Just Genetics: How far does the moratorium on the use of genetic data by insurers within the UK accord with Rawls' principles of justice?

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Abstract

Since the completion of the Human Genome Project in 2004, understanding of genetic information has expanded, opening much greater possibilities with regard to identifying, diagnosing and treating genetic conditions. Within the National Health Service, a report produced by the Human Genomic Strategy Group (HGSC) aims to roll out a comprehensive system of genetic testing within the NHS, and Department of Health discussions are underway to implement it.

Given this, more individuals are informed about their genetic status and what it may mean than ever before. As the UK insurance industry operates on principles of mutuality, there was (and is) substantial interest in the potential this data holds when it comes to identifying those who will suffer from future disease. However, currently, within the UK, there is very little use of such data, as the industry complies with a voluntary moratorium first announced in 2005, now extended until 2017. As of the publication of this thesis, the moratorium has been in place for seven years, and little progress has been made in establishing what should replace it.

It is here that this thesis makes a contribution. Using a Rawlsian methodology, this piece establishes first, under a system of mutuality such as that within the UK, what one *should* do when considering whether to use genetic information in the insurance context. This is done through examining representative conditions, and assessing whether it would be justifiable to allow insurers to access data relating to these conditions. Second, the thesis then assesses how far the current UK moratorium complies with the principles of justice, and in cases where it does not, suggests feasible changes to make it more compliant. In this way, I hope to contribute, at least in part, to answering the question of where the UK should proceed post-2017.

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Chapter One

Introduction

"Today, with genetic research continuing apace, many more conditions will be conclusively linked with a specific single gene mutation. But recent advances in medical science – in particular, the completion of the human genome – have opened up much greater possibilities, to understand the impact of genetic variation not just in a single gene but across multiple genes or even the whole genome." - **Human Genomics Strategy Group (2012)**.[1]

Since the completion of the Human Genome project in 2004, understanding of genetic information has increased significantly. A recent study by the United Health Group (part of United Health, a large US Health Insurer) shows that the cost of genetic testing for their customers alone was $500 million in 2010, and this is projected to increase year on year.[2] Using estimates from 2006 to 2009 data, and extrapolating from this, they also demonstrate that national spending within the USA on genetic testing services reached $5 billion within 2010, which constitutes approximately 8% of national spending on clinical services. Further, they suggest that this sector could see further explosive growth over the next decade, estimating national spending of between $15 billion and $25 billion by 2021.[3] Given genetic tests are also becoming cheaper to supply, this means that the market in these services is expanding year on year.

Although I am unaware of a specific study such as this within the UK context, one can see a similar trend, with the most recent report of the UK Genetic Testing Network (UKGTN) suggesting a rapidly expanding repertoire of tests and care networks.[4] Indeed, at the start of this year, Professor John Bell (in his role as chair of the Human Genomics Strategy Group) suggests a strategy for continued

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[1] Human Genomics Strategy Group, *Building on our Inheritance: Genomic Technology in Healthcare*, January 2012, Accessible at: [http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_132382.pdf], Accessed 14-08-2012.

[2] United Health, *Personalized Medicine: Trends and Prospects for the new science of genetic testing and molecular diagnostics*, Working Paper 7, March 2012, Accessible at: [http://www.unitedhealthgroup.com/hrm/UNH_WorkingPaper7.pdf] Accessed 07-04-2012.

[3] *Ibid.*

[4] UK Genetic Testing Network, *Supporting Genetic Testing in the NHS: Second Report of UKGTN*, November 2010, Accessible at: [http://www.ukgtn.nhs.uk/gtn/digitalAssets/0/929_SECONDREPORT.pdf] Accessed 07-04-2012.

development of genetic testing within the UK, stating that "Genomic technologies have the potential to transform the delivery of healthcare in the UK, providing vital insights to support more accurate diagnosis of disease and inform therapeutic decisions…"[5] This is a view also shared by the House of Lords Science and Technology Committee, who suggest that the science of human genetics will lead to real advances in medical care.[6]

With recent reports on the capability of genetic testing produced by the Department of Health and media outlets such as the BBC, and statements from political figures such as ex- Health Secretary Andrew Lansley, the public is now more aware than ever of the availability and potential importance of genetic testing.[7 and 8] In addition to this, we are beginning to see a large market develop outside of the specific healthcare context. Direct to consumer genetic testing is now easily available, with several private companies offering everything from paternity testing and ancestry tracking to determining susceptibility to certain conditions. As an example, *thatDNAcompany* offer to provide: "An accurate DNA test; for the people and by the people who care", and that they are, "…here to help you resolve family issues of fatherhood (paternity) at an affordable price. No frills, no bells, no whistles."[9] This is a service also offered by other providers such as GeneTrack UK.[10] Indeed, companies such as 23andMe offer an even wider variety of services, claiming to inform you about your risk of developing several different conditions or diseases such as diabetes or obesity.[11] This information is easily available, with the individual merely needing to order an online kit, provide them with a sample through the post, and obtaining results online or through the post within a few weeks.

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[5] Human Genomics Strategy Group, *Building on our Inheritance: Genomic Technology in Healthcare*, January 2012, *op. cit.*

[6] House of Lords Science and Technology Committee. *Genomic Medicine – Volume 1: Report. (2009)* 2nd Report of Session 2008–09.

[7] Department of Health, *Genomic Innovation will better target treatment in the NHS*, January 25th 2012, Accessible at: [http://mediacentre.dh.gov.uk/2012/01/25/genomic-innovation-will-better-target-treatment/] Accessed 07-04-2012.

[8] British Broadcasting Corporation, *Genetic testing: NHS 'must back revolution'*, January 25th 2012, Accessible at: [http://www.bbc.co.uk/news/health-16725032] Accessed: 07-04-2012.

[9] ThatDNACompany, Accessible at: [http://www.thatdnacompany.com/index.htm] Accessed 07-04-2012.

[10] GeneTrack UK, Accessible at: [http://www.genetrack.co.uk] Accessed 07-04-2012.

[11] 23andMe, Accessible at: [https://www.23andme.com/health] Accessed 07-04-2012.

All of this means that individuals are now able to be more informed about their own genetic status than ever before. As the UK insurance market operates on the principle of mutuality, within which premiums are set to accord with the risk that an insured individual brings to the pool, there is *prima facie* a clear interest for insurers to be able to access and use this data, insofar as it is relevant to an individual's future health status.

1.1 – The UK Position

Indeed, even before the rapid expansion in the availability of genetic information to individuals, there was concern over the use of this information. The Human Genetics Advisory Commission (HGAC) suggested within its February 1997 report that concern over the use of genetic data in life insurance decision making was warranted because:

> "For most people in the UK, life insurance is linked to home purchase and the protection of dependants. It matters to individuals, to society and, indeed, to the insurance industry itself that people are not excluded from life insurance without good reason."[12]

First suggested in 1998, the moratorium on the use of genetic test data was initially opposed by the industry at large, with them fearing that it paved the way for a more permanent ban on the use of such data.[13] Eventually, feeling threatened by possible legislative action, the industry voluntarily enacted a five year moratorium on the use of genetic test data, starting in November 2001. This moratorium was subsequently extended until 2011, and then again until 2014, and most recently, 2017. [14,15] This is a voluntary moratorium which the Association of British Insurers (ABI) is responsible for monitoring and enforcing.

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[12] Human Genetics Advisory Commission, *The Implications of Genetic Testing for Insurance*, Issued December 1997, Accessible from: [http://www.dh.gov.uk/ab/Archive/HGAC/index.htm] Accessed 07-04-2012.

[13] Masood, Ehsan, *UK insurers oppose moratorium plea on use of genetic data*, Nature (1998) 391 (6662) p.3

[14] Association of British Insurers, *ABI News Release: Insurance Genetics Moratorium extended to 2014*, Accessible at:
[http://www.abi.org.uk/Media/Releases/2008/06/Insurance_genetics_moratorium_extended_to_2014.aspx], Accessed 30-07-2010.

[15] Association of British Insurers, *ABI News Release: Insurance Genetics Moratorium extended to 2017*, Accessible at:

Specifically, the moratorium prohibits the use of all genetic test data for life insurance cover under £500,000 in value, or critical illness cover under £300,000 in value. This is the sum total of policies held, not the value of individual policies. As the moratorium document itself highlights, over 97% of policies held in 2004 were below this value.[16] For policies held over these values, insurers may request the results of genetic tests pre-approved by the Genetics and Insurance Committee. (GAIC) This committee (which was wound up on the 1st July 2009) was responsible for hearing applications from insurers to use specific genetic tests, and evaluating their applicability, reliability and relevance[17]. The committee only ever approved one genetic test application, for Huntington's disease in October 2000.[18] Within the UK, there is therefore very little use of genetic test data by the insurance industry. For 97% of policies, no data is used at all, and for the remaining 3%, insurers are only able to ask for the results of one specific test.

1.2 – My Question

As a reminder, the question which I seek to answer in this thesis is: *How far does the moratorium on the use of genetic data by insurers within the UK accord with the principles of justice?* Within this, there are two sub-questions. First, what is the position which would be supported by the principles of justice? And then second, how far does the current moratorium accord with this position?

As my thesis is explicitly concerned with the UK and the current moratorium, it does assume certain facts, one of which has already been identified. For the purposes of this thesis, I will be assessing the potential use of this data by the UK insurance industry as it currently exists, that is to say, a private industry based on the principle of mutuality rather than solidarity. This will be important when I come to consider the potential consequences that allowing use of certain data could have.

[http://www.abi.org.uk/Media/Releases/2011/04/Insurance_Genetics_Moratorium_extended_to_2017.aspx], Accessed 10-02-2012.

[16] Association of British Insurers, *Concordat and Moratorium on Genetics and Insurance, op. cit.*

[17] Genetics And Insurance Committee, Summary, Accessible at: [http://www.dh.gov.uk/ab/Archive/GAIC/index.htm], Accessed 10-02-2012.

[18] Genetics and Insurance Committee, *October 2000: Huntington's Disease*, Accessible at: [http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@ab/documents/digitalasset/dh_087703.pdf], Accessed 30-07-2010.

Second, I will also be assuming the presence of a social healthcare system, namely the National Health Service (NHS) which provides healthcare free at the point of service, and is funded by taxation.

The reason for this is that I aim to provide an answer which has some real-world relevance. Whilst I could base my thesis entirely in abstract theory, or consider the UK were it to adopt a life insurance system based upon solidarity, it would seem to me relatively unlikely that the UK will adopt such a system, at least in the near future, and therefore, the actual relevance of my thesis would be limited. The aim of the thesis is to provide some guidance as to what we *should* do about the use of genetic information within the current UK context, which requires consideration of the current position, not a hypothetical one. This is important, as both the use and understanding of genetic testing continues to advance. We are moving rapidly towards a position where genetic testing for many conditions will routinely be offered on the NHS (the aim of the HGSG report), and many individuals will be informed about their health status. A moratorium agreed in 2005 and simply renewed since is ill-equipped to cope with this changing situation. As Stirton argues, the moratorium has become a *de facto* inflexible and permanent response to the question, and one which is neither reflexive nor sufficiently credible.[19] Specifically, she argues that whilst compliance is currently high, this depends on there being no great gain to be made from breaching the moratorium, and that this is threatened by developing science. I agree with this assessment. Further, I agree with the idea that within the seven years since the moratorium was agreed, progress should have been made on a more permanent solution. Indeed, looking to Stirton's other work, she suggests that legislating either abstractly for 'fairness' or specifically in an exceptionalist way is bound to fail, and that engagement is needed instead with specific policy questions.[20] Whilst I feel that there are legitimate reasons to subscribe to a weak form of genetic exceptionalism (specifically, with regard to perceptions), I do agree that legislating as if all genetic information was uniquely special is misguided. Instead, I intend to contribute toward finding the answer to these difficult policy questions through the prism of justice.

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[19] Stirton, Ruth, *Insurance, Genetic Information and the Future of Industry Self-Regulation in the UK*, upcoming in Law, Innovation and Technology, 2013.

[20] Wilkinson(Stirton), Ruth, *Genetic Information: Important But Not 'Exceptional'*, Identity in the Information Society (2010) 3 (3) p. 472.

1.2.1 – Research Plan

Within the thesis, I therefore intend to do the following. Briefly within the rest of *Chapter One*, I will explore the potential reasons that could be offered for focusing on data gathered from genetic tests above all other data (such as family history). Whilst I eventually conclude that there may be reason to subscribe to a weak form of genetic exceptionalism, given I am assessing the justifiability of the moratorium; I must focus on what said moratorium deems 'genetic test data' (an exceptionalist position). Within *Chapter Two*, I will then provide an explanation of my methodology, namely how the principles of justice are derived, and what they entail. I will then explain my reasons for focusing on the second principle in particular, the inequalities which I am specifically concerned about, and defend the focus of my thesis on income and particularly wealth. Having established these things, I will proceed to analysis.

First, *Chapter Three* will offer an initial analysis of the use of genetic test data, based on income and wealth, before I recognise and demonstrate the problems with this, and proceed to a more detailed development of typologies within *Chapter Four*. Within *Chapter Five*, I will then answer the first of my two sub-questions, namely, which data (if any) could justifiably be used by insurers under a system of mutuality, and which data should be prohibited from use. Finally *Chapter Six* will then provide a re-framing of my conclusions from *Chapter Five*, and an analysis of whether the moratorium is justifiable in each specific case. Finally, I will conclude by briefly relating my work back to the wider field.

1.2.2 – Thesis Parameters

Whilst there are several different questions that *could* be answered here, it is important to note at this point the limits of the specific question I seek to answer. First, the question of whether or not we should use genetic data can be affected by considerations other than those identified within the thesis, for example, concerns over genetic privacy. However, within the thesis, I am explicitly attempting to answer the question from a Rawlsian perspective. As I will illustrate within *Chapter*

Two, these ideas are not explicitly relevant to a Rawlsian conception of justice. As such, there will be no discussion of genetic privacy within the thesis, although this could provide an interesting avenue for future work. As I illustrate above, I am also strictly considering justifiability within the existing system of mutuality, rather than solidarity. Analysing a hypothetical position of solidarity would detract from my ability to assess the status quo. It is also not at all clear that a Rawlsian would necessarily have to commit to a system of solidarity. The thesis is also not a creative re-imagining of the Rawlsian theory. With a few exceptions (notably the work of Norman Daniels and Amartya Sen), the thesis is simply an application of this theory. Fundamentally, the thesis then only seeks to answer one question. Namely, whether the current position on the use of genetic information by insurers within the UK can be justified according to Rawls' principles of justice. It is this narrowly defined question which I will now proceed to answer.

1.3 – Genetic Exceptionalism

Both the moratorium, and therefore, my thesis, place special importance on data obtained from genetic tests. The most recent definition of 'genetic tests' offered by the Association of British Insurers (ABI) is:

> "A *genetic test* is defined as a test that examines the structure of chromosomes (cytogenetic tests) or detects abnormal patterns in the DNA of specific genes (molecular tests). A genetic test can be predictive or diagnostic:
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> • a *predictive* genetic test is taken prior to the appearance of any symptoms of the condition in question
> • a *diagnostic* genetic test is taken to confirm a diagnosis based on existing symptoms. "[21]

The moratorium in particular concerns itself with the use of predictive genetic tests. This means that within the thesis, I will be assessing how far the use of certain predictive genetic tests is justifiable, using the above definition of 'genetic test'. However, there is significant debate as to whether genetic information such as this is meaningfully different from any other data. This idea that genetic data is indeed special, and worthy of different protection or consideration is often termed

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[21] Association of British Insurers, *ABI Code of Practice, Genetic Tests, June 2008,* Accessible at: [*www.abi.org.uk/consumer2/disclosure.htm] Accessed 11-10-2012.*

genetic exceptionalism. The following is a brief exploration of the literature in the area.

Objections to genetic exceptionalism can be arranged under two major headings. Suter raises these broad concerns, which she terms "over" and "under-inclusiveness"[22] First, over-inclusiveness. According to Suter,

> "Concerns about the lack of control over one's genes, the high level of predictiveness of genetic information, and its stigmatizing and hidden features do not apply equally to all genetic information."[23]

In other words, not all genetic information has these characteristics, and therefore to ground our differentiation on this concept is nonsensical. Our genetic information is broadly not unique; we share the majority of it with other individuals. Equally, Suter claims that certain 'genetic' traits such as blood type, eye colour and gender undermine genetic exceptionalism, as although genetic; they do not "seem particularly susceptible to discriminatory uses."[24] Whilst the fact that these are not susceptible to discriminatory uses is certainly open to question (especially gender), much more interesting is the question of under-inclusiveness.

Here we must confront the claim that genetic information is not at all special, and shares its most important characteristics with information that would be considered non-genetic. To subscribe to genetic exceptionalism, it is claimed, is therefore to make an arbitrary distinction between the two, and to privilege genetic information without any substantial grounding. In order to determine whether we can overcome this objection, it is necessary to examine some of the most common reasons given for subscribing to the exceptionalist position.

The first is the concept of genetic information as 'uniquely identifying'. Genetic exceptionalism is able to be justified because unlike any other information, my genetic code is absolutely unique to me. Using that code, you could identify me, and no-one else. This is not however something that is unique to genetic information.

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[22] Suter, Sonia, *The allure and peril of genetics exceptionalism: do we need special genetics legislation,* Public Law and Legal Theory Working Paper No.19, 2001, Accessible at [http://papers.sssrn.com/abstract=276875], Accessed 01/03/11
[23] *Ibid*. p. 46.
[24] *Ibid*. p.47.

In fact, given a sufficient quantity of information, almost any information could be used to identify me. Vehicle registration numbers, addresses, medical records indicating previous injuries and many other sources of information could potentially identify me just as accurately. My genetic code is not even unique in the sense that unlike the above example, it can identify me without amalgamation of data. The use of certain biometrics can allow someone to determine my identity just as accurately, and with just as little data.[25] My fingerprint is unique to me in the same way as my genetic code. Given this, it seems that the 'uniqueness' argument is utterly insufficient to ground any claim to genetic exceptionalism. To do this, we must look elsewhere.

Within the literature, we find that there are many other reasons provided for grounding a claim to genetic exceptionalism. Rothstein identifies them as the following:

" (1) genetic information has implications not only for the individual but also for family members;
(2) genetic information may have implications for reproduction and characteristics of future generations;
(3) genetic information may be predictive;
(4) genetic information often carries stigma, and the misuse of genetic information has led to eugenics, racism, and genocide;
(5) genetic information is regarded as unique by the public;
(6) there are other "special" categories of medical information for which separate protections have been adopted, including HIV/AIDS and mental illness; and
(7) the political reality is that there is greater support for genetic non-discrimination legislation than for more general and sweeping laws"[26]

He then proceeds to claim that reasons 1 through 3 can be dismissed as they are again not unique to 'genetic' data. Other aspects of my life, and indeed, other aspects of my medical record may have implications for future generations, my family members, and predict my future health. As an example, if I were HIV positive, this would have clear implications for my partner, and also be significantly predictive of my potential future health. Indeed, if we consider a woman who is HIV positive, this also fulfils reason 2, as her HIV status will have potentially important implications for any future children as her infection could be passed to the child during birth.

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[25] *Ibid.* p. 50.
[26] Rothstein, Mark, *Genetic Exceptionalism and Legislative Pragmatism*, The Journal of Law, Medicine and Ethics, (2007), 35 Supplement. 2, p. 61-62.

1.3.1 – Scope

So are there any reasons to support claims 1 through 3? Sticking with our HIV example, we can meaningfully begin to distinguish genetic information on the grounds of scope.[27] It is difficult to envision a single non-genetic example which has the same scope as a genetic one. Although HIV status may potentially fit within claims 1-3, it does not tell me nearly the same amount about others that a genetic test does. To use an example, my discovery that my father has become HIV positive would not have implications for my own health. However, my discovery that my father possesses the gene for Huntington's (an autosomally dominant disease) has a significant effect not only on my actual health, but on my future health and how I perceive it.

That genetic information is unique in scope is also a position supported by the *Report on Genetic Exceptionalism* presented to Congress in 2008. Within this, Sarata demonstrates the true scope of 'genetic' data, and is led to conclude that "…it may be argued that there is little medical or personal information that shares *all* of these characteristics with genetic information." [28] I would agree with this assessment. It is difficult to imagine any piece of non-genetic information which is able to tell us the same amount as an individual's genetic information. This is the image that Annas invokes when he refers to the concept of a "future diary".[29] He suggests that although other health information is relevant, and can certainly have important implications for the future health of individuals and their family members, when it comes to genetic information;

> "There is nothing else quite like this type of information."[30]

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[27] Friedman Ross, Lainie, *Genetic Exceptionalism vs. Paradigm Shift*: *Lessons from HIV*, The Journal of Law, Medicine and Ethics, (2001) 29 (2) p. 142.

[28] Sarata, Amanda, *CRS Report for Congress, Genetic Exceptionalism: Genetic Information and Public Policy*, 2008, Accessible at [http://www.policyarchive.org/handle/10207/bitstreams/18867.pdf], Accessed 02/03/11 p. 1.

[29] Annas, George et. al, *Drafting the Genetics Privacy Act: Science, Policy, and Practical Considerations,* The Journal of Law, Medicine and Ethics (1995), 23 360-366.

[30] Annas, George, *Genetic Privacy*, Accessible at [http://www.hks.harvard.edu/dnabook/George%20Annas%20II.doc], Accessed 05/03/11

It would seem that initially we can therefore begin to justify holding a position of weak genetic exceptionalism, at least as distinguished from other medical information such as HIV status due to its unprecedented scope. One can find support for this position from many other commentaries, including Gostin, who claims that the exceptional nature of this information stems from:

> "…the sheer breadth of information discoverable; the potential to unlock secrets that are currently unknown about the person….and the generalizability of the data to families, genetically related communities and ethnic and racial populations."[31]

However, this position begins to look weaker when we examine the case of family histories. Are we able to usefully distinguish these from the data gathered by genetic tests? Using only the justifications we have so far, this seems tenuous. There is no meaningful difference between the example above where my father has a genetic test for Huntington's, and where I know that I have a family history of Huntington's because my father has lived to an age where he is a sufferer. Anything that the results of that genetic test tell me about my own future health, my families, or my reproductive chances I can also discover from the family history.

We still perhaps have a very weak claim to genetic exceptionalism remaining, in that if we examine the characteristics of genetic information presented above, the family history does not seem to possess all of these components. It is difficult to see how a family history could impact communities in the same way as genetic information. A discovery that I am susceptible to Huntington's does not have the same impact as a hypothetical discovery that all individuals of a certain subgroup are susceptible to a specific disease or set of diseases.[32]

Indeed, if we consider reason 4 given by Rothstein, (that genetic information could carry stigma, and be used to promote racism, eugenics and genocide) we begin to find an important difference. This is a difference dismissed by many commentators, including Friedman, who claims that:

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[31] Gostin, Lawrence, *Genetic Privacy*, Journal of Law, Medicine and Ethics (1995), 23 p. 326.
[32] A good example here is the Ashkenazi Jewish population, who have been found to have a significantly higher incidence of several genetic diseases.

> "…threats of discrimination and stigmatization will exist as long as there are differences and that these differences need not have a genetic basis, as current international conflicts illustrate."[33]

It is difficult to argue that discrimination and stigmatisation would not exist if genetic differences were not brought to light. Individuals are likely to always find ways to discriminate against and stigmatise those who they feel are different. However, I do not accept this as a reason to dismiss the potential impact of genetic test data. It is apparent that this data could be used to discriminate against and stigmatise those who are different, and it could do so in a unique way. We already know that certain ethnic groups are prone to a higher incidence of genetic defects. It is not a stretch to suggest that Ashkenazi Jews could potentially suffer discrimination from merely being a part of this group. Genetic test data demonstrating that these groups suffer from certain defects could provide scientific 'justification' for discrimination. If we believe that stigmatisation and discrimination against these groups is bad, then we must account for the fact that genetic test data may provide further reason for this to happen, and not simply dismiss it by reasoning that individuals will find a reason to discriminate anyway.

Even given this, family history data possesses the majority of the characteristics above. This is unsurprising, as data gathered from family histories is a brute form of genetic data. Is there an essential difference between family history data and the data that can currently be gathered from genetic tests that justifies a distinction when we are speaking about individuals, rather than populations?

1.3.2 – Predictive Ability

We can meaningfully begin to differentiate the two on the grounds of what they are able to predict. A family history indicating my father possesses the allele for Huntington's means that I (and others) can infer that I have a 50% chance of developing the disease. If I undergo a genetic test for the allele and am found to have it, this increases to 100%. Equally, if the test proves negative, my chance of developing Huntington's drops to 0%. The genetic test is able to provide me data

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[33] Friedman Ross, Lainie, *Genetic Exceptionalism vs. Paradigm Shift*: *Lessons from HIV, op. cit.* p. 143.

which the family history simply cannot. This data is incredibly relevant when attempting to assess my future health prospects, or when I come to consider my reproductive choices.

Another good example is breast cancer. An individual who possesses a mutation of BRCA1 or BRCA 2 has a hugely increased risk of developing cancer.[34] Although this is also substantially affected by environmental or social factors (such as weight, diet and choice of contraception) the genetic contribution is still significant. Whether a woman actually carries this mutation can only be discovered through genetic testing. A woman with a family history of death from cancer may actually not possess a mutation and therefore be at no higher risk. More dangerously for the woman, she may have a relatively clear family history, and yet still be a carrier. Given the importance of early intervention in lowering cancer mortality rates, it is medically important that she be made aware of the fact that she carries a mutation. A genetic test would do this, whereas her family history would not. These two types of data can therefore be distinguished again in this way. This view is shared by Friedman Ross, who suggests:

> "The exceptional nature of genetics, if it is exceptional, is due to its *power to predict late onset, Mendelian conditions*; its power to identify genes that increase susceptibility to multifactorial conditions but are neither necessary nor sufficient for the development of disease; and its implication for reproduction."[35]

This is certainly true of some genetic information, for example Huntington's and BRCA as explained above. Equally, genetic tests seem to be able to be differentiated from family history data on the grounds of sheer predictive ability. The predictiveness (often termed penetrance) of a specific genetic marker will become relevant later when I derive my typologies.

However, the majority of conditions are multifactorial, and development of the eventual condition relies on a complex interplay between genes, environment and societal factors which we do not yet fully understand. Currently then, our claim to

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[34] Kurian, Allison et. al., *Survival Analysis of Cancer Risk Reduction Strategies for BRCA 1/2 mutation carriers*, Journal of Clinical Oncology (2010) 28(2) 222-231.
[35] Friedman Ross, Lainie, *Genetic Exceptionalism vs. Paradigm Shift*: *Lessons from HIV, op. cit.* p. 142.

genetic exceptionalism is very weak indeed, relying solely on the fact that in some cases, specific conditions can be diagnosed or identified much more accurately through genetic tests than through other means. When considering these multifactorial conditions, do we have any reason to distinguish between genetic data that might indicate an increased risk of heart disease, and obesity which may indicate an equally increased risk?

1.3.3 - Perceptions

Whether genetic information is or is not in fact special, individuals certainly perceive it to be so. As an empirical example, in a study carried out at Columbia University, two groups of participants were given identical likelihoods of suffering from disease. One group was given an estimate based on 'family history' data, whereas the other was given the same estimate through the use of data derived from a genetic test. When asked about their perceptions of their risk, 73% of the genetic group judged their risk to be lower, compared to 25% of the family history group. 67% of the members of the genetic group also reported lower anxiety about potentially suffering from the disease, compared to 26% of the family history group. The genetic group was also significantly less likely to believe that they would suffer from the disease.[36]

This perception of genetic test data as somehow more meaningful or more serious is also reported back through several policy documents produced on the issue. A report by the Human Genetics Commission (HGC) produced in contains echoes of this idea, stating that:

> "There is public concern about the extent to which existing safeguards and regulation of information may be effective in the face of what can be done with the new technologies."[37]

This perception of genetic test data as so much more important than other information begins to give us other grounds for subscribing to a weak form of

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[36] LaRusse, Susan et. al., *Genetic susceptibility testing versus family history-based risk assessment: Impact on perceived risk of Alzheimer disease*, Genetics in Medicine: January 2005 7 (1) 48-53

[37] Human Genetics Commission, *Whose Hands on Your Genes: A Discussion document on the Storage, Protection and Use of Personal Genetic Information*, (London:HGC 2000) para. 2.4.

genetic exceptionalism. The difference between this data and other data is how it is perceived. This is important, because how the information is perceived directly impacts upon how it is used, and how it is used relates to what kind of impacts it is able to have on individuals. Indeed:

> "Our current obsession with genetic-sequence information means that it is likely to be taken more seriously than other information in a medical record that could also predict future risks, like high blood pressure or cholesterol levels."[38]

Genetic testing data has taken on a level of meaning that other data simply does not possess. This is one of the important differences which justifies an idea of genetic exceptionalism. In fact:

> "DNA has also been **culturally endowed** (emphasis mine) with a power and significance exceeding that of other medical information. Much of this significance is undoubtedly misplaced, but can be justified in so far as genetic information can radically change the way people view themselves and family members, as well as the way that others view them."[39]

Because of this, an individual is likely to take the use of genetic data much more seriously when considering what impact it may have on their life. Their perception will be that any impacts which stem from genetic testing data are somehow more important, and therefore, any adverse effects will be amplified.[40]

It is for this reason that several bodies have begun to accord genetic information this special status. In their 2001 policy document, the ABI notes that:

> "…the popular and political perception appears to be that there is an important difference. In the absence of change in these perceptions insurers ought to make plans to deal with public policy interventions based on the argument that molecular genetic information is categorically different."[41]

Similarly, in a 2008 document presented to Congress, Sarata claims that when deciding whether to pursue genetically exceptionalist policies:

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[38] Roche, Patricia and Annas, George, *Protecting Genetic Privacy*, Nature Reviews Genetics (2001) 2, 392-396.
[39] Ibid. p. 393
[40] Green, Michael and Botkin, Jeffrey, *"Genetic Exceptionalism" in Medicine: Clarifying the differences between genetic and non-genetic tests*, Annals of Internal Medicine (2003), 138, p.572.
[41] Association of British Insurers, *Insurance and Genetic Information*, (London: ABI 2001) s.5.1.2

> "…it is perhaps more relevant in terms of public policy to examine how the public views genetic information."[42]

When we are assessing the effects that the use of genetic data may have on the public, we cannot disregard public perception. If the public regard genetic data as special (which they appear to do) then it is justifiable to subscribe to a form of genetic exceptionalism in according it this special status, at least in the short term.

It is not just the public perception of genetic test data that raises issues however. There are serious issues with the patchiness of genetic test data which may affect how it is applied to individuals. Unlike much other information about individuals, we do not really fully comprehend what genetic data is actually able to tell us. Our understanding of how we are able to use this data is incomplete at best. As O'Neill explains:

> "…it might (because of the patchiness of information) be a context for ascribing much higher risks to some individuals than would be ascribed to them in the light of fuller information;"[43]

We therefore have more reason for treating genetic information as exceptional. We do not really understand how we can apply genetic information properly, and as such, any application may well be misinformed. This could adversely affect individuals. Our understanding of genetic data is incomplete, and therefore, we can find reason for subscribing to a form of genetic exceptionalism in treating genetic data with the utmost care until we can determine what we are actually able to draw from it.

1.3.4 – Conclusions on Exceptionalism

In light of the analysis above, I believe there is good reason to subscribe to at least a weak form of genetic exceptionalism. This is not to be genetically determinist, clearly genetic data is not the end point of the discussion, nor does it constitute everything we are. Although the claims that genetic information is uniquely identifying or predictive appear to fail when we consider family history

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[42] *Ibid*. p. 6.
[43] O'Neill, Onora, *Genetic Information and Insurance: Some Ethical Issues,* Philosophical Transactions of the Royal Society of Biological Sciences. (1997) 352 (1357) p. 1091.

data (at least on the individual level), we can support this position using the public perception of genetic test data. It certainly seems that whatever the facts (of which we are still mostly unaware), genetic test data is viewed to be special in some way. Indeed, empirical studies show that individuals act differently when presented with risks derived from genetic data than with equal risk derived by other means. If individuals treat genetic data differently to non-genetic data, this means that genetic data may have a broader or deeper impact, and allows us to justify offering it special protection. This is especially true when we consider the limitations of what we actually know about genetic data. There is no guarantee that were genetic test data to be applied to make decisions about individuals, it would be applied in any meaningful way. Our level of understanding is compromised. It is therefore wise to subscribe to a weak form of genetic exceptionalism.

Although this justification is theoretically important, the thesis seeks to answer specifically whether the position reached by the moratorium is justified. I will therefore be using the definition used by the ABI with regard to the moratorium, which is an exceptionalist position.. This will allow me to assess whether the moratorium, as understood by the ABI, accords with the justifiable position on the use of genetic data. This material on exceptionalism will however become relevant within *Chapter Five*, when I come to analyse whether individuals will be disincentivsed from obtaining genetic tests.

Chapter Two
Methodology

The methodology I am using is Rawlsian, as initially put forward in *A Theory of Justice* and subsequent publications. I will also be making use of developments on the theory, specifically, those offered by both Norman Daniels and Amartya Sen. Within this Chapter, I will first explain the background to the theory, including how the two principles of justice are derived and supported. I will then explain why, within this thesis, the first principle of justice will not be significantly considered, before moving onto my focus, the second principle. Further, I will then present a detailed account of primary goods, explaining their relevance to the second principle of justice, and how specifically the primary good of income and wealth is relevant to my later analysis. Within this, I will also offer an explanation and brief analysis of the theory of capability sets as put forward by Sen, and the relevance of this theory to my thesis. Finally, I will then offer an examination of fair equality of opportunity, and demonstrate both how the principle operates and its relevance to my conclusions. Within this section I will also explain why it is specifically equal access to employment which I am considering within the context of this thesis. First, the theoretical background.

2.1 – Background to the Methodology

2.1.1 – The Initial Principles

Initially put forward within *A Theory of Justice* (first published 1971, revised 1975), the Rawlsian conception of justice is one which revitalised interest in liberalism as a theory, and renewed wider interest in political philosophy. The theory itself espouses ideas of 'justice as fairness', and puts forward an essentially egalitarian account of justice, where the purpose of justice is to "mitigate the arbitrariness of natural contingency and superficial fortune."[1]

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[1] Rawls, John, *A Theory of Justice: Revised Edition* (Harvard: Belknap Press 1975) p. 82.

These ideas of justice stem from the concept of the social contract, the idea that as individuals, we cede certain rights to the state in order to obtain certain benefits.[2] In a hypothetical sense, we contract to give up, for example, our ability to kill others, in return for a guarantee that the state will also endeavour to prevent others from murdering us. In this regard, we gain benefits through this contractual behaviour that outweigh any freedoms we choose to sacrifice. In this way:

> "Social cooperation makes possible a better life for all than any would have if each were to live solely by their own efforts"[3].

The central theme of social contract theories is that the power of the state is limited by the hypothetical consent of the people within it. Clearly though, we have not actually contracted to be within the state. I happened, by chance, to be born within the United Kingdom, and at no point did I explicitly agree to the laws of this country, nor sign a contract. Given this, how are we to assess what I would choose, were I to have a free choice of terms? In order to determine what individuals would choose for themselves within the social contract, we must make use of two theoretical devices. The first of these is the *original position*. This requires that, when choosing which laws to be governed by, an individual imagines themselves in a situation where there exists no law. An individual is therefore not artificially limited by the constraints of current society, but instead puts themselves in a position where they are absolutely free. They can then legitimately decide which freedoms they wish to give up to obtain which benefits.[4]

However, the use of the original position as the only theoretical device is insufficient. If an individual is only to place themselves outside current constraints and then decide on what laws they would like to apply, it is possible that such laws would be discriminatory in nature. Operating from a position of rational self-interest, if I am currently wealthy, I am unlikely to see a benefit to myself in legislation protecting the poor. Indeed, measures which do protect the poor (such as redistributive taxation, or social healthcare) directly harm me by taking my wealth

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[2] Rawls, John, *A Theory of Justice: Original Edition* (Harvard: Belknap Press 1971) p. 16.
[3] *Ibid.* p. 4.
[4] *Ibid.* p.11.

and redistributing it to others. I am therefore unlikely to approve of a system in which this taxation features. Similarly, if I am a member of the majority ethnic group, I may not see any need for explicit policies against racism or other discrimination, as I do not envisage myself being subjected to them. The original position alone leads to a system which is potentially severely discriminatory.

To overcome this, Rawls employs a second theoretical device alongside the original position, termed the *veil of ignorance*.[5] From behind the veil of ignorance, an individual has no idea of who they will become in the potential society that they are tasked with deciding upon. Every individual behind the veil is placed in the same position; they know that they will be a person within the future society, but little else. Indeed:

> "…no-one knows his place in society, his class position or social status; nor does he know his fortune in the distribution of natural assets and abilities, his intelligence and strength, and the like. Nor, again, does anyone know his conception of the good, the particulars of his rational plan of life, or even the special features of his psychology such as his aversion to risk, or liability to optimism or pessimism."[6]

Given these individuals are making decisions based upon rational self-interest; they therefore cannot have any biases either toward or against a certain set of characteristics. As I do not know whether I will be one of the poorest members of society, it no longer makes sense for me to construct a system within which the poor suffer severely. I become more willing to sacrifice some future wealth (should I be a rich person) in order to safeguard myself against the possibility of poverty. The use of these theoretical devices therefore requires a rational individual, "whatever his temporal position… [to] choose for everyone."[7]

Through the use of these two theoretical devices, Rawls then argues that it is possible for us to then derive a "unanimous…conception of justice"[8] upon which we all agree. Indeed, he in fact argues not only that we are able to come to unanimous agreement on a conception of justice, but that there exists only one conception of justice upon which we can all rationally agree, namely the *Two Principles of Justice*.

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[5] *Ibid.* p. 136-142.
[6] *Ibid.* p. 137.
[7] *Ibid.* p.140.
[8] Rawls, John, *A Theory of Justice: Revised Edition op.cit*. p.121

2.1.2 – The First Principle of Justice

It is first important to note that the principles are ranked in lexical order, with the first principle lexically prior to the second.[9] So, what is the first principle? This principle, revised within *Justice as Fairness* reads:

> "Each person has the same indefeasible claim to a fully adequate scheme of equal basic liberties which scheme is compatible with the same scheme of liberties for all"[10].

The purpose of this principle is to ensure that whatever position an individual occupies in society, their right to certain liberties is protected. As this is lexically prior, Rawls claims that this is the supreme value that any self-interested individual would choose under the required theoretical constraints. The importance of basic liberties is clear. If you do not possess certain basic liberties, then it is difficult to achieve other goals. The possession of liberty is essentially a value-neutral precondition to the ability to fulfil one's own conception of the good, and a rational, self-interested individual should therefore choose to have an adequate scheme of them.

But what are the liberties that we are concerned with protecting? Within *Political Liberalism*, Rawls provides a list of the basic liberties to which the first principle relates. These are:

> "…freedom of thought and liberty of conscience; the political liberties and freedom of association, as well as the freedoms specified by the liberty and integrity of the person; and finally, the rights and liberties covered by the rule of law."[11]

It is these liberties to which an individual has an indefeasible claim. These liberties arise from specific characteristics within individuals, which Rawls terms the "two moral powers."[12] These powers are explained as those necessary for individuals to be

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[9] Rawls, John, *A Theory of Justice: Original Edition op. cit.* p. 250.

[10] Rawls, John, *Justice as Fairness: A Restatement*, (Cambridge Massachusetts: Belknap Press, 2001) p.42.

[11] Rawls, John, *Political Liberalism*, (Columbia: Columbia University Press, 2005) p.12.

[12] *Ibid*. p.19.

"full participants in…social cooperation," and are "a capacity for a sense of justice, and a capacity for a conception of the good."[13]

A *capacity for a sense of justice* requires that an individual be able to understand, apply, and act according to the requirements of justice. To possess the *capacity for a conception of the good*, an individual must be able to "form, to revise and rationally to pursue a conception of one's rational advantage or good".[14] The conception of the good is therefore concerned with individuals being able to pursue life plans or goals. The justification for protecting the above liberties is that these are the liberties required:

> "…to guarantee equally for all citizens the social conditions essential for the adequate development and the full and informed exercise of these powers."[15]

In examining the two *moral powers* outlined above, it becomes clear how the basic liberties laid out by Rawls are necessary to preserve these powers. For example, political liberties and freedom of association are essential to allow the "full and effective exercise of citizens´ sense of justice, to the basic structure of society."[16] Without these liberties, they are unable to apply their sense of justice to society and see it enacted (for example, through mechanisms such as voting).

Equally, the liberties of conscience and freedom of association:

> "…are to secure the full and informed and effective application of citizens´ powers of deliberative reason to their forming, revising, and rationally pursuing a conception of the good over a complete life."[17]

A lack of these liberties would infringe upon this ability to form a conception of the good, and therefore infringe upon the full exercise of one of the two moral powers. In order for an individual to be able to fully exercise their two *moral powers*, they must therefore possess a fully adequate scheme of these basic liberties, fully

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[13] *Ibid.*
[14] *Ibid.*
[15] Rawls, John, *The Basic Liberties and their Priority*, The Tanner Lectures on Human Values Delivered at the University of Michigan 1981, Accessible at [http://www.tannerlectures.utah.edu/lectures/documents/rawls82.pdf] Accessed 04/03/12 p. 47.
[16] Ibid p.49.
[17] Ibid. p.50.

adequate to be understood with regards to the exercise of these powers. However, within the thesis, there is little room for first principle considerations.

2.1.3 – The First Principle and My Question

Let us take the most extreme assumption as an example. Imagine an individual, who, when their insurer discovers their genetic status, sees their premium rise so that it is no longer affordable for them. Indeed, imagine that this individual is now essentially untouchable, and cannot obtain life insurance anywhere, from any insurer, for any price. It would not appear that this individual would see their access to a fully adequate scheme of basic liberties compromised. Moving through the list, it is difficult to envisage a situation whereby the denial of life insurance restricts either their freedom of thought or liberty of conscience, as this would appear to be much more relevant in cases of political repression by the state. Second, it would seem that their political liberties would not be meaningfully compromised either. It is important to remember here that within the UK we have full provision of social healthcare through the NHS, and that we are talking about life insurance rather than health insurance. As such, an individual will still be able to access healthcare, and their health will not deteriorate to the point where they are unable to exercise their basic liberties simply through the denial of insurance. Similarly, this individual would also retain bodily integrity, and any rights or liberties granted to them by the rule of law. Therefore, even under the most severe assumptions, the use or prohibition on use of this data by insurers does not have any meaningful effect on an individual's access to a scheme of basic liberties. The first principle is therefore more concerned with restricting the actions of a totalitarian or dictatorial state, and the establishment of basic political structures. Given this, I will proceed to the second principle, as it is this which the use of genetic data may affect.

2.1.4 – The Second Principle of Justice

Having established that my question does not meaningfully engage with the first principle of justice, we must move to the second. Again, the revised principle within *Political Liberalism* reads:

"Social and economic inequalities are to satisfy two conditions: first, they are to be attached to positions and offices open to all under conditions of fair equality of opportunity; and second, they are to be to the greatest benefit of the least advantaged members of society."[18]

We must first consider whether the ability to obtain social and economic goods (hereafter termed *primary goods*) is available under conditions of fair equality of opportunity, and then second, whether these inequalities are to the benefit of the least advantaged members of society. The second principle of justice is a rejection of strict egalitarianism. It recognises that, given equal basic liberties, it may in fact be beneficial to all of us to allow inequalities in primary goods to exist, rather than to each have a strictly equal share. As an example, it may be justifiable under the second principle to pay medical professionals more than other jobs, provided that the education and skills required to obtain this job are available to all under conditions of fair equality of opportunity. The attachment of higher wages to this profession may encourage the most talented individuals to work in this field, and a society with the most talented medical professionals is beneficial to the poorest as they can obtain better medical care (assuming the presence of a social health system like that currently provided by the UK). We can also see from the second principle that the inequalities we concern ourselves with are merely "social and economic inequalities", and not every inequality *per se*. To understand this requires a very brief exploration of the literature on social and natural inequality.

2.1.5 – Social and Natural Inequality

Within *A Theory of Justice*, Rawls draws a distinction between what he terms "natural good[s]" and social goods, natural goods being those which are not "directly under [the] control" of society.[19] Within this section, he gives an individual's health status as an example of a natural good. Of course this is true; we do not control the particular natural endowments that an individual may be born with, nor their genetic status.[20] We do however control the structure of the society in which this individual

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[18] Rawls, John, *Political Liberalism, op. cit.* p.181.

[19] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.*, p. 223.

[20] Although with the rise in foetal genetic screening and pre-implantation genetic diagnosis, the distinction between natural and social inequalities continues to erode. Recent discussions of the theory focus on this erosion, and whether, in future, the principles of justice will require pre-emptively levelling natural inequality. There is much literature on this area, but for more, see: Buchanan, Allan, *Equal Opportunity and Genetic Intervention*, Social Philosophy and Policy (1995) 12 (2) 105-135.

will find themselves in for example, their access to healthcare to mitigate their disadvantage, rules against discrimination based on natural characteristics, rules on accessibility for the disabled, etc.

Indeed, even the first discussion of the theory within *A Theory of Justice* explicitly concerns itself with levelling natural inequality, searching as it does for:

> "…a conception of justice that nullifies the accidents of natural endowment ... as counters in quest for political and economic advantage..."[21]

At the very least then, even if we either *should not* or *cannot* level natural inequality in itself, we are bound to nullify their effect on either political or economic advantage. As Buchanan notes, the central purpose of the whole endeavour, especially the second principle is to ensure that:

> "…individuals be compensated for having lower life-prospects as a result of their (less fortunate or undeserved) natural or social endowments"[22]

Whether an individual is economically disadvantaged due to social or natural factors is utterly irrelevant here. If they are disadvantaged through no fault of their own, the principles of justice require us to intervene either to provide an equal distribution, or an unequal distribution which is to the benefit of members of the least advantaged group.

The question of whether we should attempt to correct natural inequality itself is outside the scope of this thesis. Instead, my specific focus within this piece is twofold. First, does the possession of a certain genetic status lead to an individual being unable to compete under conditions of fair equality of opportunity, and therefore, prevent them from obtaining equal primary goods over the course of a whole life? Second, if there is such a disadvantage, how should we approach the use of this genetic data in order to fulfil our obligations under the principles of justice? The approach I am taking within the thesis is therefore the one explicitly suggested by Rawls, the use of social structures to compensate for inequality (insurance and genetic status respectively).

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[21] Rawls, John, *A Theory of Justice: Original Edition, op.cit.* p. 15.
[22] Buchanan, Allan, *Equal Opportunity and Genetic Intervention*, *op. cit.*

As I am concerned with genetic data which may lead to the development of future illness, access to healthcare is also important here. When discussing this, I am grateful to Norman Daniels, who within *Just Health* (amongst other publications) makes the case for health and healthcare to be considered as concerns of Rawlsian justice. He does not however suggest that these should be added to the list of primary goods, as he fears to do so would leave us "likely to lose our shared political conception of the needs of citizens."[23] He identifies that several other essential goods such as food, clothing [and] shelter" are not primary goods per-se, but simply expected to be "adequately supported by fair shares of income and wealth".[24] Instead, he suggests the appropriate mechanism through which we should confront health and healthcare inequalities is that of fair equality of opportunity. If an individual is unable to compete fairly for primary goods due to their health status, they are entitled as a matter of justice to assistance rectifying this unfairness. A proper understanding of the difference principle also requires a concern for health inequality. Insofar as I am able to demonstrate that genetic status leads to ill-health, and that ill-health compromises either fair equality of opportunity or the ability to obtain primary goods, we must be legitimately concerned. Even under the most strict understanding of our obligations (dismissing any requirement to level natural inequality), we must still structure societal and political structures so as to fulfill the requirements of justice. Regulation relating to the use of genetic data is part of this.

Fair equality of opportunity is therefore as this denial of opportunity means that individuals are less able to obtain primary goods that they otherwise would be able to possess, something which violates the principles of justice. However, we must now answer two further questions before we are able to apply the principle. First, what are the primary goods we are concerned about distributing? And second, how are we to define fair equality of opportunity, and why is it relevant to the thesis question? The rest of the Chapter will be devoted to answering these two questions. First, the primary goods.

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[23] Daniels, Norman, Just *Health: Meeting Health Needs Fairly*, (Cambridge: Cambridge University Press, 2008) p. 56.
[24] *Ibid.*

2.2 – The Primary Goods

Within this section, I will first offer a very brief account of which goods we are concerned about distributing, before proceeding to explain the concept of *capability sets* and its relevance to my question. Finally, I will defend my specific focus on the good of income and wealth.

2.2.1 – What are the Primary Goods?

If we look again at the two principles, we find that both are concerned with *distribution*, to a Rawlsian, justice is all about distributing things fairly between different individuals. The currency we are concerned about distributing is that of *primary goods*.[25] So, what are these primary goods? Within *A Theory of Justice* Rawls provides a basic list, suggesting that these goods are "rights, liberties, and opportunities, and income and wealth."[26] This list is somewhat unhelpful in its brevity, but further clarification is provided within later work, specifically a list of primary goods which reads as follows:

> "
> a. basic rights and liberties, also given by a list;
> b. freedom of movement and free choice of occupation against a background of diverse opportunities;
> c. powers and prerogative of offices and positions of responsibility in the political and economic institutions of the basic structure;
> d. income and wealth; and finally
> e. the social bases of self-respect."[27]

Within this account, we have already discussed a. b. and c., as these relate to the list of basic liberties provided in *2.1.2*. Broadly speaking, the first principle is concerned with the distribution of those primary goods identified under a to c. and the second principle with the distribution of d. and e. Within the thesis, I will be primarily focused on the distribution of income and wealth rather than the social bases of self-respect.

Looking again at the list of primary goods and the second principle of justice, we know that our starting point is that individuals should possess each of these

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[25] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.*, p. 54.
[26] *Ibid.*
[27] Rawls, John, *Political Liberalism, op. cit.* p.181.

goods equally, unless an unequal distribution benefits the least advantaged group. We also know that the justification for the primary goods is that they constitute "things that every rational man is presumed to want" and which "normally have a use whatever a person's rational plan of life."[28] It is therefore claimed that, as these individuals are both self-interested and rational, that "given human nature, wanting them is part of being rational."[29] The list of goods above is therefore perhaps better expressed as a list of citizens' needs. Given that we wish to allow citizens to pursue rational plans of life, the political question then essentially becomes *what do citizens need to pursue these plans?* What the list describes is what should "be publicly recognised as citizens' needs and hence as advantageous for all."[30]

What is important here is not that an individual accepts that *primary good X* is beneficial to their life plan, because it may not be. Instead, the individual merely has to accept that it may be necessary for other individuals' life plans, and may have been necessary for a plan that they may have wished to pursue, or may wish to pursue in future. The individual does not have to value any of these specific goods; only recognise that they may indeed be valued by a different individual.

2.2.2 – Primary Goods and Capability Sets

Before we proceed to examine fair equality of opportunity, there exists a problem with the primary goods account, identified by *Amartya Sen*. Specifically, he criticises the primary goods account as one which is unable to take account of differing capability between citizens, and therefore, an account which fails to produce a just system. Indeed, he claims that:

> "…equality of holdings of primary goods or of resources can go hand in hand with serious inequalities in actual freedoms enjoyed by different persons."[31]

The focus here is on substantive freedom. Two individuals may possess the same bundle of primary goods, but one of them may be unable to convert these

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[28] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.*, p. 54.
[29] *Ibid*. p.223.
[30] *Ibid*. p.179
[31] Sen, Amartya, *Inequality Reexamined*, (Cambridge Massachusetts: Harvard University Press 1995). p.81.

bundles into actual freedoms. Given the research question is concerned with genetic markers which may be predictive of future disease, the potential for capability to be limited may be important here. As we acknowledged earlier, primary goods are simply means to ends, in that they are the basic necessities required for an individual to advance their conception of the good. Sen claims that inequalities can arise during the conversion of these all-purpose means to individual specific ends.

So what of the distribution of the primary goods under the second principle? Thinking specifically about genetic data and the potential for future ill health, what about the individual who cannot use their primary goods as well as another because they are disabled, or ill? They would seem neither to have chosen this, nor to be able to rectify this by changing their preferences. This is a concern first highlighted by Arrow in his 1973 paper, and it would seem similar to the concerns which Sen expresses.[32]

One way of beginning to solve this objection is to consider the scope of the primary goods. Earlier in the Chapter, I briefly explained how we are able to consider health and healthcare needs as facilitative of fair equality of opportunity, and the ability to obtain primary goods. Let us start with an idea of what health is. For our purposes:

> "…the basic idea is that health is the absence of disease, and diseases (I include deformities and disabilities that result from trauma) are deviations from the normal functional organization of a typical member of a species."[33]

Therefore, if an individual has sub-normal functioning for a member of their species, they are considered to have a disease. If they function normally, they are considered to be healthy. Accepting this definition, we can begin to form a claim that the second principle of justice does indeed deal with the criticisms levelled at it by Arrow and Sen. Specifically, our commitment to the idea of *fair equality of opportunity* means that a justifiable application of these principles will somewhat solve this problem.

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[32] Arrow, Kenneth, *Some Ordinalist-Utilitarian Notes on Rawls Theory of Justice*, Journal of Philosophy (1973) 70, p.253.

[33] Daniels, Norman, *Equality of What: Welfare, Resources, or Capabilities?* Philosophy and Phenomenological Research, (1990) Vol. 1 Supplement p. 275.

There is no doubt that this individual may be less able to compete for opportunities (such as employment) to which primary goods are attached due to their disease or disability. However, a proper application of these principles will necessitate provision of adequate healthcare, as the healthcare an individual receives for their condition can determine whether they are able to compete under conditions of fair equality of opportunity. A commitment to allowing these individuals to compete fairly means that we are committed to rectifying or mitigating the effects of any disease, as possession of a disease would mean that they were functioning and therefore competing sub-normally.

However, this commitment to provide healthcare does not completely satisfy this objection. As we will come to find later in the thesis when we discuss my representative conditions, certain conditions cannot be cured, and their symptoms cannot currently be mitigated. As such, there will exist individuals whose capability is compromised through no fault of their own, and who we are unable to assist through providing access to healthcare. These individuals are entitled to receive aid through the structure of society more widely, and it may therefore be unjustifiable to allow the use of their genetic data. Indeed, it would seem that our underlying commitments bind us to considering reduced capability when considering whether it is legitimate to allow the use of their data. The purpose of the second principle is to "attempt to mitigate the arbitrariness of natural contingency and superficial fortune."[34]

Indeed, as we know from earlier, this is one of the guiding principles of our entire theory. The idea of justice as fairness starts from the belief that the fact that certain individuals are advantaged or disadvantaged through the operation of natural and social lotteries is unjustifiable, and we should work to rectify this. Indeed, within *Political Liberalism*, Rawls explicitly assumes that everyone within society will be a "normal cooperating member of society" and therefore must never confront the situation where these genetically disadvantaged individuals exist.[35] This focus on normal members of society, along with the fact that it was written in the early 1970's explains why there is no explicit answer to the question of genetic inequality within

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[34] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.*, p. 82.
[35] Rawls, John, *Political Liberalism*, *op. cit.* p.184.

his work. As my thesis concerns the real world rather than the ideal, I must be able to account for these genetically disadvantaged individuals within my methodology, especially given that they are the focus of my research question.

Although the Rawlsian theory is an 'ideal theory', offering as it does a picture of the ideal world rather than strategies for dealing with the non-ideal world in which we currently live, we do have anecdotal evidence which suggests he would have approved of corrective measures such as this. Indeed, Thomas Nagel recalls Rawls expressing strong support for affirmative action programs within the United States, recognising that African-American individuals were unable to compete normally under the status-quo.[36] Corrective efforts to either return these individuals to being able to compete normally or to compensate for their disadvantage are therefore within the spirit of the Rawlsian ideal.

As such, we must consider the fact that these individuals are unable to convert their primary goods into their own conceptions of the good as effectively as others, merely through bad luck in the natural lottery. This inability to convert their goods as effectively will become important later when I begin to assess both which individuals are likely have either their fair equality of opportunity or capability to use primary goods compromised, and therefore whether individuals such as this can be legitimately disadvantaged further.

2.2.3 – Why Income and Wealth?

Within the thesis, I intend to assess the distribution of one specific primary good, that of income and wealth. This section will provide an explanation of why the other primary goods will not be considered here. We know from *2.2.1* that a., b. and c. in the list relate to the first principle rather than the second, and from *2.1.3* why I am not considering basic rights and liberties within the context of this thesis. But what of e)? The reasoning is again fairly simple, but it is worth highlighting in brief here.

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[36] Nagel, Thomas, *John Rawls and Affirmative Action*, The Journal of Blacks in Higher Education, (2003) 39 pp.82-84.

First, what are the social bases of self-respect? Within the theory, the good of self-respect serves to, in wider form, represent a "stronger variant of the Kantian idea, that is, the idea of always treating persons solely as ends and never in any way as means."[37] This idea of self-respect is fleshed out within a later paper on Kantian Constructivism, which claims these bases are:

> "…those aspects of basic institutions which are normally essential if individuals are to have a lively sense of their own worth as moral persons, and to be able to realize their higher order interests and advance their ends with zest and self-confidence."[38]

Further, within *A Theory of Justice*, we find a longer description of the reasoning behind these bases, which reads:

> "We may define self-respect . . . to have two aspects. First of all . . . it includes a person's sense of his own value [understood as] his secure conviction that his conception of the good, his plan of life, is worth carrying out. And second, self-respect implies a confidence in one's ability, so far as it is within one's power, to fulfil one's intentions."[39]

This definition of self-respect requires two things be fulfilled, first, that an individual feels that their conception of the good is seen as valid by society and second, that they are able to advance this conception with confidence. Indeed, Rawls claims that this principle requires that individuals be able to access both communities of similar shared interests, and that their individual aims are respected by others.[40] However, in much the same way as the basic liberties discussed within *2.1.3*, the use or prohibition on use of genetic test data within the insurance industry does not meaningfully impact the social bases of self-respect. Again taking the most extreme assumption from *2.1.3*, where an individual finds themselves completely unable to obtain life insurance, this is unlikely to significantly undermine their ability to live within a community which validates and confirms their conception of the good. Indeed, much like the basic liberties, this primary good would appear to concern itself with preventing the state from affirming certain conceptions of the good, or from explicitly or implicitly condemning other conceptions as wrong.

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[37] Rawls, John, *Distributive Justice: Some Addenda*, in Freeman, Samuel ed. *John Rawls: Collected Papers*, (London: Harvard University Press 1999) p.171.

[38] Rawls, John, *Kantian Constructivism in Moral Theory*, in Freeman, Samuel ed. *John Rawls: Collected Papers*, *op. cit.* p. 314.

[39] Rawls, John, *A Theory of Justice: Original Edition op. cit.* p. 440.

[40] *Ibid.* p. 442.

When considering the ability to obtain life insurance, and the potential variance in premium pricing that could result from the use or prohibition on use of genetic test data, it is the good of income and wealth which is affected. It is this which grounds the specific focus on income and wealth as the primary good of relevance within the thesis as a whole. In addition to this primary good, the ability to obtain it and compete under conditions of fair equality of opportunity is also relevant. It is to there we now proceed.

2.3 – Fair Equality of Opportunity

We know from the second principle of justice that we are also concerned with fair equality of opportunity. However, we still do not know what equality this principle requires, nor the relevance of the principle to the thesis question. It is this which will be discussed here.

2.3.1 – What is Fair Equality of Opportunity?

Again looking to *A Theory of Justice* first, we find that the concept of fair equality of opportunity is designed to ensure that:

> "In all sectors of society there should be roughly equal prospects of culture and achievement for everyone similarly motivated and endowed".[41]

Within the context of the basic liberties, this means that:

> "…the worth of the political liberties to all citizens, whatever their social or economic position, must be approximately equal, or at least sufficiently equal, in the sense that everyone has a fair opportunity to hold public office and to influence the outcome of political decisions."[42]

Moving away from the basic liberties and toward the second principle, we can see above that the principle of fair equality of opportunity is one which applies to all sectors of society. Specifically, we are concerned about an individual having an equal chance, whatever their social or economic position, to obtain the positions to

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[41] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.* p. 73.
[42] Rawls, John, *The Basic Liberties and their Priority, op. cit.* p. 42.

which primary goods are attached. Take for example, employment. If two individuals both aspire to be doctors, given the same amount of effort from both of them, fair equality of opportunity would require they have equal prospects of achieving their goals. If one of these individuals grows up in a rich household and attends private school whilst the other has parents who are unable to afford such an education, it is likely that the wealthier child will have a higher chance of success. This is a violation of fair equality of opportunity, and something which the state must act to correct. In this case it could, for example, offer tutoring or higher quality education to the poorer child funded by the state in order to increase their opportunity. The principle is essentially one which requires a *level playing field*, it does not mandate that individuals do in fact achieve equally, only that they have equal prospects to, unhindered by factors which they have not chosen (such as their parents, social background or genetic makeup).

Fair equality of opportunity (or the denial of it) is therefore capable of affecting the distribution of primary goods, in our case, income and wealth. To fulfil this requirement, individuals with roughly equal desire to achieve should be able to achieve roughly equal shares of income and wealth, regardless of their social or natural circumstances, and society should be constructed to aid them in this end.[43] As we are concerned with roughly equal access to shares of income and wealth, we must therefore concern ourselves with roughly equal access to the ability to obtain and maintain employment, as it is through employment that an individual will earn the vast majority of their lifetime share of income and wealth. An individual should therefore be roughly equally able to access the same employment regardless of their starting position within either the natural or social lottery. Given this, how is this relevant to the question I wish to answer?

2.3.2 – How does fair equality of opportunity engage with genetics?

The concept of fair equality of opportunity becomes relevant when we examine which individuals we are going to consider. These individuals are those who possess genetic markers which point to the development of future disease, as

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[43] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.* p. 73.

these are the markers which would interest insurers.[44] Because of this, there exists the possibility (or near certainty) of these individuals developing future disease through no fault of their own.

First, this means that these individuals may have their fair equality of opportunity compromised with regard to access to employment, as an individual suffering from a severe disease may be unable to compete equally with an identical individual who does not suffer from the disease. One possible way of rectifying the situation would be through provision of healthcare to the individual, curing or substantially mitigating their condition and returning their ability to compete fairly. When considering the ability of these individuals to obtain income and wealth over the course of a whole life, some of these individuals will be disadvantaged. The fact that they are no longer able to compete fairly for positions of employment means that they are also less likely to be able to obtain the income and wealth which is attached to those positions. Therefore this loss of opportunity may also lead to a loss of important primary goods. This means that they are meaningfully disadvantaged, and that it may be unjust to disadvantage them further through the use of their data. Depending on the extent of the condition and resulting disadvantage, it may even be that these individuals could meaningfully be considered members of the least advantaged group over the course of their life. Membership of this group becomes relevant when we assess whether it is justifiable to potentially disadvantage them further through allowing the use of their test data by insurers. If they are not members of this group, it may also be that preventing the use of their data is unjustifiable, as that may also operate to harm members of the least advantaged group. These are all factors I will consider within my later analysis, specifically within *Chapter Five*.

2.4 – Conclusions

Within my later analysis, there are therefore three factors to consider. These are:

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[44] This is because these markers influence actuarial decisions on risk, and therefore, influence the premiums that these individuals are charged, consistent with the principles of mutuality and actuarial fairness.

a) Whether fair equality of opportunity is compromised by possession of this genetic data, and whether this affects the ability to obtain primary goods,

b) Whether capability is compromised by possession of this genetic data, and

c) The effect of prohibiting the use of this data on members of the least advantaged group

If the possession of a certain genetic status leads an individual to be disadvantaged either in terms of fair equality of opportunity (and therefore possession of primary goods), or the capability to use them, then allowing the use of their data might be unjustifiable. However, it may also be possible that not allowing this use harms members of the least advantaged group, in violation of the *difference principle*. If members of this group are harmed through denying use of this data, it may then be that the interests of justice require us to allow insurers access to the data in question. However, before I proceed to substantially analyse fair equality of opportunity and capability in this context, allow me to first offer a preliminary assessment of justifiability premised on membership of the least advantaged group, and identify the problems with that account. This will be the substantive content of *Chapter Three*.

Chapter Three
Preliminary Assessment of Justifiability

3.1. Introduction

Within this Chapter I will provide a preliminary assessment of justifiability, focusing on the possession of income and wealth. First, I will establish both that within the insurance context, the use (or prohibition on use) of test data is capable of affecting wealth through the mechanism of premium pricing, and second, that it is possible to identify those individuals who possess high income and wealth through the use of policy value as an appropriate proxy. I will then proceed to provide a preliminary assessment of justifiability focusing on the identification of individuals who are members of the least advantaged group, and as such, those individuals who it is unjustifiable to further disadvantage through allowing the use of their data. Finally, I will identify several significant problems with the assessment of justifiability in this way. The purpose of this Chapter is both to defend my use of policy value as a proxy, and to demonstrate both the insufficiency and difficulty of assessing the issue purely through reference to membership of the least advantaged group. This will then inform the more nuanced analysis offered within *Chapter Four* and more substantially within *Chapter Five*.

3.1.1 – How is Wealth to be assessed?

From *Chapter Two*, we know that when assessing the use of genetic information by insurers, the only primary good which may be meaningfully affected is that of income and wealth. It is through the distribution of these primary goods that we assess which individuals are members of the least advantaged group, and specifically in this case, whether the distribution of wealth is in accordance with the principles of justice. The issue here is the variation in insurance policy price an individual would receive were insurers to be able to use their test data. Looking at the list of primary goods, it is only iv), income and wealth, that could be affected by a change in policy price. Even in the worst case scenario where an individual is

priced out of insurance altogether, they will not have their possession of any of the other primary goods affected. When we assess which individuals may be members of the least advantaged group, we are in this case assessing an individuals' possession of income and wealth. If a policy further disadvantages members of the least advantaged group it is unjustifiable. If it does not, it may not be. Further, it is reasonable to suggest that there exists a level of income and wealth whereby an individual can no longer be considered a member of this group. One suggested boundary is the lowest wealth quartile.[1] If we are able to identify these individuals who are not members of the least advantaged group, it is not *prima facie* unjustifiable to disadvantage them through allowing insurers to use their test data. This becomes relevant later when we discuss adverse selection, as it is possible that preventing insurers from using certain data could disadvantage all policy holders, including those who are members of the least advantaged group.

3.1.2 – Why does the use of test data affect wealth?

The issue here is one of increasing premiums. The UK insurance industry operates broadly on a system of mutuality, whereby an individual pays a differentiated premium representative of the risk that they bring to the insured pool. These individual risks are aggregated to produce wide risk categories within which an individual will fall. Assuming rational economic behavior and profit motives, the insurance company will charge a higher premium to an individual if they present a higher risk to the pool, that is, if they are more likely to make a claim (or make a larger claim) on their policy. Specifically within the life insurance context, we therefore care about how likely an individual is to die over the insured period, and how much is to be paid out as a result of death. If an individual carries genetic markers predisposing them to a certain condition, they will in many cases be at a higher risk of developing this condition than someone who does not carry said markers. If this condition is sufficiently serious, they may then be at a higher risk of death during the insured period. If insurers are allowed to use this data when

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[1] Rawls suggests one possible way of assessing membership of this group, namely "all persons with less than half of the median income and wealth", although he concedes this will always have a "certain arbitrariness" and be somewhat "ad hoc." Another way of phrasing "less than half of the median" is to refer to the lowest wealth quartile, and that is what I will be doing in the thesis. Rawls, John, *A Theory of Justice: Revised Edition, op. cit.*, p. 98.

assessing risk, the individual may be seen as more risky to insure, meaning they will pay a higher premium for the same level of cover. This directly affects the individuals' total level of wealth, a primary good. If the individual can legitimately be considered a member of the least advantaged group, further disadvantaging them in this way is unjustifiable.

Preventing the use of this genetic data also potentially raises an issue with respect to premium pricing. In theory, an individual who knows that they are at higher risk due to the results of a genetic test will likely purchase a higher level of insurance than an individual at lower risk, as they are more likely to need to claim on the policy. Equally, individuals who know they are at low risk of developing specific conditions may choose to purchase a less comprehensive policy, or not purchase insurance at all, confident that they will not need to make a claim. The insured pool then contains many individuals whose risk is higher than their risk assessment would suggest, and fewer individuals whose risk is perhaps overestimated. The insurer therefore suffers a hit to profit as they pay out on more policies than their risk assessment would suggest. This cost to the insurer is then passed to the consumer in the form of an increase in premiums. A denial of actuarially relevant information to insurers in this case leads to what the industry terms 'adverse selection'. This is a state in which the "insurer cannot determine some characteristics of the insured that are relevant to the determination of the probability of the future state of nature."[2] Essentially, this is an issue of asymmetric information where the insured individual is able to better assess their risk than the insurer.[3] This increase in premiums as a result of adverse selection means that denial of actuarially relevant information may act to harm those who are members of the least advantaged group, and therefore, that this denial of information would be unjustifiable under the *difference principle*.

Moving beyond the theoretical, empirical studies such as Cawley and Philipson's studies on the US life insurance market[4], and Cardon and Hendel's

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[2] Pauly, Mark, *Overinsurance and Public Provision of Insurance: The Roles of Moral Hazard and Adverse Selection*, The Quarterly Journal of Economics (1974) 88 (1) p. 44-45.
[3] Arrow, Kenneth, *Political and Economic Evaluation of Social Effects and Externalities*, in *The Analysis of Public Output*, Margolis, J. ed. (New York: Columbia University Press 1970) p.11.
[4] Cawley, J. and Philipson, T., *An Empirical Examination of Information Barriers to Trade in Insurance*, American Economic Review (1999) 89 (4) 827-846.

studies on US health insurance[5] both find that this positive correlation between risk and possession of insurance is not necessarily supported. Indeed, in the life insurance studies, the authors "…found evidence of bulk discounts, multiple contracting, and a negative covariance between risk and quantity. Each of these is the opposite of the predicted pattern."[6] Specifically, Cawley and Phillipson find that within the US life insurance context, there is actually a neutral or negative relationship between the risk an individual poses, and the amount of insurance they possess. Further, it is suggested that this might be because insurers act, over time, to limit coverage to high risk individuals, or that insurers may be able to overcome information asymmetry through analysis of costs of production and market trends.

In contrast, evidence of adverse selection due to asymmetric information can be found both in US Automobile insurance[7], and a substantial amount of literature relating to health insurance markets.[8] Support for this positive correlation can also be found in US studies by Ettner[9] and Finkelstein[10], and a Swiss study by Gardiol et. al.[11] Finally, an assessment of the potential impact of a specific Medicare reform found that adverse selection would lead to an increase in premium price for those remaining in the Medicare 'fee-for-service' program.[12] The empirical evidence from the US is therefore mixed.

We can find help with the UK context by examining a study of the UK Annuity market published in 2004. Within this study, we find that whilst there is little evidence that adverse selection affects the amount payable if the insured event

[5] Cardon, J. and Hendel, I., *Asymmetric Information in Health Insurance: Evidence from the National Medical Expenditure Survey*, RAND Journal of Economics (2001) 32 (3) 408-427.

[6] Cawley, J. and Philipson, T., *An Empirical Examination of Information Barriers to Trade in Insurance, op. cit.*, p. 842.

[7] Cohen, A., *Asymmetric Information and Learning in the Automobile Insurance Market*, Harvard Law School John M. Olin Center For Law, Economics and Business Discussion Paper Series, 2002, Paper 371. Accessible at: [http://lsr.nellco.org/harvard_olin/371], Accessed 06-02-2012.

[8] Cutler, D., *Health Care and the Public Sector*, in Auerbach, A. and Felstein, M. eds.*Handbook of Public Economics, Volume 4*, (Amsterdam: North Holland 2002).

[9] Ettner, S.L., *Adverse Selection and the Purchase of Medigap Insurance by the Elderly*, Journal of Health Economics, (1997) 16 (5) 543-562.

[10] Finkelstein, A., *Minimum Standards, Insurance Regulation, and Adverse Selection: Evidence from the Medigap Market*, Journal of Public Economics (2004) 88 (12) 2515-2547.

[11] Gardiol, L. Geoffard, P. and Grandchamp, C,, *Separating Selection and Incentive Effects: an Econometric Study of Swiss Health Insurance Claims Data*, CEPR Working Paper 5380, 2003, Accessible at: [http://www.cepr.org/meets/wkcn/3/3519/papers/Geoffard.pdf]. Accessed 06-02-2012.

[12] Rice, T. and Desmond, K., *The Distributional Consequences of a Medicare Premium Support Proposal*, Journal of Health Politics, Policy and Law (2004) 29 (6) 1187-1226.

occurs, there is significant evidence that both mortality rates and policy pricing do act consistently with adverse selection.[13] The study stresses the importance of studying different dimensions of the insurance contract, as some may be vulnerable to adverse selection whilst others are not. This could also offer an explanation of the divide in the literature. Again, as highlighted in the study, the amount of payment if the insured event occurs is the dimension of the contract most studied in other papers, and is the dimension that does not show adverse selection.[14]

Of the studies cited, the only one which considers multiple dimensions of the insurance contract is that performed by Finkelstein and Poterba. Equally, this is the only study which relates to the UK insurance market, and their results support the theoretical position outlined earlier, namely that a positive correlation exists between an individuals' perceived risk and their possession of insurance. If higher risk individuals purchase more insurance, and insurers cannot account for this, then adverse selection occurs. Further, this study demonstrates that the insurer response to adverse selection is a rise in premium price for all. Another 2008 study carried out on behalf of the Institute for Fiscal Studies found that "…adverse selection is present in the British private health insurance market."[15]

The risk of adverse selection is even higher within the life insurance context. Unlike health insurance, where the predicted costs of healthcare impose a soft limit upon the coverage an individual will purchase, there is no such limit on life insurance.[16] In addition, these individuals are also able to purchase coverage from more than one provider, or buy more than one policy, preventing the use of limiting mechanisms such as price quantity contracts or non-linear pricing.[17] The impact of adverse selection may therefore be considerable, as can be seen in empirical studies

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[13] Finkelstein, A. and Poterba, J, *Adverse Selection in Insurance Markets: Policyholder Evidence from the UK Annuity Market*, Journal of Political Economy (2004) 112 (1) 1. p. 206.
[14] *Ibid.*
[15] Olivella, P. and Hernandez, M., *Testing for Adverse Selection into Private Medical Insurance*, Institute for Fiscal Studies Working Paper 06/02, Accessible at: [http://www.ifs.org.uk/wps/wp0602.pdf], Accessed 06-02-2012. p. 31.
[16] Butler, James, *Adverse Selection, Genetic Testing and Life Insurance – Lessons from Health Insurance in Australia*, Agenda (2003) 10 (1) p.85.
[17] Hoy, M and Polborn, M, *The Value of Genetic Information in the Life Insurance Market*, Journal of Public Economics (2000) 78 p. 235-252.

by Subramanian et. al. which show that even under strict underwriting conditions, adverse selection (in this case, for BRCA mutations) produces premium increases.[18]

The empirical studies above suggest both a) that adverse selection is capable of occurring when information asymmetry occurs, and b) that this is responded to by an increase in premium pricing, especially in the life insurance context. Both the use of, and prohibition on the use of test data are therefore capable of affecting individual wealth, and therefore affecting a primary good. If an individual is wealthy enough to not be considered a member of the least advantaged group, it may be justifiable to disadvantage them by allowing the use of their test data in order to prevent a rise in premiums for all (and therefore for members of the least advantaged group) as a response to adverse selection.

3.2 – Policy Value as a Proxy for Individual Wealth

Of course, in order to assess whether these individuals are or are not members of the least advantaged group, I first need to be able to identify who these individuals are, and how much income and wealth they possess. A possible solution is through the use of the sum insured as a proxy for individual wealth. If this is an appropriate proxy, then policy setting may become easier, as we may be able to identify a level of coverage that cannot possibly include any members of the least advantaged group. But is this a valid proxy?

3.2.1 – Do increased policy values correlate with wealthier consumers?

First, it can be demonstrated empirically that an increase in the wealth possessed by individuals does indeed lead to increased possession of life insurance. Early studies found that an increase in life insurance correlated with an increase in income[19], as well as total assets, and net worth.[20] This correlation between increased

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[18] Subramanian, K., et. al. *'Estimating Adverse Selection Costs from Genetic Testing for Breast and Ovarian Cancer: The Case of Life Insurance'* (1999) Journal of Risk and Insurance 66 (4) p. 531-550.
[19] Ann Arbor: Survey Research Center, *Life Insurance Ownership Among American Families* (1952), Institute of Social Research, University of Michigan.
[20] Hammond, J.D. et. al., *Determinants of Household Life Insurance Premium Expenditures: An Empirical Investigation*, The Journal of Risk and Insurance (1967) 34 (3) 397-408.

individual income and increased insurance possession can also be found in surveys of consumer finances both in the past,[21] and more recently.[22] Indeed, the recent survey cited found that:

> "Ownership of cash value insurance is broadly spread across demographic groups, with a tendency toward increasing rates among families with higher levels of income and wealth and those with older family heads."[23]

The correlation is further reinforced by a 2002 study on household portfolio allocations, which demonstrates that as earnings rise, so does the tendency to invest in non-liquid assets such as insurance. Chambers and Schlagenhauf found that whilst the lowest earning quartile held approximately 70.9% of their portfolio in liquid form, this fell sharply when considering the second quartile of income, and even further beyond that.[24] Therefore, as individual wealth rises, the likelihood of an individual possessing life insurance at all also rises. This correlation is also suggested by industry publications on the demand for health insurance, such as those by Deloitte.[25] In addition to the early empirical studies, survey data and industry publications, we can also find comprehensive academic work demonstrating the link between individual incomes and an increase in the sum insured, such as those offered by Berekson,[26] Anderson,[27] and Greene.[28] However, as later studies demonstrate, wealth is not the only determinant of the sum held.

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[21] Katona, G. et. al., *1964 Survey of Consumer Finances* (1965) Ann Arbor: Survey Research Center, Institute of Social Research, University of Michigan.

[22] Bucks, B. et. al., *Recent Changes in US Family Finances: Evidence from the 2001 and 2004 Survey of Consumer Finances*, Accessible at: [http://www.federalreserve.gov/pubs/bulletin/2006/financesurvey.pdf] Accessed 08-02-12. p. A11.

[23] Bucks, B. et. al., *Recent Changes in US Family Finances: Evidence from the 2001 and 2004 Survey of Consumer Finances*, *op. cit.*, p. A17.

[24] Chambers, M. and Schlagenhauf, D., *Household Portfolio Allocations, Life Cycle Effects and Anticipated Inflation*, (2002) Working Paper, Accessible at: [http://mailer.fsu.edu/~dschlage/garnet-dschlage/money.pdf] Accessed 08-02-12.

[25] Deloitte Access Economics, *Macroeconomic drivers of private health insurance coverage*, (2011), Accessible at: [https://www.deloitteaccesseconomics.com.au/uploads/File/nib%20report%20WEB%20final%20010911.pdf] Accessed 08-02-12.

[26] Berekson, L., *Birth Order, Anxiety, Affiliation and the Purchase of Life Insurance*, The Journal of Risk and Insurance (1972) XXXIX. (1) 93-108.

[27] Anderson, D. and Nevin, J., *Determinants of Young Marrieds' Life Insurance Purchasing Behaviour: An Empirical Investigation*, The Journal of Risk and Insurance, (1975) XLII. (3) 375-387.

[28] Greene, M., *Attitudes Toward Risk and a Theory of Insurance Consumption*, The Journal of Insurance, (1963) XXX (2) 165-182.

For example, several publications also highlight other factors. Anderson identifies that both the level of education of the purchaser, and the behaviour of the insurance agent both influence the amount of insurance purchased.[29] Further, individuals who rely upon an insurance agent demonstrate different purchasing behaviour than those who do not.[30] In a later study on married couples, Ferber and Lee find that while "The primary determinant of life insurance purchases tends to be some measure of financial status"[31]; other characteristics are also influential, including whether the husband is in control of the family finances, and the attitudes of the couple towards saving.[32] Other studies find correlations between an increase in insurance purchase and factors such as city size, education, occupation, and age. [33,34]

So far then, it would certainly appear that an individual may hold a larger policy value for many reasons, only one of which being an increase in their wealth. We now need to determine how significant a contributor wealth is to this purchasing decision in order to be able to tell whether the amount of life insurance held is a legitimate proxy for individual wealth. A 1984 study by Burnett and Palmer can help. Within the study, they examined the relation of differing levels of life insurance ownership to the presence of certain demographic and psychographic characteristics. These characteristics were then assessed using multiple classification analysis in order to determine the significance of their contribution to the amount of insurance held. At the conclusion of this study, Burnett and Palmer found that of the nine demographic variables studied, three were of statistical significance, namely income, level of education and number of children.[35] Meanwhile, amongst the twenty-two psychographic characteristics considered, fourteen were statistically significant, including whether the individual classified themselves as a risk taker,

[29] Anderson, D. and Nevin, J., *Determinants of Young Marrieds' Life Insurance Purchasing Behavior: An Empirical Investigation*, *op. cit.*

[30] Burnett, J and Palmer, B., *Reliance on Life Insurance Agents: A Demographic and Psychographic Analysis of Consumers*, The Journal of Risk and Insurance (1983) 50 (3) 510-520.

[31] Ferber, R. and Lee, L.C., *Acquisition and Accumulation of Life Insurance in Early Married Life*, The Journal of Risk and Insurance (1980) XLVII (4) 713-734.

[32] *Ibid.* p.727.

[33] Kreinin, M.E. et. al., *Analysis of Life Insurance Premiums*, The Review of Economics and Statistics (1957) 39 (1) 46-54.

[34] Hammond, J.D. et. al., *Determinants of Household Life Insurance Premium Expenditures: An Empirical Investigation*, The Journal of Risk and Insurance (1967) 34 (3) 397-408.

[35] Burnett, J. and Palmer, B., *Examining Life Insurance Ownership through Demographic and Psychographic Characteristics*, The Journal of Risk and Insurance (1984) 51 (3) p. 458.

valued religion, or considered themselves assertive.[36] This significant statistical correlation between both income and number of children supports the HGAC position that life insurance purchase is important for the protection of dependents.[37]

These statistically significant variables were then classified according to relative importance. Through this process, we find that the five most significant contributing factors to the amount of life insurance held are all psychographic, namely Work Ethic, Fatalism, Socialisation Preference, Religion Salience and Assertiveness, ranging in score from .98 to .80.[38] Of the three statistically significant demographic indicators, Income ranks the lowest, at a relative score of .39.[39] At first glance, it would therefore certainly appear that an individual's level of income, whilst a statistically significant contributor, is far less important than other factors. It is however possible to make more of individual wealth than the relative score income receives would indicate. Of the other demographic factors, Education receives a relative score of .59, indicating that those who possess higher levels of education are more likely to possess higher levels of life insurance. As educational attainment correlates with increased individual income and wealth, both in the UK[40] and the US,[41] it is reasonable to suggest this also be used as predictive of individual wealth. From this study, individual income and wealth can be understood as a significant contributor to the amount of life insurance possessed, but not the only one, nor indeed the most important one. In addition, this study is somewhat limited, both because it only considered a small sample size in the Midwest US, and because it excluded the "extremes of [the] income continuum"[42] both the very rich and the

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[36] *Ibid.*

[37] Human Genetics Advisory Commission, *The Implications of Genetic Testing for Insurance*, Issued December 1997, Accessible from: [http://www.dh.gov.uk/ab/Archive/HGAC/index.htm] Accessed 07-04-2012.

[38] Burnett, J. and Palmer, B., *Examining Life Insurance Ownership through Demographic and Psychographic Characteristics*, *op.cit.* p. 461.

[39] *Ibid.*

[40] Blundell, R. et. al., *Evaluating the Impact of Education on Earnings in the UK: Models, Methods and Results from the NCDS*, CEEDP 47, Centre for the Economics of Education, London School of Economics and Political Science, Accessible at: [http://eprints.lse.ac.uk/19451/] Accessed 08-02-2012.

[41] US Census Bureau, *The Big Payoff: Education Attainment and Synthetic Estimates of Work-Life Earnings*, Special Study, July 2002, Accessible at: [http://www.census.gov/prod/2002pubs/p23-210.pdf] Accessed 08-02-2012.

[42] Burnett, J. and Palmer, B., *Examining Life Insurance Ownership through Demographic and Psychographic Characteristics*, *op. cit.* p. 462.

very poor. It must also be noted that this study is now relatively old, having been carried out in the early 1980s. So, is this conclusion borne out by more recent work?

Looking at a 2007 study of OECD[43] countries, we again find a positive correlation between wealth and sum insured. When examining life insurance demand across these countries, Li and others find that "…Disposable income and financial development are positively associated with life insurance demand"[44] and "…income is highly correlated with life insurance purchases"[45] They also offer a possible theoretical explanation for this, suggesting that individuals who are higher earners stand to suffer a greater loss of utility upon losing their income stream than individuals who earn lower amounts.[46] Indeed, this study returns similar results to the 1980 one, finding that:

> "There appears to be a combination of wealth and preference effects as life insurance consumption almost doubles over the two central quartiles, whereas little difference is observed in terms of average income. On the other hand, the difference between the upper and lower quartiles reflects a strong wealth effect. In fact, average demand in the upper quartile is 3 times higher than in the next quartile for average incomes only 50 percent higher. Likewise, life insurance demand falls to an average of only US$71.8 [in the lowest quartile] revealing a high income elasticity at low income levels."[47]

From this, it would appear that the central two quartiles of insurance demand are largely influenced by factors other than income, as insurance demand rises rapidly whereas income remains relatively similar. However, at the extremes of the continuum (those excluded in the 1980 study), wealth would appear to have a much greater effect. Demand for life insurance is significantly lower amongst those in the bottom income quartile, and significantly higher within the highest income quartile. This seems reasonable, as the wealthy may suffer the greater cost of utility highlighted earlier, whereas the very poor may just be unable to afford premiums past a certain level, and therefore, policies above a certain amount. Indeed, in this

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[43] Organisation for Economic Co-operation and Development. Established in 1947, the organisation concerns itself with global development. It has 34 members which span the globe, and include developed Western states such as the USA, UK and France, as well as developing economies like Mexico, Chile and Turkey. [www.oecd.org] Accessed 09-02-12.
[44] Li, Donghui et. al., *The Demand for Life Insurance in OECD Countries*, The Journal of Risk and Insurance (2007) 74 (3) p. 638.
[45] *Ibid.* p. 645.
[46] *Ibid.* p. 640.
[47] *Ibid.* p. 645-646.

study, demand for insurance amongst the lowest income quartile rapidly falls off, suggesting this is plausible.

In conclusion, the empirical studies suggest that, whilst not the most important contributor, a rise in individual income and wealth correlates with an increase in the amount of insurance held. For the median earner it would appear that although income is still relevant, psychographic factors far more strongly correlate and may go a significant way to explaining the amount of insurance held. However, at the extremes of the income continuum, we see that individual income and wealth becomes more dominant, and could be said to be the dominant causal factor. Within the thesis, I am therefore able to use amount of insurance held as a legitimate proxy for their individual wealth, as higher premiums correlate significantly with wealthier consumers, especially at the extremes of the income and wealth continuum. As I am explicitly attempting to identify those who are members of the least advantaged group, the proxy is even more appropriate. These individuals will reside toward the lower extreme of the income and wealth continuum, and only be able to afford premiums below a certain amount, and therefore only possess a certain value of policy. Again, within *Chapter Two*, one suggested boundary for membership of the least advantaged group is membership of the lowest income quartile. If we are to use this as our boundary, it would appear that this proxy becomes more useful, as possession of life insurance rapidly tails off with wealth upon reaching this quartile, which indicates a strong correlation between wealth and insurance demand. This has implications when I come to consider justifiability, as it may be possible to use data for policies over a certain amount, where this policy value is only accessible by those individuals who find themselves within the top three wealth quartiles. In this way, we may be able to use genetic test data while avoiding harm to members of the least advantaged group, either through use of their data, or through adverse selection.

3.3 – Is the *status quo* justifiable?

In order to determine whether the current moratorium is justifiable, and whether it could be made more so, we must first identify which individuals are members of the least advantaged group. The following sub-section will provide a

brief summary of the efforts made so far. Having established this, I will attempt to assess how far the moratorium accords with the ideal position derived from the principles of justice.

3.3.1 – Who are the members of the least advantaged group?

When we discuss membership of this group, we are not looking for the absolute most disadvantaged individual, but instead are aggregating this to a group level; we are looking for the least advantaged *class* of individuals, the "average taken over this whole class."[48] However, in the absence of comprehensive empirical work (something outside the scope of this thesis) it is impossible to offer a claim as to which specific individuals are members of the least advantaged class. The fact that this method of identifying the least advantaged group is somewhat arbitrary is something explicitly conceded by Rawls himself.[49] The primary good we are concerned with in this case is income and wealth, and as such it is legitimate to suggest that the individuals who are the least advantaged with regard to this primary good reside in the lowest wealth quartile, as Rawls does.[50]

Although this is somewhat arbitrary, for now, let us accept the idea that the members of the least advantaged group are those individuals who find themselves in the lowest wealth quartile. Given the correlation between the sum of insurance held and individual income and wealth, I would then suggest that there exists a policy which no members of the least advantaged class will be able to obtain, likely because they would be unable to afford the premiums this policy would demand.

3.3.2 – Assessment of Justifiability

We are now able to assess whether the status quo within the UK is justifiable, and whether it could be made more so. In order to do this, I am essentially asking

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[48] Rawls, John, *A Theory of Justice*: *Original Edition, op. cit.* p.98.
[49] *Ibid.*
[50] *Ibid.*

one question, namely, does the status quo provide benefit to, or avoid harming, members of the least advantaged group?

We know that a failure to allow insurers use of certain genetic information may lead to adverse selection and that this leads to a rise in premium prices across the board. This would then lead to the least advantaged individuals either paying increased premiums for the same policy, or being priced out of the insurance market altogether. Both of these mean that these individuals are further disadvantaged with regard to possession of primary goods. It therefore follows that we must only prevent the use of actuarially relevant data by insurers where the harm to members of the least advantaged group would be greater than the harm caused by the insurers' response to adverse selection. In other words, we should not use data which relates to members of the least advantaged group (as this would raise their premiums directly) and we should use data relating to individuals who are not in this group (to prevent indirect premium rises). The question then essentially becomes:

> *Which individuals can never be considered members of the least advantaged group,* and as such*, which individuals may we be able to justifiably disadvantage in order to protect those who are members of the least advantaged group?*

First, let us consider the issue of policy value. Larger insurance policies are held by individuals who possess larger amounts of income and wealth, something which becomes even more true at the extreme ends of the income continuum. It is therefore reasonable to make the claim that there exists a policy value which no member of the least advantaged group could hold, likely because they could not afford the premiums which this policy would require. Let us term this policy *value X*.

Following on from this, it would therefore be justifiable to allow the use of genetic test data for policy values greater than *value X*. Allowing this use would disadvantage the holders of these policies, whilst benefiting the individuals who hold lower value policies by preventing significant information asymmetry, and therefore adverse selection. This then protects them from the premium rises that would occur

as a response to this phenomenon. As the holders of these higher value polices cannot be considered members of the least advantaged group, disadvantaging them is not unjustifiable under the second principle, and indeed, the interests of justice demand we do this to avoid disadvantaging members of the least advantaged group who are likely to reside in the group of policies below *value X*. Whilst it is not possible in the absence of significant empirical work to suggest what *X* might be or at the exact level it may be set, it must exist at some level.

3.3.3 – Problems with the assessment thus far.

There are however two key problems with the account thus far. First, as we know from *Chapter Two*, we are concerned with individuals over the course of a whole life,[51] and that we are examining the "lifetime *expectations* of the least advantaged *representative* persons."[52] As we are examining income and wealth, this becomes an issue, as this is likely to change over the course of a lifetime. Currently, my analysis only assesses their income and wealth up to the point they purchase insurance. Over the course of their lifetime, they may earn far more or less than they currently do, and as such, possess a significantly different amount of lifetime wealth than this assessment would suggest. Specifically, it is this concern which motivates my development of a typology and analysis of representative conditions within *Chapter Four*. Due to the nature of genetic conditions and health and life insurance more widely, the individual will always be purchasing insurance in advance of the event occurring (in this case, the development of the genetic condition). As such, when they develop said condition, this may operate to change either their level of income, or their possession of wealth, to the point where they move to being a member of the least advantaged class (when considered over the course of a whole life). Further, the assessment so far takes no account of the effect the development of a genetic condition may have either on fair equality of opportunity, or an individual's capability to use their existing primary goods. As we know from *Chapter Two*, both of these need to be considered to provide an adequate account of justifiability.

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[51] Rawls, John, "Social Unity and Primary Goods," in *Utilitarianism and Beyond*, Amartya Sen and Bernard Williams (eds.) (Cambridge: Cambridge University Press, 1982), p. 164.
[52] Schaller, Walter, *Rawls, The Difference Principle, and Economic Inequality*, Pacific Philosophical Quarterly (2003) 79 (4) p.371.

Different genetic predispositions will also have different effects on individuals, and affect their risk profiles in different ways. To treat all genetic data as the same is a key failing of the account so far. This is also dealt with within *Chapter Four*, through the development of a typology, and identification of specific representative conditions. As different predispositions affect risk profiles in different ways, they also differ in their potential to cause adverse selection. This means that allowing or denying the use of some genetic data may be justifiable, whereas it may not for another type. This will also need to be considered to provide a fully defensible conclusion.

Also, and perhaps most fatally to the account so far, I am not performing large empirical studies. I therefore have significant difficulty with determining the level at which an individual is or is not a member of the least advantaged group, something which is key to the whole account. In the absence of significant data relating to wealth distribution within the UK, I am unable to identify which individuals may be members of the least advantaged group. Even if we accept the somewhat arbitrary conclusion that the members of the least advantaged group are those who find themselves in the bottom wealth quartile (which is questionable in itself), we are unable to identify exactly where the boundary of this quartile falls, and therefore what *value X* should be. Even more concerning, we are (again as we know from *Chapter Two*) actually concerned with an individual's actual capability to use their primary goods, not merely whether they possess them. As such, the more appropriate approach is perhaps to identify members of the least advantaged group as those individuals who fall within the bottom quartile when assessing their capability of using their primary goods. This is all but impossible in the absence of significant empirical work, and my methodology is not equipped to help me identify these individuals.

3.3.4 – Problems with Policy Value 'X'

Let us accept for the moment that the individuals we are concerned about have genetic predispositions which do develop into future disease. Depending on the genetic condition the individual possesses, the policy value at which they can be considered a member of the least advantaged group also changes. Allow me to

illustrate. The median individual income within the UK is approximately £419 per week, or £21,788 per annum.[53] Therefore, income at the boundary of the lowest quartile is approximately £10,894 p/a. Assuming continual employment at this level for 49 years (age 16 to retirement at age 65), an individual stands to earn lifetime earnings of £533,806 at the boundary of the lowest wealth quartile. Under the metric above then, *value X* would be the policy that is unaffordable by the individual who earns £209.50 a week, but is affordable by those who earn more than this.

However, when we look at the development of genetic conditions, the purpose of lifetime earnings becomes apparent. Let us take a comparative individual who earns the median income. When they come to purchase insurance, they would likely purchase a policy above *value X*, as they would be able to afford it, earning twice as much a week as our previous individual. However, this second individual also carries a genetic mutation which means they will become seriously ill and unable to continue employment from age 40. This means that:

Individual A - £10,894 p/a for 49 years = **£533,806**

Individual B – £21,788 p/a for 24 years = **£522,912**

As we can see, Individual B therefore earns less over the course of his life than Individual A. With regard to lifetime possession of the primary good of wealth, it is no longer clear that Individual A is a member of the least advantaged group, whilst B is not. If we consider the first individual a member of this group, it seems consistent to regard any individual who earns less than them as also a member of this group. This is true even under the assumption that Individual B also begins employment at 16, something which is unlikely as higher incomes correlate with more time in education, and therefore a later start to employment. If however Individual B does not suffer from the condition until age 45, then the situation is as follows:

Individual B (at age 45) - £21,788 p/a for 29 years = **£631,852**

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[53] Institute for Fiscal Studies, *Living Standards, poverty and inequality in the UK: 2012*, IFS Commentary C124, June 2012, Accessible at: [http://www.ifs.org.uk/comms/comm124.pdf], Accessed 16-09-2012.

and the individual moves out of the lowest wealth quartile again. The legitimate metric for measuring membership of the least advantaged group is still wealth, but assuming we are using the boundary of the lowest wealth quartile as our line, it is important to assess the lifetime wealth an individual will likely obtain rather than their current income. This makes the problems with specificity and identification of the least advantaged group harder, as it means we must extrapolate likely future wealth.

Further, at the point an individual purchases insurance, the policy they will be able to access does not depend upon their lifetime wealth earnings, but upon their current income. Individual B would be able, even in the first scenario, to purchase a policy of higher value than Individual A, even though they are less advantaged over the course of their whole life. This means that if we are to use *value X* as a metric, that this metric shifts depending upon the condition the individual is likely to suffer from. In simple terms, the more severe the conditions effect on income and wealth long-term, the higher *value X* must be, in order to account for the future effects of the disease on lifetime wealth. In the absence of substantial empirical work, it is impossible to reasonably speculate where any of these policy values may be set. If we are to use this metric, this will be an exercise which requires significant co-operation between government, groups such as the Institute for Fiscal Studies and insurers.

3.3.5 – Where do we go from here?

The account so far therefore has several key weaknesses, specifically with regard to identification of individuals who are members of the least advantaged group over the course of a whole life. It is therefore difficult to draw a clean line around the data we *should not* use under the difference principle. All we know so far is that we cannot use the information of those individuals who are members of the least advantaged group, as this would cause their premiums to rise and disadvantage them contrary to the principles of justice.

In order to provide more specific and useful conclusions, I will now examine the problem from a different perspective, and determine which data we should

legitimately be able to use when looking at individuals who are not members of this group. This will allow me to classify data into two broad groups:

a) Data which can legitimately be used providing it does not relate to an individual who is a member of the least advantaged group, and

b) Data which should not be used, regardless of whether the individual is a member of the least advantaged group.

Illustrated through the use of specific representative conditions, generalisable to conditions (and therefore data) of a similar type. Given the moratorium currently prevents almost all use of genetic data, I will then be able to suggest both areas in which the moratorium should be re-assessed to allow some use (data within a)) and those where the moratorium is likely correct (data within b)). This will allow me to provide meaningful conclusions re: the justifiability of the current moratorium position from a Rawlsian perspective.

In order to do this, instead of focusing on membership of the least advantaged group, I am going to first ground my analysis in the first part of the second principle of justice, the idea of fair equality of opportunity, specifically in relation to employment. If an individual has this equality of opportunity compromised, they are entitled to action to remedy this inequality, as a denial of fair equality of opportunity leaves them unable to compete for and thus obtain income and wealth in the same way as an unaffected individual. I will also provide some thoughts on whether the development of their condition is likely to compromise their capability in a meaningful way, and therefore whether they may also be considered legitimately disadvantaged due to this. All of this will then allow me to assess whether they are disadvantaged in a way which is unjust, and therefore, whether use of their data should be allowed. I will then proceed to assess whether denying insurers the use of this information would lead to adverse selection, and an increase in premiums for all (including members of the least advantaged group). If significant adverse selection would occur, it is justifiable to allow use of their data to protect members of this group, and the *difference principle* demands this. However, if adverse selection would not occur in a specific case, it may be that the interests of justice require denying access to this data, in order to not further disadvantage an

individual already disadvantaged either in terms of equality of opportunity or capability.

However, before I am able to proceed to analyse this, I must first solve the other two problems with the account identified earlier, namely that not all genetic predispositions are identical in their potential effect, and that because of this income (and therefore wealth) may change dramatically over the course of a whole life. It is this which motivates the work in the next Chapter, and informs the development of the following typology.

Chapter Four
Representative Conditions and Consequences of Use

Mindful of the differing effects of genetic conditions (and therefore possession of genetic data), I will now establish a typology which allows me to assess the potential effects of the possession of different genetic markers on future disadvantage. Specifically, I will be assessing the potential of genetic data to affect either:

a) Fair equality of opportunity, or
b) Capability

Second, as it is clearly impractical to assess every possible genetic marker and subsequent condition, I will then identify representative conditions along the scale, and provide an evidence base for where these conditions fit along my typology. Within *Chapter Five*, I will then be able to analyse the effects of these conditions on these key areas, and reach conclusions generalisable to conditions of a similar type. Finally, I will then briefly outline the two major individual consequences that will result from the use of genetic test data by insurers, those being:

- a change in individual premium prices and,
- the creation of a potential disincentive for that individual to obtain the results of genetic tests,

both of which may disadvantage the individual whose data is to be used. This will again feed into the analysis within *Chapter Five*, where I will assess both whether an individual is significantly disadvantaged due to their possession of certain genetic makers (and the effect that this may have on their fair equality of opportunity and capability to convert their primary goods), and second, whether it is then legitimate to further disadvantage them through the use of their test data. This will then allow me to reach conditions on justifiability, and therefore within *Chapter Six*, assess whether the moratorium is justifiable.

4.1 – Typologies

4.1.1 – Introduction

In order to assess the effect of genetic conditions on both fair equality of opportunity and capability, I have identified three characteristics of genetic test data and conditions which may be relevant. These closely mirror the criteria which were used by the GAIC in order to assess whether a genetic test should be approved for use.[1] My characteristics are:

- *Severity of Disease or Disorder* (If managed appropriately according to clinical guidelines)
- *Level of Penetrance* (How likely it is that those with the gene will go on to manifest the disease or disorder)
- *Time of Onset* (Pre or Post Retirement, with Age 65 as a proxy)

The following section of the Chapter will provide explanation for each of these criteria, before I use them to identify the representative conditions which I will later analyse.

4.1.2 – Severity

The first characteristic I am concerned with is severity, specifically, the severity of the condition which results (or may result) from the possession of certain genetic data. Within this, I am assessing severity as (assuming the individual receives appropriate medical treatment for their condition) either:

a) how far the development of the condition negatively impacts upon their ability to compete for or maintain employment under conditions of fair equality of opportunity, or

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[1] Association of British Insurers, *Concordat and Moratorium on Genetics and Insurance*, March 2005, *op. cit.* p. 3.

b) their capability to use their existing primary goods, namely wealth.

If a condition is capable of infringing upon either of these significantly, it will be judged relatively severe in my taxonomy. It is important to note at this point that there is a spectrum of severity, along which I will be considering representative points.

As an example of the types of impact I am considering, let us consider the following. At one end of the severity spectrum, we may have a disease which is degenerative, terminal, and the effects of which cannot be substantially mitigated. Such a disease might significantly affect both an individual's ability to compete under conditions of fair equality of opportunity and their capability to convert their primary goods. First, equality of opportunity. Such an individual would be unable to compete for, obtain, or maintain employment in the same way as an unaffected individual, nor use their goods as effectively. It may therefore be unjustifiable to further disadvantage them through allowing use of their data. At the other end of the spectrum, we may have a condition which is transient, and easily managed through relatively minor medical intervention. This condition would not meaningfully compromise either an individual's ability to compete for, obtain or maintain employment, nor their capability to use their existing primary goods. Such an individual is therefore not meaningfully disadvantaged by their condition, and as such, the use of their data may be justifiable in order to protect individuals who are more disadvantaged from the insurers' response to adverse selection. I will later be assessing conditions at different points along this spectrum.

4.1.3 – Penetrance

As highlighted briefly above, the penetrance of genetic data is the likelihood that the possession of that genetic data will result in the development of the condition in question. Data with a high degree of penetrance will be that which correlates with a high chance of developing the condition. Data with a low degree of penetrance the opposite. As an example, the genetic markers for Huntington's would have a penetrance of close to 100%, as the vast majority of those who possess them

will go on to develop the condition. At the lower end of the penetrance scale falls genetic data which predisposes individuals to certain conditions, such as high blood pressure or atherosclerosis.

This matters because (assuming some degree of severity) penetrance will affect whether the condition does in fact develop, and therefore, whether the individual will suffer the effects associated with it.[2] Individuals who carry genetic data with high penetrance are more likely to suffer from the condition in question over the course of their life, and therefore, more likely to suffer these effects both to their ability to compete, and their capability to convert existing primary goods. As penetrance decreases, the likelihood that the individual suffers these negative consequences at all also decreases. The potential of an individual to be relevantly disadvantaged therefore decreases as penetrance does. Penetrance therefore potentially affects whether the use of the data could be said to be justifiable and is important to consider when drawing my conclusions. How penetrant the condition is also affects the usefulness of the information to insurers, as it affects how predictive it is. The more predictive information is, the larger the potential increase in premium if insurers are able to use this data. Again, the conditions identified later will represent several different points along the penetrance spectrum.

4.1.4 – Time of Onset

To explain this final criterion, let us return to the idea that employment is one of the major areas to which an important primary good is attached, and as such, an area which much be open to the requirements of equality of opportunity. It therefore becomes important to consider time of onset as there is a distinction to be made between conditions which onset pre-retirement, when an individual is still likely to be engaged in employment, and post-retirement when they are not.[3] Assuming some degree of both severity and penetrance, individuals with conditions which are likely to onset pre-retirement age are more likely to suffer the loss of the ability to compete under conditions of fair equality of opportunity, and the subsequent loss of primary

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[2] Rawls, John, "Social Unity and Primary Goods," in *Utilitarianism and Beyond*, Amartya Sen and Bernard Williams (eds.) (Cambridge: Cambridge University Press 1982)
[3] With 65 as a proxy for retirement age.

goods such as income and wealth. This is true even if the individual is self-employed, as the condition may inhibit their ability to work and therefore, their ability to earn income and wealth. In contrast, these effects are not nearly as pronounced (and in some cases, entirely absent) in individuals whose conditions onset post-retirement. As these individuals are no longer working, they suffer little meaningful loss of fair equality of opportunity in relation to competition for employment, as they are no longer competing. Equally, they suffer no loss of income and wealth, as they would not be earning a wage anyway. The only relevant effect may be a compromise of their capability to use primary goods already obtained, although this depends on the severity and penetrance of the genetic marker in question. It may therefore be justifiable to allow the use of certain test data for conditions which onset post-retirement age in a way that it would not be should the condition onset earlier.

4.1.5 – Complete Typology

The previous analysis now provides me with a system for meaningfully classifying different types of genetic data. Both severity and penetrance exist on a spectrum, the extremes of which have been identified, with time of onset a binary between pre and post-retirement age. Tests and conditions which fall toward the top end of both spectra, and which onset pre-retirement will have a very serious effect upon an individual's fair equality of opportunity and capability, and as such, potentially make it unjustifiable to allow the use of their test data. Equally, data falling toward the lower end of the spectra, or which onset post-retirement could perhaps justifiably be used, given that failing to allow use of this data may disadvantage members of the least advantaged group. This will be explored in detail within *Chapter Five*.

It is however impractical for me to consider every possible permutation of these criteria within the thesis. Therefore, I intend to limit my analysis to specific representative points along these spectra, points which will be illustrated through the use of specific genetic conditions. The use of specific representative conditions allows me to gather an evidence base relating to severity, penetrance and time of

onset, and provide meaningful conclusions about the use of test data for real world conditions. This will then also be generalisable to conditions of a similar type, where the data considered is representative of all alike data. The conditions which I am going to consider are:

- Alzheimer's Disease
- Huntington's Disease
- BRCA1 and BRCA2 Mutations
- Type 1 Haemochromatosis
- Hypertension

with their position within the typology explained and defended within the following section.

4.2 – Representative Conditions

Before I am able to analyse whether the use of data relating to a specific condition is justifiable, I must first be able to accurately place each condition within my typology. To do this, I need to know the severity, penetrance and time of onset of each condition I am studying. The following sections provide this evidence base.

4.2.1 – Alzheimer's Disease

Severity

Alzheimer's disease is a progressive form of dementia, characterised by a gradual loss of memory at onset. Individuals may misplace items, become disorientated or begin to have problems with language. The condition can also lead to psychological issues such as depression or mood swings. After the initial stages, the individual continues to decline, and will begin to suffer a loss of sensory and

motor function towards the later stages of the disease.[4] In the late stages, the disease can fairly be characterised as very severe, with the individual losing the ability to recognise even close family members, the ability to communicate, and eventually becoming bed-bound.[5] The rate of progression is variable, but the average progression of symptoms spans a decade.[6]

The disease is eventually terminal, and there is currently no known cure. The average life expectancy for an Alzheimer's patient after the onset of symptoms is between eight and ten years.[7] As such, the treatment plan for the disease is based upon management of the condition, and seeks to "minimize behavioural disturbances, maximize functioning and independence, and foster a safe and secure environment."[8] The degenerative progression of the disease and the fact that symptoms cannot be prevented from developing both mean that the development of the condition has a significant effect on both fair equality of opportunity and an individual's capability to convert their primary goods. Indeed, an individual suffering from Alzheimer's would be entirely unable to compete for employment under conditions of fair equality of opportunity, and less capable of using their existing primary goods. As such, the condition would be placed toward the top end of the severity spectrum. In approximately 90% of cases, the diagnosis can be made on the basis of a general medical and psychiatric evaluation, so genetic testing is not needed to perform a diagnosis.[9]

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[4] Small GW, et. al. *Diagnosis and treatment of Alzheimer disease and related disorders. Consensus statement of the American Association for Geriatric Psychiatry, the Alzheimer's Association, and the American Geriatrics Society.* JAMA. (1997) 278 (16) 1363–1371.

[5] Alzheimer's Association. *Alzheimer's disease facts and figures 2007: a statistical abstract of U.S. data on Alzheimer's disease.* Accessible at:
[http://alz.org/national/documents/Report_2007FactsAndFigures.pdf] Accessed 10-02-2012.

[6] Small GW, et. al. *Diagnosis and treatment of Alzheimer disease and related disorders. Consensus statement of the American Association for Geriatric Psychiatry, the Alzheimer's Association, and the American Geriatrics Society. op. cit.*

[7] *Ibid.*

[8] *Ibid.*

[9] Rasmusson, D. X. et al. *Accuracy of clinical diagnosis of Alzheimer disease and clinical features of patients with non-Alzheimer neuropathology*, Alzheimer Disease & Associated Disorders (1996) 10 180-188.

Penetrance

The risk factors for the development of Alzheimer's are multifactorial. Within this there exists a strong genetic component, although its penetrance is debateable. For example, the development of Late Onset (Post 60 Years) Alzheimer's is strongly influenced by the allele Apolipoprotein Epsilon Four (APOE-4). The possession of certain APOE-4 statuses confers a dramatically increased risk of developing the disease, and at an earlier age.[10] Indeed, studies demonstrate that an increasing number of APOE-4 alleles correlates with an increasing incidence of the disease, with homozygosity "virtually sufficient to cause AD by age 80".[11]

Further, in a study of individuals over the age of seventy-five, we find that:

> "Family history of dementia was associated with an increased risk of dementia and AD in this very old population, but only among APOE epsilon4 carriers"[12]

which suggests that an individual's APOE-4 status may be an important indicator for assessing whether they will go on to develop Alzheimer's disease. However, the same study also concluded that "other familial (genetic or environmental) risk factors for dementia and AD might be active among APOE-4 carriers."[13] Indeed, work done by Ashford in 2004 suggests that APOE-4 contributes approximately 50% of the attributable risk for Alzheimer's.[14] Certain other studies implicate genetic contributions located on Chromosome Twelve[15], which is supported by other sources.[16,17] The genetic contribution towards Alzheimer's is neatly summarised by Tanzi, who explains that:

[10] Roses, A.D. et. al., *Influence of the susceptibility genes apolipoprotein E-epsilon 4 and apolipoprotein E-epsilon 2 on the rate of disease expressivity of late-onset Alzheimer's disease*, Arzneimittelforschung (1995) 45 (3A) 413-417.

[11] Corder, E.H. et. al., *Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families*, Science (1993) 13 (261) 921-923.

[12] Huang,W. et. al., *APOE genotype, family history of dementia, and Alzheimer disease risk: a 6 year follow-up study*, Archives of Neurology (2004) 61 (12) 1930-1934.

[13] *Ibid.*

[14] Ashford, J, *APOE Genotype Effects on Alzheimer's Disease Onset and Epidemiology*, Journal of Molecular Neuroscience (2004) 23 (3) pp. 157-165.

[15] Pericak-Vance, M.A. et. al., *Complete genomic screen in late-onset familial Alzheimer disease. Evidence for a new locus on chromosome 12*, JAMA (1997) 278 (15) 1237-41.

[16] Schellenberg, G.D. et. al., *The genetics of Alzheimer's Disease*, Current Psychiatry Reports (2000) 2 (2) 158-164.

> "While *APP*, the presenilins, and *APOE* represent the only firmly established AD genes to date, the other genes described in this review remain at best functional and/or positional *candidates*…[however] some of these loci exhibit genetic linkage and/or association with AD across independent datasets and are thus worthy of further investigation at both the genetics and functional levels."[18]

It is therefore clear that certain genetic markers predispose individuals towards the development of Alzheimer's disease. However, we do not yet have a clear picture as to the exact contribution of this genetic status. For help, we can look to Skeehan's summary of the contribution of APOE-4. He states:

> "Persons with APOE ε4/ ε4 have increased risk—more than sixteen-fold higher among Caucasian males at peak relative risk—and they have earlier age of onset than individuals with only one ε4 (three-fold higher risk in Caucasian males). Individuals with only one ε4 have a higher risk and earlier onset, in turn, than those with no ε4 alleles."[19]

We therefore know that APOE-4 (and possibly other markers) contributes significantly to the risk of developing Alzheimer's disease, possibly in the region of 50% attributable risk. The presence of these markers therefore provides a decent indication as to whether an individual will go on to develop the condition. The condition is therefore relatively penetrant, and would be relevant in an actuarial context. This relevance is strengthened by Skeehan's second conclusion, that possession of certain combinations of APOE-4 also causes earlier onset of the condition. Given that this can range from before seventy to over ninety years of age, it is reasonable to suggest that this is also of actuarial relevance. If an individual has genetic markers which indicate an age of onset before or around seventy years of age, they will develop the condition over the course of an average lifespan. If the median age of onset suggested by their genetic markers is over ninety years of age, it is far less likely they will survive long enough to suffer from the condition. As such, it is less likely that a life insurer would view this genetic predisposition as relevant to their risk calculation, as the individual is likely to die from another cause first.

Given that genetic markers are thought to contribute approximately 50% of the attributable risk, it is fair to conclude that genetic markers (and thereby, genetic

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[17]Yu, C.E. et. al*., The apolipoprotein E/CI/CII gene cluster and late-onset Alzheimer disease*, American Journal of Human Genetics (1994) 54 (4) 631-642.

[18] Tanzi, R. and Bertram, L., *Twenty Years of the Alzheimer's Disease Amyloid Hypothesis: A Genetic Perspective*, Cell (2005) 120 545-555.

[19] Skeehan, K. et. al., *Impact of Gene Patents and Licensing Practices on Access to Genetic Testing for Alzheimer's Disease*, Genetic Medicine (2010) 12 (4 Suppl.) S71-S82.

testing) can provide us a reasonable indication of an individual's level of risk with regard to the development of Alzheimer's disease, and it seems appropriate to place it towards the middle of the penetrance spectrum.

Time of Onset

As Alzheimer's disease is a form of degenerative dementia, it is primarily a disease of the old. We can already see from the studies cited earlier, that the majority of individuals within those studies were over the age of 60. Indeed, late-onset Alzheimer's is defined as that which onsets post sixty years of age, and the majority of cases onset post retirement.[20]

Location within typology

Alzheimer's disease is therefore an example of a condition that is somewhat penetrant, and is capable of having severe effects on both fair equality of opportunity and an individuals' capability to use their existing primary goods. The specific relevance of this condition is that it is the only one of my representative conditions which onsets post-retirement age. Further, with regard to severity, both the onset of the condition and its subsequent symptoms are similar to those caused by Huntington's, one of my other representative conditions. Using this condition, I will therefore later be able to examine how far the fact that a condition onsets post-retirement affects my conclusions regarding whether use of data is justifiable in the insurance context. This is the relevance of this specific condition, and the reason for its selection.

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[20] Alzheimer's Association. *Alzheimer's disease facts and figures 2007: a statistical abstract of U.S. data on Alzheimer's disease*. Accessible at:
[http://alz.org/national/documents/Report_2007FactsAndFigures.pdf]

4.2.2 – Huntington's disease

Severity

Much like Alzheimer's, Huntington's disease is a degenerative, neurological disease characterised by an initial onset of clumsiness, small involuntary movements, unsteadiness and depression.[21] As the disease progresses, chorea worsens, as does cognitive ability. Middle-disease symptoms include further motor degeneration which begins to interfere with daily life, such as trouble walking, psychological issues such as delusions and hallucinations, and a further decline in memory.[22] Late-stage Huntington's is characterised by severe ataxia leading to severely impaired mobility, and a progressive worsening of both dysarthria (problems with speech and enunciation) and dysphagia (difficulty swallowing). This development of symptoms can lead to an individual being unable to communicate or feed themselves without assistance in the later stages of the disease.[23] Severe neurological degeneration can also lead individuals to lose bowel and bladder control.[24] Due to the severe nature of the condition, individuals with late-stage Huntington's often require round-the-clock care as they are unable to support themselves.

In terms of progression, a study of 510 patients suggests that symptoms progressively worsen for between fifteen and twenty years.[25] The most common cause of death is infection, at an average age of fifty-four.[26] Kirkwood provides more detail, suggesting that middle stage symptoms tend to develop anywhere between 2 to 10 years, progressing toward late stage symptoms at the end of that scale.[27] Again, like Alzheimer's, the condition is terminal, and there is no known cure. Further,

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[21] Di Maio, L. et. al., *Onset symptoms in 510 patients with Huntington's Disease*, Journal of Medical Genetics (1993) 30 (4) 289-292.

[22] Kirkwood S.C. et. al., *Progression of symptoms in the early and middle stages of Huntington disease*. Archives of Neurology (2001) 58 (2) 273-278.

[23] Sturrock, A. and Leavitt, B., *The Clinical and Genetic features of Huntington's Disease*, Journal of Geriatric Psychiatry and Neurology (2010) 23 (4) p. 243-259.

[24] Kirkwood S.C. et. al., *Progression of symptoms in the early and middle stages of Huntington disease*. op. cit.

[25] Di Maio, L. et. al., *Onset symptoms in 510 patients with Huntington's Disease*, op. cit.

[26] Myers, R. et. al., *Late onset of Huntington's Disease*, Journal of Neurology, Neurosurgery and Psychiatry (1985) 48 530-534.

[27] Kirkwood S.C. et. al.,. *Progression of symptoms in the early and middle stages of Huntington disease*. op. cit.

treatment is currently limited entirely to the management of symptoms, as "no drug therapy has yet been shown to modify the disease course."[28] Drugs and therapies are available to attempt to mitigate the effects of the disease, with varying effect, although the symptoms are still severe.[29] The condition would therefore be placed at the extreme end of the severity continuum, as it severely affects both fair equality of opportunity and capability.

Penetrance

Unlike severity, the penetrance of the genetic markers for Huntington's disease is markedly different to those for Alzheimer's. Huntington's disease is a trinucleotide repeat disorder, characterised by excessive repeats of the cytosine-adenine-guanine (CAG) group along chromosome four. A repeat count of below 35 means that the individual will be unaffected whereas a repeat size of between 35 and 39 indicates reduced penetrance. Adult onset of the disease is generally associated with repeat sized between 36 and 50. Certain excessively long repeat sizes (>60) are associated with the development of Huntington's as a juvenile, but this is extremely rare. Within the thesis I am concerned with adult onset Huntington's. Within the range for adult onset, "…there is a trend to increasing penetrance with increasing repeat length in the 36-41-repeat range: <90% for 39 CAG repeats and 99% for 41 CAG repeats"[30]

As we can see, penetrance is extremely high, especially for individuals who possess a CAG repeat number over 40. Further, our understanding of how penetrance and the development of the condition relates to an individual's genetic status means that genetic markers (and therefore testing) can provide an extremely accurate assessment of individual risk. This is especially true as the condition is Mendelian Dominant, and monogenetic. Much like with severity, the condition can therefore be placed at the extreme end of the penetrance continuum, as possession of the genetic markers in question almost guarantees development of the condition.

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[28] Sturrock, A. and Leavitt, B., *The Clinical and Genetic features of Huntington's Disease*, Journal of Geriatric Psychiatry and Neurology (2010) 23 (4) p. 243-259.
[29] *Ibid.*
[30] Brinkman R.R, et. al., *The likelihood of being affected with Huntington disease by a particular age, for a specific CAG size*. American Journal of Human Genetics (1997) 60 1202-1210.

Time of Onset

Myers suggests that the onset of the disease usually begins in mid-life (between 21 and 50), with the mean age of onset being 41. The condition then follows the progression highlighted earlier, leading to death at an average of 54.[31] This study is supported by further empirical studies performed by Myers, Wendt, and Newcombe. [32,33,34] With a repeat number of 40, onset occurs at a median of 59, with 45 repeats a median of 37, and with 50 repeats, a median of 27.[35] Studies of Huntington's amongst the certain northern European populations suggest a mean onset age of the mid-40.[36] The condition therefore onsets pre-retirement, when an individual is likely to still be engaged in employment.

Location within typology

Much like Alzheimer's, Huntington's is a condition which falls at the highest end of the severity scale, with the symptoms both extremely likely to compromise fair equality of opportunity and capability to use primary goods, and unable to be significantly mitigated or treated. In addition, Huntington's disease is an example of a condition which is extremely penetrant, and as such, carriers are very likely to suffer from the above effects. Taking this, and the fact that the condition onsets pre-retirement, this condition is essentially a *worst-case scenario*, with an individual extremely likely to suffer extremely severe effects on their fair equality of opportunity at a time in their life when they are engaged in employment. If in my analysis I am able to justify the use of test data relating to this condition, it would seem unlikely that any test data could not be justifiably used.

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[31] Myers, R. et. al., *Late onset of Huntington's Disease*, *op. cit.*

[32] Myers R.H and Martin J.B. *Huntington's disease*, Seminars in Neurology (1982) 2 p. 365-372.

[33] Wendt G,G, et. al., *Das Erkrankungsalter bei der Huntintongschen Chorea*, Acta Genet Med (1959) 9 p. 18-32.

[34] Newcombe R.G, *A lifetable for onset of Huntington's chorea*, Annals of Human Genetics (1981) 45 p. 375-385.

[35] Brinkman R.R, et. al., *The likelihood of being affected with Huntington disease by a particular age, for a specific CAG size*. American Journal of Human Genetics (1997) 60 1202-1210.

[36] Maat-Kievit A, et al. Predictability *of age at onset in Huntington disease in the Dutch population*. Medicine (2002) 81 (4) 251-259.

4.2.3 – BRCA1 and BRCA2 Mutations.

Breast Cancer Susceptibility Gene 1 (BRCA1) and Breast Cancer Susceptibility Gene 2 (BRCA2) are both genes which encode for proteins responsible for the repair of DNA. If these genes are mutated, DNA repair is compromised, increasing the risk of developing breast and other cancers. Specifically, mutations of these genes significantly increase the risk of both breast and ovarian cancer. It is these conditions which I will be considering.

Severity

The severity of cancers is assessed through reference to five year survival statistics, that is, the number of patients alive five years after the identification of the disease. These statistics are separated according to the stage of development of the cancer in question.

For breast cancer, the five year survival statistics are as follows:

- Localised – 98%
- Regional – 81%
- Distant – 26%
- All stages (average) – 88%.[37]

Further statistics indicate a total risk of recurrence of 11% for five years after completion of treatment, and 20% for 10 years. Divided by stage, recurrence rates are 7%, 11% and 13% for stage i. ii. and iii. cancers respectively.[38]

Five year survival for Ovarian cancer is much lower, sitting at 35%, although this is because the majority of suffers present with advanced forms. Earlier stages

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[37] Jemal A, et al. *Cancer Statistics*, CA Cancer Journal for Clinicians (2006) 56 106-130 Fig 8.
[38] Brewster, A. et. al., *Residual risk of Breast Cancer Recurrence 5 years after Adjuvant Therapy*, Journal of the National Cancer Institute (2008) 100 (16) 1179-1183.

have much higher five year survival rates.[39] Amongst this late-stage majority, 50%-75% of these cases will relapse within 18 months and require further treatment.[40] 10 year survival rates for ovarian cancer are high (80%-95%) for early stage cancers, but long term survival for advanced cancer is only 10%-30%.[41] For this reason, preventative oophorectomy is advised for high risk patients carrying BRCA mutations. [42] If diagnosed early, it is therefore likely that these conditions can be treated, and unlikely that the condition will recur. Whilst the condition is likely to in some way affect either fair equality of opportunity or the capability of an individual to use their primary goods, an early diagnosis means that the condition is not terminal, and that these effects will be relatively short-term. I explicitly concern myself with early-stage cancers as the only relevant individuals in this case are those who possess information about their BRCA status, as it is only those individuals who could have their data used by insurers. If an individual does not possess this data, an insurer cannot obtain it. These individuals who do possess this genetic information are placed in high-risk groups and monitored, receiving more frequent mammograms, and in some cases, preventative mastectomy or oophorectomy. Due to this, it is likely that these individuals would receive diagnosis of any subsequent breast or ovarian cancer at an early stage, and that they are less likely to suffer cancer at all (in cases of preventative mastectomy or oophorectomy). Given my focus on early-stage cancers, it would seem appropriate to locate this condition toward the lower end of the severity continuum. Whilst it is capable of having some relevant effect, these effects are short-term, and the individual is likely to recover their prior function.

If however, I find that allowing the use of this data by insurers disincentivises these individuals from obtaining information relating to their BRCA status, the position changes. These individuals are now no longer likely to receive an early

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[39] Ovarian Cancer, *The recognition and initial management of ovarian cancer*, Nice Clinical Guideline CG/122, (April 2011), Accessible at [http://www.nice.org.uk/nicemedia/live/13464/54194/54194.pdf], Accessed 11-02-2012.
[40] McGuire W.P., et. al., *Cyclophosphamide and cisplatin compared with paclitaxel and cisplatin in patients with stage III and stage IV ovarian cancer.* New England Journal of Medicine (1996) 334 1-6.
[41] Hennessey, B et. al., *Ovarian Cancer*, Lancet (2009) 374 (9698) 1371-1382.
[42] Burke, W et. al., *Recommendations for follow-up care of individuals with an inherited predisposition to cancer. II. BRCA1 and BRCA2. Cancer Genetics Studies Consortium,* Journal of the American Medical Association (1997) 277 pp. 997–1003.

diagnosis of their condition, and as such, more likely to suffer the more severe effects associated with later stage cancers. This means that the condition would become one more appropriately placed toward the severe end of the continuum. This will again be explored in further detail within *Chapter Five*.

Penetrance

BRCA mutations are by no means the only contributors to the development of cancer. Indeed, they account for a relatively small amount by percentage.[43] Recent studies demonstrate that BRCA mutations account for approximately 11% and 4.9% (BRCA1 and 2 respectively) of breast cancer amongst populations.[44] These figures are lower in Britain, possibly due to population differences in mutation frequency.[45] Indeed, BRCA mutations do not even account for all hereditary breast cancer, other genetic factors may be at play.[46] There are many other suggested loci for cancer risk genes, including p53 mutations,[47] hCHK2,[48] CDKN2A,[49,50], 13q21,[51] and 6q22.33[52] The exact risk which each of these contributes is unclear. A recent twin study suggests that taking all suspected genetic risk factors together, 27% of breast cancer may be genetic (also 42% of prostate, and 35% of colorectal.)[53]

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[43] Frank, T.S. et. al., *Hereditary susceptibility to breast cancer. Significance of age of onset in family history and contribution of BRCA1 and BRCA2*, Disease Markers (1999) 15 89-92.

[44] Loman, N, et. al., *Family History of Breast and Ovarian Cancers and BRCA1 and BRCA 2 Mutations in a Population-based series of Early-Onset Breast Cancer*, Journal of the National Cancer Institute (2001) 93 (16) 1215-1223.

[45] Peto, J. et. al., *Prevalence of BRCA1 and BRCA2 gene mutations in patients with early-onset Breast Cancer*, Journal of the National Cancer Institute (1999) 91 (11) 943-949.

[46] Loman, N, et. al., *Family History of Breast and Ovarian Cancers and BRCA1 and BRCA 2 Mutations in a Population-based series of Early-Onset Breast Cancer*, op. cit.

[47] Malkin, D. et. al., *Germ Line p53 mutations in a familial syndrome of breast cancer, sarcomas, and other neoplasms*, Science (1990) 250 (4985) 1233-1238.

[48] Bell, D. et. al., *Heterozygous Germ Line hCHK2 mutations in Li-Fraumeni Syndrome*, Science (1999) 286 (5449) 2528-2531.

[49] Borg, Ake et. al., *High Frequency of Multiple Melanomas and Breast and Pancreas Carcinomas in CDKN2A Mutation-Positive Melanoma Families*, Journal of the National Cancer Institute (2000) 92 (15) 1260-1266.

[50] Debniak, T. et. al., *A common variant of CDKN2A (p16) predisposes to breast cancer*, Journal of Medical Genetics (2005) 42 (10) 763-765.

[51] Kainu, T. et. al*., Somatic deletions in hereditary breast cancers implicate 13q21 as a putative novel breast cancer susceptibility locus*, Proceedings of the National Academy of Sciences (2000) 97 (17) 9603-9608.

[52] Gold, B. et. al*., Genome-wide association study provides evidence for a breast cancer risk locus at 6q22.33*, Proceedings of the National Academy of Sciences (2008) 105 (11) 4340-4345.

[53] Lichtenstein, P. et. al., *Environmental and heritable factors in the causation of cancer- analyses of cohorts of twins from Sweden, Denmark and Finland*, New England Journal of Medicine (2000) 343 (2) 78-85.

Returning to BRCA, we can show that the lifetime penetrance of breast cancer amongst people with BRCA1 mutations is between 45% and 74%, depending on the study.[54,55] It has been suggested by a more recent population study that the lifetime penetrance for breast cancer amongst those with BRCA mutations is approximately 68%[56] The same study suggests the penetrance of ovarian cancer amongst those with BRCA mutations to be approximately 36%.[57] When taken together, this means that "…the estimated penetrance by age 80 years, of cancer of any type among female carriers of *BRCA1* mutations, is thus nearly 100%."[58] When we compare this to the non-mutated population, we find a significant decrease. The general population risk for the development of breast cancer is 4.81%, and for ovarian cancer, 0.7%.[59] We therefore see that being a carrier of a BRCA mutation leaves an individual 63% more likely to develop breast cancer, and 35% more likely to develop ovarian cancers. Penetrance is therefore significant, and genetic test data could therefore be useful in actuarially identifying high risk individuals.

Indeed, testing for BRCA mutations has proved to be useful diagnostically, even above and beyond family history. Studies show that:

> "…ovarian cancers occurring among carriers of *BRCA1* mutations were diagnosed, on average, ~4–5 years earlier than those among women not found to have mutations and ~7 years earlier than sporadic invasive ovarian cancer."[60]

Further, excluding patients from screening who did not have a first degree relative affected with cancer would have missed over 50% of the mutations.[61] It is therefore important that these individuals are not disincentivsed from obtaining their BRCA

[54] Whittemore, A.S. et. al., *Prevalence and contribution of BRCA1 mutations in breast cancer and ovarian cancer: results from three US population-based case-control studies of ovarian cancer*, American Journal of Human Genetics (1997) 60 (3) 496-504.
[55] Antoniou, A.C. et. al., *Risk models for familial ovarian and breast cancer*, Genetic Epidemiology (2000) 18 (2) 173-190.
[56] Risch, H.A. et. al., *Prevalence and penetrance of germline BRCA1 and BRCA2 mutations in a population series of 649 women with ovarian cancer*, American Journal of Human Genetics (2001) 68 (3) 700-710.
[57] *Ibid.*
[58] *Ibid.*
[59] American Cancer Society, *Global Cancer: Facts and Figures 2007*, Atlanta (2007) Accessible at [http://www.cancer.org/acs/groups/content/@nho/documents/document/globalfactsandfigures2007rev 2p.pdf] Accessed 12-02-2012.
[60] Risch, H.A. et. al., *Prevalence and penetrance of germline BRCA1 and BRCA2 mutations in a population series of 649 women with ovarian cancer*, *op. cit.*
[61] *Ibid.*

status, as otherwise they may not be aware of their increased risk. Given this, the condition can be located towards the more penetrant end of the spectrum, somewhere between Alzheimer's and Huntington's. Whilst clearly less penetrant than Huntington's, possession of certain genetic markers leaves an individual 63% more likely to develop breast cancer, 35% more likely to develop ovarian cancer, and almost 100% likely to develop cancer of any type by age 80. The likelihood of the individual developing a condition which may potentially infringe upon either their fair equality of opportunity or their capability is therefore high, and the level of this infringement depends on whether an individual is disincentivised from obtaining a test.

Time of Onset

Looking again at the data, we find that amongst those diagnosed with cancer, the highest frequency of mutations were found in those aged between 40 and 50, suggesting that the time of onset is between these ages.[62] Another study suggests that BRCA mutations lower the mean age of onset for cancer by several years, depending on the mutation.[63] Again, the population study by Risch suggests that, *"BRCA1-associated cases typically occur during a patient's 40s and 50s, with <10% being diagnosed at age <40 years."*[64] Some cancers which occur pre-forty have also been linked to BRCA mutations. BRCA mutations can therefore be linked to onset of cancer in middle age. This makes the condition one which clearly onsets pre-retirement.

Location within typology

As I explained earlier, it may be the case that for some conditions, a genetic test is extremely helpful in obtaining an early diagnosis, and further, that an early diagnosis is relevant to an individual's prognosis. This is broadly the purpose of this representative condition. As we see above, both breast and ovarian cancers have high

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[62] *Ibid*.

[63] Loman, N, et. al., *Family History of Breast and Ovarian Cancers and BRCA1 and BRCA 2 Mutations in a Population-based series of Early-Onset Breast Cancer, op. cit.*

[64] Risch, H.A. et. al., *Prevalence and penetrance of germline BRCA1 and BRCA2 mutations in a population series of 649 women with ovarian cancer, op. cit.*

survival rates if diagnosed and appropriately treated at an early stage, falling rapidly the later the condition is identified. If the use of test data by insurers is shown to disincentivise individuals from taking genetic tests, it is likely that these individuals would receive tests (and therefore diagnoses) later than they otherwise would, and that therefore, their condition would be more severe. This case study will serve to illustrate the potential effect of disincentivisation on the justifiability of the use of data.

4.2.4 – Haemochromatosis

Haemochromatosis is an autosomal recessive condition which manifests as an excess of iron in the bloodstream. There are several forms of the condition, and they can be divided into Adult Onset (HFE, or Classic) Haemochromatosis, also termed *Type 1* and Juvenile Haemochromatosis, also termed *Type 2*. Similarly to Huntington's, I will only be considering the adult onset form of the condition, as juvenile onset conditions are irrelevant to the purchase of life insurance, as individuals will likely already be symptomatic by the time they come to purchase.

Severity

Within adults, the onset of symptoms is usually non-specific, and patients often present with complaints of fatigue, or joint pain. Untreated, this can develop into liver disease (including cirrhosis) endocrine disorders such as diabetes, cardiac problems (including heart failure) and joint diseases.[65] Many of these later complications could prove fatal. Actual patient prognosis varies, and depends on the degree of development of complications. If treatment is started before the development of severe secondary complications, the survival chances of a patient are very good. These rapidly deteriorate as secondary complications develop.[66,67,68]

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[65] Pietrangelo, A., *Medical Progress: Hereditary Haemochromatosis – A New Look at an Old Disease*, New England Journal of Medicine (2004) 350 (23) 2383-2397.
[66] Niederau, C, Strohmeyer, G and Stremmel, W., *Epidemiology, clinical spectrum and prognosis of hemocrhomatosis*, Advances in Experimental Medicine and Biology (1994) 356 293-302.
[67] Yang, Q. et. al., *Hemochromatosis-associated mortality in the United States from 1979 to 1992: an analysis of Multiple-Cause Mortality Data*, Annals of Internal Medicine (1998) 129 (110 946-953.

Obtaining an early diagnosis is therefore important to prognosis.[69] If early diagnosis is obtained, and treatment begins long before secondary complications develop, survival rates are comparable to the general population.[70] In terms of absolute risk, the chance of severe liver damage is approximately 5% in men, and 1% in women, so morbidity is also generally low.[71]

If started before secondary complications develop, treatment is relatively simple. Regular phlebotomy can keep iron loads under control, and prevent the development of secondary complications.[72] The condition therefore falls at the bottom end of the severity spectrum, with appropriate treatment all but preventing any meaningful symptoms from developing. Much like with breast and ovarian cancer, evidence of significant disincentivisation may increase the severity, as the condition is difficult to diagnose otherwise.

Penetrance

Originally, Haemochromatosis was thought to stem entirely from a mutation of the HFE gene.[73] There are now however several different types of Haemochromatosis, and at least four genes are implicated, of which, HFE-linked Haemochromatosis is the most common form.[74] The exact penetrance of this mutation is however unclear. In a population study of 16 homozygous individuals, 8 of them were found to have clinical presentation of Haemochromatosis, a penetrance

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[68] Milman, N. et. al., *Clinically overt hereditary hemochromatosis in Denmark 1948-1985: epidemiology, factors of significance for long-term survival, and causes of death in 179 patients*, Annals of Hematology (2001) 80 (12) 737-744.

[69] British Society for Haematology, *British Committee for standards in Haematology: Guidelines on diagnosis and therapy, Genetic Haemochromatosi*s (2000) Accessible at: [http://www.bcshguidelines.com/documents/haemochromotosis_2000.pdf] Accessed 14-02-2012.

[70] Van Bokhoven, M.A et. al., *Diagnosis and Management of hereditary haemochromatosis*, BMJ (2011) 342:c7251, Accessible at: [http://www.bmj.com/content/342/bmj.c7251] Accessed 14-02-2012.

[71] Asberg, A. et. al.. *Screening for hemochromatosis—high prevalence and low morbidity in an unselected population of 65 238 persons.* Scandinavian Journal of Gastroenterology (2001) *36 1108-1115.*

[72] Pietrangelo, A., *Medical Progress: Hereditary Haemochromatosis – A New Look at an Old Disease*, *op. cit.*

[73] Feder J.N. et. al., *A novel MHC class I-like gene is mutated in patients with hereditary haemochromatosis*, Nature Genetics (1996) 13 (4) 399-408.

[74] Hemochromatosis. In: Online Mendelian Inheritance in Man, (2000) OMIM. Baltimore: McKusick-Nathans Institute for Genetic Medicine, Accessible at: [http://www.ncbi.nlm.nih.gov/omim/], Accessed 14-02-2012.

of 50%[75] However, another wider population study of 410 homozygotes found that 110 presented with no clinical symptoms, whereas 300 did. Further, it suggested that some of these 110 individuals could simply be presymptomatic.[76] This study therefore suggests a penetrance of at least 73%, possibly higher. The penetrance of the condition is therefore likely to reside between 50% and 75%. I am inclined to suggest it resides toward the higher end of the scale, as the second study has a far higher sample size than the first, and will therefore be more representative. HFE-related Haemochromatosis is therefore relatively penetrant and genetic test data a reasonable indicator of risk, although it is difficult to suggest exactly if or when the mutation will be expressed.[77]

Genetic testing is however often helpful in effectively diagnosing the condition. This is especially true as non-genetic diagnosis of haemochromatosis is not easy. Patients often present with nonspecific complaints or symptoms, and the biological indicators can be linked with other diseases. This is why The British Society for Haematology suggest in their standard setting document that "a genetic test offers the best approach to early detection."[78] This need to test is something also highlighted by Adams, who claims that genetic screening is important as symptoms may go unrecognised and lead to the development of life threatening complications.[79] The condition would appear to fall toward the top end of the penetrance spectrum, with individuals approaching a 75% likelihood of developing the condition.

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[75] Olynyk, J. et. al., *A population-based study of the clinical expression of the haemochromatosis gene*, New England Journal of Medicine (1999) 341 (10) 718-724.
[76] Adams, P.C. et. al., *The relationship between iron overload, clinical symptoms, and age in 410 patients with genetic hemochromatosis* Hepatology (1997) 25 (1) 162-166.
[77] Pietrangelo, A., *Medical Progress: Hereditary Haemochromatosis – A New Look at an Old Disease*, *op. cit.*
[78] British Society for Haematology, *British Committee for standards in Haematology: Guidelines on diagnosis and therapy, Genetic Haemochromatosi*s (2000) Accessible at: [http://www.bcshguidelines.com/documents/haemochromotosis_2000.pdf] Accessed 14-02-2012.
[79] Adams, P.C. et. al., *The relationship between iron overload, clinical symptoms, and age in 410 patients with genetic hemochromatosis*, *op. cit.*

Time of Onset

Empirical studies suggest that HFE-related Haemochromatosis tends to onset in middle-age.[80] It therefore falls on the pre-retirement side of my binary.

Location within typology

The purpose of Haemochromatosis is to provide an example of a strongly penetrant genetic condition which can be effectively treated, and therefore potentially has low severity. This will allow me to briefly explore the duties that the state owes to these individuals with regard the provision of healthcare, and how this affects the justifiability of the use of their data.

4.2.5 – Hypertension

Unlike the other conditions I have selected, hypertension is often used to describe several different clinical conditions. Within the thesis, I am specifically referring to a generalised state of elevated blood pressure.

Severity

Unmanaged hypertension can lead to various secondary complications. At the less significant end of the scale, individuals with high blood pressure may be unable to complete strenuous exercise, or suffer from shortness of breath, or transient dizziness. More severe complications can include a substantially increased risk of cardio-vascular diseases, and a higher risk of cardiac events such as myocardial infarction.[81,82]

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[80] Pietrangelo, A., *Medical Progress: Hereditary Haemochromatosis – A New Look at an Old Disease*, *op. cit.*

[81] Mosterd, A. et. al. *Trends in the prevalence of hypertension, antihypertensive therapy, and left ventricular hypertrophy from 1950 to 1989,* New England Journal of Medicine (1999) 340 (16) 1221–1227.

[82] Kannel, W.B. *Elevated systolic blood pressure as a cardiovascular risk factor* American Journal of Cardiology, (2000) 85 (2) 251–255.

There are however many effective management strategies. The NICE management pathway indicates lifestyle changes should be suggested first, and that this can often substantially reduce hypertension and associated morbidity and mortality.[83] Should lifestyle changes not reduce hypertension, or the individual fail to actually carry them out, several pharmaceutical options are also available. Beta-Blockers, Thiazides, ACE inhibitors and ARB's can all prescribed to the patient and are effective at managing symptoms, controlling risk and reducing severity of outcome.[84,85,86,87]

The condition would therefore seem to be appropriately placed somewhere between Breast Cancer and Haemochromatosis, on the lower end of the severity spectrum. Whilst it is capable of having meaningful effects on both fair equality of opportunity and capability, there are several treatment options available which have proved effective at managing the condition, and reducing both morbidity and mortality.

Penetrance

The causes of hypertension are multifactorial. Genetic, lifestyle and environmental factors all affect an individual's risk of developing the condition. To provide a very brief and non-exhaustive list, hypertension has been found to be significantly affected by age, gender, smoking status, and body mass index,[88] as well as salt consumption, and other dietary effects.[89] Indeed, demographic factors and

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[83] NICE Pathways, *Management of hypertension*, NICE (2011) Accessible at: [http://pathways.nice.org.uk/pathways/hypertension/management-of-hypertension] Accessed 15-02-2012.

[84] Chen, J.M et. al. *Blood pressure lowering efficacy of beta-blockers as second-line therapy for primary hypertension*, Cochrane Database of Systematic Reviews (2010) 20 (1) CD007185.

[85] Ramsey, L.E, Yeo, W.W and Jackson, P.R. *Thiazide Diuretics: first-line therapy for hypertension*, Journal of Hypertension: Supplement (1992) 10 (3) S29-32.

[86] Heran, B.S et. al. *Blood pressure lowering efficacy of angiotensin converting enzyme (ACE) inhibitors for primary hypertension*, Cochrane Database of Systematic Reviews (2008) 8 (4) CD003823.

[87] Heran, B.S et. al. *Blood pressure lowering efficacy of angiotensin receptor blockers for primary hypertension*, Cochrane Database of Systematic Reviews (2008) 8 (4) CD003822.

[88] Stanton, J.L et. al., *Demographic, dietary, life style, and anthropometric correlates of blood pressure*, Hypertension (1982) 4 (5 Pt. 2) III135–III142.

[89] Appel, L.J. et. al., *A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group*, New England Journal of Medicine (1997) 336 (16) 1117–1124.

lifestyle choices are the main determinants of whether an individual develops hypertension.

There is however evidence that a variety of genes contribute significantly. Several genes have been hypothesised to have the ability to exert modest effects upon blood pressure, and many of these have been positively identified to date.[90,91,92,93,94] Taken together, what is the extent of this effect? Although the exact contribution is still unclear, a study of almost 24,000 women found that:

> "…an increase of one standard deviation in the genetic risk score was associated with a 23% increase in the odds of hypertension. Among individuals in the top decile of the risk score, the prevalence of hypertension was 29% compared with 16% in the bottom decile."[95]

It is therefore possible to conclude that although genetic factors may not be the major influence on whether an individual develops hypertension, higher risk individuals can be identified using genetic markers. In terms of actual use, an individual who knows they are in the top decile for risk may be more likely to undertake risk reducing lifestyle changes than an individual in the bottom decile. This is especially true given the material on perceptions presented within *Chapter One*, as the individual is likely to treat a genetic indication that they are at risk of hypertension more seriously than lifestyle or demographic data due to its perceived importance. Getting this information to individuals may therefore be important in reducing severity of outcome. The condition is therefore appropriately located toward the lowest end of the penetrance spectrum. Whilst genetic factors contribute to the development of the condition, they are by no means the most significant contributor.

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[90] Jeunemaitre, X. et. al. *Molecular basis of human hypertension role of angiotensinogen,* Cell (1992) 71 (1) 169–180a.

[91] Corvol, P. et. al. *Seven lessons from two candidate genes in human essential hypertensionangiotensinogen and epithelial sodium channel* Hypertension (1999) 33 (6) 1324–1331.

[92] Staessen, J.A. et. al. *M235T angiotensinogen gene polymorphism and cardiovascular renal risk,* Journal of Hypertension (1999) 17 (1) 9–17.

[93] Fornage, M. et. al. *Variation in the region of the angiotensin-converting enzyme gene influences interindividual differences in blood pressure levels in young white males* Circulation (1998) 97 (18) 1773–1779.

[94] O'Donnell C.J. et. al. *Evidence for association and genetic linkage of the angiotensin-converting enzyme locus with hypertension and blood pressure in men but not women: the Framingham Heart Study,* Circulation (1998) 97 (18) 1766–1772.

[95] The International Consortium for Blood Pressure Genome-Wide Association Studies, *Genetics variants in novel pathways influence blood pressure and cardiovascular disease risk*, Nature (2011) 478 (7367) 103-109.

Time of Onset

Hypertension related complications usually onset in late middle age, progressing into old age. Studies suggest the development ranges between ages 45 and 75 depending on the exact combination of risk factors an individual possesses. The majority of individuals will therefore develop this condition pre-retirement, although some may certainly only become symptomatic post age 65. As the majority will develop the condition pre-65, it is appropriately located on the pre-retirement side of my binary.

Location within typology

Finally, Hypertension is, within my typology, an example of a condition where the penetrance of the genes in question is doubtful. The condition is certainly multifactorial, and within that strong environmental and lifestyle factors are implicated. This condition will illustrate the duties that the state owes to individuals who may have made their condition worse through their own actions, and more widely, the justifiability of using data whose penetrance is questionable.

4.2.6 – Representative Conditions Summarised

Below is a table summarising the evidence base above for analysis within *Chapter Five*. The conclusions offered later for these conditions should be generalisable to all similar conditions, that is to say, those conditions which share a similar position in this table.

Condition	Severity	Penetrance	Time of Onset
Alzheimer's Disease	High	Mid	Post Retirement
Huntington's Disease	High	High	Pre Retirement
Breast and Ovarian Cancers	Mid to Low – Early High – Late	Mid to High	Pre Retirement
Haemochromatosis	Low	High	Pre Retirement
Hypertension	Low	Low	Pre Retirement

Having established this evidence base, I must now briefly assess the potential individual consequences of allowing the use of genetic test data by insurers, namely:

a) An increase in premium price as a result of increased risk, and

b) The potential to disincentivise individuals from obtaining genetic test results.

Later, within *Chapter Five*, I will then be able to assess whether it is justifiable to allow these to occur based whether an individual has either their fair equality of opportunity or capability compromised.

4.3 – Consequences of Use

4.3.1 – Policy pricing

The first important consequence of allowing the use of genetic test data is a change in policy pricing. As I have already highlighted, the UK insurance industry operates on principles of mutuality and actuarial fairness. An individual is therefore charged a premium representative of the risk they bring to the insured pool, with higher risk individuals attracting higher premiums. If an individual possesses genetic markers which indicate they are likely to suffer from a serious disease, they present a higher risk for both health and life insurance, as they are more likely to become ill, or in some cases to die. An insurer being able to access this data means that the insurer will likely consider these individuals a higher risk than would be the case could they not access the data in question, although this will obviously depend upon both the result of the test and the type of genetic data. However, there certainly exists the potential for these individuals to receive higher premiums than would be the case were the use of their data by insurers prohibited.

But what about those who do not possess genetic markers for certain conditions, and are able to present an insurer a relatively clear genetic test record? There is a case to be made that these individuals would benefit from a situation where insurers were allowed to use their test data, as they could be classified as

lower risk customers and receive lower premiums as a result. It is however doubtful as to how much this would occur. First, it seems plausible to suggest that as companies with profit motives, there exists a reason for insurers to charge the highest premiums they possibly can, and less incentive to allow discounts. This is not persuasive on its own, as one insurer offering lowered premiums for those with lower risk genetic data would mean that they would be more attractive to those individuals, and potentially obtain a greater market share as a result. However, if we look to the ABI Code of Practice on Genetic Tests, within the code, we see that insurers:

> "…must not offer individuals lower than standard premiums on the basis of their predictive genetic test results: that is, genetic test results cannot be used as a trigger for allowing preferred life underwriting terms."[96]

Given this code is agreed by the ABI, which represents the British Insurance Industry, it would seem that there currently exists an agreement not to use genetic test data to offer premiums and policies more favourable than the standard. The justifiability of using genetic data in this way will also be discussed within *Chapter Five*. Although only the current Code of Practice, and therefore subject to change, this provides us a useful insight into the current thinking of the insurance industry, and their potential position moving forward. There does however exist another way in which allowing the use of genetic test data might provide an advantage to some individuals. Indeed, in the same Code, we find that:

> "Insurers may use a normal (negative) predictive genetic test result to reduce the impact of loading which would otherwise have applied due to the applicant's known medical or family history."[97]

As an example, if an individual's family history suggests that one of their parents possesses a dominant gene mutation for a condition, it is likely that the individual in question will be assessed as having a 50% chance of developing the condition in question (through basic Mendelian Genetics). If an individual can provide the insurer a genetic test demonstrating that they have not inherited this condition their premium is not loaded in the same way.

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[96] ABI Code of Practice for Genetic Tests, June 2008, Accessible at:
[*www.abi.org.uk/consumer2/disclosure.htm], Accessed 24/08/10.*
[97] *Ibid.*

Within my analysis, I must therefore consider two different effects on premium pricing. First, it may be that upon production of a genetic test which indicates an increased risk, an individual may see their premium rise. Second, possession of a negative test may result in a fall in premium for the individual concerned. This reduction in premium may also have important effects on other individuals within the insurance pool. Most obviously, a widespread use of negative genetic test results would allow for identification by exclusion. If an insurer makes it clear that a negative test for a condition (for example, Huntington's) will attract a reduced premium, then consumers who have such a test would be motivated to provide it. The insurer may then assume that anyone who refuses to provide such a negative result must therefore have something to hide, and act to load premiums accordingly. Allowing widespread use of negative tests in this way could operate to harm individuals with Huntington's in the same way as mandating them to disclose their own data would.

Even if the insurer does not conclude that everyone who does not provide this data is, *de facto,* a carrier of a condition which increases their risk profile, these individuals are still likely to see their premiums rise. By providing negative test results, less risky individuals remove themselves from the aggregate pool and create their own 'low risk' pool of insured individuals. The aggregate risk of the remaining pool therefore increases because low-risk individuals have been withdrawn from it, and premiums rise for those who remain within it. Within my analysis, I will therefore have to consider not only the effect positive test results may have on those who hold them, but how far insurers should be allowed to take account of the negative tests, as this may also operate to disadvantage others.

In summary, allowing insurers to use the genetic test data of individuals therefore affects the setting of policy prices. For some individuals, this may result in increased premiums due to them being assessed as more risky than they otherwise would be. For others, and even in the absence of explicitly lower premiums offered for favourable genetic status, allowing the use of test data may help remove loading from their premium by allowing them to demonstrate that they possess a lower risk than had previously been assumed. This will be explored further within the analysis in *Chapter Five*.

4.3.2 – Disincentivisation

In order to assess whether allowing insurers to use genetic test data may disincentivise individuals from taking genetic tests, we must first answer a few preliminary questions. Given insurance is, in general, a policy taken out to guard against risk, we must explore how individuals make decisions about risk, and how and why they choose to take genetic tests. A summary of the literature in this area follows, and will help determine whether allowing the use of this data by insurers does carry a risk of disincentivisation.

4.3.3 – How do people make decisions about risk?

Possibly the pre-eminent theory of individual decisions about risk originates in the late 1970's, with Kahneman and Tversky.[98] In their account of what they term *Prospect Theory*, they provide a model of decision making which has several important aspects. The first, and perhaps most important of these is that individuals weight probabilities in a non-linear way.[99] Take for example a bet where you have a 50% chance of losing £10, and a 50% chance of gaining £20. Previous theories would suggest that an individual would (and should) always take the gamble, as the potential payoff would result in an increase in utility. However, *Prospect Theory* introduces another element. It suggests that an individual is much less likely to take the gamble if, for example, they are down to their last £10. A reduction in your amount of money from £10 to £0 is more significant to you than a reduction from £100 to £90. Individuals therefore weight the exact same probability of occurrence higher, dependant on their circumstances. Indeed:

> "…people look not at the levels of final wealth they can attain but at gains and losses relative to some reference point, which may vary from situation to situation…"[100]

Because of this, *Prospect Theory* provides an account of individuals as *loss-averse*. As Plous describes:

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[98] Kahneman, Daniel and Tversky, Amos, *Prospect Theory: An Analysis of Decision under Risk*, Econometrica (1979) 47 (2) 263-291.
[99] *Ibid.*
[100] Shleifer, Andrei, *Inefficient Markets: An Introduction to Behavioural Finance,* (Oxford: Oxford University Press 2000) pp. 10-11.

> "Moreover, the value function for losses is different than the value function for gains. [...] the value function for losses (the curve lying below the horizontal axis) is convex and relatively steep. In contrast, the value function for gains (above the horizontal axis) is concave and not quite so steep. These differences lead to several noteworthy results. Because the value function for losses is steeper than that for gains, losses "loom larger" than gains. For instance, a loss of $500 is felt more than a gain of $500."[101]

Prospect Theory therefore provides us with two important characteristics of individual decision making. First, it suggests that individuals weight probability non-linearly, and that the same probability is capable of having different importance dependant on relative circumstances. Circumstances such as a significant loss of health or risk of death are therefore likely to be considered more weighty than a simple financial loss. Second, it provides an account of individuals as *loss-averse*, in that they are less willing to gamble on losing something than they are to gamble for a gain.

This position is also supported by several empirical studies. First we have some simple studies of individual risk aversion. Although both studies found substantial heterogeneity, they both conclude that risk aversion is the most frequent situation.[102] Indeed, the study by Barksy et. al. suggests that "…low risk tolerance characterises most of the population."[103] Because individuals are *loss-averse*, it would therefore appear they are also *risk-averse*, in that they avoid taking risks due to the possibility of loss. Again, individuals possess differing levels of risk aversion, and some are certainly *risk-seeking*, but as a population trend (amongst adult Americans), individuals would appear to be averse to risk. This also would not appear to be hugely affected by culture. In a study of Rural India, Binswanger found that individuals were again generally risk averse, with wealthier individuals demonstrating slightly lower risk aversion than poorer ones.[104] I am unaware of a significant study having been performed on the UK population, but this is not problematic. First, I would suggest that UK culture is similar enough to that within

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[101] Plous, Scott, *The Psychology of Judgment and Decision Making*, (New York: McGraw-Hill 1993) p. 95 – 96.
[102] Hartog, J. Ferrer-i-Carbonell, A. and Jonker, N. *On A Simple Survey Measure Of Individual Risk Aversion* (November 2000). CESifo Working Paper Series No. 363. Accessed at SSRN: [http://ssrn.com/abstract=260938], Accessed 20-02-2012.
[103] Barksy, R. et. al. *Preference Parameters and Behavioural Heterogeneity: An Experimental Approach in the Health and Retirement Study*, (1997) 112 (2) 537-580.
[104] Binswanger, H. *Attitudes toward Risk: Theoretical Implications of an Experiment in Rural India*, Economic Journal (1980) 91 (364) 867-890.

the USA that the results of the first study would likely replicate. Second, as we know from the Binswanger study, this effect is largely cross-cultural anyway.

We can also see that individuals are risk-averse from other larger empirical studies. A study of contestants on the gameshow 'Deal or No Deal' found that whilst some individuals exhibited risk seeking behaviour (possibly due to chasing a loss, or the perception that they were not playing with their own money); most contestants were still moderately risk averse.[105] Indeed, this modification of behaviour with regard to the chasing of losses also demonstrates that decisions about risk are influenced by previous circumstances, and probability is not always assessed in a linear way. A further study of the same gameshow in Italy found that whilst individuals were what they termed *risk-neutral* at smaller stakes, higher stakes lead to the individuals again demonstrating risk-aversion.[106]

Studies also suggest that an individual's level of risk aversion is affected by their wealth, possibly because an individual's attitude to risk determines their earning potential. For example, those who are risk averse are less likely to invest in risky investments which may have large payoffs. Indeed, we find that risk aversion is highest amongst the poorest in society[107] and decreases with increases in wealth.[108] There is however limited evidence to suggest that the very poorest are actually less risk averse than those on average incomes, possibly because at that level one has to take some risks in order to survive.[109]

As a general trend, we therefore know two things of relevance. Individuals are generally both loss and risk averse, in that they fear losses more than they anticipate gains, and as such, are often reluctant to take risks. Further, risk aversion

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[105] Post, T. et. al. *Deal or No Deal? Decision Making under Risk in a Large Payoff Game Show*, American Economic Review (2008) 98 (1) 38-71.

[106] Bombardini, Matilde, and Trabbi, Francesco, *Risk Aversion and Expected Utility Theory: An Experiment with Large and Small Stakes*, Accessible at: [http://www.eui.eu/Personal/Guiso/Courses/Lecture3/Bombardini-Trebbi2007.pdf] Accessed: 21-02-2012.

[107] Riley Jr, W.B. and Chow, K.V. *Asset Allocation and Individual Risk Aversion*, Financial Analysts Journal (1992) 48 (6) 32-37.

[108] Hartog, J. Ferrer-i-Carbonell, A. and Jonker, N. *On A Simple Survey Measure Of Individual Risk Aversion*, op. cit.

[109] Barksy, R. et. al. *Preference Parameters and Behavioural Heterogeneity: An Experimental Approach in the Health and Retirement Study*, (1997) 112 (2) 537-580.

shows a negative correlation with individual wealth and a positive one with the stakes at play. We do not however yet know how this can be applied to genetic testing. In order to be able to answer this question, first we must look at why individuals choose to take genetic tests.

4.3.4 – Why do people take genetic tests?

In order to be able to conclude whether the above analysis on risk applies, we must now discover why these individuals might take these tests in the first place and whether a disincentivisation effect will exist. First, we know that whether an individual obtains a genetic test is affected by their risk of developing a genetic condition, with those who perceive themselves to be higher risk more likely to obtain tests.[110,111] Possibly the biggest reason for this is that those thought to be at high risk of developing genetic conditions will often be offered genetic tests within the healthcare context as a useful diagnostic tool. This could also be due to the prevailing perception of genetic information as a panacea which provides unique insight into future health status.[112] Further, we see that a willingness to take a genetic test increases with the medical usefulness of the test. For a condition such as Huntington's, where a cure is not available, and development of the condition cannot be slowed, test uptake is approximately 20%.[113,114] Uptake for genetic tests is much higher for conditions such as breast cancer, where early diagnosis is far more useful.[115] In brief, individuals are more likely to take tests when they a) perceive themselves to be at high risk of suffering from a condition, and b) feel that the test result would be useful in obtaining early diagnosis or appropriate treatment. These

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[110] Picone, G Sloan, F. and Taylor Jr., D. *Effects of Risk and Time Preference and Expected Longevity on Demand for Medical Tests*, The Journal Of Risk and Uncertainty (2004) 28 (1) 39-53.

[111] Witt, J. *The Effect of Information in the Utilization of Preventive Health Care Strategies: An Application to Breast Cancer,* Unpublished mimeo, Melbourne Institute of Applied Economic and Social Research, The University of Melbourne, Australia.

[112] Struewing J.P. et. al. *Anticipated uptake and impact of genetic testing in hereditary breast and ovarian cancer families*, Cancer Epidemiol Biomarkers Prev. (1995) 4 (2) 169–173.

[113] Craufurd D. et. al. *Uptake of presymptomatic predictive testing for Huntington's disease,* Lancet. (1989) 2 (8663) 603–605.

[114] Quaid K.A and Morris M. *Reluctance to undergo predictive testing: the case of Huntington disease,* American Journal of Medical Genetic (1993) 45 (1) 42–45.

[115] Lerman C, et. al. *BRCA1 testing in families with hereditary breast-ovarian cancer. A prospective study of patient decision making and outcomes*. JAMA. (1996) 275 (24) 1885–1892

factors are independent, and an increase in either leads to an individual being more likely to take a test.[116]

4.3.5 – How does this all relate to my question?

Given what we know so far, how does individual risk-aversion play out within the insurance context, and how is this relevant to genetic testing? First, insurance provides an excellent example of how individuals are actually generally risk averse. Despite the statistically small chance of making a significant claim, many individuals choose to take out insurance against a particular event because they are risk averse to it. Indeed, the insurance industry depends on an individual's risk-averse nature. As Crocker explains, the industry relies upon a "continuum of risk-averse consumers"[117] This is explained by *Prospect Theory*. Although we stand a statistically low chance of losing our house in a fire, we value not losing our house to the point where we are in most cases willing to pay for home insurance. In this way "…prospect theory…predicts prevailing risk aversion in insurance, which mostly concerns small-probability losses…"[118]

Insurance also appeals to an individual's risk-averse nature. Through the presentation of insurance as a gain, and the comparison with the loss of something important to you such as your health, or a house, uptake of insurance is high. As Hershey found:

> "…individuals focus on protective aspects when the situation is presented in an insurance context so that this is perceived as a gain."[119]

In addition to the theory, we can also see empirically that individuals weight probabilities non-linearly in automobile, home and health insurance.[120,121] Further,

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[116] Marteau, T and Croyle, R, *The New Genetics: Psychological Responses to Genetic Testing*, British Medical Journal (1998) 316 (7132) 693-696.

[117] Crocker, K. and Snow, A. *The Efficiency Effects of Categorical Discrimination in the Insurance Industry*, Journal of Political Economy (1986) 94 (2) p. 323.

[118] Wakker, P. et. al. *The Effects of Statistical Information on Risk and Ambiguity Attitudes, and on Rational Insurance Decisions*, Journal of Management Science (2007) 53 (11) 1170-1784.

[119] Hershey, J. et. al. *Sources of Bias in Assessment Procedures for Utility Functions*, Management Science (1982) 28 (8) 936-954.

[120] Barseghyan, Levon, et. al. *The Nature of Risk Preferences: Evidence from Insurance Choices* (June 30, 2011) Accessible at SSRN: [http://ssrn.com/abstract=1646520] Accessed 22-02-2012.

when considering insurance purchasing decisions, an individual tends to either be completely dismissive of the risk, or to be risk averse and over-insure, with a tendency toward the latter.[122] We also see loss aversion happening within the insurance context. In a study comparing the effect of price rises and price reductions, it was found that a price increase lead to far more defection to other companies than did other companies reducing their prices. This defection was attributed to the price increase being perceived as a loss as compared to a gain.[123] This is the risk that we are concerned about here. The above study by Dawes suggests that individuals perceive a price increase as a loss. If individuals are *loss-averse*, they may therefore be averse to taking a genetic test through fear of their premium increasing. However, they may also be averse to the adverse health effects that may result from not obtaining a genetic test. This is something which will be discussed within *Chapter Five*.

So far we are able to suggest that due to the generally risk-averse nature of individuals, insurance is an important commodity, and one that many individuals would like to possess. But why is this relevant to the use of genetic tests? If insurers are able to use this data, premium prices could increase as I have previously demonstrated. Doherty and Thistle suggest that because individuals currently exist in a situation where their exact risk status is uncertain, a situation whereby insurers are able to mandate the disclosure of genetic data creates a disincentive effect because:

> "…when insurers can access this data…the value of information to the uninformed is negative and they would never choose to become informed."[124]

In essence, an individual who does not possess a genetic test will, according to Doherty and Thistle, lack any incentive to obtain a genetic test. Through obtaining a genetic test, the consumer essentially enters into a lottery, where they either do not possess a relevant genetic condition and therefore become low risk and receive lower

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[121] Marquis, S and Holmer, M. *Alternative Models of Choice under Uncertainty and Demand for Insurance*, The Review of Economics and Statistics (1996) 78 (3) 421-427.

[122] McClelland, G. et. al. *Insurance for low-probability hazards: A bimodal response to unlikely events*, Journal of Risk and Uncertainty (1993) 7 (1) 95-116.

[123] Dawes, J. *Price Changes and Defection levels in a Subscription-type Market* Journal of Services Marketing (2004) 18 (1) 35-44.

[124] Doherty, N. and Thistle P. *Adverse selection with Endogenous Information in Insurance Markets*, Journal of Public Economics (1996) 63 (1) 83-102.

premiums, or they become high risk, and receive higher premiums. This is as compared to the mid-level premium that they likely currently receive in a state of uncertainty. Due to this:

> "…risk averse consumers prefer the 'uninformed' policy with certainty. Rational policyholders fail to invest in information…"[125]

As individuals are both loss and risk averse, they tend to prefer paying the mid-level premium over risking the possibility of a large loss through receiving a rise in premium price upon disclosure of a genetic test. Even Hoel et. al. who put forward an argument for allowing genetic test data to be used concede that risk aversion to economic consequences means that some individuals will not take tests.[126] This conclusion is consistent with Hoy's assessment of the situation, which is that individuals would prefer uncertain categorisation to the possibility of being classified in a higher risk category.[127] This is what Tabarrok terms *premium risk*. Given that people are both risk and loss averse, and the possibility of being classified at higher risk presents the chance of a relatively large financial loss for relatively little financial gain, moving from a situation of uncertain information to a certain one has negative utility, and a rational individual would never choose to do it.[128]

This is only the economic case however. An individual may be incentivised to take a test for other reasons, such as to achieve a better or earlier diagnosis, or to allow more effective treatment of their condition. The exact balance between incentives and disincentives will depend on the type of genetic data in question, and the condition or conditions to which it relates, something which will again be discussed within *Chapter Five*. Having established my typology, the evidence base for analysis, and the potential consequences of allowing use of genetic data by insurers, I can now proceed to analysis.

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[125] *Ibid.*

[126] Hoel, M. et. al. *Genetic Testing and Repulsion from Chance*, (April 2004). CESifo Working Paper Series No. 1181. Accessible at SSRN: [http://ssrn.com/abstract=542362] Accessed 22-02-2012.

[127] Hoy, M. *The Impact of Imperfectly Categorizing Risks on Income Inequality and Social Welfare*, The Canadian Journal of Economics (1984) 17 (3) 557-568.

[128] Tabarrok, A. *Genetic Testing: An Economic and Contractarian Analysis*, Journal of Health Economics (1994) 13 (1) 75-91.

Chapter Five
Can the use of genetic data be justified?

Within this Chapter, I will now proceed to analyse whether use of data relating to these conditions is justifiable. Below is a reminder of the conditions and their place within my typology, re-ordered to reflect their order of consideration within this Chapter.

Condition	Severity	Penetrance	Time of Onset
Alzheimer's Disease	High	Mid	Post Retirement
Huntington's Disease	High	High	Pre Retirement
Haemochromatosis	Low	High	Pre Retirement
Breast and Ovarian Cancers	Mid to Low – Early High – Late	Mid to High	Pre Retirement
Hypertension	Low	Low	Pre Retirement

First, I will assess whether the use of this information by insurers is likely to cause a disincentive effect, as it may be the case that a significant disincentive effect affects the development of the condition, for example, through the individual no longer being able to access the same treatment options. This may in turn affect the severity of the condition, and therefore the effect it is able to have on either fair equality of opportunity with regard to accessing employment, or an individual's capability to convert their existing primary goods. I will then proceed to analyse whether the development of the subsequent condition will disadvantage the individual, either with regard to their fair equality of opportunity, or their capability to convert their existing goods. This will then allow me to determine whether it is unjustifiable to further disadvantage these individuals through allowing use of their data.

Finally, I will assess the potential for adverse selection in each case. This is relevant, as if there is no potential for adverse selection, denying insurers' use of this specific information no longer results in premium price increases across the board. It may therefore be justifiable to deny access to this information due to concerns about

fair equality of opportunity or an individual's capability, even if this individual is not a member of the least advantaged group. However, if significant adverse selection is likely to occur, it becomes harder to justify keeping this information from insurers. This is something which will vary based upon the condition being examined.

In terms of Chapter structure, I will first examine Alzheimer's, as it is my only example of a condition which onsets post-retirement. This will be followed by Huntington's and Haemochromatosis, as both of these conditions are important due to their place on the severity spectrum, of which they occupy opposite ends. Fourthly, I will then assess BRCA as an example of a condition to which disincentivisation may be extremely significant, before finally assessing Hypertension, and the issues that this condition raises with regard to penetrance.

5.1 – Alzheimer's Disease

As we know from the evidence base presented earlier, Alzheimer's is a condition of high severity and mid-level penetrance which onsets on the post-retirement side of my binary. So, is the use of this data by insurers justifiable in this case?

5.1.1 – Does a disincentive effect matter here?

The disincentivisation effect is likely to be particularly strong in this case, as the individual does not obtain any diagnostic nor medical benefit from this test. Faced with a situation where obtaining a test means an individual will suffer a financial loss for no medical or health benefit, it would seem unlikely that an individual would choose to obtain a genetic test due to the reasoning provided within *Chapter Four*. This is especially true given uptake of tests for degenerative, terminal conditions is low anyway. However, although a disincentive effect exists, it is unlikely to matter. As the test is not medically useful and progression of the condition cannot be slowed, an individual will suffer similar effects on both their fair equality of opportunity and capability regardless of when (or indeed, whether) they become aware that they possess the condition prior to developing it. Whether an

individual is disincentivised from obtaining a test is therefore, in this case irrelevant as there are no treatment options an individual is able to access with this genetic information that they would not otherwise be able to. The treatment offered and progression of the condition remains the same, and therefore, all relevant effects on both fair equality of opportunity and capability also remain the same.

5.1.2 – Is fair equality of opportunity compromised?

So, does the development of Alzheimer's disease meaningfully inhibit an individual's fair equality of opportunity? Specifically, given what was said earlier about the attachment of income and wealth to positions of employment, the appropriate question is whether the development of the condition limits an individual's ability to obtain or maintain employment under these conditions.

Given the condition is one of high severity; development of symptoms therefore significantly limits an individual's ability to compete under conditions of fair equality of opportunity. We also know that the condition is one of middle or moderate penetrance, and as such, it is likely that an individual will develop the condition over the course of a whole life. Indeed, in *Chapter Four*, we find that for certain combinations of APOE-4, median onset occurs before seventy years of age, which means that an individual is likely to develop the condition over the course of an average lifespan.[1] Possession of these specific allele combinations therefore means that the individual is likely to suffer the severe effects associated with the symptoms of the condition. We also know that the progression of the condition cannot be slowed, and there is no known cure. As such, the provision of health care does little to mitigate the effects of this condition, and the condition remains severe. However, when we come to consider time of onset, the usefulness of this particular representative condition becomes apparent. As we know from earlier, the condition is one which occurs on the post-retirement side of my binary distinction. Even those conditions which onset at age 60 will not progress to the more severe late-stage until after age 65.

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[1] Skeehan, K. et. al., *Impact of Gene Patents and Licensing Practices on Access to Genetic Testing for Alzheimer's Disease*, Genetic Medicine (2010) 12 (4 Suppl.) S71-S82.

Although an individual does undoubtedly depart from normal functioning here, this does not occur until after retirement. [2] As such, the development of this condition will not significantly affect an individual's fair equality of opportunity. By the time symptoms progress to the point where they will compromise an individual's ability to access or maintain employment, it is likely that the individual will have retired. They will therefore have been able to compete for a whole working life in the same way as someone who does not develop this condition. This means that the share of wealth they are able to obtain from employment will be the same as an identical individual who does not develop Alzheimer's. The fact that they are no longer able to compete therefore does not matter in this sense.

5.1.3 – Is capability compromised?

Although an individual does not have their fair equality of opportunity compromised, and so can access similar primary goods as an unaffected individual, their capability to convert their primary goods into ends might still be compromised. According to the approach outlined within *Chapter Two*, we know that there can exist variability in an individual's capability to convert primary goods into ends. If they are unable to convert these goods as effectively as others through no fault of their own, they are meaningfully disadvantaged. The idea of capability as the morally relevant factor is the *capabilities approach* as described by Sen.[3] We also know from *Chapter Two* that we have a moral duty to provide healthcare services to attempt to mitigate this, and indeed, both health and social services are provided free at the point of service under the *status quo*.[4]

However, earlier within *Chapter Four*, we learn that this is condition for which there is no available cure, and the progression of which cannot be significantly slowed. As such, the provision of these services is not radically helpful, although it may mitigate this disadvantage in two ways. First, because an individual in the late stages of the condition (and who may be bed-bound) might otherwise have to use

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[2] With regard to species functioning. See *Chapter Two*, specifically *Daniels*.
[3] Sen, Amartya, *Inequality Reexamined*, (Cambridge Massachusetts: Harvard University Press 1995)
[4] Daniels, Norman, *Just Health: Meeting Health Needs Fairly*, (Cambridge: Cambridge University Press, 2008)

some of their wealth in order to purchase necessary healthcare and social support, assuming they lack a supportive family. The presence of the care system within the *status quo* at the very least prevents these individuals from losing primary goods through the costs associated with managing their condition. Secondly, the care provided seeks to "maximise functioning and independence", and as such, helps these individuals retain capability that they may not possess was this care not provided.[5] The situation could certainly be worse.

However, as compared to the individual who does not develop Alzheimer's, it would seem difficult to argue that the individual with the condition does not have their capability compromised. An individual who has severe problems with their memory (to the point of being unable to recognise close family) and who will eventually be bed-bound, is unable to actualise their interests and goals in the same way as an individual who does not suffer from these symptoms.

As the entire purpose of the theory is to "attempt to mitigate the arbitrariness of natural contingency and superficial fortune" it would seem we must be concerned here.[6] However, the relevance of this loss of capability is somewhat mitigated by the time of onset of the condition. As the condition does not develop until post-retirement, and the most severe symptoms between eight and ten years later, our comparative is that of an individual aged between 70 and 80 who does not suffer from Alzheimer's. It is likely that, by this age, the majority of individuals will have developed some form of illness or disability which interferes with their ability to convert their primary goods into ends. As such, whilst the individual with Alzheimer's is certainly disadvantaged, it would appear that they are disadvantaged less than they would be if their condition developed earlier.

We also know from *Chapter Four* that allowing the use of this test data would disadvantage these individuals with regard to primary goods. If an individual must disclose possession of these genetic markers to insurers, their premiums will rise, consistent with the principles of mutuality and actuarial fairness. Immediately,

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[5] Small GW, et. al. *Diagnosis and treatment of Alzheimer disease and related disorders. Consensus statement of the American Association for Geriatric Psychiatry, the Alzheimer's Association, and the American Geriatrics Society*. JAMA. (1997) 278 (16) 1363–1371.

[6] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.*, p 82.

they suffer a loss of wealth as compared to an individual who does not have to disclose such data. Whilst in many cases this premium increase is likely to be relatively small (due to family history data already increasing their premium) those without family histories of Alzheimer's are likely to see their premiums rise significantly. Allowing insurers to use this data would therefore disadvantage these individuals with regard to possession of the primary good of wealth. Equally, it would seem that allowing insurers to take account of negative test results would also harm those individuals who possess positive ones, either through identification via exclusion, or through increasing the aggregate risk of the pool which they occupy.

However, whether it is justifiable to disadvantage these individuals in this way not only depends on whether their fair equality of opportunity is compromised, or whether their capability is, but the effect that the denial of this information may have on other policy holders. In order to assess whether allowing use of this data is justifiable, I must first determine the effect that the prohibition on use of this data may have on others. This is where the material on adverse selection becomes important.

5.1.4 – What level of adverse selection is likely?

Assuming individuals possess tests for the condition, there are two elements relevant to the risk of adverse selection, the prevalence of the mutation within the population, and the degree to which the genetic marker modifies an individual's risk status. If a mutation is more prevalent, more individuals will possess said mutation, and will therefore have their risk mis-assessed. The more individuals who have their risk underestimated, the stronger the adverse selection effect becomes, as the difference between the actual risk the pool presents, and the risk that the insurer think that the pool presents grows. The degree to which an individual's risk is underestimated is also important. If the mutation does not place the individual at a significantly higher risk than other data (such as family history) would indicate, the adverse selection risk is somewhat minimised, as an insurer is already able to access that data, and use it within actuarial calculations. However, if it does, adverse

selection is much stronger, as insurers assess individuals as much less risky than they actually are.

Using these criteria, adverse selection is likely to be moderate here. Although the possession of certain genetic markers (such as APOE-4 mutations) increases an individual's risk of developing Alzheimer's, the disease is multifactorial. Indeed, as we know from above, genetic markers contribute approximately 50% of the attributable risk.[7] Within this genetic component of risk, an insurer will therefore likely not hugely underestimate the risk an individual presents. Certain individuals may possess family history data which suggests they are at high risk for developing the disease, something which would already be incorporated into their risk profile. As such these individuals would not significantly contribute towards adverse selection.

However, the analysis so far assumes that all individuals possess information about their condition at the point they purchase insurance. For adverse selection to operate, these individuals need to *know* that they are at higher risk at the point they purchase insurance. As there is a disincentive to test here, it is unlikely that a large number of individuals possess data relating to their genetic status, as there is no medical incentive to obtain a test, and tests are not routinely offered in the medical context (consistent with *Chapter Four*). As such, adverse selection is likely to be small and controllable, and the insurer likely to adapt without increases in premiums across the board.

5.1.5 – Conclusions

In conclusion, I find the following. First, it is unlikely that fair equality of opportunity will be significantly compromised. Although the condition is certainly severe enough to substantially interfere with employment prospects, and certain combinations of genetic markers are significantly penetrant, it is not likely to onset until post-retirement, and as such, an individual's employment prospects would not be affected. An individual who develops Alzheimer's will therefore, over the course

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[7] Skeehan, K. et. al., *Impact of Gene Patents and Licensing Practices on Access to Genetic Testing for Alzheimer's Disease*, Genetic Medicine (2010) 12 (4 Suppl.) S71-S82.

of a whole life, earn similar wealth to an identical individual who does not develop the condition.

Second, whilst the affected individual's ability to convert their primary goods is certainly compromised, this is significantly mitigated in several ways. First, social and health care is focused on maintaining independence and function, and will in part mitigate this loss of capability. Secondly, the majority of individuals of this age are likely to have their capability compromised. As such, capability is not hugely compromised as compared to an identical individual without genetic markers for Alzheimer's disease.

The potential for adverse selection in this case is, whilst present, also small. Few individuals will hold data relating to their Alzheimer's risk status, as it is not diagnostically nor medically useful, and therefore would not be offered as a matter of medical course. Individuals are unlikely to seek out their genetic status from private providers, as it would not be useful to them, and they have little incentive to do so. Further, many individuals will already possess family history suggesting a risk of developing the disease, and their premiums will already be appropriately loaded. Information asymmetry between insurers and the insured is therefore small, and the risk of significant adverse selection is low. As such, the insurer is able to cope without raising premiums.

Given the above, it is justifiable to prevent the use of data relating to this condition. Without a risk of adverse selection, there no longer exists a risk that preventing the use of this data would act to harm members of the least advantaged group through a rise in premiums across the board. Without this harm, we are left purely considering those individuals who do go on to develop Alzheimer's. Although their fair equality of opportunity is not compromised, their capability to use their existing primary goods is somewhat compromised as compared to an identical individual (even accounting for age), and as such, it would be justifiable to prevent the use of their data for this reason. Equally, it would be justifiable to prevent the use of negative test results, as this would also operate to harm the individuals who will go on to develop the condition either through identification by exclusion, or through changing the composition of the risk pool.

This conclusion is strengthened by other concerns relating to the use of genetic information. Given the sensitivity of this data and the fact that individuals view it as especially meaningful or important, there may be significant privacy concerns when we begin to talk about disclosure. There is no room within this thesis to provide a detailed account of the material relating to genetic privacy, but good starting points can be found here.[8,9] For a specific discussion on the tension between insurance and medical research using genetic data (although health, rather than life), see Townend.[10] Given the fact that denying the use of this data raises no issue of adverse selection, and therefore means that denial of this information cannot negatively impact those who are members of the least advantaged group, it is legitimate to prevent use of this data out of concern both for the reduced capability of these individuals, and wider privacy and accuracy concerns. As I established within *Chapter One*, our understanding of genetic information is still at a formative stage, and absent significant harm, it is sensible to pursue a precautionary approach.

5.2 – Huntington's disease.

Second, let us consider another severe condition, Huntington's. Again, as we know from *Chapter Four*, Huntington's is a condition which can be characterised as extremely severe, extremely penetrant, and which onsets pre-retirement in middle-age.

5.2.1 – Does a disincentive effect matter here?

Much as with Alzheimer's, the disincentive effect would appear to be strong in this case, as the test is not medically nor diagnostically useful, and premiums would rise. Indeed, studies suggest that uptake of genetic tests for Huntington's is only around 20% even in a situation where use is prohibited.[11] It is therefore unlikely

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[8] Laurie, Graham, *Genetic Privacy: A Challenge to Medico-Legal Norms*, (Cambridge: Cambridge University Press 2002)

[9] Taylor, M.J, and Townend, D, *Issues in Protecting Privacy in Medical Research Using Genetic information and Biobanking: The PRIVILEGED Project,* Medical Law International, 2010, 10 (4) pp. 253-268.

[10] Townend, D, *Privacy, health insurance and medical research: tensions raised by European data protection law,* New Genetics and Society, 2010 29 (4) pp. 477-493.

[11] Craufurd D. et. al. *Uptake of presymptomatic predictive testing for Huntington's disease, op. cit.*

that many individuals will possess information about their genetic status. However, again as above, it is unlikely that this matters. Given that the disease is terminal and progression cannot be slowed, knowledge of whether the individual is or is not a sufferer of this condition is not useful with regard to mitigating the effects. Again, an individual will suffer the same severity of condition, and thus, the same effects on both their fair equality of opportunity and capability, regardless of whether they obtain a test result. The condition and symptoms progress the same in either case. As such, the fact that an individual may be disincentivised from taking a test (and therefore prevented from knowing their genetic status) does not have any meaningful effects either on their fair equality of opportunity, nor their capability to convert their primary goods. In either case, the appropriate condition to consider remains one of extreme severity.

5.2.2 – Is fair equality of opportunity compromised?

We know from above that the condition is essentially a *worst-case scenario*, being both extremely severe and extremely penetrant, and occurring in middle-age. When compared with an individual who does not possess this genetic marker, we see a clear difference. After the onset of the condition, the individual with Huntington's will be unable to fairly compete for employment, as they will begin to suffer from the physical and mental decline characteristic of the disease. Progression of the disease further compromises this ability to compete. Dysphagia and dysarthria, combined with the development of the chorea from which the condition draws its name mean that the individual is unlikely to be able to maintain employment at all. They certainly are unable to compete fairly for employment when compared to an individual who does not suffer from Huntington's. This is a meaningful inhibition as (unlike Alzheimer's), it manifests in middle age, a time of life when an individual is likely to still be in employment. It can certainly be said that possession of this mutation leads to a situation whereby an individual's inability to compete means that their "….shares [shrink] below what is fair" as they become unable to compete through no fault of their own.[12] As compared to an individual who does not develop

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[12] Daniels, Norman, *Equality of What: Welfare, Resources, or Capabilities?*, *op. cit.*, p. 283.

Huntington's, the individual who does is substantially less able to obtain or maintain employment, and as such, has a lower expectation of lifetime wealth.

5.2.3 – Is capability compromised?

In addition to the inability to obtain wealth through employment under conditions of fair equality of opportunity, the individual with Huntington's may also have their capability to convert their primary goods compromised through the development of their condition. Similar to the analysis within *5.1.3*, we find that although we have a moral duty to provide healthcare services, they are not helpful in this case.[13] The condition is again one which is progressive and terminal, and for which there is no effective treatment. Social care does in some cases mitigate this, for example, the individual with late-stage Huntington's who is bed-bound would receive social care funded by the state. This prevents them from losing wealth through having to find and fund care from other sources. There is however little that can currently be done to restore or retain capability.

As compared to the individual who does not develop Huntington's, this individual's capability is clearly compromised. Toward the middle stages of the disease (likely between ages 40 and 50) the individual with Huntington's is beginning to suffer symptoms such as difficulty walking, hallucinations, and worsening chorea. This development of symptoms will substantially inhibit their ability to convert their primary goods. This capability is only further infringed by the development of late stage symptoms, especially as the individual often becomes bed-bound and requires round-the-clock care. There is little we can do to mitigate this, and as such, we must consider this individual relevantly disadvantaged. Unlike Alzheimer's, this lack of capability also develops in middle-age, when our comparative individual is likely to still be fully functional. The individual with Huntington's is therefore even further disadvantaged.

The individual who possesses the mutation linked to Huntington's will again present a higher risk to the insured pool than the individual who does not. As such,

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[13] Daniels, Norman, *Just Health: Meeting Health Needs Fairly*, (Cambridge: Cambridge University Press, 2008)

their premiums will rise if insurers are allowed to use their data. Even if they possess family history suggesting that they have the allele for Huntington's, a genetic test both confirms this diagnosis, and allows the insurer to obtain further data such as CAG repeat numbers, useful in assessing time of onset. This rise in premium price means disadvantages them with regard to possession of an important primary good, wealth. However, consistent with my earlier analysis, this increase in premiums is likely to be relatively small, as in the majority of cases; it is likely that family history information would mean that these individuals' premiums would already be loaded to account for the risk of them developing the condition. However, for those individuals who do not possess prior family history data, premium rises are likely to be very large, as their risk of death will substantially increase. It would therefore seem difficult to justify allowing the use of genetic data relating to the development of Huntington's. Similar analysis also applies with regard to negative test results, again, either through allowing identification by exclusion, or increasing aggregate risk. However, before I am able to conclude, I must again assess the potential for adverse selection in this case, and therefore, the effect that prohibition may have on other policy holders.

5.2.4 – What level of adverse selection is likely?

Again, we are first considering two things here. First, the prevalence of the genetic status within the population, and the degree to which it modifies an individual's risk. With regard to the first consideration, it is important to note that Huntington's is a rare condition, with a prevalence of between five and seven people per 100,000 affected in Western Countries.[14] There is however some suggestion that this represents a significant underassessment with regard to the U.K. As of June 2010, the Huntington's disease Association had 6376 symptomatic individuals on its books, a prevalence of 12.4 per 100,000.[15] An All-Party Parliamentary group has been set up to investigate prevalence, however, even if the true figure rests somewhere closer to 12.4 per 100,000, the condition is still rare. Indeed, within the

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[14] World Health Organisation, *Genomic Resource Centre*, Accessible at: [http://www.who.int/genomics/public/geneticdiseases/en/index2.html#HD] Accessed 07-03-2012.
[15] Spinney, Laura, *Uncovering the true prevalence of Huntington's Disease*, The Lancet Neurology (2010) 9 (8) 760-761.

European Union, a rare disease is defined as one which affects fewer than five people per 10,000, or fifty per 100,000.[16] The prevalence of Huntington's falls within these boundaries. As such, the effect of adverse selection is small, as few individuals will have their risk mis-assessed in this case. Actuarial work by MacDonald indicates that the insurance industry would be able to cope with the risk of adverse selection presented here, suggesting:

> "The former [Monofactoral Conditions] are rare enough that solutions outside of the free market should be sought…"[17]

According to MacDonald, the rarity of the condition means that the industry is likely to respond in a way other than with an increase of premium. As the adverse selection risk is small, the industry would not suffer a substantial hit to profit, and could adapt.

Further, Huntington's disease is an autosomally dominant condition, and as such, any carrier of the mutation is guaranteed to develop the condition. As such, there exists strong family history data here, as for an individual to possess such a mutation, one of their biological parents must have possessed it, and therefore must have suffered from the condition. Whilst there will exist situations where an individual's family history cannot be adequately traced, or an individual's parents have died before they could develop symptoms, in most cases family history will be present. As such, risk will not be hugely miss-assessed, as premiums will already be loaded based on the risk of inheritance of the mutation. This means that the risk of the insured pool is not hugely distorted and further reduces the risk of adverse selection, making it more likely that solutions other than premium increases will be found by insurers.

Finally, much like Alzheimer's, few individuals will possess information on their risk status in this case, as individuals are unlikely to obtain a genetic test. Therefore, the chance of significant information asymmetry is negligible, and as such adverse selection is unlikely to occur. Given all of this, the effect of adverse

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[16] European Commission, *Rare Diseases*, Accessible at: [http://ec.europa.eu/health-eu/health_problems/rare_diseases/index_en.htm] Accessed 07-03-2012.
[17] MacDonald, A, *Genetics and health costs: some actuarial models*, Law, Probability and Risk (2002) 1(2) 97-118.

selection in this case is minimal, and actuarial work suggests that there would be no premium increase were this information to be withheld from insurers.

5.2.5 – Conclusions

In this case, I find first that fair equality of opportunity is meaningfully compromised. The condition is both extremely severe and extremely penetrant, and as such, the individual is likely to suffer from the effects of the condition over the course of their life, specifically, in middle-age. The condition is therefore one which meaningfully interferes with an individual's ability to compete for employment, and therefore, to compete for the primary good of wealth. Over their life, the individual with Huntington's will be able to compete for and earn substantially less wealth than an identical individual without the condition, not only because their equality of opportunity is inhibited, but also because they will die much earlier.

In addition, the individual also has their capability to use their primary goods significantly compromised. Although social care is provided, and will act to prevent the condition imposing financial costs upon the individual, the development of symptoms would, in this case, act to substantially inhibit the individual's ability to convert their wealth into ends. Again, as compared to the individual without the condition, they spend a significant portion of their life being unable to effectively convert the wealth that they do obtain. The individual therefore not only possesses less wealth, but is less able to use it than an individual who does not develop Huntington's.

The potential for adverse selection in this case is, whilst present, also very small. It is unlikely that many individuals will hold genetic information relating to their risk status (as the test is not medically useful) and as such, the effect of information asymmetry will be limited. This means that the insured pool does not become significantly distorted. Further, premiums will already be loaded with family history data which is likely to be present in this case, which means that premium rises would be small anyway. Finally, the condition is also rare enough that insurers

are unlikely to raise premiums overall, and instead look for solutions outside the free market anyway.

Assuming the above, the conclusion is relatively simple. In this case study, we have on one side an individual who is severely disadvantaged both in terms of fair equality of opportunity and capability through no fault of their own. Allowing access to their data would act to further disadvantage them through a likely rise in their life insurance premium. These individuals are also amongst those who are most likely to need life insurance, as they are guaranteed to die at an early age, and therefore more likely to need to support any dependents they leave behind through the mechanism of insurance. On the other hand, preventing the use of this data carries no third party harms, and would not see premiums rise for members of the least advantaged group. Therefore, the just approach would be to prevent use of this data by insurance companies in setting premiums.

5.3 – Haemochromatosis

At the other end of the severity spectrum is Haemochromatosis. Whilst the development of complications can prove fatal if untreated, effective management of the condition through regular phlebotomy can prevent secondary complications.[18] The condition is therefore one of low severity, relatively high penetrance and onsets on the pre-retirement side of the binary. Again, before I can assess justifiability I must briefly discuss disincentivisation. This is relevant because in this case, a genetic test is incredibly useful in diagnosing the condition. An individual who is disincentivised from obtaining one may then not know that they have the condition. This in turn means that they would not receive regular phlebotomies, and severe complications could develop. The severity of the condition therefore depends on how effectively it is able to be diagnosed, and whether individuals are likely to obtain a genetic test result is part of that.

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[18] Pietrangelo, A., *Medical Progress: Hereditary Haemochromatosis – A New Look at an Old Disease*, *op. cit.*

5.3.1 – Is there likely to be a disincentive to obtain a test?

Unlike both Alzheimer's and Huntington's, there is a large incentive to test for this condition. Without a genetic test, an individual is unlikely to discover they suffer from the condition at an early stage, as the onset of symptoms is extremely non-specific, often manifesting in general complaints such as joint pain or fatigue. By the time more specific symptoms develop, the patient will already be suffering from the effects of secondary complications. Further, there is a very effective and relatively non-invasive treatment option available which can prevent the development of complications. We know from within *Chapter Four* that individuals become more likely to obtain genetic tests both when they feel at risk, and when they feel that something is able to be done. Given the penetrance of the condition, it might be that an individual possesses a family history of Haemochromatosis, meaning they feel at risk and are likely to take a test. Even in the absence of this, the fact that the test is extremely useful in effectively diagnosing the condition early means that individuals are likely to obtain it, especially as it is offered in a medical context. Consistent with earlier analysis within *Chapter Four*, it is therefore likely that individuals choose to undergo genetic testing within the medical context, as they know it will be medically beneficial for them.

It is unlikely that allowing the use of test data by insurers does anything to change the likelihood an individual will obtain a test. On one side of the calculation, obtaining a test is beneficial to the individual, as it allows them to receive an early diagnosis of the condition. This in turn means that the condition can be properly managed through regular phlebotomy, and the complications associated with the condition prevented. On the other side, the increase in premium pricing is likely to be extremely small for these individuals, as properly treated; the condition does not substantially increase the risk of mortality relative to the general population. As such, there exists on one side a small disincentive effect created by a small increase in premium prices. On the other, there exists a large incentive to test as an early diagnosis hugely affects eventual prognosis and the development of complications.

The risk of loss from not undertaking a test is therefore much higher than the risk from undertaking one, as the comparison is of a very small financial loss set against the potential to suffer life-threatening complications as a result of being unable to receive early diagnosis and thus early treatment. As individuals are both *loss* and *risk averse*, it seems likely that they would therefore act to prevent the large potential loss by obtaining a test. This is again consistent with the analysis presented within *Chapter Four*. It would therefore seem that there exists no relevant disincentive to test here, and the appropriate level of severity to consider is that of the condition if diagnosed early and properly treated. The condition therefore remains one of low severity.

5.3.2 – Is fair equality of opportunity compromised?

Again, as elsewhere in the Chapter, the appropriate question here is whether an individual's ability to compete fairly for employment is compromised, as this is one of the positions to which an individual's ability to obtain wealth is most commonly attached.

Given what was said above about disincentivisation, it is likely that the individual will obtain a diagnosis and therefore receive appropriate treatment. The relevant severity is therefore low. With appropriate treatment, the individual is therefore unlikely to suffer from any complications that would inhibit their ability to compete for, obtain or maintain employment in any way. Further, the treatment for the condition is relatively simple, and the need to undergo phlebotomies will not affect the ability to compete. The fact that the condition is of high penetrance does not matter in this case, as the severity is so low. Even though the individual in question is extremely likely to develop the condition, the condition does not infringe upon their fair equality of opportunity. The same is true of time of onset. Although the condition onsets pre-retirement, and therefore would in theory be capable of affecting employment, the fact that severity is so low means that this is also irrelevant. As compared to an identical individual without Haemochromatosis, the individual who does develop this condition has the same lifetime expectations with regard to competition for employment, and thus, all else being equal, the same

lifetime expectation of wealth. As such, the individual does not appear to be meaningfully disadvantaged. This condition is useful in illustrating the importance of severity to the overall analysis. If severity is sufficiently low, neither time of onset nor penetrance matter, as the development of the condition does not meaningfully infringe fair equality of opportunity.

5.3.3 – Is capability compromised?

Unlike both previous conditions, this individual does not suffer a significant loss to their capability to convert their primary goods either. The individual who suffers from Haemochromatosis is entitled to medical services (such as their regular phlebotomies) which the state is duty bound to provide. This is not an issue within the UK context, as the NHS does currently exist, and does currently provide this service. Was the thesis to be set within the US context, an interesting debate could be had here about provision of healthcare, and access to health insurance.[19] However, currently under the *status quo*, this healthcare is already provided.

Given that the individual is able to access adequate and effective healthcare free at the point of service, they first do not have wealth costs imposed upon them through having to purchase healthcare. Further, the provision of treatment in this case means that they do not then go on to develop complications, and their prognosis and life-expectancy is similar to an individual who does not develop Haemochromatosis. For this reason, the individual does not have their capability affected. When compared to an individual without Haemochromatosis, both individuals (again, all else being identical) will have the same capability to convert any primary goods they possess, as any symptoms which could infringe upon capability are prevented by regular phlebotomy. The fact that this individual happens to have Haemochromatosis therefore has no significant effect on capability.

Further, as regards premium pricing, we have already established that these increases are likely to be small. Appropriately treated, these individuals do not

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[19] For those interested in these issues, Norman Daniels again provides a good entry into the debate from a Rawlsian perspective. Specifically, Daniels, Norman, Just *Health: Meeting Health Needs Fairly*, (Cambridge: Cambridge University Press, 2008)

present a significantly increased risk to the insured pool, as they have life expectancy similar to the general population. As we are talking about life insurance, the relevant risk calculation is how likely the individual is to die from their condition and these individuals have, at worst, a very small increased chance of death. They therefore, they only suffer a mild financial disadvantage. Given these individuals suffer neither a loss to their fair equality of opportunity, nor to their capability to convert their primary goods, it would it seem initially that the use of data relating to this condition is able to be justified. Before I am able to conclude however, I must again briefly examine the potential for adverse selection in this case in order to assess the effect of prohibiting the use of this data.

5.3.4 – What level of adverse selection is likely?

Given the large incentive to test, and the fact that the condition is relatively common (approximately 1:200), one might expect the risk of adverse selection to be high, as many individuals possessing information about their genetic status leads to large information asymmetry.[20] However, adverse selection is actually minimal here. Although information asymmetry is certainly high, this asymmetry does not lead to substantial adverse selection. The possession of a genetic test result which is positive for HFE related Haemochromatosis operates to reduce the risk an individual presents rather than to increase it. An individual who receives an early diagnosis can undergo treatment for the condition, and therefore, not go on to develop complications which may make them more likely to die. Therefore, an individual who possesses a genetic test for this condition presents little more risk to the insured pool than an individual who does not have Haemochromatosis, as they have a similar life expectancy to the general population.

As such, even though there is likely to be large information asymmetry here, there exists little risk of adverse selection, as the insurer will not significantly underestimate the risk of the insured pool. Further, individuals are unlikely to feel that they are at high risk, and therefore engage in the purchasing behaviour necessary to cause adverse selection (purchasing more insurance). Even if the insurer were to

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[20] Haemochromatosis Society UK, *Prevalence of Haemochromatosis*, Accessible at: [http://www.haemochromatosis.org.uk/] Accessed 08-03-2012.

be aware of the individual's genetic status, the increase in premium price would be extremely modest. This in turn means that adverse selection will be manageable in this case, and therefore, a rise in premium prices across the board will not occur. As such, there is no harm to the least advantaged group through prohibiting the use of this data.

5.3.5 – Conclusions

In conclusion, it is unlikely that an individual will be significantly disincentivised from obtaining a genetic test. If insurers are able to use this data and an individual obtains a test, the worst-case scenario is that they receive a small increase in the cost of their life insurance premium. In contrast, if an individual does not obtain a test, their condition is significantly harder to diagnose, and therefore there is a risk that they are unable to obtain adequate treatment at an early stage. This could then lead to the development of significant complications, including life-threatening ones. As individuals are both *risk* and *loss-averse* they would likely act to avoid the large risk of life-threatening symptoms developing and instead obtain a test and accept the small premium increase. The appropriate severity to consider is therefore extremely low.

If complications are prevented, this individual does not have their fair equality of opportunity compromised; as they are no less able to compete for employment than they would be did they not have Haemochromatosis. The treatment required is relatively non-invasive, and is will not interfere with the ability of the individual to obtain nor maintain employment. As such, they are able to access the same amount of wealth as an identical individual who did not possess the condition over a whole life. Second, their capability to convert their existing primary goods is not compromised. Treatment is free at the point of access and as such, accessible to all. An individual who receives appropriate treatment will not suffer a loss of capability when compared to an individual without the condition. They are therefore not disadvantaged in this way either.

The individual is therefore not meaningfully disadvantaged either in terms of fair equality of opportunity, nor capability, meaning that it is not unjustifiable to use their data. The individual with Haemochromatosis would only receive a small increase in premium price upon disclosure of this data, if indeed they received one at all. Indeed, the fact that possession of a genetic test makes diagnosis and subsequent treatment easier, and the incredibly good prognosis one is able to obtain with this early treatment means that the individual who possesses and discloses this data might actually be viewed as a lower risk, and receive a premium reduction. Equally, there is no reason to necessitate disclosure of this data, as the risk of adverse selection being significant enough to lead to premium price increases appears incredibly small. This means that prohibiting the use of this data is incapable of harming members of the least advantaged group through premium rises across the board. The position on use of this specific data is irrelevant from the view of the principles of justice.

Much as with Alzheimer's, absent compelling reasons to allow use of data, I would suggest a precautionary approach. Given our compromised understanding of genetic data, the public perception of it as especially meaningful, personal and important, and the privacy concerns that this generates, I would suggest that use continue to be prohibited until compelling reason to allow use arises.

5.4 – Breast and Ovarian Cancer

Within the analysis on Haemochromatosis, we begin to see the possible effect of disincentivisation on the severity of the resulting condition. The creation of a disincentive to test through allowing insurers use of genetic data is particularly relevant when we come to consider BRCA mutations and their relation to breast and ovarian cancer. First, let us again establish the background of the conditions in question. The conditions in question are of variable severity, mid-level penetrance, and onset on the pre-retirement side of my binary.

Whether an individual receives an early diagnosis, and therefore, the severity of the cancer an individual suffers, is significantly linked to whether they obtain a

genetic test result indicating their BRCA status. If individuals are disincentivised from obtaining these tests through fear that they will be used prejudicially by insurers, it is significantly more likely that they are then diagnosed at a later stage. As I highlighted within *Chapter Four*, the severity of the condition to be studied therefore depends on whether this disincentive effect exists. This is what I will now explore.

5.4.1 – Is there likely to be a disincentive to obtain a test?

Taking into account the analysis earlier, I am now able to determine whether the use of this specific data by insurers is likely to have a disincentive effect. First, we know from *Chapter Four* that this test is diagnostically useful, even above and beyond family history data. For example, use of genetic tests meant that ovarian cancers were diagnosed at least half a decade earlier.[21] Genetic testing is therefore important, as earlier diagnosis of these cancers means that individuals can receive treatment at an earlier stage. Not only is treatment of early stage cancers usually less invasive, but as we already know, it has far higher five and ten year survival rates, and is far less severe in its effects on both fair equality of opportunity and capability.

Testing for BRCA1 and BRCA2 in the medical context therefore has high diagnostic value with regard to treating conditions which develop. If we look back at section *4.3.4* we can see that the likelihood that an individual will take a diagnostic test rises sharply with the diagnostic usefulness of the test, possibly because more useful tests are more likely to be suggested by medical professionals, and provided as a matter of medical course.[22] In addition to this, and because of the penetrance of the mutation, a significant number of these individuals will have previous family history of breast cancer, increasing their risk. Again, as we know from earlier, individuals who perceive themselves to be at higher risk of developing conditions are also more likely to obtain a genetic test. [23,24] As a starting point, these individuals

[21] Risch, H.A. et. al., *Prevalence and penetrance of germline BRCA1 and BRCA2 mutations in a population series of 649 women with ovarian cancer*, *op. cit.*

[22] Lerman C, et. al. *BRCA1 testing in families with hereditary breast-ovarian cancer. A prospective study of patient decision making and outcomes. op. cit.*

[23] Picone, G Sloan, F. and Taylor Jr., D. *Effects of Risk and Time Preference and Expected Longevity on Demand for Medical Tests*, *op. cit.*

could therefore be said to be highly likely to obtain genetic testing for BRCA mutations.

So how might the use of this test by insurers change the situation? Given individuals are both *risk* and *loss averse*, and disclosure of data which indicates possession of these mutations will raise premium prices, it is possible that some individuals will be disincentivised from taking these tests. However, I would suggest that it is unlikely. First, it is again important to mention family history. As many of these individuals will have previous family history of breast cancer, their premiums will already likely be loaded as they will be assessed as at risk of possessing BRCA mutations. Confirming this perception would lead to an increase in premium price, but it is likely to be small. On the other hand, a failure to obtain a test may lead to their cancer not being effectively diagnosed, and them therefore suffering from late-stage cancer rather than early stage cancer. This means that they are significantly more likely to die. Therefore, similar to the Haemochromatosis example, we have a comparison between a relatively small increase in premium, versus a large increased risk of serious illness or death. The *loss averse* individual would therefore likely choose to be tested to guard against the large risk, consistent with *Chapter Four*.

It would therefore seem unlikely that allowing insurers to use genetic test data relating to BRCA1 and BRCA2 mutations would significantly disincentivise individuals from taking tests within the medical, diagnostic context, as *loss averse* individuals would likely guard against the large increased risk of death or serious illness, and be willing to accept a small rise in premium. The appropriate severity to consider is therefore that of early-stage cancer, as early diagnosis is likely to occur with individuals obtaining tests. The conditions are therefore of mid to low severity.

5.4.2 – Is fair equality of opportunity compromised?

So, does the possession of BRCA mutations suggest that an individual will have their fair equality of opportunity compromised? From the analysis above, we know the disease is appropriately categorised as one of mid to low severity, as a lack

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[24] Witt, J. *The Effect of Information in the Utilization of Preventive Health Care Strategies: An Application to Breast Cancer, op. cit.*

of significant disincentive effect means that high-risk individuals are likely to be screened, receive earlier diagnostic intervention, and therefore receive treatment when their cancer is at an early stage. We know from *Chapter Four* that early stage cancer is treatable, and has very high survival statistics and low rates of recurrence. Therefore, it is likely that, even if the condition compromises fair equality of opportunity, this will only be short term.

In addition to the use of this genetic marker as an example of why the disincentive effect may matter (although, as outlined above, there is likely no disincentive here), these representative conditions are also useful as conditions of mid to low severity. Although unlike both Alzheimer's and Huntington's, the conditions are short-term, the individual is likely to suffer some compromise to their fair equality of opportunity. Patients are likely to receive surgical intervention (either mastectomy or oophorectomy) followed by adjuvant therapies such as chemotherapy or radiotherapy.[25] The surgery, radiotherapy and chemotherapy associated with the treatment of cancer may mean that, while they are receiving treatment, they are unable to compete equally with an identical individual who is not receiving this treatment. The individual with the condition will be required to attend hospital on a regular basis, at least in the early stages. This may infringe upon their ability to maintain employment, although the existence of both statutory and occupational sick pay schemes mean that this effect will be limited.[26] Indeed, the individual is likely to only be absent from work for a short period during and immediately after surgery, something which would be covered by these existing provisions. Secondly, the individual may suffer side effects including fatigue and nausea associated with their chemotherapy, and this may again mean that they require time away from work.[27] However, these side effects are again short term and likely to be covered by existing provision.

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[25] NICE Pathways, *Early and locally advanced breast cancer overview*, NICE (2011) Accessible at: [http://pathways.nice.org.uk/pathways/early-and-locally-advanced-breast-cancer] Accessed 24-02-2012.
[26] DirectGov, *Company sick pay rights*, Accessible at: [http://www.direct.gov.uk/en/Employment/Employees/Sicknessabsence/DG_10027238] Accessed 28-05-2012.
[27] Macmillan Cancer Support, *Possible side effects of some chemotherapy drugs*, Accessible at: [http://www.macmillan.org.uk/Cancerinformation/Cancertreatment/Treatmenttypes/Chemotherapy/Sideeffects/Possibleside-effects.aspx] Accessed 28-05-2012.

Whilst these are by no means small interventions, it is unlikely that they would significantly affect an individual's ability to obtain or maintain employment. An individual would be absent from work for a brief period during and immediately after surgery (and possibly for chemotherapy) something which could be covered by existing state and contractual provision for absence from work due to illness. Ongoing adjuvant therapies can then be arranged around an individual's working life. Unlike Alzheimer's and Huntington's, these conditions are not degenerative, and individuals do not lose permanent functionality which might prevent them from being able to perform duties they performed previously.

Again, recall that we are concerned with departures from normal functioning that shrink shares of primary goods below what is fair.[28] In this case, although the individual does undoubtedly depart from normal functioning here, they do not depart in a way which significantly affects their ability to obtain nor maintain employment long-term.[29] As such, their departure is unlikely to shrink their share of primary goods, and fair equality of opportunity is not meaningfully compromised, they still have roughly equal prospects of achievement as compared to an individual who has not suffered from the condition. Specifically, their expectation of wealth over the course of a whole life is similar to an identical individual who does not suffer from the condition.

5.4.3 – Is capability compromised?

Again, although an individual's fair equality of opportunity is not compromised by possession of a BRCA mutation and subsequent development of cancer, it may be that their capability to actualise their goods into ends is. Much like with Haemochromatosis, the individual can receive effective treatment in this case, meaning that their long term prognosis is good, and they receive no permanent compromise to their ability to convert primary goods. However, what differentiates this example, and one of the reasons for its consideration, is the fact that the individual does suffer significant short term symptoms. Not only must they attend hospital regularly, and often undergo surgery, but the side effects associated with

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[28] Daniels, Norman, *Equality of What: Welfare, Resources, or Capabilities?*, *op. cit.*, p. 283.
[29] With regard to species functioning. See Chapter Two, specifically *Daniels*.

adjuvant therapy such as chemotherapies mean that the individual may be somewhat unwell for a significant period of time. This will compromise their ability to fully utilise their primary goods, and to convert them into ends.

It would therefore appear that their capability is somewhat compromised here, albeit for a short period of time. Further, this disadvantage again occurs through no fault of their own, and therefore, we owe them assistance to rectify this disadvantage (which we provide through the NHS). It may also be illegitimate to further disadvantage them through allowing use of their data.

Let us now consider how far this individual would actually be disadvantaged through allowing insurers the use of their data. If an individual must disclose a positive BRCA test result to insurers, their premiums will rise, however this price rise is likely to be relatively small. Individuals who carry BRCA mutations often possess family history or other demographic data which increases their risk profile. Even in the absence of genetic test data, their insurer therefore already likely has a relatively accurate picture of the risk they present to the pool, and they are already charged a loaded premium. Confirming this bias will only make them a slightly more risky prospect rather than a radical one, and premium pricing will reflect that. Therefore, they only suffer a small financial disadvantage.

So far then, we have an individual whose fair equality of opportunity is not meaningfully compromised, but whose capability is, at least in the short term. This individual therefore has the same expectation of lifetime wealth as an identical individual without the mutation, but a reduced capacity to use it during the period they suffer from cancer. Further, we know any disadvantage this individual suffers from allowing the use of their test data by insurers is likely to be small. Whether the use of their data can be justified depends significantly on the effect of adverse selection here. If there is no significant adverse selection effect, it may be justifiable to prevent the use of their data, as their capability is somewhat compromised. Equally, a large adverse selection effect may mean that their data is justifiably used in order to protect members of the least advantaged group. It is this that I will now explore.

5.4.4 – What level of adverse selection is likely?

In this case, the prevalence of BRCA1 and BRCA2 mutations is surprisingly low. Although penetrant amongst those who possess the mutation, BRCA mutations are only responsible for approximately 6% of breast cancer within the UK (3.5% BRCA1, 2.4% BRCA2).[30] Indeed, BRCA mutations only account for a small number of hereditary cancers.[31] One might therefore expect the effect of adverse selection to be low.

However, unlike the other conditions I am considering, there is market simulation data available here. Hoy and Witt simulated a standard economic model of the life insurance market, assuming that insurance companies offer nonexclusive contracts and price linearly. The situations compared were between allowing the individual to hold this genetic information privately, and allowing insurers to use it to set premiums in a laissez-faire market. Within the study, they found that in situations where 5% or 20% of individuals possessed test data privately, adverse selection occurred to a level of approximately a 15% increase in price across the board. A situation where 100% of people possessed test data lead to substantial adverse selection, increasing prices by a factor of almost three (dependant on risk group). As access to data such as family history decreases, individuals are likely to cause more adverse selection.[32] This can however be somewhat mitigated by insurers employing strict underwriting rules, for example, being diligent in requesting cancer history and onset age for all first degree relatives.[33] Subramanian et. al. suggest that under these strict underwriting conditions, adverse selection may be able to be controllable, however, controllable in this context merely means that the insurance industry would survive. The study still suggests that under these strict underwriting terms, adverse selection could lead to an increase in portfolio cost, somewhere below

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[30] Peto, J. et. al., *Prevalence of BRCA1 and BRCA2 gene mutations in patients with early-onset Breast Cancer*, Journal of the National Cancer Institute (1999) 91 (11) 943-949.

[31] Frank, T.S. et. al., *Hereditary susceptibility to breast cancer. Significance of age of onset in family history and contribution of BRCA1 and BRCA2*, Disease Markers (1999) 15 89-92.

[32] Hoy, M. and Witt. J, *Welfare Effects of Banning Genetic Information in the Life Insurance Market: The Case of BRCA1/2 Genes*, The Journal of Risk and Insurance (2007) 74 (3) pp. 537.

[33] Subramanian, K., et. al. *'Estimating Adverse Selection Costs from Genetic Testing for Breast and Ovarian Cancer: The Case of Life Insurance'* op. cit.

10%.[34] This still likely means an increase in premiums across the board. Even if this increase is small, it may have a large effect upon those who are poorest, the members of the least advantaged group. There is also doubt as to how far strict underwriting terms can account for risk sufficiently to prevent adverse selection. If we look again at the evidence base within *Chapter Four*, we know that as many as 50% of BRCA mutations occur in individuals who do not have a first-degree relative affected with cancer.[35] Strict underwriting terms asking for cancer history and age of onset for first degree relatives would therefore seem an insufficient mechanism to account for adverse selection.

Even under the NHS where tests for the condition are offered free at the point of service, it would seem unlikely that we reach a level where 100% of individuals hold data about their BRCA status, as testing is expensive and only offered to those who already thought to be high risk. However, the fact that these individuals are aware of their genetic status will cause some adverse selection as they purchase more insurance due to being high risk. This is exacerbated by the fact that, as established earlier, individuals are likely to want to undergo testing in this case, and are unlikely to be disincentivised from doing so, which means more individuals will be aware of their genetic status. As such, information asymmetry is likely to be relatively high. Even 20% of insured individuals possessing information about their genetic status could cause premiums to increase by 15%, therefore harming those who are members of the least advantaged group. The effect of adverse selection is therefore strong.

5.4.5 – Conclusions

In conclusion, it is again in this case unlikely that an individual will be disincentivised from obtaining a genetic test result. If they obtain a test and insurers are allowed to use the resulting data, they will only receive a small increase in their life insurance premium, first because they likely possess family history or other demographic data allowing accurate risk assessment, and second, because possession

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[34] *Ibid.*

[35] Risch, H.A. et. al., *Prevalence and penetrance of germline BRCA1 and BRCA2 mutations in a population series of 649 women with ovarian cancer*, *op. cit.*

of a test allows them to be streamed into receiving care such as more frequent mammograms or preventative surgery. This means that their risk of mortality is lower than it would be had they not acquired the test, something relevant to life insurance risk assessment. If the individual does not choose to obtain a test, they will not receive this enhanced care, and as such, are more likely to suffer from a later stage cancer, for which mortality is significantly higher. A *risk* and *loss averse* individual would therefore likely choose to be tested. This means that the appropriate severity of condition to consider is that of early stage cancer.

The individual therefore does not have their fair equality of opportunity significantly compromised. Although they will likely need surgery, both statutory and contractual measures exist which mean that an individual is able to obtain paid leave from employment during this period. Ongoing adjuvant therapies (such as chemotherapy) can be arranged around an individuals working life. As such, the individual will not be absent from work for an extended period, and similarly able to obtain or maintain employment when compared to an identical individual who does not develop cancer. As such, they have the same expectation of wealth over the course of a whole life. However, unlike Haemochromatosis, it would appear that capability is compromised here, albeit temporarily. While receiving adjuvant therapies, the individual is likely to suffer side effects (such as fatigue and nausea) that mean they are unwell for a significant period of time. This will inhibit their ability to convert their primary goods into ends. When compared to the identical individual without the condition, their capability is therefore compromised, and they are disadvantaged in this way.

When we come to consider adverse selection, I find it plausible that significant adverse selection will occur here. First, the large incentive to be tested, when combined with the lack of disincentive effect means that, in a situation where use is prohibited, information asymmetry is high. These women are then likely to engage in purchasing behaviour which distorts the risk of the insured pool, and means that the insurer suffers a hit to profit. Market simulation data suggests that adverse selection will in this case lead to significant premium rises, even if the studies disagree as to the extent. Even the small premium rises projected by the Subramanian study would operate to significantly harm those members of the least

advantaged group who find themselves just barely able to afford their current insurance plans. It would therefore be justifiable to allow insurers use of this data in order to prevent adverse selection from operating to harm members of the least advantaged group contrary to the *difference principle*. However, in accordance with this principle, any use needs to also be limited so as to exclude using the data of members of the least advantaged group, as this would also harm them (through direct premium rises) contrary to the requirements of justice. If we are to allow use, we therefore need to establish what *value X* is in the case of individuals with BRCA. As I explained earlier, this is incredibly difficult to define specifically, but it should be possible to take a precautionary approach, and set the threshold for use of data significantly above where we think the value may be, and yet still at a level which allows the use of enough data to prevent adverse selection effects. Determining this value will require significant cooperation between government, policy institutes such as the Institute for Fiscal Studies and the insurance industry. This could be an interesting avenue for further work.

Were the market simulations not to bear out, and adverse selection turn out to be controllable, then the conclusion changes. Much as with the Alzheimer's case, we then have a situation where third party harms to the least advantaged group are prevented, and the appropriate concern is now for the individual whose capability is limited by the development of a condition for which they are blameless. Should insurers find strict underwriting terms which operate to prevent this adverse selection risk, then use should be prohibited in order to protect these individuals who possess mutations linked to the development of cancer.

5.5 – Hypertension

Finally, we must consider Hypertension, the general state of elevated blood pressure. We know from *Chapter Four* that, properly treated, the condition is one of relatively low severity, low penetrance, and one which onsets on the pre-retirement side of my binary. Further, the condition can, in this case, be diagnosed easily through the use of a syphgmomanometer, and blood pressure is frequently checked by doctors. A genetic test result is not required to diagnose this condition and

provide appropriate treatment. As such, disincentivisation does not matter in this case, as even if no individual ever undergoes genetic testing due to the disincentive effect, this will have no effect on severity of outcome, and therefore no effect on either fair equality of opportunity or capability. Indeed, the penetrance for this condition is so low that genetic testing is not offered in the medical context, and is only available through outside private providers. The number of individuals who undergo testing even in a situation where there is no disincentive is therefore incredibly small.

5.5.1 – Is fair equality of opportunity compromised?

Again, the relevant question is how far the individual's ability to access or maintain employment may be compromised by the possession of the genetic markers in question. We know from above that the severity of the condition is low, and that treatment is available which reduces both risk and severity of outcome. Whilst risk is not quite equivalent to the general population (unlike with Haemochromatosis), the various management options available mean that the treated condition is one of low severity.

It would seem unlikely that there is any meaningful compromise of fair equality of opportunity here, for several reasons. First, the fact that effective treatment options are available means that should hypertension develop, it is unlikely to be severe enough to inhibit an individual's ability to obtain or maintain employment. Mild symptoms such as brief periods of dizziness or shortness of breath will not compromise the ability to compete fairly. Indeed, it would seem an individual is able to obtain or maintain employment at a similar level to an individual who does not have hypertension with symptoms of such low severity. Secondly, penetrance becomes important for two reasons. First, due to the condition being of such low penetrance, there is only a 23% increased risk that the individual in question develops hypertension when an identical individual without the genetic markers would not. 75%+ of individuals will not develop hypertension due to their genetic characteristics. When considered alongside the low severity, it seems

unlikely that the possession of these genetic markers could be said to significantly increase the risk of fair equality of opportunity being compromised.

Also with regard to penetrance, it is important to note that lifestyle factors are by far the most significant contributors to whether an individual develops hypertension. If an individual eats unhealthily, has high levels of cholesterol and does not undertake exercise, they are likely to develop hypertension, whether or not they are genetically predisposed to it. In contrast, an individual who does eat healthily and exercises regularly is unlikely to have hypertension, again regardless of their genetic status. As such, the major contributor to the development of hypertension is one which individuals can be held responsible for. Remember that one of the purposes of the theory is to "attempt to mitigate the arbitrariness of natural contingency and superficial fortune."[36] Unlike the other conditions, the development of hypertension is primarily associated with individual behavior rather than bad luck in either the natural or social lottery.[37]

It is therefore first unlikely that an individual does in fact have their fair equality of opportunity compromised, as the severity of the condition is too low, and they are unlikely to develop the condition specifically due to their genetic makeup. Secondly, if the individual does develop the condition, it is likely due to actions they have taken, either through an unhealthy diet or avoiding exercise, which means that they can legitimately be held responsible for their condition, and therefore we owe them no assistance as a matter of justice.

5.5.2 – Is capability compromised?

Much like with the several of the other conditions studied, it may again be the case that although fair equality of opportunity is not meaningfully compromised

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[36] Rawls, John, *A Theory of Justice: Revised Edition, op. cit.*, p 82.
[37] It is possible to construct a claim that a bad diet is, in part, a result of bad luck in the social lottery. Specifically, the creation of what are termed '*food deserts*' sometimes means that poorer individuals are unable to access healthy foods easily. For an introduction see: Wrigley, Neil, *'Food Deserts' in British Cities: Policy Context and Research Priorities*, Urban Studies (2002) 39 (11) 2029-2040. Development of hypertension however requires a poor diet over the course of an entire life, not just in childhood, and assistance with both diet and exercise is available and offered as part of the treatment plan for hypertension. Further, complications do not develop until late middle age. It is therefore legitimate to hold these individuals responsible for the development of their condition.

here, an individual's capability to actualise their primary goods may be. We again know that we have a duty to provide healthcare services, and they are again provided adequately in this context.

With adequate treatment, the symptoms associated with the condition are not severe, and will not compromise an individual's capability to convert their primary goods. As compared to an identical individual without the condition, this individual will have the same capability. Further, it is questionable to what extent the individuals are meaningfully different, as the penetrance is also quite low. It is unlikely that an individual will develop hypertension purely due to their genetic characteristics. This means that their capability is even less likely to be compromised, as they may not actually suffer from the mild symptoms associated with the treated condition at all. It is in fact more likely that the individual who does develop hypertension does so due to lifestyle factors. As these are not a result of either the natural or social lottery, but instead choices that an individual has made, we can again hold them responsible for their disadvantage. We therefore owe them no duty under the principles of justice to rectify this lack of capability. As the individual is not meaningfully disadvantaged, much like Haemochromatosis, there is no *prima facie* reason why the use of their genetic data by insurers would be unjustifiable. In order to assess whether the principles of justice require us to allow use of this data, we must again examine the potential for adverse selection.

5.5.3 – What level of adverse selection is likely?

Taking into account the lack of incentive to test, and the fact that they would not usually be provided in the medical context, it is unlikely that a large percentage of individuals will know their genetic status in this case. The fact that few individuals know their genetic status limits the possibility of information asymmetry, and thus, the possibility of adverse selection.

Second, the fact that the development of the condition is so influenced by environmental and lifestyle factors also hugely limits adverse selection. Any increase in premium through the use of this test for insurance purposes is likely to be

extremely small, as the test is not hugely predictive as compared to lifestyle and environmental factors which the company already possesses. Further, genetic risk can be (and indeed, is) somewhat already accounted for by taking a family history of cardiac events. It is likely that the risk of these individuals developing hypertension is already accurately assessed by insurers, and their premiums are unlikely to change much. Indeed, there is question as to whether this is even information which insurers would use, given it is less predictive than lifestyle and environmental data. When discussing significantly multifactorial conditions, MacDonald suggests that these:

> "…probably will not provide clear and reliable estimates of lifetime risk, distinguishable from lifestyle and environmental factors; they might therefore not meet criteria of accuracy and reliability such as those that govern discriminatory pricing in respect of disability."[38]

The possibility of adverse selection leading to price increases across the board is therefore almost nil. It is incredibly unlikely that insurers would even use this data, and even if they did, the effect of the data on the risk assessment means that substantial adverse selection would not occur. As such, there is almost no risk of premium rises across the board as a result of denying insurers this information.

5.5.4 – Conclusions

Briefly, we can therefore conclude the following. First, due to the relatively small influence that these genetic markers would appear to exert, an individual who possesses them is not guaranteed to develop hypertension, absent other environmental or lifestyle factors. Whilst these factors do influence risk, they are by no means the most important contributor. Further, we know that disincentives are irrelevant in this case, as the condition is easily diagnosed in the absence of genetic data, and treatment can be provided. This means severity is low. Given the fact that tests are unlikely to be offered in the medical context (as they are not useful); the number of individuals who are aware of their genetic status is likely to be relatively small. Following from this, information asymmetry between individuals and insurers is therefore limited, and so is the potential for adverse selection. Adverse selection is

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[38] MacDonald, A, *Genetics and health costs: some actuarial models*, Law, Probability and Risk, (2002) 1(2) 97-118.

limited further both by the fact that premiums (if they rise) are likely to only rise by a small amount, and there exists question as to whether insurers would even use this data at all. There is therefore no harm to the least advantaged group through denying insurers the use of this data.

However, we also know that the individual who does possess these markers is not meaningfully disadvantaged compared to the individual who does not. First, as I outlined earlier, the mere possession of certain genetic markers does not guarantee one individual will develop the condition when the other will not, especially if they share similar lifestyle and environmental factors. Moving beyond this, even if this were true, the relatively mild symptoms present at onset, and the fact that effective treatment options are available means that the individual who does suffer from hypertension does not have their fair equality of opportunity in terms of access to employment compromised. They also do not have their capability compromised either, again due to the lack of severity of the condition. Insofar as they do, it is likely to be due to their own actions, and therefore we owe them no duty under the principles of justice.

Much as with Haemochromatosis, use of data relating to this condition is neither justifiable nor unjustifiable, but irrelevant. There exists no compelling reason why this data must be used, as adverse selection does not in this case appear to operate to the detriment of members of the least advantaged group. Equally, there exists no reason why this data could not be legitimately used, as the individuals are not relevantly disadvantaged. As with Haemochromatosis, I would suggest in this case a precautionary approach, and maintain the prohibition on use unless there is a compelling reason to remove it. This is not only due to the privacy and perception concerns outlined within *5.1.5*, but also the lack of understanding of genetic data explored within *Chapter One*. Given this data is of such low penetrance, the fact that this data is not fully understood, and the fact that genetic data is culturally endowed with almost prophetic potential, it is possible that this information could be misused and treated with more relevance than it truly deserves. This could operate to disadvantage individuals through unfair revisions of their risk status and therefore, premium price. In the absence of reason to use this data, I see no reason to take these risks.

Chapter Six

Final Conclusions

Within this final Chapter of the thesis, I will offer conclusions which follow from my analysis of representative conditions within *Chapter Five*. Specifically, I will explore and answer the final part of my thesis question, whether the position of the moratorium accords with the justifiable position outlined within the previous Chapter. I will first offer conclusions on conditions where the *status quo* is able to be justified, followed by those where it is irrelevant, and finally and most interestingly, those conditions where the current UK position is unjustifiable.

Before I proceed, below is a table summarising the conclusions reached within the previous Chapter, again re-ordered to reflect the order of consideration within this final section:

Condition	Fair Equality of Opportunity	Capability	Use Justifiable?
Alzheimer's Disease	Not Compromised	Compromised	No
Haemochromatosis	Not Compromised	Not Compromised	Irrelevant
Hypertension	Not Compromised	Not Compromised	Irrelevant
BRCA Mutations	Not Compromised	Compromised (short-term)	Yes
Huntington's Disease	Compromised	Compromised	No

6.1 – Conditions where the *status quo* is justifiable

Of the five conditions I have studied, there is only one where the current UK position is certainly correct from the perspective of the principles of justice. This is Alzheimer's disease, and I will present my conclusions on this first.

6.1.1 – Alzheimer's Disease

We know from the previous Chapter that there exists no great harm to the least advantaged through prohibiting the use of this data, as adverse selection is likely to be minimal. Given this, and give that the individual who develops the condition is at least somewhat less capable of converting their primary goods than an equivalent individual without the condition, it would seem justifiable to prohibit the use of genetic data in this case, out of moral concern for this reduced capability.

As a reminder, the current UK position prohibits the use of all genetic data for life insurance policies under £500,000 in value, and critical illness cover under £300,000 in value.[1] Over this amount, only one test was ever approved for use by the GAIC, that for Huntington's.[2] The current position within the moratorium therefore allows no use of genetic data relating to these individuals who possess genetic markers suggesting the development of Alzheimer's disease. As I explained earlier, allowing the use of this data by insurers would be unjustifiable under the principles of justice. Therefore, the *status quo* is, in this case, justifiable.

With regard to this specific condition, my conclusions do not differ radically from others in the area. Indeed, one of the applications received by the GAIC was for the use of a test relating to Alzheimer's disease, an application which was never granted.[3] During the *Whose Hands on Your Genes?* consultation, the GAIC received several pieces of feedback relating to the use of this data, from groups such as the Alzheimer's society and Alzheimer Scotland.[4] Whilst it is unsurprising that these pieces of feedback supported a prohibition on the use of the data, they share my view that adverse selection is likely to be small and controllable, citing evidence brought

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[1] Association of British Insurers, *Concordat and Moratorium on Genetics and Insurance*, March 2005, Accessible at: [http://www.abi.org.uk/Information/Consumers/Health_and_Protection/528.pdf], Accessed 10-02-2012.
[2] Genetics and Insurance Committee, *October 2000: Huntington's Disease*, Accessible at: [http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@ab/documents/digitalasset/dh_087703.pdf], Accessed 30-07-2010.
[3] Human Genetics Commission, *Whose Hands on Your Genes: A Discussion document on the Storage, Protection and Use of Personal Genetic Information*, (London:HGC 2000) para. 2.4.
[4] Human Genetics Commission, *Responses to the 'Whose Hands on Your Genes?' Consultation*, Accessible at: [http://www.hgc.gov.uk/Client/Content_wide.asp?ContentId=419], Accessed 13-10-2012.

before the Science and Technology Committee.[5] The responses also suggest that a disincentive effect may be relevant here with regard to participation in research, something which could harm efforts to understand the condition.[6] Looking specifically at Rawlsian approaches to the use of genetic information in the insurance industry, many of the concerns highlighted within my analysis are shared by Robert Card. Essentially; he concludes that genetic information should not be required to be disclosed to insurers, except where an individual is put in an unfair bargaining position through knowledge of the data. This is motivated by a concern for the effects of adverse selection.[7] As he notes in the article, at that time, it was difficult to assess whether significant adverse selection would occur. With regard to this condition, I feel I have made a substantial case as to why it would not, and as such, he would agree with me in prohibiting the use of this data.

6.2 – Conditions where the *status quo* is irrelevant

Within my analysis, there are two conditions where use and prohibition are both justifiable, meaning that whilst the position of the moratorium on these issues is justifiable, a removal of the prohibition on use would not necessarily be a problem either. It is these conditions I will consider next. First, Haemochromatosis.

6.2.1 – Haemochromatosis

Again, within *Chapter Five*, we learned that neither their fair equality of opportunity nor capability to convert their existing primary goods is significantly compromised by the condition. As such, it would not necessarily be illegitimate to allow the use of their data. However, the risk of adverse selection is, in this case, also very minimal, and likely to be controllable by insurers without necessitating premium price increases across the board. Therefore, there exists no disadvantage to the least advantaged group through prohibiting the use of this data by insurers. As far as the principles of justice are concerned, the question is therefore somewhat

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[5] Ibid. Specifically *Alzheimer's Society*, para 3.5.
[6] Ibid. Para 5.4
[7] Card. R, *Genetic Information, Health Insurance and Rawlsian Justice*, in Card, R. (ed) *Critically thinking about Medical Ethics*, (Pearson/Prentice Hall, 2004) 288-294.

irrelevant. The *status quo* currently prevents the use of this data and is justifiable in doing so; however equally, a change in position to allow the use of this data would also be justifiable. Whether this data should be disclosed to insurers might therefore be affected by other concerns, such as privacy concerns, and this may be an interesting avenue for further work.

There is little material from others specifically discussing Haemochromatosis. A consultation on rare genetic disorders performed by the HGC in 2001 included the condition, and found that there were two examples of unfair discrimination occurring, arbitrary and harsh treatment with regard to travel insurance, and a general failure to take account of developments in treatment.[8] However, this was carried out over a decade ago, an incredibly long time in the field of genetics. In the absence of a follow-up study, it is impossible to know if these problems persist. Further, unlike for Alzheimer's, the GAIC never received an application to use a diagnostic test for Haemochromatosis, indicating that insurers never considered its use essential. This would suggest that my conclusions re: the extremely low likelihood of adverse selection have some weight behind them. There is also no mention of the condition in the "Whose Hands on Your Genes?" consultation, nor any significant mention throughout the operation of neither the GAIC nor the HGC.[9] Whilst not necessarily supportive of the idea that people are ambivalent about the use of this data, this lack of concern does suggest that the current moratorium position on this condition (use prohibited) is not one which anyone is strongly advocating changing. As the use or prohibition on use of this data is irrelevant from a Rawlsian perspective, I would suggest the *status quo* simply remain, especially given the potential concerns about privacy and confidentiality mentioned earlier.

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[8] Human Genetics Commission, *Rare Genetic Disorders and Insurance: An Empirical Study*, 2001 Accessible at:
[http://www.hgc.gov.uk/UploadDocs/Contents/Documents/iievidence_raredisorders_gigreport.pdf] Accessed 13-10-2012.
[9] Human Genetics Commission, *Responses to the 'Whose Hands on Your Genes?' Consultation*, *op. cit.*

6.2.2 – *Hypertension*

Much as with Haemochromatosis, neither fair equality of opportunity nor ability to convert existing primary goods is significantly compromised here, and it is justifiable to use their data, should an insurer deem it relevant. However, again similar to Haemochromatosis, it is would also be legitimate to prohibit the use of this data, as adverse selection would be controllable, and as such, the least advantaged group would not be harmed by this prohibition. The *status quo* is justified in preventing use of this data, but whether this data is in fact used by insurers is again irrelevant from the perspective of the principles of justice.

Conditions such as this have also been treated as an irrelevance by the community at large. From the perspective of the insurers, data relating to these multifactorial conditions provides such low specificity and accuracy that:

> "…they might therefore not meet criteria of accuracy and reliability such as those that govern discriminatory pricing in respect of disability."[10]

There is therefore no great pressure from the industry to allow the use of this test, as it is one that they likely would not use anyway. They are already able to use lifestyle data, and it is this which is the predominant risk factor for Hypertension and associated complications. In the absence of demand from the industry, nor any evidence of adverse selection, I would recommend that the *status quo* again simply remain as a matter of precaution given our understanding of genetic data. However, without either significant adverse selection, or evidence that the condition compromises fair equality of opportunity or capability, the regulatory position is irrelevant from a Rawlsian perspective.

6.3 – Conditions where the *status quo* is unjustifiable

Finally, within the thesis there are two conditions for which the approach taken by the moratorium is unjustifiable under the principles of justice. The current UK position is unjustifiable for different reasons for each of these, in one case

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[10] MacDonald, A, *Genetics and health costs: some actuarial models*, *op. cit.*

allowing use of data which should justly be prohibited from use, and in another prohibiting the use of data which must justly be used. It is here that reform of the moratorium is required, and these conditions I will consider last.

6.3.1 – BRCA

When considering BRCA mutations, an individual's fair equality of opportunity is not meaningfully compromised, and although they suffer a loss of capability, this is likely transient and full capability will be restored. A prohibition on the use of this data may in fact operate to harm those who could legitimately be considered the least advantaged due to the operation of adverse selection. It is therefore justifiable to allow the use of this data by insurers. The *status quo* is therefore unjustifiable in this case, as it prohibits the use of this data. A prohibition on the use of this data means that significant adverse selection occurs, and this harms policy holders who are members of the least advantaged group through increasing their premiums. As a matter of justice, the moratorium should therefore allow the use of this data.

However, it is essential that, in allowing this use, we do not allow the use of data relating specifically to individuals who are members of the least advantaged group. Doing so would cause their premiums to rise in violation of the *difference principle*. If we are to allow use of this data, it is therefore essential that we discover where *value X* lies, and therefore, the policy value at which we are able to use data, secure in the knowledge that members of the least advantaged group do not purchase those policies. As we know from *Chapter Three*, finding this value is likely to be incredibly difficult, if not impossible. However, even a heavily precautionary approach where we only allow use for the top 10% of policies or so (similar to the current approach to Huntington's) could operate to significantly reduce adverse selection, and therefore harm to members of the least advantaged group. This is because as policy value increases, the financial consequences of mis-assessing risk for the insurer also increase, and therefore their response is larger.[11] Whilst the ideal

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[11] Subramanian, K., et. al. *'Estimating Adverse Selection Costs from Genetic Testing for Breast and Ovarian Cancer: The Case of Life Insurance' op. cit.*

position requires that we prevent any premium increases affecting the least advantaged, it will at the very least take significant time and work to discover what *value X* is in this case. A precautionary approach to using this data would, in the meantime, allow us to prevent significant harm at no risk to the individuals within the least advantaged group.

As we know from *Chapter Five*, this conclusion is heavily dependent on the existence or absence of a disincentive effect. If an individual is disincentivised from obtaining a genetic test, they are far less likely to be diagnosed and treated at an early stage, and therefore more likely to suffer significant effects on both their fair equality of opportunity and capability. We know from *Prospect Theory* (and the empirical studies cited within *Chapter Four*) that individuals are both risk and loss-averse. In this case, such an individual would prioritise obtaining a test result and avoiding severe cancer over a small increase in premium price. This also links back to the genetic exceptionalism discussion within *Chapter One*. As we know from there, individuals perceive genetic information to be uniquely powerful or important, and studies demonstrate that they are more likely to make health decisions with this information than with information such as family history.[12] When faced with a decision between obtaining information they consider incredibly important to their future health, and a loss of a relatively small amount of wealth, it is unlikely that a significant disincentive effect exists, and therefore, the above conclusion is the correct one.

An application for the use of genetic test data relating to BRCA status was made by the ABI, and subsequently sent for resubmission by the GAIC.[13] The application was never resubmitted, and as such, was never explicitly rejected. Several responses to the GAIC consultation echo concerns present within my analysis, such as the idea that allowing use of tests may disincentivise women from undergoing testing.[14] However, I believe that the analysis provided within *Chapters Four* and *Five* provides a convincing account of why a loss and risk averse

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[12] LaRusse, Susan et. al., *Genetic susceptibility testing versus family history-based risk assessment: Impact on perceived risk of Alzheimer disease*, Genetics in Medicine, (2005) 7 (1) 48-53
[13] Human Genetics Commission, *Responses to the 'Whose Hands on Your Genes?' Consultation*, *op. cit.* Specifically *Association of British Insurers* para. 39.
[14] Ibid. Specifically *Breakthrough Breast Cancer*, para. 9.5 (ii).

individual would still choose to undergo testing in a situation where use was allowed. It was also put forward to the GAIC that insurers are able to gather enough information from family history, and this made genetic testing essentially irrelevant.[15] There is however no empirical data provided to back up this claim. In light of this, I would again argue that studies such as those cited earlier by Hoy and Witt support my claim that significant adverse selection may occur should use continue to be prohibited.[16] A 2003 study by Armstrong et. al. also supports this position, demonstrating that women informed of increased breast cancer risk demonstrate behaviour consistent with adverse selection, namely, an increase in life insurance purchasing.[17]

Of course, should adverse selection prove to be controllable, then the moratorium position would be justifiable. In that case, the prohibition would no longer harm members of the least advantaged group, and the fact that individuals who develop cancer suffer from reduced capability would become the issue of primary concern. Much as with Alzheimer's, it would then be justifiable to prevent the use of this data out of moral concern for this reduction in capability. More empirical studies on the effect of prohibition on adverse selection is also needed.

6.3.2 – Huntington's disease

With regard to justifiability, the *status quo* would appear somewhat justifiable as it prevents the use of genetic test data relating to Huntington's in most cases, consistent with the principles of justice. However, as we know from *Chapter One*, for policies over a certain value, genetic data relating to this condition is approved for use.[18] So, is this use justifiable? First, it would seem that the individuals whose data can be used under the *status quo* are certainly not members of the least advantaged group, as they are looking to purchase large life insurance

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[15] Ibid. Specifically *Breast Cancer Care*.

[16] Hoy, M. and Witt. J, *Welfare Effects of Banning Genetic Information in the Life Insurance Market: The Case of BRCA1/2 Genes*, *op. cit.*

[17] Armstrong et. al. *Life insurance and breast cancer risk assessment: adverse selection, genetic testing decisions, and discrimination*. American Journal of Medical Genetics (2003) 120A (3) 359-364.

[18] Genetics and Insurance Committee, *October 2000: Huntington's Disease*, Accessible at: [http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@ab/documents/digitalasset/dh_087703.pdf], Accessed 30-07-2010.

policies, a policy value that only 3% of the population hold.[19] Given the link between policy value and individual wealth established in *Chapter Three*, it is reasonable to suggest that these are relatively wealthy individuals, and not likely to be found within the bottom wealth quartile. It is certainly not unjustifiable to disadvantage them for this reason.

However, we also discover within *Chapter Five* that it is unlikely that prohibiting the use of this type of genetic data would lead to adverse selection, and as such, premiums are unlikely to rise across the board. Therefore, preventing the use of this data does not act to harm members of the least advantaged group. Additionally, even the wealthiest individuals still have their capability severely limited by the development of the condition, and therefore are less able to use their primary goods through no fault of their own. Given that members of the least advantaged group will not be harmed by preventing the use of this data as adverse selection is controllable, the interests of justice require us to prevent the use of this data even for these wealthy individuals. As such, the *status quo* is in fact unjustifiable, as it allows the use of this data.

Should adverse selection prove to be uncontrollable, and premium rises across the board become necessary, the position changes. At that point, it would still be unjustifiable to use the data of those individuals with Huntington's who could legitimately be considered members of the least advantaged group. It would however become justifiable to use the information relating to those few wealthy individuals, as it would be legitimate to disadvantage them in order to protect the least advantaged group. In a situation where adverse selection does occur, and lead to premium increases, the *status quo* is the justifiable position. Indeed, under these assumptions, use would likely need to be expanded, as within the 97% of policy-holders currently protected there will be many individuals who cannot legitimately be considered members of the least advantaged group, and who it would therefore be legitimate to disadvantage in order to protect the least advantaged group.

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[19] Association of British Insurers, *Concordat and Moratorium on Genetics and Insurance, op. cit.*

We therefore have broadly two justifiable positions. Under the most likely scenario, where adverse selection proves to be controllable without premium prices rising across the board, the *status quo* is unjustifiable, as it allows the use of some data relating to this condition, when the justifiable position is to prevent all use. However, should adverse selection prove to be uncontrollable, the *status quo* is more justifiable, preventing the use of most data, whilst allowing the use of data relating to wealthy individuals in order to protect the least advantaged group. This is subject to the difficulties identified within *Chapter Three*, specifically, the difficulty of identifying where the boundary between the least advantaged group and everyone else lies. Of these positions, it is overwhelmingly likely that the first is the true position, as unless there is a huge uptake of genetic testing for this condition, information asymmetry is very low, and adverse selection therefore unlikely.

It is this which I consider my most interesting conclusion, explicitly contradicting as it does the decision of the GAIC to allow the use of a test relating to this condition. This is however unsurprising, as the criteria considered by the GAIC when deciding whether to approve a test were merely the following:

> "• **Technical Relevance:** Does the test accurately measure the genetic information?
> • **Clinical Relevance:** Does a positive result in the test have likely future adverse implications for the health of the individual?
> • **Actuarial Relevance:** Does a positive result justify increased premiums?"[20]

Of these, it is difficult to argue that the test does not fulfil the first two. The condition is highly penetrant, and therefore the test can accurately determine whether an individual will suffer from the condition. Further, a positive test result does not adversely affect the health of the individual, as they already carry a degenerative and terminal disease. I do however have significant difficulty with the third criteria, specifically, the understanding of 'justify' that the GAIC chose to adopt. If we examine the announcement approving the use of the test, the concern appears to be primarily for those whose premiums are adversely loaded due to a family history of

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[20] Genetics and Insurance Committee, *Fifth Report from January 2006 to December 2006*, Annex C, Accessible at:
[http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_074086.pdf] Accessed 14-10-2012 p.13.

Huntington's, even though they do not suffer from the condition.[21] There appears to be little concern within the announcement for those individuals who will suffer from the condition, and who may receive increased premiums through the approval of this test.

According to the GAIC consultation those individuals whose premiums are loaded inaccurately, "…find it very difficult, or in some cases impossible, to obtain insurance at affordable rates, based on their family history…"[22] Whilst this is certainly a matter of concern, I would suggest that we should concern ourselves more with those individuals who have the condition. It is fair to assume that if these individuals who do not suffer from the condition and yet have family history find it difficult to obtain premiums, then those who actually carry the condition also find this difficult. However, unlike those whose premiums are loaded inaccurately, these individuals also have a much higher need for life insurance, suffering as they do from a terminal, degenerative illness which will substantially limit their earning potential, and condemn them to an early death. Further, the current position acts to further disadvantage these individuals who (although wealthy) have significantly reduced capability to use their primary goods from middle-age onwards. Given the absence of harms associated with prohibiting the use of this data wholesale, it is correct to fully prohibit use out of moral concern for this reduced capability.

6.4 – Summary of Conclusions

Mindful of the difficulties identified with identifying the least advantaged group, and the assumptions outlined within both *Chapter Five* and *Six*, my conclusions are the following. Under the most likely scenarios, the moratorium is justifiable when examining Alzheimer's disease. Through the use of the typology, I am also able to suggest that the moratorium is justifiable when considering

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[21] Genetics and Insurance Committee, *October 2000, Huntington's Disease (GAIC/0.1 1)*, Accessible at:
[http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@ab/documents/digitalasset/dh_087703.pdf] Accessed 14-10-2012.
[22] Human Genetics Commission, *Responses to the 'Whose Hands on Your Genes?' Consultation*, *op. cit.* Specifically *Genetics and Insurance Committee* para 9.4

conditions which share similar characteristics to Alzheimer's, those conditions which are severe, of mid penetrance, and onset post-retirement.

For two of my conditions, Haemochromatosis and Hypertension, the current UK position is justifiable, but also somewhat irrelevant. Under the principles of justice, it would also be justifiable to allow use of this data. This is useful again because the claim can be expanded to conditions of a similar type. Therefore, should there exist a compelling reason to, it would be justifiable to allow insurers the use of data relating to conditions which are of low severity, and which onset pre-retirement, even if their penetrance is high. However, in the absence of reason to reverse the current position, I suggest a precautionary approach should be adopted, out of concern for both privacy and the general lack of understanding associated with genetic data.

Finally, we find that the current position is unjustifiable in two key ways. First, when considering BRCA mutations, the moratorium acts to prohibit the use of data relating to conditions of mid to low severity, and causes large adverse selection. For cases such as BRCA, where a large number of individuals are likely to know their genetic status, it may be in the interests of justice to allow insurers the use of this data in order to protect members of the least advantaged group. The *status quo* is unjustifiable in this way. However, this use would have to ensure that those individuals who possess data relating to BRCA and who may also be considered members of the least advantaged group were adequately protected. This may be done through a mechanism similar to that already applied to Huntington's, where information is allowed to be used for policies over a certain value, where that value is inaccessible by anyone who might be within the least advantaged group. This is a position suggested by Subramanian as one which may act to significantly limit the harm caused by adverse selection.[23]

Second, the status quo is also unjustifiable when we consider the one condition for which the use of data is actually authorised. Although the policy limit is set at a high level, and therefore only data relating to the wealthiest 3% of

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[23] Subramanian, K., et. al. *'Estimating Adverse Selection Costs from Genetic Testing for Breast and Ovarian Cancer: The Case of Life Insurance' op. cit.*

individuals is used, the lack of adverse selection leading to price increases means that there is no harm to the least advantaged group from prohibiting the use of this data. Therefore, the justifiable position is to prohibit all use out of concern for the significantly reduced capability that even these wealthy individuals suffer from.

Importantly, it is unjustifiable to use data relating to the wealthiest individuals only because doing so does not cause harm to members of the least advantaged group through the mechanism of adverse selection. The conclusions therefore act within a kind of hierarchy, where preventing harm to members of the least advantaged group is of absolute moral concern. As we know from the principles of justice, structuring inequality so that members of this group would be harmed runs explicitly contrary to our obligations. Therefore, if prohibition on use would harm members of the least advantaged group, the justifiable position is to allow use of this data. For example, I argue that data relating to BRCA status should be allowed to be used due to the potential for adverse selection. Whilst I accept that individuals who suffer from cancer will have somewhat compromised capability, and that some of them may then be denied insurance and subsequently harmed, the principles of justice do not allow their interests to be prioritised over the interests of members of the least advantaged group. Where it is possible to protect both groups (Huntington's), it is both justifiable and correct to do so. However, where circumstances force one group to be sacrificed in order to protect the other, it is always the least advantaged that must be protected, as they are by definition the worst off.

6.5 – Relation to the wider field

Finally, it is important to again pull back from the specific examples I have studied and examine developments within the wider field. Only a month ago, the Presidential Commission for the study of Bioethical Issues published a report on whole genome sequencing. Although not explicitly concerned with the use of specific genetic test data, many of their concerns echo those within my analysis above.

When discussing the justifiable use of genetic data, the Commission express

concerns about genetic privacy and what whole genome sequencing could mean for knowledge of future disease. Amongst their suggested responses, the Commission consider it important that explicit commitments are made to justice and fairness. Specifically:

> "A commitment to justice and fairness is a commitment to ensuring that the
>
> unavoidable burdens of technological advances do not fall disproportionately on any particular individual or group, and that the benefits are widely and equitably distributed."[24]

The work I have undertaken above begins to flesh out what form this commitment to justice and fairness should take when we consider the use of genetic test data within the insurance context. By structuring the system in such a way as to protect the disadvantaged from adverse selection, we ensure that they are not disproportionately burdened by advances in genetic technology.

The idea that we should protect the vulnerable from disadvantage caused by technological advancement is present throughout the recommendations; as they consider it important to "ensure that the risks are not disproportionately borne by any particularly vulnerable or marginalized group."[25] Ensuring that the system is both just and fair is important when it comes to encouraging participation, another key feature of the report. Indeed, they suggest that in order to realise the potential that whole genome sequencing holds, we need large scale public participation and a public willingness to share genetic information (for example, through biobanks.) Such sharing is far less likely to occur if the public distrust the system, or feel that they would not be treated justly or fairly within it.[26] My work is therefore important in ensuring that the system regulating the use of this information is one which is just and fair, and which is perceived as such.[27]

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[24] Presidential Commission for the Study of Bioethical Issues, *Privacy in Progress in Whole Genome Sequencing*, Oct 2012, Accessible at: [http://bioethics.gov/cms/sites/default/files/PrivacyProgress508.pdf] Accessed 31-10-2012. p.42.

[25] *Ibid.* p.62.

[26] *Ibid.* p.39.

[27] Adequate privacy protection also forms a large part of obtaining and fostering public trust, and is one of the reasons I suggest a precautionary approach for those conditions where use is irrelevant from the perspective of the principles of justice. For more, specifically in relation to biobanks, see: Taylor, M.J, and Townend, D, *Issues in Protecting Privacy in Medical Research Using Genetic information and Biobanking: The PRIVILEGED Project,* Medical Law International, (2010), 10 (4) pp. 253-268.

This is especially important given the growth of genetic testing within the NHS. The UK Genetic Testing Network are assisting with the development and use of this technology within the service, and working closely with NICE to approve tests for use.[28] The UKGTN work programme, the UKGTN commissioning working group and specialised commissioning groups have been (and are) engaged in developing national targets for test use, promoting equity of access, and dealing with test costs.[29] Indeed, recently the UKGTN Steering Group have endorsed recommendations to allow tests for foetal sex determination for certain conditions, and have developed best practice guidelines and care pathways.[30] It is clear therefore that, as we saw at the beginning of *Chapter One*, the use of genetic testing within the NHS continues to grow and develop. The report which opens this thesis, *Building on our Inheritance*, not only highlights the importance of genetic data, but provides a comprehensive future plan for the NHS, and recommendations on how to integrate genetic testing fully into existing clinical plans and programmes. This includes the development of a network of Genomic Technology Centres, Biomedical Diagnostic Hubs and Regional Genetics Centres to cover research, deliver diagnostic testing at an affordable scale, and engage with patients suffering genetic disease.[31] Indeed, the Emerging Science and Bioethics Advisory Committee (ESBAC) recently met to discuss how to carry the recommendations within this report forward into the wider NHS.

In this situation, the relevance and use of genetic information continues to grow, and an ever greater number of individuals possess information about their genetic status. Given this, the potential for adverse selection within the insurance

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[28] UK Genetic Testing Network, *Supporting Genetic Testing in the NHS: Second Report of the UKGTN*, Accessible at: [http://www.ukgtn.nhs.uk/gtn/digitalAssets/0/929_SECONDREPORT.pdf] Accessed 31-10-2012.
[29] UK Genetic Testing Network, *Review of Commissioning Arrangements for Genetic Services and Strategic Recommendations*, March 2011, Accessible at: [http://www.ukgtn.nhs.uk/gtn/digitalAssets/1/1034_UKGTNReviewofCommissioningArrangements. pdf] Accessed 31-10-2012.
[30] UK Genetic Testing Network, *Non-Invasive Prenatal Diagnosis (NIPD): UKGTN Approval of Gene Dossiers for fetal sex determination for Congenital Adrenal Hyperplasia (CAH) and X-linked Conditions (excluding haemophilia)*, Accessible at: [http://www.ukgtn.nhs.uk/gtn/What_s_New?contentId=443], Accessed 31-10-2012.
[31] Human Genomics Strategy Group, *Building on our Inheritance: Genomic Technology in Healthcare*, January 2012, Accessible at: [http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/d h_132382.pdf], Accessed 14-08-2012 pp. 49.

context grows, and the 'wait and see' approach taken by the moratorium begins to look increasingly outdated. Given the importance of genetic data, it is time we move to a fully justifiable and comprehensive approach to the use of genetic data in the insurance context. It is here where I hope to have made a small contribution.

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