal care +1			
Independence	0.091***	0.002	(0.087, 0.095)
OLS	0.079***	0.003	(0.073, 0.085)
PSM	0.078***	0.003	(0.072, 0.084)
IPW+OLS	0.08***	0.003	(0.074, 0.086)
NNM	0.072***	0.003	(0.066, 0.078)
EBW	0.079***	0.003	(0.073, 0.085)
RDD	0.139***	0.035	(0.07, 0.208)
Antenatal care +3			
Independence	0.108***	0.004	(0.1, 0.116)
OLS	0.082***	0.005	(0.072, 0.092)
PSM	0.07***	0.006	(0.058, 0.082)
IPW+OLS	0.079***	0.005	(0.069, 0.089)
NNM	0.075***	0.006	(0.063, 0.087)
EBW	0.091***	0.004	(0.083, 0.099)
RDD	0.129***	0.050	(0.031, 0.227)
Postnatal care for mother			
Independence	0.086***	0.004	(0.078, 0.094)
OLS	0.105***	0.005	(0.095, 0.115)
PSM	0.094***	0.006	(0.082, 0.106)
IPW+OLS	0.106***	0.005	(0.096, 0.116)
NNM	0.102***	0.006	(0.09, 0.114)
EBW	0.106***	0.004	(0.098, 0.114)
RDD	0.071	0.051	(-0.029, 0.171)
Postnatal care for baby			
Independence	0.054***	0.004	(0.046, 0.062)
OLS	0.076***	0.004	(0.068, 0.084)
PSM	0.068***	0.005	(0.058, 0.078)
IPW+OLS	0.077***	0.004	(0.069, 0.085)
NNM	0.073***	0.004	(0.065, 0.081)
EBW	0.071***	0.004	(0.063, 0.079)
RDD	0.113***	0.044	(0.027, 0.199)
Iron folic acid supplement intakes			
Independence	0.14***	0.004	(0.132, 0.148)
OLS	0.099***	0.005	(0.089, 0.109)
PSM	0.091***	0.006	(0.079, 0.103)
IPW+OLS	0.096***	0.005	(0.086, 0.106)
NNM	0.085***	0.006	(0.073, 0.097)
EBW	0.105***	0.004	(0.097, 0.113)
RDD	0.145***	0.051	(0.045, 0.245)
Tetanus toxoid injections			
Independence	0.099***	0.003	(0.093, 0.105)
OLS	0.088***	0.003	(0.082, 0.094)
PSM	0.088***	0.004	(0.08, 0.096)
IPW+OLS	0.088***	0.003	(0.082, 0.094)
NNM	0.078***	0.004	(0.07, 0.086)
EBW	0.09***	0.003	(0.084, 0.096)
RDD	0.17***	0.039	(0.094, 0.246)

Source: DLHS-4. Note: Number of observations is 67,595. The choice of covariates follows Rahman and Pallikadavath (2018). 95% confidence intervals are shown. PSM=Propensity score matching; IPW+OLS=OLS adjusted with propensity score inverse probability weighting; NNM=Nearest neighbour matching; and EBW=Entropy balance weighting; and RDD=Regression discontinuity design. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Figure 4.2: Discontinuities in the probabilities of treatment participation and healthcare use at parity more than two



Source: DLHS-4. Note: ANC=Antenatal care; PNC=Postnatal care; IFA=Iron and folic acid; and TT=tetanus toxoid.

MTR+MTS and (4) MTR+MTS+MIV. Table 4.7 also reports the confidence intervals for the respective ATE bound estimates. We observe that the widths of the confidence intervals are just slightly wider than those of the estimated ATE bounds. Compared with the widths of the bound estimates, the additional widths due to sampling variability are very small, which is thanks to the large sample sizes. This suggests that the identification problem is the dominant concern for inference in relation to the impacts, and sampling variance poses a much less serious problem for inference (Manski and Nagin, 1998).

Institutional delivery

First, for institutional delivery, in the worst-case scenario where we do not impose any assumption, the lower bound is -0.650 and the upper bound is 0.350. In the worst case, the bound width is always 1, and the bound always includes 0. This bound just ensures that the true ATE can never be outside of this bound as long as the ATE is estimated from these data. Although this

Table 4.7: ATE bounds

	Estimates		Confidence intervals	
	Lower bound	Upper bound	Lower bound	Upper bound
Institutional delivery				
No assumption	−0.652	0.348	−0.654	0.351
MTR	0.000	0.348	0.000	0.351
MTR+MTS	0.126	0.348	0.122	0.351
MTR+MTS+MIV	0.262	0.331	0.255	0.335
Skilled delivery attendance				
No assumption	−0.702	0.298	−0.705	0.301
MTR	0.000	0.298	0.000	0.301
MTR+MTS	0.081	0.298	0.078	0.301
MTR+MTS+MIV	0.176	0.294	0.170	0.297
Antenatal care +1				
No assumption	−0.685	0.315	−0.688	0.319
MTR	0.000	0.315	0.000	0.319
MTR+MTS	0.000	0.091	0.000	0.095
MTR+MTS+MIV	0.000	0.042	0.000	0.050
Antenatal care +3				
No assumption	−0.587	0.413	−0.590	0.417
MTR	0.000	0.413	0.000	0.417
MTR+MTS	0.000	0.108	0.000	0.115
MTR+MTS+MIV	0.000	0.014	0.000	0.029
Postnatal care for mother				
No assumption	−0.558	0.442	−0.561	0.445
MTR	0.000	0.442	0.000	0.445
MTR+MTS	0.086	0.442	0.078	0.445
MTR+MTS+MIV	0.196	0.430	0.187	0.434
Postnatal care for baby				
No assumption	−0.644	0.356	−0.647	0.358
MTR	0.000	0.356	0.000	0.358
MTR+MTS	0.054	0.356	0.048	0.358
MTR+MTS+MIV	0.158	0.349	0.150	0.352
Iron folic acid supplement intakes				
No assumption	−0.570	0.430	−0.573	0.433
MTR	0.000	0.430	0.000	0.433
MTR+MTS	0.000	0.140	0.000	0.146
MTR+MTS+MIV	0.000	0.065	0.000	0.081
Tetanus toxoid injections				
No assumption	−0.664	0.336	−0.668	0.339
MTR	0.000	0.336	0.000	0.339
MTR+MTS	0.000	0.099	0.000	0.104
MTR+MTS+MIV	0.000	0.046	0.000	0.056

Source: DLHS-4. Note: Number of observations is 67,595. Note: 95% confidence intervals are calculated following Imbens and Manski (2004) by bootstrap with 200 repetitions.

bound has a wider negative range, it does not necessarily mean that the ATE is more likely to be negative. Also, the centre of the bound (-0.15 in this case) is not necessarily the most probable ATE.

When we impose the MTR assumption, it truncates the lower bound of the ATE at 0, because the MTR assumes that the ATE is non-negative. We then add the MTS assumption to the MTR assumption. As the ATE estimated by OLS is larger than the ATE estimated by mean comparisons, we assume the negative MTS assumption here. Negative selection bias assumes that individual observable and unobservable characteristics are correlated with participation in the programme and institutional delivery in the opposite directions. One possible case would be that richer mothers who seek high-quality healthcare at private hospitals may have lower motivation to participate in the JSY. Imposing the MTS assumption, we find that the lower bound becomes even larger, from 0 to 0.126. Furthermore, adding the MIV assumption makes the ATE bound even narrower. The lower bound has become 0.262 and the upper bound has become 0.331.

After adding the MIV assumption, we find that all point-identified ATE estimates are outside of the ATE bound. ATEs on institutional delivery and estimated under the CIA are smaller than the lower ATE bound estimated under the MTR, MTS and MIV assumptions, thereby suggesting that their maintained assumptions are not compatible with the MTR, MTS and MIV assumptions used in the partial identification approach.[14] This incompatibility occurs because (1) the CIA is not valid, (2) functional-form assumptions are not correct and/or (3) the MTR, MTS and MIV assumptions are not valid. The finding that the non-parametric EBW estimate is outside of the bound implies that regardless of functional-form assumptions, the point-identified ATE is outside of the ATE bound.

Moreover, as discussed in the previous section, the identification assumptions used for the bound estimation are supported by empirical and observational evidence. Given that the CIA is hard to justify for the case of the JSY, it would be reasonable to think that the incompatibility between assumptions for the point-identification and those for the partial-identification mainly comes from the possibly unacceptable CIA and/or functional form specification errors.[15] Also,

[14] Strictly speaking, even without the MTR assumption, all the point-identified ATEs are found to be outside of the bound estimates (see Figure 4.4).

[15] We estimated the propensity score differently with the probit and linear probability model, but we still obtained similar results.

we found that the ATE estimated by the fuzzy RDD is also smaller than the lower bound of the ATE, which implies that the compliers are very much different from the other populations and/or the assumption of discontinuity is not valid.

Skilled birth attendance

For skilled birth attendance, in the worst case, the ATE bound runs from -0.702 to 0.298. The MTR assumption truncates the lower bound at 0. As the ATE estimated by OLS is larger than the ATE estimated by mean comparisons, we assume the negative MTS assumption. We speculate that most of the rich mothers who can afford to give birth in the presence of skilled birth attendants may not have been eligible for the JSY. Adding the negative MTS assumption raises the lower bound up to 0.081. Furthermore, adding the MIV assumption makes the band narrower. Under the three assumptions, the lower and upper bounds become 0.176 and 0.294, respectively.

All point-estimated ATEs are smaller than the lower limit of this ATE bound, which suggests that they could be under-estimated under the maintained assumptions. The confidence interval of the ATE bound under the MTR, MTS and MIV assumptions is between 0.170 and 0.297, which does not include the point-identified ATEs.

ANC use at least once

For ANC use at least once, in the worst case, the ATE bound is from -0.685 to 0.315. The MTR assumption makes the bound non-negative. As the ATE estimated by OLS is smaller than the ATE estimated by mean comparisons, we add the positive MTS assumption to the MTR, which thus implies that mothers with higher awareness of the importance of maternal healthcare may be more likely to participate in the JSY and receive ANC. Adding the MTS assumption substantially narrows the upper bound of ATE to 0.091, implying that the assumption regarding the direction of selection bias has strong identifying power. The MIV assumption further narrows the upper bound to 0.042.

We observe that ATEs under the independence assumption and the CIA lie beyond the upper limit of this ATE bound. We also observe that the ATE estimated by the RDD is outside of this bound, thereby implying that the compliers are very much different from the entire pop-

ulation and/or the assumption of discontinuity is not valid. We do not observe the overlapping of confidence intervals between point-estimated ATEs and the bound-estimated ATE.

ANC use at least three times

We also observe similar shrinkage in the ATE bound for ANC use three times and more. Its worst-case bound ranges from -0.587 to 0.413, and the MTR assumption truncates the lower bound at 0. As the ATE estimated by OLS is smaller than the ATE estimated by mean comparisons, we assume the positive MTS assumption, which narrows the upper bound from 0.413 to 0.108. The MIV assumption further narrows it up to 0.014. After adding the MIV assumption, the point-identified ATEs are again found beyond this upper bound. We do not observe the overlapping of confidence intervals between point-estimated ATEs and the bound-estimated ATE.

PNC for mothers

For PNC for mothers, the worst-case bound is -0.558 to 0.442, and the MTR assumption makes the lower bound non-negative. As the ATE estimated by OLS is larger than the ATE estimated by mean comparisons, we assume the negative MTS assumption, which suggests that those mothers who have greater difficulty in accessing healthcare may have been more strongly encouraged by the accredited social health activists to join the JSY programme and/or that mothers who can afford to receive PNC may not have been eligible for the JSY. We find that the negative MTS has strong identifying power and succeeds in increasing the lower bound up to 0.086. The MIV assumption contributes to further narrowing the bounds of the ATE.

Under the three assumptions, we observe that the ATE is no less than 0.193 and no larger than 0.430. Under the three assumptions, we observe that the point-identified ATEs are all smaller than the lower limit of the ATE bound, suggesting the possibility of under-estimating ATEs under the CIA (Figure 4.5). The ATE under the RDD assumption lies also outside the bound. We do not observe the overlapping of confidence intervals between point-estimated ATEs and the bound-estimated ATE.

PNC for babies

For PNC for babies, the worst-case bound runs from -0.644 to 0.356. Along with PNC for mothers, as the ATE estimated by OLS is larger than the ATE estimated by mean comparisons,

we assume the negative MTS assumption. The MTR and negative MTS assumptions raise the lower limit of the ATE bound up to 0.054, and the MIV assumption further narrows the lower bound to 0.158 and the upper bound to 0.349. We find that ATEs under the CIA and the RDD assumption lie below this lower bound, thereby suggesting that they could be under-estimated. Even when considering sampling variability, the confidence interval of the ATE bound does not include the point-estimated ATEs.

Iron and folic acid supplement intakes

For iron and folic acid supplement intakes, we find that in the worst case, the ATE ranges from -0.570 to 0.430. As the ATE estimated by OLS is smaller than the ATE estimated by mean comparisons, we impose the positive MTS assumption, implying that mothers who understand the importance of maternal healthcare are more likely to participate in the programme and receive the supplement. The MTR assumption makes the bound non-negative, and the positive MTS assumption substantially narrows the upper bound to 0.140. Adding the MIV assumption further narrows the upper bound up to 0.065. We find that all point-estimated ATEs are greater than the upper bound, thereby suggesting their over-estimation. Although the confidence interval of the ATE bound does not include the point-identified ATEs, we observe the overlapped confidence intervals of the ATEs point-estimated by PSM, NNM and RDD with that of the bound-estimated ATE.

Tetanus toxoid injections

Finally, for the tetanus toxoid injections, the worst-case bound is from -0.664 to 0.336, and the MTR assumption truncates the lower bound at 0. As for the case with iron and folic acid supplements, as the ATE estimated by OLS is smaller than the ATE estimated by mean comparisons, we assume the positive MTS assumption. We hypothesise that more risk-averse mothers are more likely to join the JSY and access treatment. We observe that the positive MTS assumption substantially contributes to narrowing the upper bound up to 0.099, and adding the MIV assumption contributes to narrowing it further up to 0.046. We find that the point-estimated ATEs are once again all outside of the bound, indicating the evidence of over-estimation. We do not observe the overlapping of confidence intervals between point-estimated ATEs and the bound-estimated ATE.

4.5.3 Result summary

Figure 4.3 visually summarises the results presented above. The bounds shown in Figure 4.3 are the sharp bounds under the MTR, MTS and MIV assumptions. We find that the point-identified ATEs are below the lower ATE bound of institutional delivery, skilled birth attendance and PNC for mothers and children. On the other hand, for ANC at least once and ANC at least three times, the point-identified ATEs are over the upper limit of the ATE bounds. In particular, a larger deviation from the lower bound is observed for institutional delivery. Even considering the uncertainty of sampling variability, we do not observe the overlapping of the confidence intervals between point-identified and partially-identified ATEs for institutional delivery, ANC at least once, PNC for mothers and tetanus toxoid injections.

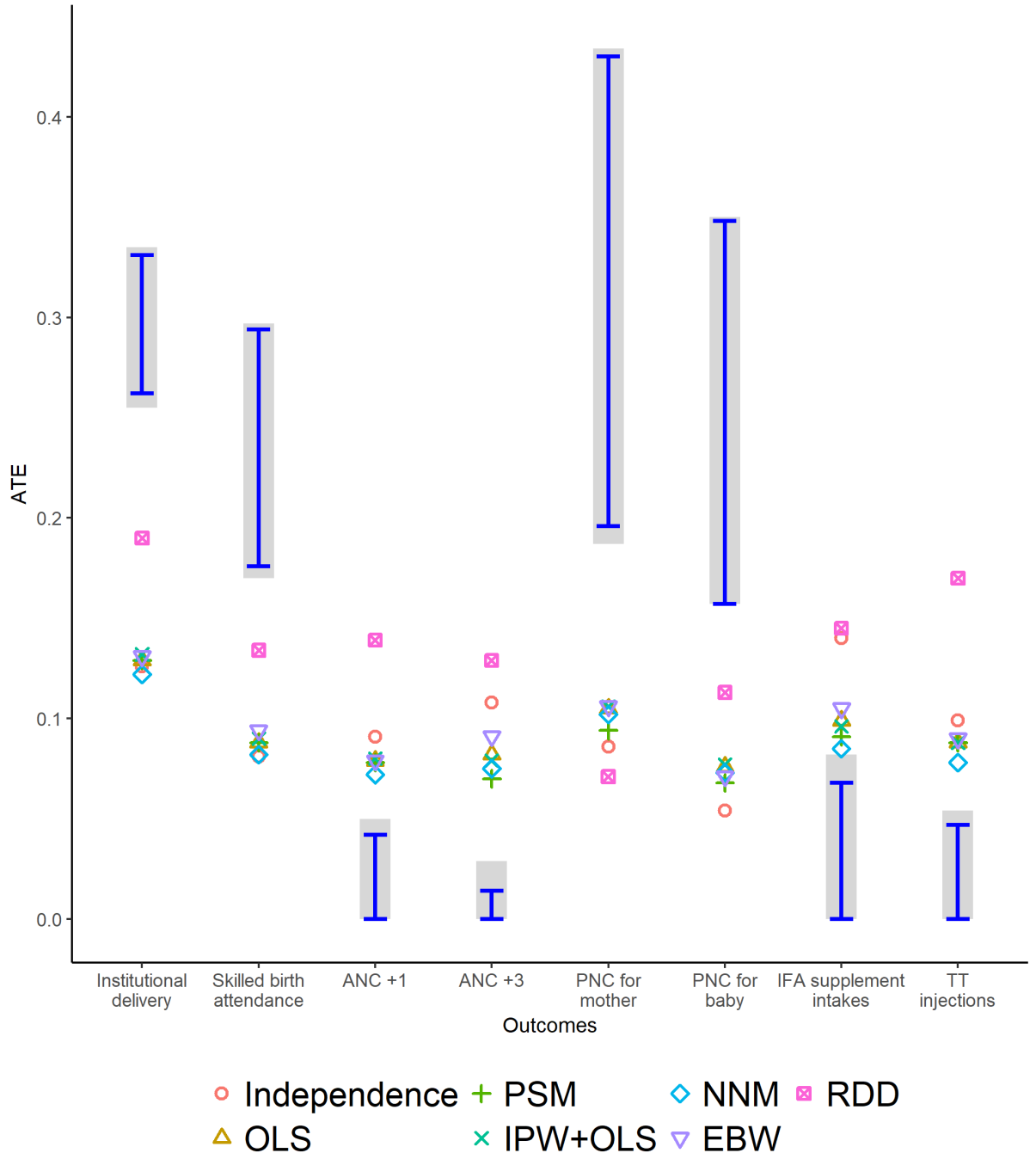
Next, we explore how each of the three assumptions contributes to estimating the ATE bounds for each outcome. Figure 4.4 and Figure 4.5 illustrate the sharp ATE bounds for each outcome estimated under the eight different combinations of assumptions. By comparing the bound widths, we can infer the identification power of each assumption. The joint imposition of the MTS and MIV assumptions leads to our main finding that point-estimated ATEs are outside the bound-estimated ATEs. Interestingly, even without the MTR assumption, we could have reached the same finding, although the estimated ATE bound can have a larger width. Another interesting finding is that the MTR assumption is not binding in the presence of the negative MTS assumption, because the negative MTS assumption itself made the lower limits of the ATE bounds positive. Nevertheless, the MTR assumption significantly contributes to narrowing the ATE bounds, unless the negative MTS assumption is imposed.

4.6 Robustness checks and further analysis

4.6.1 Robustness check: choice of covariates

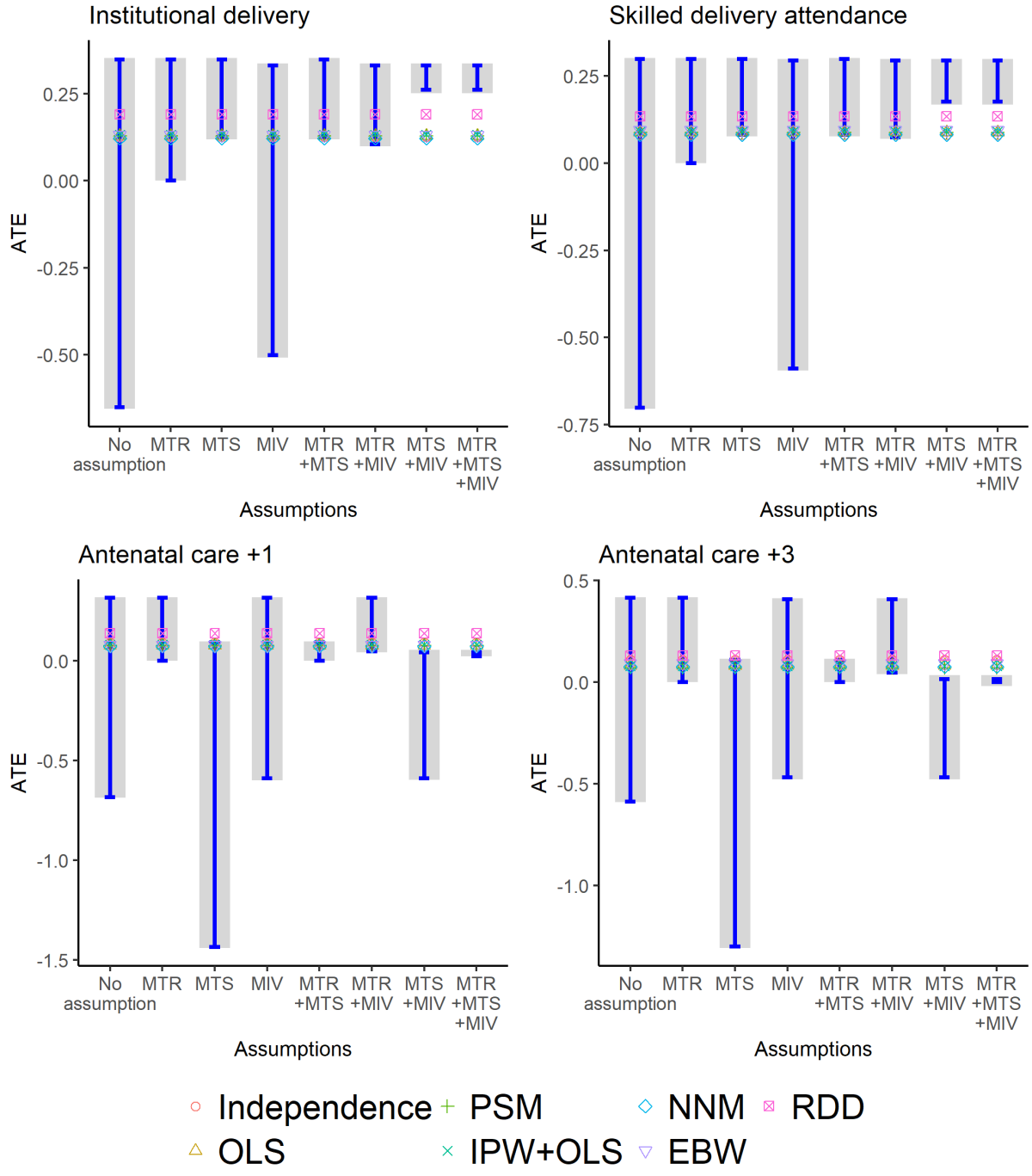
As robustness checks with respect to the choice of covariates, we point-estimate ATEs again with different covariate sets used in the previous studies and the full covariate sets, in order to see whether the choice of covariates substantially affects our findings. Note that as an ATE bound is unconditional on covariates, it is invariant across the covariate sets. First, we closely follow Lim et al. (2010) and use the following covariate sets: state of residence, urban residence, below-the-poverty-line card ownership, wealth quintile, scheduled caste, education, parity and maternal age.

Figure 4.3: Point-estimated ATE and bound-estimated ATE under the MTR, MTS and MIV assumptions



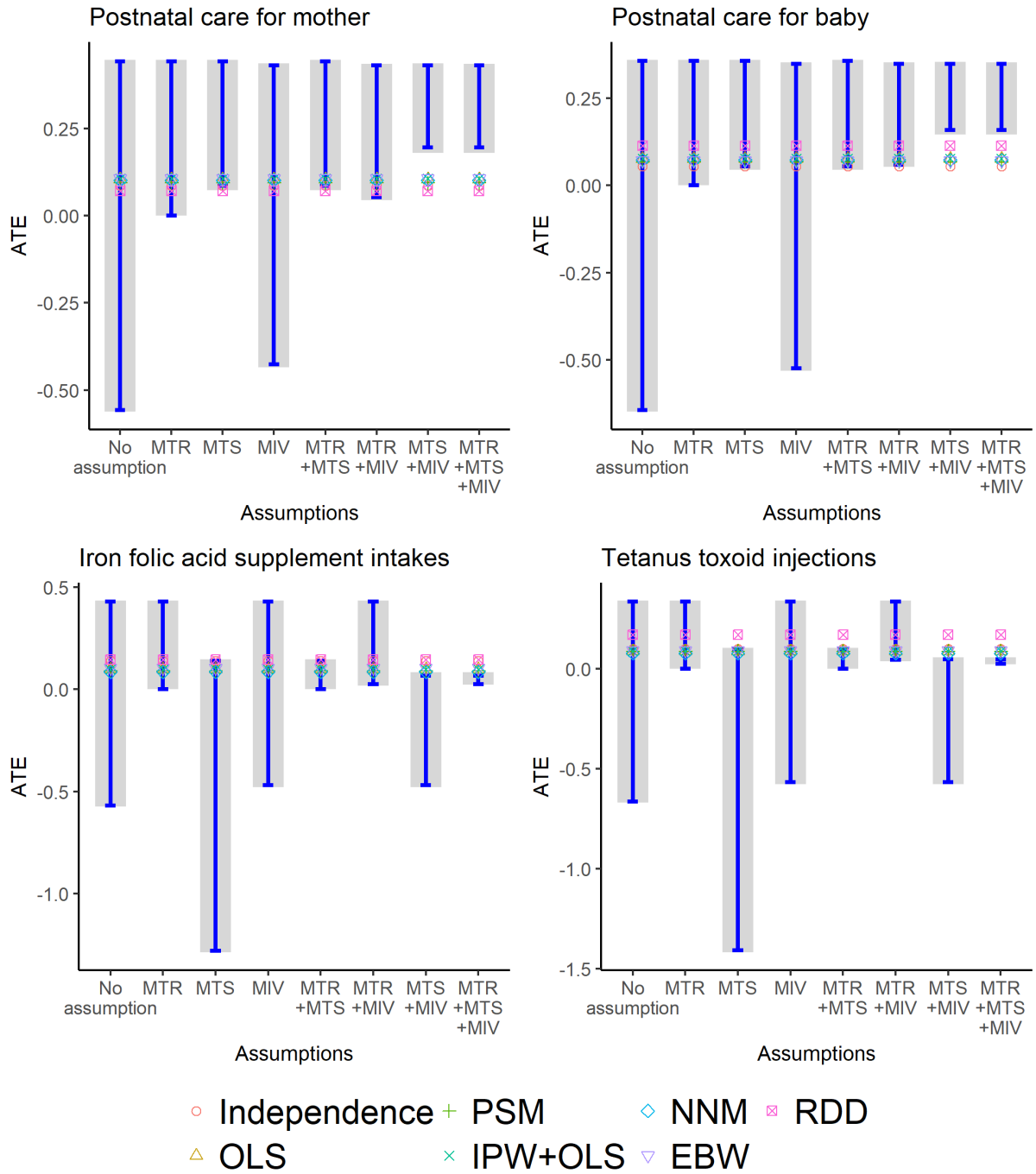
Source: DLHS-4. Note: The choice of covariates follows Rahman and Pallikadavath (2018). PSM=Propensity score matching; IPW+OLS=OLS adjusted with propensity score inverse probability weighting; NNM=Nearest neighbour matching; EBW=Entropy balance weighting; RDD=Regression discontinuity design; ANC=Antenatal care; PNC=Postnatal care; IFA=Iron and folic acid; and TT=Tetanus toxoid. The bounds shown here are sharp bounds estimated under the MTR, MTS and MIV assumptions. The shaded areas show the 95% level confidence interval of the ATE bound under the MTR, MTS and MIV assumptions.

Figure 4.4: ATE bounds for institutional delivery, skilled birth attendance and antenatal care



Source: DLHS-4. Note: The choice of covariates follows Rahman and Pallikadavath (2018). PSM=Propensity score matching; IPW+OLS=OLS adjusted with propensity score inverse probability weighting; NNM=Nearest neighbour matching; EBW=Entropy balance weighting; and RDD=Regression discontinuity design. The bounds shown are sharp bounds under the MTR, MTS and MIV assumptions. The shaded areas show the 95% level confidence interval of the ATE bound under the MTR, MTS and MIV assumptions.

Figure 4.5: ATE bounds for postnatal care, iron and folic acid supplement intakes and tetanus toxoid injections



Source: DLHS-4. Note: The choice of covariates follows Rahman and Pallikadavath (2018). PSM=Propensity score matching; IPW+OLS=OLS adjusted with propensity score inverse probability weighting; NNM=Nearest neighbour matching; EBW=Entropy balance weighting; and RDD=Regression discontinuity design. The bounds shown are sharp bounds under the MTR, MTS and MIV assumptions. The shaded areas show the 95% level confidence interval of the ATE bound under the MTR, MTS and MIV assumptions.

Next, we closely follow Sengupta and Sinha (2018), who used the following variables as determinants of JSY participation and healthcare use: residence in North-Western states, urban residence, below-the-poverty-line card ownership, number of live births, scheduled caste, scheduled tribe, whether pregnancy was known within three months, any previous stillbirth, any previous miscarriage, any child who died, age at which living with a husband, age at time of birth, ratio of male to female members in a household, being Hindu, wealth quintiles, living in a *kachcha* (made of natural materials) house and maternal and paternal education dummies.[16] As well as these, following Sengupta and Sinha (2018), we use the following variables that affect healthcare use only: sex of baby, number of brothers and sisters, whether any household member is covered by health insurance and having no toilet facility or water treatment for drinking.

Third, we control for more variables, to take account of the selection bias as best as we can manage. We control for the following covariates: birth-year fixed effects, birth-month fixed effects, state-fixed effects, urban residence, below-the-poverty-line card ownership, number of live births, scheduled caste, scheduled tribe, whether pregnancy was known within three months, any previous stillbirth, any previous miscarriage, any child who died, age at which living with a husband, age at time of birth, ratio of male to female members in a household, wealth quintiles, maternal and paternal education levels and being Hindu. This comprehensive set of covariates is expected to remove more effectively the selection bias attributable to measurable characteristics. If the point-estimated ATEs estimated with this full covariate set are outside of the ATE bounds, then it supports our finding that the CIA is implausible.

The results estimated with these three sets of covariates are shown in Table 4.8 and summarised visually in Figure 4.6. We find that all point-identified ATEs lie outside of ATE bounds in three sets of covariates. We reconfirm that the point-identified ATEs are likely to be over-estimated for ANC, iron and folic acid supplement intakes and tetanus toxoid injections. Also, we observe that the point-identified ATEs are likely to be under-estimated for institutional delivery, skilled birth attendance and PNC. Overall, these findings are consistent with our main results in Figure 4.3, implying that regardless of the choice of covariate set, the CIA and the functional form assumption are likely to be invalid.

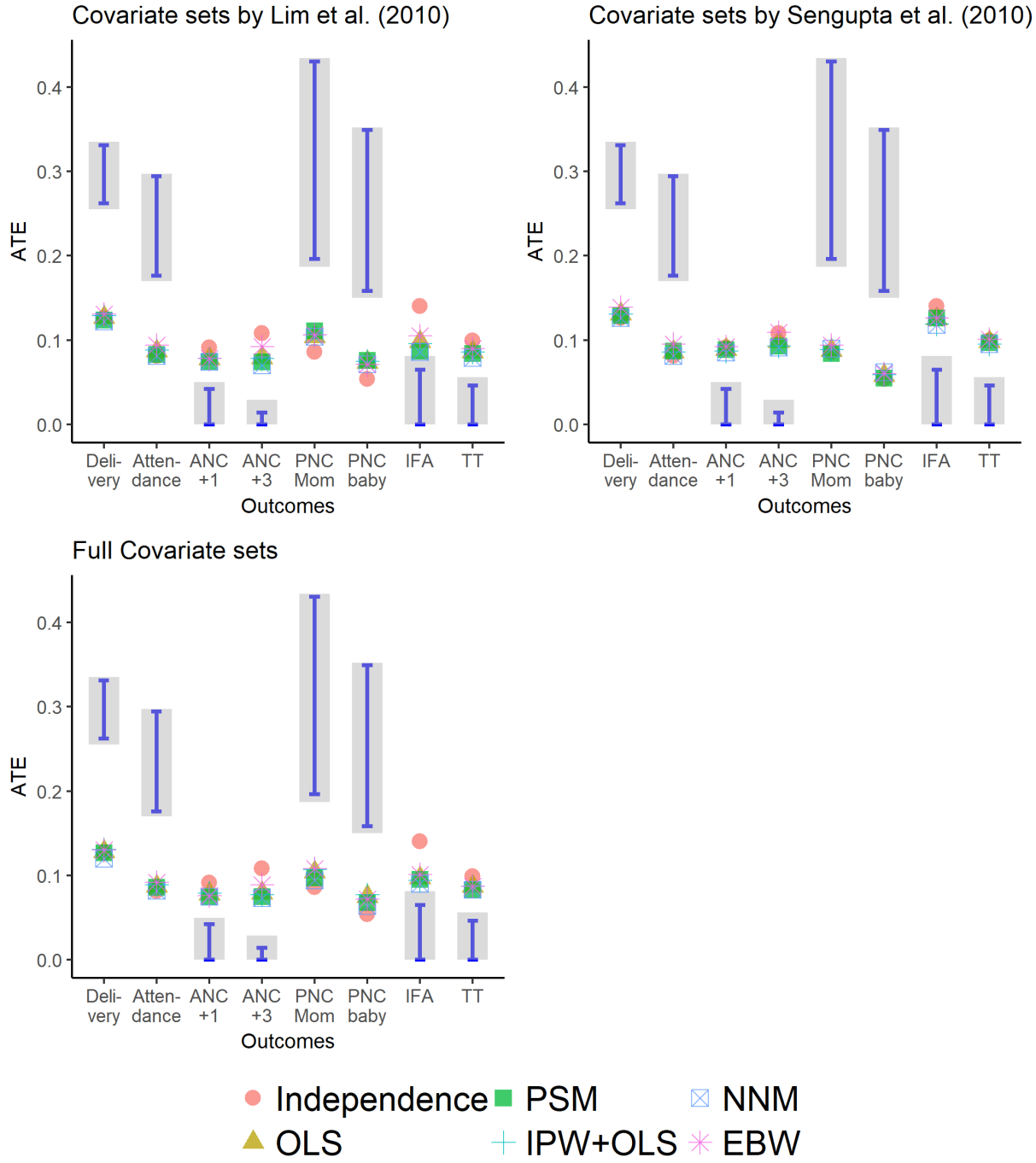
[16] We defined the following three educational levels for mothers and fathers, respectively: 1-6 years of education, 7-12 years of education and more than 12 years of education.

Table 4.8: Point-estimated ATE with different covariate sets

	Lim et al. (2010)		**Sengupta et al. (2018)**		**Full sets**	
	Estimates	SEs	Estimates	SEs	Estimates	SEs
Institutional delivery						
Independence	0.432***	0.005	0.432***	0.005	0.432***	0.005
OLS	0.127***	0.003	0.131***	0.002	0.128***	0.003
PSM	0.124***	0.003	0.129***	0.003	0.127***	0.003
IPW+OLS	0.129***	0.003	0.131***	0.003	0.131***	0.003
NNM	0.122***	0.003	0.126***	0.003	0.12***	0.003
EBW	0.131***	0.003	0.139***	0.003	0.13***	0.003
Skilled delivery attendance						
Independence	0.349***	0.005	0.349***	0.005	0.349***	0.005
OLS	0.087***	0.002	0.086***	0.002	0.087***	0.002
PSM	0.083***	0.002	0.087***	0.002	0.086***	0.002
IPW+OLS	0.088***	0.002	0.086***	0.002	0.089***	0.002
NNM	0.081***	0.002	0.081***	0.002	0.082***	0.002
EBW	0.094***	0.002	0.095***	0.002	0.092***	0.002
Antenatal care +1						
Independence	0.105***	0.005	0.105***	0.005	0.105***	0.005
OLS	0.078***	0.003	0.089***	0.002	0.078***	0.003
PSM	0.075***	0.004	0.089***	0.003	0.074***	0.004
IPW+OLS	0.078***	0.003	0.087***	0.003	0.079***	0.003
NNM	0.074***	0.004	0.085***	0.003	0.075***	0.004
EBW	0.078***	0.003	0.092***	0.002	0.076***	0.003
Antenatal care +3						
Independence	0.101***	0.007	0.101***	0.007	0.101***	0.007
OLS	0.079***	0.005	0.098***	0.004	0.079***	0.005
PSM	0.074***	0.006	0.093***	0.006	0.075***	0.006
IPW+OLS	0.078***	0.005	0.093***	0.004	0.077***	0.005
NNM	0.07***	0.006	0.091***	0.005	0.073***	0.005
EBW	0.092***	0.004	0.109***	0.004	0.089***	0.004
Postnatal care for mother						
Independence	0.178***	0.007	0.178***	0.007	0.178***	0.007
OLS	0.104***	0.005	0.088***	0.005	0.104***	0.005
PSM	0.111***	0.006	0.084***	0.006	0.097***	0.006
IPW+OLS	0.106***	0.005	0.089***	0.005	0.107***	0.005
NNM	0.104***	0.006	0.09***	0.006	0.094***	0.006
EBW	0.106***	0.004	0.094***	0.004	0.108***	0.004
Postnatal care for baby						
Independence	0.065***	0.007	0.065***	0.007	0.065***	0.007
OLS	0.074***	0.004	0.058***	0.004	0.075***	0.004
PSM	0.076***	0.005	0.055***	0.005	0.068***	0.005
IPW+OLS	0.075***	0.004	0.059***	0.004	0.077***	0.004
NNM	0.071***	0.004	0.062***	0.004	0.064***	0.004
EBW	0.071***	0.004	0.06***	0.004	0.072***	0.004
Iron folic acid supplement intakes						
Independence	0.104***	0.006	0.104***	0.006	0.104***	0.006
OLS	0.098***	0.005	0.125***	0.004	0.097***	0.005
PSM	0.087***	0.006	0.126***	0.005	0.095***	0.006
IPW+OLS	0.096***	0.005	0.119***	0.004	0.094***	0.005
NNM	0.086***	0.006	0.118***	0.005	0.09***	0.006
EBW	0.105***	0.004	0.126***	0.004	0.101***	0.004
Tetanus toxoid injections						
Independence	0.056***	0.004	0.056***	0.004	0.056***	0.004
OLS	0.086***	0.003	0.098***	0.003	0.087***	0.003
PSM	0.084***	0.004	0.097***	0.004	0.083***	0.004
IPW+OLS	0.086***	0.003	0.095***	0.003	0.087***	0.004
NNM	0.079***	0.004	0.095***	0.004	0.083***	0.004
EBW	0.09***	0.003	0.101***	0.003	0.087***	0.003

Source: DLHS-4. Note: 95% confidence intervals are shown. PSM=Propensity score matching; IPW+OLS=OLS adjusted with the propensity score inverse probability weighting; NNM=Nearest neighbour matching; and EBW=Entropy balance weighting. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Figure 4.6: ATE bound and point-estimated ATE with different covariate sets



Source: DLHS-4. Note: PSM=Propensity score matching; IPW+OLS=OLS adjusted with propensity score inverse probability weighting; NNM=Nearest neighbour matching; EBW=Entropy balance weighting; ANC=Antenatal care; PNC=Postnatal care; IFA=Iron and folic acid; and TT=tetanus toxoid. The bounds shown are sharp bounds estimated under the MTR, MTS and MIV assumptions. The shaded areas show the 95% level confidence interval of the ATE bound under the MTR, MTS and MIV assumptions.

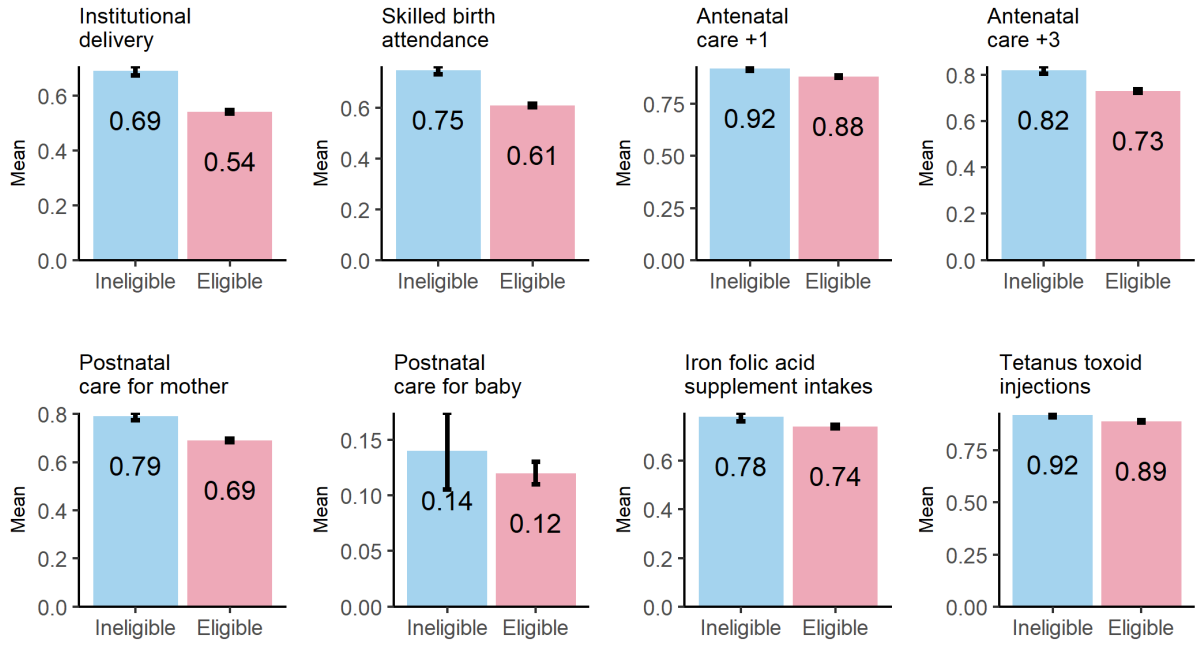
4.6.2 Robustness check: validity of the MIV assumption

Manski (2003) argues that the credibility of estimates decreases in line with the strength of the assumptions maintained (*The Law of Decreasing Credibility*). We observe that the MIV assumption played a key role in our finding that point-identified ATEs lie outside the bound-estimated ATEs, and so consequently it is important to examine the credibility of the MIV assumption. Although a large volume of literature reports that socio-economic status is negatively associated with maternal and child healthcare use in developing countries (Pathak et al., 2010; Pathak and Mohanty, 2010; Kesterton et al., 2010; Balarajan et al., 2011), we cannot directly test the validity of MIV assumption formalised in equations (4.27) and (4.28) in principle, because these are assumptions on latent probabilities. Nevertheless, we can still explore the validity of equation (4.27), using National Family Health Survey data, which were collected in 2005-2006. By focusing only on women who delivered before 12 April 2005, we can observe both $P(Y_0 = 1|v = 0)$ and $P(Y_0 = 1|v = 1)$ among women who gave birth before the introduction of the JSY. Figure 4.7 shows that $P(Y_0 = 1|v = 1)$ is larger than $P(Y_0 = 1|v = 0)$ for all outcomes ($p < 0.01$), indicating that in the absence of treatment, ineligible women are more likely to use healthcare services than eligible women. This supports the validity of equation (4.27) and provides confidence in our results.

4.7 Conclusion

In India, the CCT programme, i.e. the JSY, was introduced in 2005 and promotes the use of maternal and child healthcare with cash incentives, in order to reduce infant and neonatal mortalities. In contrast to other countries with CCT programmes, a rigorous RCT was not conducted in India for the JSY (Joshi and Sivaram, 2014), and hence a valid instrumental variable has not been available for researchers. Lim et al. (2010) conducted a first estimate of the causal impacts of the JSY, and this has attracted much attention for a number of years. To date, several studies have attempted to estimate its causal impacts on various healthcare services and health outcomes. However, according to Das et al. (2011), the JSY was immature until 2007, and the dataset analysed by Lim et al. (2010) and others (i.e. DLHS-3) was not reliable enough to estimate the causal impact. The latest wave of the DLHS (DLHS-4) has become available only recently, and Rahman and Pallikadavath (2017) and Rahman and Pallikadavath (2018) re-estimated the causal impacts, acknowledging the potential serious errors that had occurred in

Figure 4.7: Comparison in the mean outcomes between eligible and non-eligible mothers without treatment



Source: National Family Health Survey (2004-05). Note: 95% confidence intervals are shown. Mean outcomes are calculated among women in HPSs aged between 15 and 49 who delivered in the five years prior to the survey but before 12 April 2005. The left bars show $P(Y_0 = 1|v = 1)$, and the right bars show $P(Y_0 = 1|v = 0)$.

DLHS-3.

Up until now, however, no study has ever tried to assess the validity of the identification assumptions employed in previous studies. For example, Lim et al. (2010), Rahman and Pallikadavath (2018) and Sengupta and Sinha (2018) all relied on the CIA requiring that participation in the JSY programme could be regarded as random if we control for observable household and individual characteristics. However, the CIA is extremely hard to justify for the case of JSY. The utilisation of maternal and child healthcare use, and participation in the JSY, is likely to be dependent on individual unobservable characteristics such as the degree of being risk-averse and awareness of the importance of maternal and child healthcare. If there exist some selection mechanisms that cannot be controlled for by the observable characteristics, the estimated ATE under the CIA suffers selection bias, potentially resulting in flawed and conflicting conclusions (Manski, 2013). This study assessed the validity of the identification assumptions used in previous studies and provided new evidence through a partial identification approach. The partial identification provided herein yielded an honest and credible bound of ATE, which is valuable when we are not confident of the conventionally imposed identification assumptions.

If the imposed assumptions are valid, any point-estimate of ATE should lie within the ATE bound. However, we find that ATEs under the CIA lie beyond ATE bounds, suggesting the invalidity of identification assumptions and the possibility of over- or under-estimation. Especially, we find that the point-identified ATEs are below the lower ATE bounds of institutional delivery, skilled birth attendance and PNC for mothers and children. On the other hand, for ANC at least once and ANC at least three times, the point-identified ATEs are over the upper limits of the ATE bounds. For institutional delivery, the largest deviations of the point-estimated ATEs from the lower limit of its ATE bound is observed.[17] We find consistent results even when we use different covariate sets that have been used in two other previous studies.

Overall, this study provided sufficiently strong evidence that point-estimated ATEs could have been biased in previous studies. It re-estimated causal impacts through a partial identification approach and showed the conservative bounds of ATEs. Also, this study quantified how much at least the point-identified ATEs under questionable identification assumptions could be far from the true ATE. Certainly, the ATE bounds themselves do not give definite values for the causal impacts. We believe, however, that the honest estimation of the bounds based on the credible assumptions could be more useful and valuable in policy-making than definitive ATE estimates that heavily rely on unpalatable or untenable assumptions. We hope the results of this study contribute to evaluating the JSY thoroughly.

[17]However, it does not necessarily mean that the actual under-estimation size for the institutional delivery is the largest; it just means that its lower limit of potential under-estimation is the largest.

Appendix

4.A Appendix – Additional results

4.A.1 Additional evidence in LPSs with the National Family Health Survey (NFHS)

We complement the analysis by providing additional evidence from within LPSs with the latest National Family Health Survey (NFHS-4) conducted in 2015-2016. In contrast to the DLHS-4, the NFHS is nationally representative, which allows us to estimate the impacts in LPSs. The NFHS is a nationwide household survey that provides information on health, health-related behaviours and household socio-economic status, and it is the Indian version of the Demographic Health Survey conducted in more than 85 low- and middle-income countries (Corsi et al., 2012). The NFHS-4 has 19,906 female observations in LPSs.[18] After dropping observations with missing information[19], our final sample size was 13,121. Descriptive statistics for the NFHS-4 are shown in Table 4.A.1.

Point-identification in LPSs

In this subsection, we show the results for the LPSs obtained with the NFHS-4 data. In contrast to the DLHS-4, the NFHS-4 collected data from LPSs as well as HPSs (Figure 4.1). To make the results comparable as much as possible to those obtained from the DLHS-4 data, we use the same covariate sets used for the DLHS-4, i.e. the covariate set used in Rahman and Pallikadavath (2018).[20] The only difference in terms of covariates is the component of household wealth. In both datasets, wealth is created from the principal component analysis from indicators of asset ownership, housing characteristics and water and sanitation facilities, but their components are different. In the NLHS-4, we use wealth officially derived in collaboration with the World Bank, and it has been shown to be a consistent proxy for household income and expenditure (Rutstein and Staveteig, 2014; Montgomery et al., 2000).[21] Although in LPSs all women are eligible for the JSY regardless of their age, socio-economic status or number of children, we continue to use

[18] In the NFHS data, not all women in the samples were interviewed about their husbands' characteristics. In this study, we use the nationally representative female samples in which information about a woman's husband is also collected.

[19] We dropped 67 observations with missing information about the JSY status, and 68 observations that did not have information about paternal educational background.

[20] We also estimate the ATEs with the covariate sets in Lim et al. (2010) and Sengupta and Sinha (2018), and we obtain very similar results.

[21] The list of indicators can be downloaded from the official website (https://www.dhsprogram.com/programming/wealth%20index/India%20DHS%202015-16/IndiaNFHS4.pdf)–Accessed in 14/02/2019–.

Table 4.A.1: Descriptive statistics in LPSs of the NFHS-4

	Non-participants LPSs mean	Participants LPSs mean
Institutional delivery	0.80	1.00
Skilled birth attendance	0.82	0.99
Antenatal care at least once	0.86	0.95
Antenatal care (≥3 times)	0.76	0.87
Postnatal care for mother	0.72	0.87
Postnatal care for baby	0.35	0.48
Iron and folic acid (IFA) supplement	0.83	0.92
Tetanus toxoid (TT) injection	0.89	0.94
Below the poverty line card	0.31	0.38
Scheduled caste	0.16	0.24
Scheduled tribe	0.25	0.30
Rural	0.65	0.69
Birth order (parity)	2.18	1.90
Hindu	0.64	0.65
Maternal age	27.62	26.88
Maternal education years	3.55	3.72
Paternal education years	3.73	3.74
Wealth	3.38	2.03
Observations	10783	2338

Source: NFHS-4.

the same eligibility criteria that are applied in HPSs as a monotone instrument, which makes the results comparable with the previous results in HPSs.

Table 4.A.2 reports the ATEs estimated by: (1) Independence, (2) OLS, (3) PSM, (4) IPW+OLS, (5) NNM and (6) EBW. All point-estimated ATEs are positive and significant ($p < 0.01$). We find much larger ATEs in LPSs on institutional delivery and skilled birth attendance than ATEs in HPSs estimated from the DLHS-4 data, which could be attributed to the observation that in LPSs the initial outcome levels are far lower than those in HPSs. The lower initial level and wider coverage of the programme may have led to larger ATEs (Dongre, 2012).

Partial identification in LPSs

First, we explore the direction of the selection bias. Comparing the ATE under the independence assumption and the ATE estimated by OLS, we find that ATEs under the independence assumption are larger than those estimated by OLS for ANC at least once, PNC for babies, iron and folic acid supplement intakes and tetanus toxoid injections. Hence, for these outcomes, we

Table 4.A.2: Point-estimated ATE in LPSs with the NFHS-4

	Estimates	SEs	Confidence intervals
Institutional delivery			
Independence	0.432***	0.005	(0.422, 0.442)
OLS	0.433***	0.004	(0.425, 0.441)
PSM	0.435***	0.005	(0.425, 0.445)
IPW+OLS	0.433***	0.004	(0.425, 0.441)
NNM	0.428***	0.005	(0.418, 0.438)
EBW	0.429***	0.005	(0.419, 0.439)
Skilled delivery attendance			
Independence	0.349***	0.005	(0.339, 0.359)
OLS	0.35***	0.005	(0.34, 0.36)
PSM	0.35***	0.005	(0.34, 0.36)
IPW+OLS	0.35***	0.005	(0.34, 0.36)
NNM	0.346***	0.005	(0.336, 0.356)
EBW	0.345***	0.005	(0.335, 0.355)
Antenatal care +1			
Independence	0.105***	0.005	(0.095, 0.115)
OLS	0.105***	0.005	(0.095, 0.115)
PSM	0.104***	0.006	(0.092, 0.116)
IPW+OLS	0.105***	0.005	(0.095, 0.115)
NNM	0.097***	0.006	(0.085, 0.109)
EBW	0.095***	0.005	(0.085, 0.105)
Antenatal care +3			
Independence	0.101***	0.007	(0.087, 0.115)
OLS	0.104***	0.007	(0.09, 0.118)
PSM	0.105***	0.008	(0.089, 0.121)
IPW+OLS	0.104***	0.007	(0.09, 0.118)
NNM	0.093***	0.008	(0.077, 0.109)
EBW	0.099***	0.007	(0.085, 0.113)
Postnatal care for mother			
Independence	0.178***	0.007	(0.164, 0.192)
OLS	0.18***	0.007	(0.166, 0.194)
PSM	0.176***	0.008	(0.16, 0.192)
IPW+OLS	0.18***	0.007	(0.166, 0.194)
NNM	0.178***	0.008	(0.162, 0.194)
EBW	0.178***	0.007	(0.164, 0.192)
Postnatal care for baby			
Independence	0.065***	0.007	(0.051, 0.079)
OLS	0.061***	0.007	(0.047, 0.075)
PSM	0.055***	0.008	(0.039, 0.071)
IPW+OLS	0.06***	0.007	(0.046, 0.074)
NNM	0.058***	0.008	(0.042, 0.074)
EBW	0.064***	0.007	(0.05, 0.078)
Iron folic acid supplement intakes			
Independence	0.104***	0.006	(0.092, 0.116)
OLS	0.095***	0.006	(0.083, 0.107)
PSM	0.093***	0.007	(0.079, 0.107)
IPW+OLS	0.095***	0.006	(0.083, 0.107)
NNM	0.086***	0.007	(0.072, 0.1)
EBW	0.091***	0.006	(0.079, 0.103)
Tetanus toxoid injections			
Independence	0.056***	0.004	(0.048, 0.064)
OLS	0.052***	0.004	(0.044, 0.06)
PSM	0.052***	0.004	(0.044, 0.06)
IPW+OLS	0.052***	0.004	(0.044, 0.06)
NNM	0.05***	0.004	(0.042, 0.058)
EBW	0.047***	0.003	(0.041, 0.053)

Source: NFHS-4. Note: Number of observations is 13,121. The choice of covariates follows Rahman and Pallikadavath (2018). PSM=Propensity score matching; IPW+OLS=OLS adjusted with propensity score inverse probability weighting; NNM=Nearest neighbour matching; and EBW=Entropy balance weighting. *$p < 0.10$, **$p < 0.05$, ***$p < 0.01$.

impose the positive MTS assumption. For the other outcomes, namely institutional delivery, skilled birth attendance, ANC three times and more and PNC for mothers, the negative MTS assumption is imposed.

Table 4.A.3 shows the ATE bounds in LPSs estimated with the NFHS-4 data. Figure 4.A.1 visually summarises the bound-estimated ATEs and the point-estimated ATEs. Overall, we find that the point-identified ATEs are outside of the ATE bounds for institutional delivery, skilled birth attendance, ANC at least once, ANC three times and more and PNC for mothers. Compared with the results in HPSs, deviations in the point-identified ATEs from the upper/lower limits of ATE bounds are substantially smaller in LPSs, suggesting the lower limits of the potential selection bias in LPSs. For PNC for babies, iron and folic acid supplement intakes and tetanus toxoid injections, some of the ATEs estimated under the CIA are included within the ATE bounds. In particular, for the iron and folic acid supplement intakes, all the point-identified ATEs are within the bound, which is attributable to the weak identification power of the MIV assumption. The additional imposition of the MIV contributes little to tightening its ATE bound.

Overall, we find that most of the ATEs under the CIA are outside the ATE bounds. The exception is the effect on the iron and folic acid supplement intakes in which all the point-identified ATEs are inside the partially-identified ATE bound. Comparing the results in both HPSs and LPSs, we observe that deviations from the ATE bounds are larger in HPSs. The contrast between HPSs and LPSs suggests that we are more likely to suffer larger selection bias when the eligibility of the JSY is restricted to marginalised mothers.

4.B Appendix – Note on methodology

4.B.1 Entropy balance weighting

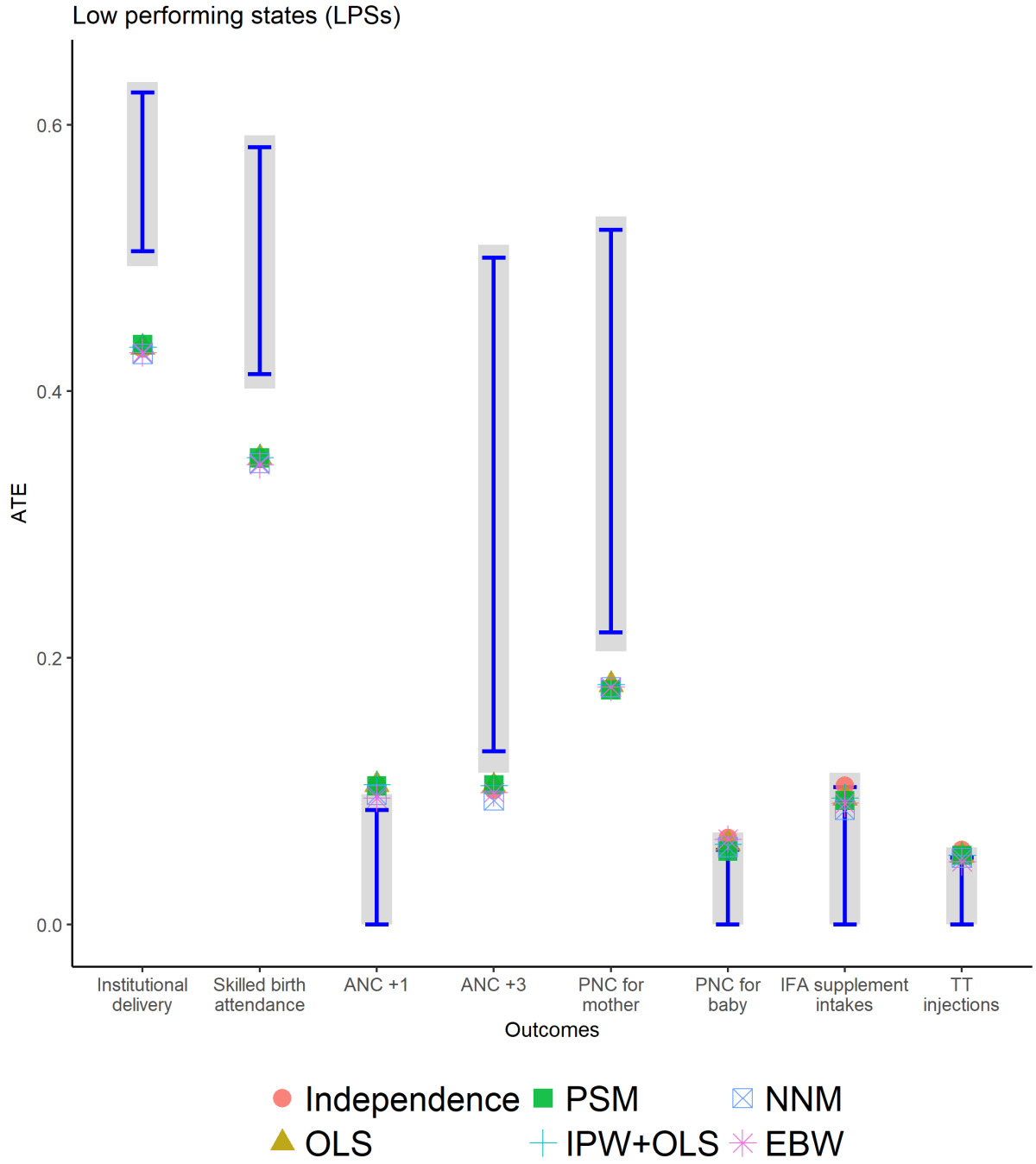
In the non-experimental study, wherein participation in treatment is not randomised, the pure comparison between treated people and untreated people does not generally provide the causal treatment effects because those who are treated can be fundamentally different from the untreated people with regard to their characteristics, such as the educational background. An entropy balance weighting, developed by Hainmueller (2012), perfectly balances the moments of the covariate distributions.

Table 4.A.3: ATE bounds in LPSs estimated with the NFHS-4

	Estimates		**Confidence intervals**	
	Lower bound	Upper bound	Lower bound	Upper bound
Institutional delivery				
No assumption	−0.319	0.681	−0.325	0.686
MTR	0.000	0.681	0.000	0.686
MTR+MTS	0.432	0.681	0.425	0.686
MTR+MTS+MIV	0.505	0.624	0.494	0.632
Skilled delivery attendance				
No assumption	−0.364	0.636	−0.369	0.642
MTR	0.000	0.636	0.000	0.642
MTR+MTS	0.349	0.636	0.341	0.642
MTR+MTS+MIV	0.413	0.583	0.402	0.592
Antenatal care +1				
No assumption	−0.487	0.513	−0.493	0.519
MTR	0.000	0.513	0.000	0.519
MTR+MTS	0.000	0.105	0.000	0.114
MTR+MTS+MIV	0.000	0.086	0.000	0.098
Antenatal care +3				
No assumption	−0.460	0.540	−0.466	0.547
MTR	0.000	0.540	0.000	0.547
MTR+MTS	0.101	0.540	0.089	0.547
MTR+MTS+MIV	0.130	0.500	0.114	0.510
Postnatal care for mother				
No assumption	−0.432	0.568	−0.437	0.574
MTR	0.000	0.568	0.000	0.574
MTR+MTS	0.178	0.568	0.167	0.574
MTR+MTS+MIV	0.219	0.521	0.205	0.531
Postnatal care for baby				
No assumption	−0.449	0.551	−0.455	0.557
MTR	0.000	0.551	0.000	0.557
MTR+MTS	0.000	0.065	0.000	0.075
MTR+MTS+MIV	0.000	0.056	0.000	0.069
Iron folic acid supplement intakes				
No assumption	−0.477	0.523	−0.483	0.529
MTR	0.000	0.523	0.000	0.529
MTR+MTS	0.000	0.104	0.000	0.114
MTR+MTS+MIV	0.000	0.103	0.000	0.114
Tetanus toxoid injections				
No assumption	−0.526	0.474	−0.532	0.480
MTR	0.000	0.474	0.000	0.480
MTR+MTS	0.000	0.056	0.000	0.061
MTR+MTS+MIV	0.000	0.050	0.000	0.058

Source: NFHS-4. Note: Number of observations is 13,121. 95% confidence intervals are calculated following Imbens and Manski (2004) by bootstrap with 200 repetitions.

Figure 4.A.1: Bound-estimated and point-estimated ATE in LPSs under the MTR, MTS and MIV assumptions with the NFHS-4



Source: NFHS-4. Note: The choice of covariates follows Rahman and Pallikadavath (2018). PSM=Propensity score matching; IPW+OLS=OLS adjusted with propensity score inverse probability weighting; NNM=Nearest neighbour matching; EBW=Entropy balance weighting; ANC=Antenatal care; PNC=Postnatal care; IFA=Iron and folic acid; and TT=tetanus toxoid. The bounds shown are sharp bounds estimated under the MTR, MTS and MIV assumptions. The shaded areas show the 95% level confidence interval of the ATE bound under the MTR, MTS and MIV assumptions.

The entropy balance weighting method assigns weights such that the covariate distributions in the weighted data satisfy a set of moment conditions set by a researcher. Specifically, weights are assigned to the control group so that the moments of the weighted covariate distributions in the control group match those in the treatment group. The entropy balance weighting method has a few advantages over the propensity score weighting. Firstly, the entropy balance weighting can "perfectly" balance a set of moments of the covariate distributions between two groups, while the propensity score methods often fail to jointly balance all the covariate distributions in practice partly because of the misspecification of the propensity score. In order to improve the balance of some covariates, the propensity score method usually needs to sacrifice the balance of the other covariates (Iacus et al., 2012). Thanks to the perfect balancing obtained with the entropy balancing method, manual balance checking is no longer necessary. Hence, we don't have to go back and forth between estimating the propensity score and manually checking the balance of the covariates (Hainmueller, 2012).

Each observation in the untreated group gets a weight satisfying a set of balance constraints set by the researcher a priori. Entropy balancing estimates the weights directly from a set of balance constraints and the normalisation and non-negativity constraints. The optimal weights for the untreated group are chosen in such a way that they minimise the following entropy distance metric:

$$H(w) = \sum_{\{i|Untreated\}} w_i ln(w_i/q_i), \tag{4.B.1}$$

subject to the following balance and normalisation constraints:

$$\sum_{\{i|Untreated\}} w_i c_{ri}(X_i) = m_r \; with \; r \in \{1, ..., R\}, \tag{4.B.2}$$

$$\sum_{\{i|Untreated\}} w_i = 1, \; and \tag{4.B.3}$$

$$w_i \geq 0, \; \forall i \in (Untreated). \tag{4.B.4}$$

Equation (4.B.2) is a set of balance constraints. Equations (4.B.3) and (4.B.4) are the normalisation constraint and the non-negativity constraint respectively. The part $w_i c_{ri}(X_i) = m_r$ in equation (4.B.2) describes a set of R balance constraints imposed on the moments of the weighted distributions of the covariates in the control group. Each balance constraint equates a certain order moment of the weighted covariate distributions in the control group to the corresponding moment of the covariate distributions in the treated group. As the entropy balancing can balance the higher order moments of the distributions, the moment constraints can be not only the

mean (first moment), but also the variance (second moment), the skewness (third moment), and beyond them. For example, if we are interested in estimating the weights for balancing the rth order moment of a specific covariate, say X_p, we set $c_{ri}(X_{ip}) = (X_{ip})^r$ or $c_{ri}(X_{ip}) = (X_{ip} - \mu_p)^r$ with mean μ_p. The choice of the moment order can vary across the covariates and largely depends on the researcher's a priori knowledge about their distribution types. For example, if the covariates are binary variables, the first order moment balancing is sufficient, whereas in the case of the variables that are normally distributed, balancing the first and second order moments is sufficient to balance their entire distributions. In this study, we will balance the covariate distributions up to their second moments.

4.B.2 MTS sharp bound and independence

This note shows why the upper bound of ATE under the positive MTS assumption corresponds to the ATE under the independence assumption. This note is based on the explanation made by McCarthy et al. (2015). Since the upper bound of ATE is, by definition, the difference between the upper bound of $P(Y_1 = 1)$ and the lower bound of $P(Y_0 = 1)$, we firstly discuss in what circumstance the upper bound of $P(Y_1 = 1)$ and the lower bound of $P(Y_0 = 1)$ are attained.

Applying the law of total probability to $P(Y_1 = 1)$,

$$\begin{aligned} P(Y_1 = 1) &= P(Y_1 = 1|D = 1)P(D = 1) + P(Y_1 = 1|D = 0)P(D = 0) \\ &= P(Y_1 = 1|D = 1)\{1 - P(D = 0)\} + P(Y_1 = 1|D = 0)P(D = 0) \\ &= \{P(Y_1 = 1|D = 0) - P(Y_1 = 1|D = 1)\}P(D = 0) + P(Y_1 = 1|D = 1). \quad (4.B.5) \end{aligned}$$

Under the positive MTS assumption, the term in the bracket is non-positive. The upper bound of $P(Y_1 = 1)$, which is $P(Y_1 = 1|D = 1)$, is achieved when the term in the bracket becomes 0, where the independence assumption, i.e. $P(Y_1 = 1|D = 0) = P(Y_1 = 1|D = 1)$, is satisfied.

Next, applying the law of total probability to $P(Y_0 = 1)$,

$$\begin{aligned} P(Y_0 = 1) &= P(Y_0 = 1|D = 1)P(D = 1) + P(Y_0 = 1|D = 0)P(D = 0) \\ &= P(Y_0 = 1|D = 1)P(D = 1) + P(Y_0 = 1|D = 0)\{1 - P(D = 1)\} \\ &= \{P(Y_0 = 1|D = 1) - P(Y_0 = 1|D = 0)\}P(D = 1) + P(Y_0 = 1|D = 0), \quad (4.B.6) \end{aligned}$$

Under the positive MIS assumption, the term in the bracket is non-negative. The lower bound of $P(Y_0 = 1)$, which is $P(Y_0 = 1|D = 0)$, is achieved when the term in the bracket becomes 0,

where the independence assumption, i.e. $P(Y_0 = 1|D = 1) = P(Y_0 = 1|D = 0)$, is satisfied.

Combined them together, the upper limit of ATE, which is $P(Y_1 = 1|D = 1) - P(Y_0 = 1|D = 0)$, is achieved when $P(Y_t = 1|D = 1) = P(Y_t = 1|D = 0) = P(Y_t = 1)$ holds for $t \in \{0, 1\}$. This condition is indeed the independence assumption. In the same way, we can show that the lower bound of ATE under the negative MTS assumption corresponds to the ATE estimated under the independence assumption.

4.B.3 Derivation of the MIV bounds in equations (4.29) and (4.30)

First, for $t \in \{0, 1\}$ and $t' \neq t$, the bound of the conditional probability, $P(Y_t = 1|v = u)$ is given from equations (4.9) and (4.10) by

$$\begin{aligned} & P(Y_t = 1|D = t, v = u)P(D = t|v = u) \\ \leq\ & \boldsymbol{P(Y_t = 1|v = u)} \\ \leq\ & \{P(Y_t = 1|D = t, v = u)P(D = t|v = u) + P(D = t'|v = u)\}. \end{aligned} \tag{4.B.7}$$

Under the MIV assumption, for $u_1 \leq u \leq u_2$,

$$\begin{aligned} & \max_{u_1 \leq u} P(Y_t = 1|D = t, v = u_1)P(D = t|v = u_1) \\ \leq\ & \boldsymbol{P(Y_t = 1|v = u)} \\ \leq\ & \min_{u \leq u_2} \{P(Y_t = 1|D = t, v = u_2)P(D = t|v = u_2) + P(D = t'|v = u_2)\}. \end{aligned} \tag{4.B.8}$$

Applying the law of total probability, we obtain the bounds of $P(Y_t = 1)$ as follows:

$$\begin{aligned} & \sum_{u \in V} P(v = u) \max_{u_1 \leq u} P(Y_t = 1|D = t, v = u_1)P(D = t|v = u_1) \\ \leq\ & \boldsymbol{P(Y_t = 1)} \\ \leq\ & \sum_{u \in V} P(v = u) \min_{u \leq u_2} \{P(Y_t = 1|D = t, v = u_2)P(D = t|v = u_2) + P(D = t'|v = u_2)\}. \end{aligned} \tag{4.B.9}$$

When an instrument is binary, we can simplify this. As u, u_0 and u_1 take the values of 0 or 1,

$$\begin{aligned}
& P(v=0)P(Y_t=1|D=t,v=0)P(D=t|v=0) \\
& +P(v=1)\max\{P(Y_t=1|D=t,v=0)P(D=t|v=0), P(Y_t=1|D=t,v=1)P(D=t|v=1)\} \\
\leq\ & \boldsymbol{P(Y_t=1)} \qquad (4.B.10) \\
\leq\ & P(v=0)\min\{P(Y_t=1|D=t,v=0)P(D=t|v=0)+P(D=t'|v=0), \\
& P(Y_t=1|D=t,v=1)P(D=t|v=1)+P(D=t'|v=1)\}+ \\
& P(v=1)[P(Y_t=1|D=t,v=1)P(D=t|v=1)+P(D=t'|v=1)].
\end{aligned}$$

This leads to equations (4.29) and (4.30).

4.B.4 Finite-sample bias in the presence of maxima and minima operators

First, for $t \in \{0,1\}$ and $t' \neq t$, we show the estimate of the lower bound of $P(Y_t=1|v=u)$ has an upward bias.

When the estimate of $P(.)$ is denoted by $\widehat{P}(.)$ from equation (4.B.8), the estimated lower bound is expressed as

$$\max_{u_1 \leq u} \widehat{P}(Y_t=1|D=t,v=u_1)\widehat{P}(D=t|v=u_1). \qquad (4.B.11)$$

Since a maximum operator is a convex function, by the Jensen's inequality,

$$\begin{aligned}
& E[\max_{u_1 \leq u} \widehat{P}(Y_t=1|D=t,v=u_1)\widehat{P}(D=t|v=u_1)] \\
\geq\ & \max_{u_1 \leq u} E[\widehat{P}(Y_t=1|D=t,v=u_1)\widehat{P}(D=t|v=u_1)] \\
=\ & \max_{u_1 \leq u} \widehat{P}(Y_t=1|D=t,v=u_1)\widehat{P}(D=t|v=u_1). \qquad (4.B.12)
\end{aligned}$$

Equality in the last holds due to the unbiasedness of $\widehat{P}(.)$. Hence equation (4.B.12) shows that the estimate of the lower bound can have an upward bias.

Next, we show the estimate of the upper bound of $P(Y_t=1|v=u)$ has a downward bias. From equation (4.B.8), the estimated upper bound is expressed as

$$\min_{u \leq u_0} \{\widehat{P}(Y_t=1|D=t,v=u_0)\widehat{P}(D=t|v=u_0)+\widehat{P}(D=t'|v=u_0)\}. \qquad (4.B.13)$$

Since a maximum operator is a concave function, by the Jensen's inequality,

$$\begin{aligned} & E[\min_{u\leq u_0}\{\widehat{P}(Y_t=1|D=t,v=u_0)\widehat{P}(D=t|v=u_0)+\widehat{P}(D=t'|v=u_0)\}] \\ \leq & \min_{u\leq u_0} E[\{\widehat{P}(Y_t=1|D=t,v=u_0)\widehat{P}(D=t|v=u_0)+\widehat{P}(D=t'|v=u_0)\}] \\ = & \min_{u\leq u_0}\{P(Y_t=1|D=t,v=u_0)P(D=t|v=u_0)+P(D=t'|v=u_0)\}. \end{aligned} \tag{4.B.14}$$

Equation (4.B.14) shows that the estimate of the upper bound can have a downward bias.

4.C Appendix – Derivations of ATE bounds under joint assumptions

4.C.1 MTR+MTS

We consider the case in which the MTR and MTS assumptions are jointly imposed. Imposing the MTR assumption and the MTS assumption jointly means that we are assuming the intersection of the bounds of $P(Y_1=1)$ and $P(Y_0=1)$ that are derived under each assumption.

(i) MTR+ Positive MTS: If we combine the MTR assumption and the positive MTS assumption, the bounds of $P(Y_1=1)$ and $P(Y_0=1)$ become

$$\underbrace{P(Y=1)}_{MTR}\leq \quad \boldsymbol{P(Y_1=1)} \quad \leq \underbrace{P(Y_1=1|D=1)}_{Positive\ MTS} \tag{4.C.1}$$

$$\underbrace{P(Y_0=1|D=0)}_{Positive\ MTS}\leq \quad \boldsymbol{P(Y_0=1)} \quad \leq \underbrace{P(Y=1)}_{MTR}, \tag{4.C.2}$$

where for $t\in\{0,1\}$ the bound of $P(Y_t=1)$ is an intersection of the corresponding bounds under the MTR assumption and the positive MTS assumption. The new bound of ATE is given by

$$0\leq ATE\leq P(Y_1=1|D=1)-P(Y_0=1|D=0). \tag{4.C.3}$$

The upper bound of ATE is given by the upper bound under the positive MTS assumption, while the lower bound is 0 thanks to the MTR assumption.

(ii) MTR+ Negative MTS: The combination of the MTR assumption and the negative MTS assumption can be obtained in the same fashion.

$$\max\{\underbrace{P(Y=1)}_{MTR},\underbrace{P(Y_1=1|D=1)}_{Negative\ MTS}\}\leq \quad \boldsymbol{P(Y_1=1)} \quad \leq \underbrace{P(Y_1=1|D=1)P(D=1)+P(D=0)}_{Worst\ case} \tag{4.C.4}$$

$$\underbrace{P(Y_0=1|D=0)P(D=0)}_{Worst\ case}\leq \quad \boldsymbol{P(Y_0=1)} \quad \leq \min\{\underbrace{P(Y=1)}_{MTR},\underbrace{P(Y_0=1|D=0)}_{Negative\ MTS}\} \tag{4.C.5}$$

and the ATE is given by

$$\begin{aligned} & \max\{P(Y=1), P(Y_1=1|D=1)\} - \min\{P(Y=1), P(Y_0=1|D=0)\} \\ \leq \; & ATE \\ \leq \; & \{P(Y_1=1|D=1)P(D=1) + P(D=0)\} - P(Y_0=1|D=0)P(D=0), \end{aligned} \tag{4.C.6}$$

where the upper bound corresponds to that in the worst-case.

4.C.2 MTR+MTS+MIV

We estimate the bounds under the MTR, MTS and MIV assumptions. When we impose the MIV assumption, the sharp bounds of $P(Y_1=1)$ and $P(Y_0=1)$ cannot be expressed simply by the intersection of the bounds estimated under each of assumption. We firstly derive the bounds for conditional probabilities $P(Y_1=1|v=u)$ and $P(Y_0=1|v=u)$ under the three assumptions. This is followed by the derivations of the bounds of $P(Y_1=1)$ and $P(Y_0=1)$.

(i) MTR+ positive MTS+ MIV: Under the MTR and positive MTS assumptions, the bounds of the conditional probabilities $P(Y_1=1|v=u)$ and $P(Y_0=1|v=u)$ are give by

$$\underbrace{P(Y=1|v=u)}_{MTR} \leq \; \boldsymbol{P(Y_1=1|v=u)} \; \leq \underbrace{P(Y_1=1|D=1,v=u)}_{Positive\ MTS} \tag{4.C.7}$$

$$\underbrace{P(Y_0=1|D=0,v=u)}_{Positive\ MTS} \leq \; \boldsymbol{P(Y_0=1|v=u)} \; \leq \underbrace{P(Y=1|v=u)}_{MTR}. \tag{4.C.8}$$

Under the MIV assumption, for $u_1 \leq u \leq u_2$,

$$\max_{u_1 \leq u} P(Y=1|v=u_1) \leq \; \boldsymbol{P(Y_1=1|v=u)} \; \leq \min_{u \leq u_2} P(Y_1=1|D=1,v=u_2) \tag{4.C.9}$$

$$\max_{u_1 \leq u} P(Y_0=1|D=0,v=u_1) \leq \; \boldsymbol{P(Y_0=1|v=u)} \; \leq \min_{u \leq u_2} P(Y=1|v=u_2). \tag{4.C.10}$$

Applying the law of total probability, we obtain the bounds of $P(Y_1=1)$ and $P(Y_0=1)$.

$$\begin{aligned} & \sum_{u \in V} P(v=u) \max_{u_1 \leq u} P(Y=1|v=u_1) \\ \leq \; & \boldsymbol{P(Y_1=1)} \\ \leq \; & \sum_{u \in V} P(v=u) \min_{u \leq u_2} P(Y_1=1|D=1,v=u_2) \end{aligned} \tag{4.C.11}$$

and

$$\begin{aligned} & \sum_{u\in V} P(v=u) \max_{u_1 \le u} P(Y_0=1|D=0,v=u_1) \\ \le\ & \boldsymbol{P(Y_0=1)} \\ \le\ & \sum_{u\in V} P(v=u) \min_{u \le u_2} P(Y=1|v=u_2). \end{aligned} \tag{4.C.12}$$

When an instrument is binary, we can simplify them. As u, u_0 and u_1 take the values of 0 or 1, arranging these equations lead to equations (4.C.13) and (4.C.14).

$$\begin{aligned} & P(v=0)P(Y=1|v=0) + P(v=1)\max\{P(Y=1|v=0), P(Y=1|v=1)\} \\ \le\ & \boldsymbol{P(Y_1=1)} \\ \le\ & P(v=0)\min\{P(Y_1=1|D=1,v=0), P(Y_1=1|D=1,v=1)\} + P(v=1)P(Y_1=1|D=1,v=1) \end{aligned} \tag{4.C.13}$$

and

$$\begin{aligned} & P(v=0)P(Y_0=1|D=0,v=0) + P(v=1)\max\{P(Y_0=1|D=0,v=0), P(Y_0=1|D=0,v=1)\} \\ \le\ & \boldsymbol{P(Y_0=1)} \\ \le\ & P(v=0)\min\{P(Y=1|v=0), P(Y=1|v=1)\} + P(v=1)P(Y=1|v=1). \end{aligned} \tag{4.C.14}$$

The sharp bound of the ATE can be obtained via equation (4.8).

(i) MTR+ negative MTS+ MIV: Under the MTR, negative MTS and MIV assumptions, the bounds of the conditional probabilities $P(Y_1=1|v=u)$ and $P(Y_0=1|v=u)$ are

$$\begin{aligned} & \max\{\underbrace{P(Y=1|v=u)}_{MTR}, \underbrace{P(Y_1=1|D=1,v=u)}_{Negative\ MTS}\} \\ \le\ & \boldsymbol{P(Y_1=1|v=u)} \\ \le\ & P(Y_1=1|D=1,v=u)P(D=1|v=u) + P(D=0|v=u) \end{aligned} \tag{4.C.15}$$

and

$$\begin{aligned} & P(Y_0=1|D=0,v=u)P(D=0|v=u) \\ \le\ & \boldsymbol{P(Y_0=1|v=u)} \\ \le\ & \min\{\underbrace{P(Y=1|v=u)}_{MTR}, \underbrace{P(Y_0=1|D=0,v=u)}_{Negative\ MTS}\}. \end{aligned} \tag{4.C.16}$$

Under the MIV assumption, for $u_1 \leq u \leq u_2$,

$$\begin{aligned} & \max_{u_1 \leq u} \max\{P(Y=1|v=u_1), P(Y_1=1|D=1,v=u_1)\} \\ \leq \; & \boldsymbol{P(Y_1=1|v=u)} \\ \leq \; & \min_{u \leq u_2} [P(Y_1=1|D=1,v=u_2)P(D=1|v=u_2) + P(D=0|v=u_2)] \end{aligned} \tag{4.C.17}$$

and

$$\begin{aligned} & \max_{u_1 \leq u} P(Y_0=1|D=0,v=u_1)P(D=0|v=u_1) \\ \leq \; & \boldsymbol{P(Y_0=1|v=u)} \\ \leq \; & \min_{u \leq u_2} \min\{P(Y=1|v=u_2), P(Y_0=1|D=0,v=u_2)\}. \end{aligned} \tag{4.C.18}$$

Applying the law of total probability,

$$\begin{aligned} & \sum_{u \in V} P(v=u) \max_{u_1 \leq u} \max\{P(Y=1|v=u_1), P(Y_1=1|D=1,v=u_1)\} \\ \leq \; & \boldsymbol{P(Y_1=1)} \\ \leq \; & \sum_{u \in V} P(v=u) \min_{u \leq u_2} \{P(Y_1=1|D=1,v=u_2)P(D=1|v=u_2) + P(D=0|v=u_2)\} \end{aligned} \tag{4.C.19}$$

and

$$\begin{aligned} & \sum_{u \in V} P(v=u) \max_{u_1 \leq u} P(Y_0=1|D=0,v=u_1)P(D=0|v=u_1) \\ \leq \; & \boldsymbol{P(Y_0=1)} \\ \leq \; & \sum_{u \in V} P(v=u) \min_{u \leq u_2} \min\{P(Y=1|v=u_2), P(Y_0=1|D=0,v=u_2)\}. \end{aligned} \tag{4.C.20}$$

As u, u_1 and u_2 take the values of 0 or 1, arranging them leads to equations (4.C.21) and (4.C.22).

$$\begin{aligned} & P(v=0)\max\{P(Y=1|v=0), P(Y_1=1|D=1,v=0)\} + \\ & P(v=1)\max\{P(Y=1|v=0), P(Y_1=1|D=1,v=0), P(Y=1|v=1), P(Y_1=1|D=1,v=1)\} \\ \leq \; & \boldsymbol{P(Y_1=1)} \\ \leq \; & P(v=0)\min\{P(Y_1=1|D=1,v=0)P(D=1|v=0) + P(D=0|v=0), \\ & P(Y_1=1|D=1,v=1)P(D=1|v=1) + P(D=0|v=1)\} \\ & + P(v=1)\{P(Y_1=1|D=1,v=1)P(D=1|v=1) + P(D=0|v=1)\} \end{aligned} \tag{4.C.21}$$

and

$$
\begin{aligned}
& P(v=0)P(Y_0=1|D=0,v=0)P(D=0|v=0) \\
& +P(v=1)\max\{P(Y_0=1|D=0,v=0)P(D=0|v=0), P(Y_0=1|D=0,v=1)P(D=0|v=1)\} \\
\leq\ & \boldsymbol{P(Y_0=1)} \qquad (4.C.22) \\
\leq\ & P(v=0)\min\{P(Y=1|v=0), P(Y_0=1|D=0,v=0), P(Y=1|v=1), P(Y_0=1|D=0,v=1)\} \\
& +P(v=1)\min\{P(Y=1|v=1), P(Y_0=1|D=0,v=1)\}.
\end{aligned}
$$

The sharp bound of the ATE can be obtained via equation (4.8).

4.D Appendix – Derivations of ATE under the other combinations of assumptions

4.D.1 MTS+MIV

(i) Positive MTS+MIV: Under the positive MTS assumption, the bounds of the conditional probabilities $P(Y_1=1|v=u)$ and $P(Y_0=1|v=u)$ are given from equations (4.19) and (4.20) by

$$
\begin{aligned}
& P(Y_1=1|D=1,v=u)P(D=1|v=u) \\
\leq\ & \boldsymbol{P(Y_1=1|v=u)} \qquad (4.D.1) \\
\leq\ & P(Y_1=1|D=1,v=u)
\end{aligned}
$$

and

$$
\begin{aligned}
& P(Y_0=1|D=0,v=u) \\
\leq\ & \boldsymbol{P(Y_0=1|v=u)} \qquad (4.D.2) \\
\leq\ & P(Y_0=1|D=0,v=u)P(D=0|v=u)+P(D=1|v=u).
\end{aligned}
$$

Under the MIV assumption, for $u_1 \leq u \leq u_2$,

$$
\begin{aligned}
& \max_{u_1\leq u} P(Y_1=1|D=1,v=u_1)P(D=1|v=u_1) \\
\leq\ & \boldsymbol{P(Y_1=1|v=u)} \qquad (4.D.3) \\
\leq\ & \min_{u\leq u_2} P(Y_1=1|D=1,v=u_2)
\end{aligned}
$$

and

$$\begin{aligned} & \max_{u_1 \leq u} P(Y_0 = 1|D = 0, v = u_1) \\ \leq \; & \boldsymbol{P(Y_0 = 1|v = u)} \\ \leq \; & \min_{u \leq u_2} \{P(Y_0 = 1|D = 0, v = u_2)P(D = 0|v = u_2) + P(D = 1|v = u_2)\}. \end{aligned} \tag{4.D.4}$$

Applying the law of total probability, we obtain the bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ as follows:

$$\begin{aligned} & \sum_{u \in V} P(v = u) \max_{u_1 \leq u} P(Y_1 = 1|D = 1, v = u_1)P(D = 1|v = u_1) \\ \leq \; & \boldsymbol{P(Y_1 = 1)} \\ \leq \; & \sum_{u \in V} P(v = u) \min_{u \leq u_2} P(Y_1 = 1|D = 1, v = u_2) \end{aligned} \tag{4.D.5}$$

and

$$\begin{aligned} & \sum_{u \in V} P(v = u) \max_{u_1 \leq u} P(Y_0 = 1|D = 0, v = u_1) \\ \leq \; & \boldsymbol{P(Y_0 = 1)} \\ \leq \; & \sum_{u \in V} P(v = u) \min_{u \leq u_2} \{P(Y_0 = 1|D = 0, v = u_2)P(D = 0|v = u_2) + P(D = 1|v = u_2)\}. \end{aligned} \tag{4.D.6}$$

When an instrument is binary, we can simplify them. As u, u_0 and u_1 take the values of 0 or 1,

$$\begin{aligned} & P(v = 0)P(Y_1 = 1|D = 1, v = 0)P(D = 1|v = 0) + \\ & P(v = 1) \max\{P(Y_1 = 1|D = 1, v = 0)P(D = 1|v = 0), P(Y_1 = 1|D = 1, v = 1)P(D = 1|v = 1)\} \\ \leq \; & \boldsymbol{P(Y_1 = 1)} \\ \leq \; & P(v = 0) \min\{P(Y_1 = 1|D = 1, v = 0), P(Y_1 = 1|D = 1, v = 1)\} + \\ & P(v = 1)P(Y_1 = 1|D = 1, v = 1) \end{aligned} \tag{4.D.7}$$

and

$$\begin{aligned} & P(v = 0)P(Y_0 = 1|D = 0, v = 0) + \\ & P(v = 1) \max\{P(Y_0 = 1|D = 0, v = 0), P(Y_0 = 1|D = 0, v = 1)\} \\ \leq \; & \boldsymbol{P(Y_0 = 1)} \\ \leq \; & P(v = 0) \min\{P(Y_0 = 1|D = 0, v = 0)P(D = 0|v = 0) + P(D = 1|v = 0), \\ & P(Y_0 = 1|D = 0, v = 1)P(D = 0|v = 1) + P(D = 1|v = 1)\} + \\ & P(v = 1)\{P(Y_0 = 1|D = 0, v = 1)P(D = 0|v = 1) + P(D = 1|v = 1)\}. \end{aligned} \tag{4.D.8}$$

The sharp bound of the ATE can be obtained via equation (4.8).

(ii) Negative MTS+MIV: Under the negative MTS assumption, the bounds of the conditional probabilities $P(Y_1 = 1|v = u)$ and $P(Y_0 = 1|v = u)$ are given from equations (4.24) and (4.25) by

$$\begin{aligned} & P(Y_1 = 1|D = 1, v = u) \\ \leq\ & \boldsymbol{P(Y_1 = 1|v = u)} \\ \leq\ & P(Y_1 = 1|D = 1, v = u)P(D = 1|v = u) + P(D = 0|v = u) \end{aligned} \tag{4.D.9}$$

and

$$\begin{aligned} & P(Y_0 = 1|D = 0, v = u)P(D = 0|v = u) \\ \leq\ & \boldsymbol{P(Y_0 = 1|v = u)} \\ \leq\ & P(Y_0 = 1|D = 0, v = u). \end{aligned} \tag{4.D.10}$$

Under the MIV assumption, for $u_1 \leq u \leq u_2$,

$$\begin{aligned} & \max_{u_1 \leq u} P(Y_1 = 1|D = 1, v = u_1) \\ \leq\ & \boldsymbol{P(Y_1 = 1|v = u)} \\ \leq\ & \min_{u \leq u_2} \{P(Y_1 = 1|D = 1, v = u_2)P(D = 1|v = u_2) + P(D = 0|v = u_2)\} \end{aligned} \tag{4.D.11}$$

and

$$\begin{aligned} & \max_{u_1 \leq u} P(Y_0 = 1|D = 0, v = u_1)P(D = 0|v = u_1) \\ \leq\ & \boldsymbol{P(Y_0 = 1|v = u)} \\ \leq\ & \min_{u \leq u_2} P(Y_0 = 1|D = 0, v = u_2). \end{aligned} \tag{4.D.12}$$

Applying the law of total probability, we obtain the bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ as follows:

$$\begin{aligned} & \sum_{u \in V} P(v = u) \max_{u_1 \leq u} P(Y_1 = 1|D = 1, v = u_1) \\ \leq\ & \boldsymbol{P(Y_1 = 1)} \\ \leq\ & \sum_{u \in V} P(v = u) \min_{u \leq u_2} \{P(Y_1 = 1|D = 1, v = u_2)P(D = 1|v = u_2) + P(D = 0|v = u_2)\} \end{aligned} \tag{4.D.13}$$

and

$$\begin{aligned} & \sum_{u\in V} P(v=u)\max_{u_1\le u} P(Y_0=1|D=0,v=u_1)P(D=0|v=u_1) \\ \le\ & \boldsymbol{P(Y_0=1)} \\ \le\ & \sum_{u\in V} P(v=u)\min_{u\le u_2} P(Y_0=1|D=0,v=u_2). \end{aligned} \tag{4.D.14}$$

When an instrument is binary, we can simplify them. As u, u_0 and u_1 take the values of 0 or 1,

$$\begin{aligned} & P(v=0)P(Y_1=1|D=1,v=0)+ \\ & P(v=1)\max\{P(Y_1=1|D=1,v=0),P(Y_1=1|D=1,v=1)\} \\ \le\ & \boldsymbol{P(Y_1=1)} \\ \le\ & P(v=0)\min\{P(Y_1=1|D=1,v=0)P(D=1|v=0)+P(D=0|v=0), \\ & P(Y_1=1|D=1,v=1)P(D=1|v=1)+P(D=0|v=1)\}+ \\ & P(v=1)\{P(Y_1=1|D=1,v=1)P(D=1|v=1)+P(D=0|v=1)\} \end{aligned} \tag{4.D.15}$$

and

$$\begin{aligned} & P(v=0)P(Y_0=1|D=0,v=0)P(D=0|v=0)+ \\ & P(v=1)\max\{P(Y_0=1|D=0,v=0)P(D=0|v=0),P(Y_0=1|D=0,v=1)P(D=0|v=1)\} \\ \le\ & \boldsymbol{P(Y_0=1)} \\ \le\ & P(v=0)\min\{P(Y_0=1|D=0,v=0),P(Y_0=1|D=0,v=1)\}+ \\ & P(v=1)P(Y_0=1|D=0,v=1). \end{aligned} \tag{4.D.16}$$

The sharp bound of the ATE can be obtained via equation (4.8).

4.D.2 MTR+MIV

Under the MTR assumption, the bounds of the conditional probabilities $P(Y_1=1|v=u)$ and $P(Y_0=1|v=u)$ are given from equations (4.14) and (4.15) by

$$\begin{aligned} & P(Y=1|v=u) \\ \le\ & \boldsymbol{P(Y_1=1|v=u)} \\ \le\ & P(Y_1=1|D=1,v=u)P(D=1|v=u)+P(D=0|v=u) \end{aligned} \tag{4.D.17}$$

and

$$\begin{aligned} & P(Y_0 = 1|D = 0, v = u)P(D = 0|v = u) \\ \leq \; & \boldsymbol{P(Y_0 = 1|v = u)} \\ \leq \; & P(Y = 1|v = u). \end{aligned} \tag{4.D.18}$$

Under the MIV assumption, for $u_1 \leq u \leq u_2$,

$$\begin{aligned} & \max_{u_1 \leq u} P(Y = 1|v = u_1) \\ \leq \; & \boldsymbol{P(Y_1 = 1|v = u)} \\ \leq \; & \min_{u \leq u_2} [P(Y_1 = 1|D = 1, v = u_2)P(D = 1|v = u_2) + P(D = 0|v = u_2)] \end{aligned} \tag{4.D.19}$$

and

$$\begin{aligned} & \max_{u_1 \leq u} P(Y_0 = 1|D = 0, v = u_1)P(D = 0|v = u_1) \\ \leq \; & \boldsymbol{P(Y_0 = 1|v = u)} \\ \leq \; & \min_{u \leq u_2} P(Y = 1|v = u_2). \end{aligned} \tag{4.D.20}$$

Applying the law of total probability, we obtain the bounds of $P(Y_1 = 1)$ and $P(Y_0 = 1)$ as follows:

$$\begin{aligned} & \sum_{u \in V} P(v = u) \max_{u_1 \leq u} P(Y = 1|v = u_1) \\ \leq \; & \boldsymbol{P(Y_1 = 1)} \\ \leq \; & \sum_{u \in V} P(v = u) \min_{u \leq u_2} [P(Y_1 = 1|D = 1, v = u_2)P(D = 1|v = u_2) + P(D = 0|v = u_2)] \end{aligned} \tag{4.D.21}$$

and

$$\begin{aligned} & \sum_{u \in V} P(v = u) \max_{u_1 \leq u} P(Y_0 = 1|D = 0, v = u_1)P(D = 0|v = u_1) \\ \leq \; & \boldsymbol{P(Y_0 = 1)} \\ \leq \; & \sum_{u \in V} P(v = u) \min_{u \leq u_2} P(Y = 1|v = u_2). \end{aligned} \tag{4.D.22}$$

When an instrument is binary, we can simplify them. As u, u_0 and u_1 take the values of 0 or 1,

$$
\begin{aligned}
& P(v=0)P(Y=1|v=0) + \\
& P(v=1)\max\{P(Y=1|v=0), P(Y=1|v=1)\} \\
\leq\ & \boldsymbol{P(Y_1=1)} \qquad (4.D.23) \\
\leq\ & P(v=0)\min\{P(Y_1=1|D=1,v=0)P(D=1|v=0) + P(D=0|v=0), \\
& P(Y_1=1|D=1,v=1)P(D=1|v=1) + P(D=0|v=1)\} \\
& + P(v=1)\{P(Y_1=1|D=1,v=1)P(D=1|v=1) + P(D=0|v=1)\}
\end{aligned}
$$

and

$$
\begin{aligned}
& P(v=0)P(Y_0=1|D=0,v=0)P(D=0|v=0) + \\
& P(v=1)\max\{P(Y_0=1|D=0,v=0)P(D=0|v=0), P(Y_0=1|D=0,v=1)P(D=0|v=1)\} \\
\leq\ & \boldsymbol{P(Y_0=1)} \qquad (4.D.24) \\
\leq\ & P(v=0)\min\{P(Y=1|v=0), P(Y=1|v=1)\} + \\
& P(v=1)P(Y=1|v=1).
\end{aligned}
$$

The sharp bound of the ATE can be obtained via equation (4.8).

Part III

Conclusion

This thesis has contributed to an understanding of health inequality in low- and middle-income countries in Asia. It has investigated two important topics in health economics. The first two chapters focused on the inequality of opportunity (IOp) in health. Chapter 1 explored the ex-ante IOp in child health in ten developing countries in Asia, i.e. Bangladesh, Nepal, Pakistan, Maldives, India, Cambodia, Myanmar, East Timor, Tajikistan and Kyrgyzstan. We observed significant health disparities between children from households with advantaged socio-economic status and those from households with disadvantaged socio-economic status in all ten countries. A decomposition analysis yielded strong evidence that the observed health disparities are strongly associated with households' living standards and suggested that priority should be given to protecting children from marginalised households. Chapter 2 proposed a new approach to measuring the ex-post IOp. The proposed approach employed the statistical property of copulas to model the distributional dependency between effort and circumstance variables. Combined with the semi-parametric distributional regression, the proposed method estimated a conditional distribution function of health and then quantified the IOp. We applied the method to the inequality in body mass in Indonesia associated with its intergenerational transmission from parents to grown-up offspring. We found that the non-negligible proportion of overall inequality in body mass is related to its intergenerational transmission.

The last two chapters evaluated the conditional cash transfer programmes conducted in Indonesia and India. Chapter 3 evaluated the impacts of the *Program Keluarga Harapan* (PKH) on child nutritional status and household expenditure in Indonesia. This chapter has contributed to the existing evidence by estimating the distributional impacts and by investigating the pathways through which the PKH influences child health. A significant improvement in weight for age z-score was observed only among children aged 25-36 months, and its improvement was not explained by the rise in household expenditure due to the PKH. We investigated why health improvement was not observed among younger age groups and discussed how the programme could be improved to maximise its potential on the basis of the evidence in Latin America. Chapter 4 re-visited the impact of the *Janani Suraksha Yojana* (JSY) programme in India, on maternal and child healthcare use. In contrast to major conditional cash transfer programmes in Lain America, the randomised controlled trial designed for the programme evaluation was not conducted in India. We reviewed the existing non-experimental evidence through the partial identification

approach and assessed the validity of the identification assumptions employed in the previous studies. The results indicated that the average treatment effects (ATEs) estimated under the identification assumptions used in previous studies lie outside of the ATE bounds estimated under weaker but more credible assumptions. These findings suggested that the identification assumptions imposed in the previous studies could have been invalid and estimated treatment effects had been over- or under-estimated because unobserved confounding factors were not fully controlled for.

As a concluding remark, I would like to discuss future research extensions of each chapter here. First, for chapter 1, the dynamic changes of IOp in child health across time would be worth exploring. Asian developing countries have been experiencing strong economic growth, rapid urbanisation and relentless globalisation, which has been substantially changing the food systems, including the processing, distribution and marketing thereof, and made more food choices available. Furthermore, hand-in-hand with economic development comes more income, which in turn can be spent more not only on foods but also on housing and early education, thereby dramatically changing the lifestyles of the people. Also, diversification in family composition has been observed by a growing middle class, with a rise in females participating in the labour force and the advent of dual-income nuclear families. Exploring the trend of health inequality and its changes over time would complement the cross-sectional findings of this study and foster an understanding of health inequality dynamics in the 21st century in Asia.

Exploring the inequality in the life-course is also worth looking into, in order to better understand the mechanism by which the child health inequality takes place and evolves across the developmental stages. Life-course perspectives on the evolution of inequalities allow us to explore the dynamic link between early-life circumstances and later consequences and to identify the optimal timing of when circumstances and events matter the most (Ben-Shlomo and Kuh, 2002; Lynch et al., 1997; Lynch and Smith, 2005). Future research would be well served by investigating how the trajectory of inequality in children's development associated with early-life circumstances diverges from early in life through to early adulthood and at what age such inequality peaks and whether and how it changes across the developmental stages.

Next, in the applied economics, with the accumulation of big-data, how we can handle the

high dimensional data has been widely discussed recently. Research into IOp is no exception; as in-depth genetic information is becoming available, high dimensional data is receiving attention these days in health economics. As genetic characteristics are totally beyond our control and any health disparity attributable to them can be considered inequitable. Hence, ultimately tens of thousands of genetic factors might also be considered to be non-ignorable circumstances. However, economists rarely know what genetic characteristics matter or how they are related to our health a priori. Furthermore, too much genetic information may often build up the model which has more circumstance variables than total sample sizes, resulting in the curse of dimensionality in a regression analysis. At the end of chapter 2, we discussed the potential limitations of copulas in the presence of high dimensional circumstance and effort variables. Recently, Brunori et al. (2019) argue that estimating the IOp can be understood as a prediction problem and suggest incorporating some of the machine learning techniques in quantifying the IOp. Exploring the potential of the machine learning approach in the study of the IOp would be beneficial for future research into health inequality in the era of big data.

For chapter 3, as we touched upon in the conclusion, estimating the much longer-term effects would complement our findings. In contrast to the healthcare utilisation, it requires much longer periods for the effect on child nutritional status to appear, in particular, increase in height. We observed little improvement in height-for-age z-score and weight-for-age z-score among children aged 0-12 months and children aged 13-24 months, 26-30 months after the implementation of the PKH. Estimating even longer-term effects with further follow-up surveys would definitely contribute to a more thorough policy evaluation. Also, evaluating effectiveness for various intermediate outcomes beyond household total expenditure and investigating the effects on household's resource allocation would further enrich our understanding of the behavioural changes of beneficiaries.

Finally, for chapter 4, another important investigation to be conducted is to explore the heterogeneity of the ATE bounds across the population. As is often carried out in empirical studies with the traditional point-identification approach, testing the existence of heterogeneity is no less important in the partial-identification approach than in the point-identification approach. Due to space limitations, we did not implement sample-splitting in the study, but it is intriguing to estimate the ATE bounds across states, religions and ethnicities of households and maternal

education levels. Through the comprehensive exploration into the heterogeneity, we can infer which population could potentially be most subjected to the selection bias due to unobservable confounding factors.

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