

ASMR, from Theory to Therapeutics:

Exploring the neural mechanism and clinical potential of
Autonomous Sensory Meridian Response.

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Abstract

Autonomous Sensory Meridian Response (ASMR) is a sensory-affective phenomenon in which specific auditory cues, such as whispering, brushing, or tapping, elicit pleasant tingling sensations, felt across the scalp and neck in listeners, along with parasympathetic calm. Despite its widespread popularity and self-reported benefits to anxiety, sleep, and pain, its underlying neural mechanisms and therapeutic potential remain relatively underexplored. This thesis develops and empirically tests the Proximity Prediction Hypothesis (PPH), a novel predictive coding model proposing that near-ear auditory cues engage the brain's Peripersonal Space network, generating top-down expectations of affective touch in ASMR. When these predictions are instantiated, the model predicts beta-band desynchronisation (reflecting sensorimotor disinhibition), gamma-band enhancement (reflecting precision-weighted sensory integration), and cascade towards parasympathetic calm. Study 1 formulated the PPH theory, integrating evidence from affective touch physiology, Peripersonal Space activation, and interoceptive predictive coding. Illustrative behavioural data (N = 64) confirmed that pleasantness, rather than arousal, drives the likelihood and intensity of ASMR tingling, supporting the model's valence-based mechanism. Study 2 used cross-modal EEG (N = 64) and MEG (N = 30) experiments to test these predictions empirically; here, ASMR sounds evoked robust beta-band desynchronisation and gamma-band enhancement relative to controls, providing the first cross-modal neurophysiological evidence for ASMR's oscillatory signature and support for its proposed hierarchical predictive coding cascade. Study 3, a preregistered, two week longitudinal trial in a chronic pain sample (N = 64), demonstrated that daily ASMR exposure significantly reduced fatigue and produced near-significant improvements in pain interference, consistent with affective-autonomic recalibration hypotheses. Together, these studies establish ASMR as a reproducible oscillatory and physiological state arising from predictive simulation of affiliative touch. The findings advance a unified account linking perception, emotion, and bodily regulation, and position ASMR as a scalable, non-pharmacological means of modulating vagal tone, as well as highlighting its potential to improve affective-autonomic balance in chronic pain.

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For my dad.

Author's Declaration

I, Josephine Flockton, declare that this thesis is a presentation of original work, and I am the sole author, under the supervision of Dr Catherine Preston, Professor Cade McCall, and Prof Daniel Baker. This work has not previously been presented for a degree or other qualification at this University (University of York, UK) or elsewhere. All sources are acknowledged as references. All the studies contained within this thesis were conducted in accordance with the ethical standards of the Department of Psychology at the University of York and, for the study in Chapter 3, with York Neuroimaging Centre's Ethics Committee, too. The work presented in Chapter Two of this thesis has been published in a peer-review journal and was co-authored by my primary and secondary supervisors, Dr Catherine Preston, and Prof Cade McCall:

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Chapter 1: Introduction

1.1 Overview

Autonomous Sensory Meridian Response (ASMR) refers to a specific sensory-affective experience marked by a pleasant tingling sensation that is typically felt on the scalp and neck and may spread down the spine. These tingles are usually elicited by quiet, repetitive auditory or visual cues, such as whispering, tapping, or brushing, and are often accompanied by deep relaxation, reduced physiological arousal, and a sense of calm (Barratt & Davis, 2015; Poerio et al., 2018). Unlike frisson, which is characterised by brief, arousing sympathetic activation (Goldstein, 1980; Salimpoor et al., 2011), ASMR is reliably associated with parasympathetic engagement and subsequent positive affect that lasts some time after the experience.

Although ASMR has become a widespread digital phenomenon with millions of users consuming ASMR-inducing content online for relaxation and sleep, among other reported benefits, its underlying neural mechanisms and clinical applications remain underexplored. Initial psychophysiological and neuroimaging evidence suggests that ASMR engages brain systems implicated in affective touch, interoception, and autonomic regulation, including the posterior insula, secondary somatosensory cortex, and medial prefrontal cortex (Lochte et al., 2018; Poerio et al., 2018). These same regions are central to parasympathetic control and emotion regulation (Craig, 2002; Bushnell, Čeko, & Low, 2013), making ASMR a

valuable naturalistic model for studying how sensory signals can modulate internal bodily states and affective homeostasis.

The present thesis develops and tests a unified theoretical account of ASMR, the Proximity Prediction Hypothesis (PPH), which proposes that near-ear auditory cues trigger expectations of affiliative touch, leading to a biphasic neural cascade involving simulated tactile processing and a shift toward vagal calm (Flockton et al., 2025). Chapter 2 introduces this framework in detail, grounding it in predictive coding and affective neuroscience. Chapter 3 tests its neurophysiological predictions using a novel combination of EEG and MEG sensor-space analysis to characterise the time-frequency dynamics of ASMR relative to matched control sounds. Chapter 4 extends these insights into an applied setting: a preregistered, longitudinal intervention study examining whether daily ASMR exposure can improve symptoms of chronic pain and its comorbidities. Finally, Chapter 5 synthesises the findings across theoretical, neural, and clinical levels, identifying broader implications for the study of sensory-affective phenomena and affective-autonomic regulation.

Together, these chapters aim to advance ASMR research from descriptive accounts to an integrated, mechanistically grounded framework, one that links the subjective experience of pleasant paresthesia (a tingling sensation felt on the skin but not elicited by direct physical contact), to a predictive coding process with corresponding neural correlates, and explores its translational potential for mental and physical health.

1.2 What ASMR is and is not: Comparisons with Parallel Phenomena

1.2.1 Phenomenology and Triggers

ASMR (autonomous sensory meridian response) manifests as a pleasant tingling sensation typically localised to the scalp, which can flow down the neck and back. Listeners most often describe the state as calming, sleep-promoting, and stress relieving rather than exciting or energising (Barratt & Davis, 2015; Poerio et al., 2018). Across surveys and laboratory studies, the most reliable elicitors are near-field, slow, intimate cues, whispered or soft-spoken speech, gentle brushing or tapping, paper handling, and close personal attention roleplays such as spa or hairdresser scenarios (Barratt & Davis, 2015; Fredborg, Clark & Smith, 2017).

Although diverse in content, ASMR triggers share key acoustic and spatial properties that categorise them as proximal, near-ear stimuli to the brain. These sounds typically contain large interaural level differences (ILDs) and sub-millisecond interaural time differences

(ITDs), which are core binaural cues used by the auditory system to localise sound sources in three-dimensional space. Specifically, neurons in the medial and lateral superior olives detect disparities in arrival time and intensity between the two ears; when these disparities are maximal, they are interpreted as indicating a sound source extremely close to the head (Grothe, Pecka, & McAlpine, 2010; Middlebrooks & Green, 1991; Blauert, 1997). The combination of exaggerated ILDs, minimal reverberation, and slow amplitude modulation in ASMR recordings thus conveys the percept of an intimate, nearby presence, effectively activating the brain's near-field, or peripersonal, auditory processing systems. ASMR recordings typically use binaural or dummy-head microphones with artificial pinnae and ear canals that preserve these cues; playback through headphones, used by about 90% of ASMR consumers (Barratt & Davis, 2015), creates the illusion that a hand, brush, or whisper is positioned directly at the listener's ear. Second, the spectral profile is colour-shifted by head shadowing, with frequencies above roughly 8 kHz rolling off steeply in the contralateral ear, another potent cue to spatial closeness (Begault, 1994). Third, ASMR content is characterised by slow amplitude envelopes and low overall sound pressure levels: intensity rises and falls gradually over hundreds of milliseconds rather than in abrupt, percussive bursts. Whispered phrases or soft taps thus form smooth, rounded waveforms that maintain a sense of gentle motion. These acoustic choices sustain a relaxed, parasympathetic state; louder or sharper transients would recruit middle-ear reflexes and sympathetic alerting responses, preventing the calm associated with ASMR (Borg, 1968; Graham, 1975; Bradley & Lang, 2000; Møller, 2006).

Physiologically, moments when listeners report tingles have been shown to be accompanied by heart rate deceleration and increased high-frequency heart-rate variability (HF-HRV), a profile consistent with vagal-parasympathetic engagement (Poerio et al., 2018). This aligns with subjective reports of feeling comfort and sleepiness, suggesting that ASMR involves coordinated sensory, affective, and autonomic processes rather than a purely perceptual anomaly.

A consistent feature across ASMR research is the striking individual variability in both susceptibility and trigger preference. Not everyone experiences tingles, and those who do often respond idiosyncratically. Using an ASMR Trigger Checklist, Poerio et al. (2022) found that trigger responses are stable within individuals but highly diverse across the population; whispering and soft tapping are reliably effective triggers, whereas chewing or eating sounds can be pleasant for some and aversive for others. This heterogeneity parallels findings in the phenomenon of misophonia, where similar sound cues that elicit ASMR in some listeners, often related to human mouth sounds, evoke defensive sympathetic arousal in others

(Edelstein et al., 2013; McGeoch & Rouw, 2020). Within a predictive coding framework, such variability likely reflects differences in affective priors. In cognitive and affective neuroscience, 'priors' refer to the brain's predictive models of the world. Predictive coding theories propose that perception is an inferential process in which sensory input is continuously compared against these top-down expectations, with any mismatch generating a prediction error that the system seeks to minimise (Friston, 2010; Clark, 2013). Emotional and bodily feelings likewise arise from predictions about internal states; interoceptive priors that shape how sensory cues are interpreted (Barrett & Simmons, 2015). Within this framework, ASMR can be understood as a case where near-ear auditory cues engage predictions of affective touch and safety, producing pleasant tingling when those expectations are maintained rather than violated. An exploration of this proposed predictive coding perspective on the mechanism behind ASMR will be discussed in depth, in Chapter 2, when the Proximity Prediction Hypothesis will be presented. For some listeners, near-ear intimacy is encoded as affiliative and soothing; for others, it carries aversive or intrusive associations. Accordingly, ASMR is best conceptualised as a polymorphic trait (Lochte et al., 2018), shaped by individual histories, expectations, and interoceptive-affective processing differences.

1.2.2 ASMR versus Misophonia

Misophonia is a condition in which certain human-generated sounds, typically those related to oral or nasal functions such as chewing, breathing, sniffing, or lip-smacking, elicit intense negative emotional reactions, including anger, disgust, or anxiety. These responses are often accompanied by pronounced autonomic arousal and avoidance behaviours, which can lead to significant distress and functional impairment (Schröder, Vulink, & Denys, 2013; Rouw & Erfanian, 2018). Physiological studies have shown that misophonic triggers induce elevated skin conductance and increased heart rate, consistent with defensive sympathetic activation (Edelstein et al., 2013). Neuroimaging research further implicates heightened activation in regions associated with salience detection and affective reactivity, such as the anterior insula, amygdala, and hubs of the default mode network, during exposure to misophonic sounds (Kumar et al., 2017; Schröder et al., 2019).

Importantly, some auditory cues overlap across ASMR and misophonia - most notably mouth and eating sounds. For some listeners these same cues are experienced as pleasant and tingle-inducing, while for others they provoke aversive tension or anger. This polarity of valence and physiology, affiliative calm versus defensive arousal, illustrates that identical acoustic inputs can map to opposite autonomic outcomes depending on individual priors and contextual interpretation (McGeoch & Rouw, 2020).

Recent comparative accounts propose that ASMR and misophonia may occupy opposing ends of an auditory-affective spectrum; both involve heightened sensitivity to social or interpersonal sound cues, but differ in how those cues are evaluated and integrated into interoceptive and emotional systems (Rouw & Erfanian, 2018; Valtakari et al., 2019; Janik McErlean & Banissy, 2018). Misophonia appears to engage the anterior insula's salience and interoceptive network in a hyper-reactive, threat-based mode (Kumar et al., 2017; Schröder et al., 2019), whereas ASMR recruits overlapping insular and somatosensory regions within a hypo-aroused, affiliative configuration linked to vagal engagement and safety prediction (Lochte et al., 2018; Smith et al., 2019; Poerio et al., 2018). Explaining why this divergence arises requires a mechanistic framework linking auditory context, affective expectation, and autonomic regulation, developed in the following chapter through the Proximity Prediction Hypothesis (PPH).

1.2.3 ASMR versus Frisson

ASMR is often compared to musical frisson (also known as 'aesthetic chills'), a sensory response involving piloerection (goosebumps) in listeners, triggered by emotionally powerful passages in music (Zentner, Grandjean, & Scherer, 2008). However, research suggests they are distinct phenomena with different triggers and physiological profiles. For instance, frisson is typically elicited by emotionally charged moments in music, such as unexpected harmonic shifts, crescendos, or the entrance of a solo voice (Grewe et al., 2007; Salimpoor et al., 2011). It can also be triggered by awe-inspiring or suspenseful scenes, but in all cases, the stimulus is abrupt and/or emotionally salient. In contrast, ASMR is evoked by subtle, near-field acoustic cues such as whispering, soft tapping, brushing, and personal attention roleplay (Barratt & Davis, 2015; Fredborg et al., 2017). These triggers are intentionally soft, slow, and intimate, often recorded with binaural microphones to simulate close spatial proximity (Begault, 1994). Rather than eliciting shock or awe, they aim to cultivate calm.

Phenomenologically, listeners usually describe frisson as constituting a pleasant surprise (Goldstein, 1980; Panksepp, 1995; Guhn et al., 2007; Grewe et al., 2007; Meyer, 1956), lasting around ten seconds on average and reportedly felt as brief 'waves of pleasure' (Blood & Zatorre, 2001) rather than an intentionally sought after state like ASMR. The latter has been reported to build gradually into a wave-like tingling sensation, most often described as pleasant and soothing rather than exhilarating, consistent with its association with parasympathetic activation and the fact that listeners seek it out for stress reduction and sleep facilitation (Barratt & Davis, 2015; Poerio et al., 2018).

Aside from the subjective experience reports, frisson is characterised by sympathetic nervous system activation, with physiological studies showing increased skin conductance, elevated heart rate, respiration changes, and piloerection during episodes, consistent with peak arousal (Benedek & Kaernbach, 2011; Laeng et al., 2016). Conversely, ASMR is associated with opposing parasympathetic responses; Poerio et al. (2018) found that participants exhibited heart rate deceleration and increased high frequency heart rate variability (HF-HRV) while watching ASMR videos, i.e, markers of vagal activation and physiological calm rather than arousal.

Interestingly, ASMR may involve a biphasic autonomic pattern; an initial orienting response to a near-ear auditory cue, followed by a sustained shift toward parasympathetic dominance (Flockton et al., 2025). This brief sympathetic activation may reflect attentional engagement or salience detection, but it is rapidly overridden by cues of safety and incoming affective touch, culminating in a calming, vagal state. In contrast, frisson appears to involve a different trajectory, beginning with an affectively charged aesthetic chill and progressing into piloerection (hair standing on end), and heightened arousal, without transitioning into calm. This divergence may partly reflect the interpretive context and content of the triggering stimuli. Maruskin et al. (2016) distinguish between chills, goosebumps, and tingles as distinct affective somatosensory phenomena. While ASMR may also begin with a perceptual chill-like initial arousal during the alerting phase, the expectation of affiliative, non-threatening human-evoked interaction (e.g., whispering or soft brushing) leads the system to downregulate arousal and engage calming touch networks. Frisson, by contrast, arises from emotionally intense or awe-inducing stimuli that amplify sympathetic arousal without such social predictions, hence never leading to vagal upshift, but either stopping with the chill or ending with further arousal, that manifests as actual piloerection, ‘feeling goosebumps’.

This distinction, its mechanistic implications, and exactly how it results in the tingling sensation specific to ASMR, is clarified in depth in Chapter 2 through the formalisation of the Proximity Prediction Hypothesis.

1.2.4 The mechanistic explanatory gap

The current literature establishes a coherent profile of what ASMR feels like and how it differs from related phenomena. Unlike frisson, which culminates in a brief sympathetic rush and often goosebumps, or misophonia, which triggers defensive arousal and aversion, ASMR is reliably described as a calming, parasympathetic experience, often elicited by near-field, intimate auditory cues. However, a key explanatory gap remains: there is no comprehensive mechanistic account of how such auditory cues, specifically those rich in

spatial proximity information, are transformed into a somatosensory experience (i.e., tingling), nor why this cascade reliably terminates in vagal calm rather than sympathetic arousal.

Chapter 2 addresses this gap by proposing the Proximity Prediction Hypothesis (PPH), a predictive coding model in which near-ear auditory signals activate peripersonal space and affective touch representations, generating tingles as a somatosensory echo and shifting the autonomic state toward parasympathetic regulation. This theoretical framework is then empirically explored in the remainder of the thesis, as Chapter 3 tests its neural plausibility using EEG and MEG sensor-space data, while Chapter 4 examines its translational potential in a preregistered intervention study with chronic pain sufferers.

1.3 Sociocognitive and affective touch perspectives

1.3.1 The social nature of triggers

A consistent finding in ASMR research is that the most potent triggers have a distinctly social and affiliative quality. Surveys reveal that ‘close personal attention’ scenarios, i.e. whispers or soft-spoken voices directed at the listener, gentle hand movements simulated to occur near the head, and one-on-one social interaction roleplays like haircuts or medical exams, are among the highest-rated elicitors among ASMR experiencers (Barratt & Davis, 2015; Fredborg et al., 2017). In one of the earliest large-scale studies, Barratt and Davis (2015) found that around 75% of ASMR respondents listed whispering as an effective trigger, followed closely by personal attention (around 69%), crisp sounds (e.g. tapping or crinkling, around 64%), and slow movements (e.g. watching someone methodically perform a task, around 53%). These interpersonal or carefully attentive triggers are perceived as calming and intimate, often simulating the feeling of being groomed or cared for. Indeed, a more recent tool for classifying ASMR stimuli, the ASMR Trigger Checklist, showed that physical touch (e.g. light stroking or face touching in videos) was the single most universally endorsed ASMR trigger, reported by 98% of responders (Poerio et al., 2023). Soft speaking and gentle personal interactions form the core of most ASMR content, aligning with the idea that simulated social interaction is key to the ASMR experience (Poerio et al., 2022). ASMR video creators commonly use techniques like binaural recording (two-ear microphones) to preserve subtle spatial cues in sound, which enhances the illusion of the speaker’s proximity. This technical trickery, a whisper panned from ear to ear, or the sound of hair brushing right next to the microphone, reinforces a sense of someone else being right there

with the listener, thereby heightening feelings of safety and interpersonal closeness. Large-scale content analyses confirm that roleplay scenarios (e.g. spa treatments, salon visits, doctor check-ups) and other intimate soundscapes consistently rank among the most popular ASMR themes, alongside repetitive quiet sounds like brushing, tapping, or scratching (Barratt & Davis, 2015; Poerio et al., 2022). Again, such triggers all serve to create an atmosphere of personalized care and gentle attention, which appears integral to eliciting the tingling relaxation of ASMR.

Experimental studies further underscore the importance of this affiliative, social context for ASMR. Experimental studies further highlight the potential role of social and tactile context in ASMR. For example, Helge Gillmeister et al. (2022) examined whether individuals who experience ASMR differ in their sensitivity to vicarious and interpersonal touch. They found that ASMR responders reported more frequent and intense vicarious tactile experiences when observing touch, as well as a higher incidence of mirror-touch synaesthesia (MTS), the trait where seeing someone else being touched evokes a sensation of touch on one's own body, compared to non-responders. Within the ASMR group, there was some evidence that stronger ASMR experiences were associated with more pronounced vicarious touch, although this relationship was not consistently supported across statistical approaches. ASMR responders also reported greater reactivity to positive, but not negative, interpersonal touch, suggesting a potential link between ASMR and sensitivity to socially relevant tactile experiences. These findings indicate that tactile and socially relevant cues may play a role in shaping the ASMR experience. However, the study does not directly address underlying mechanisms, and the authors emphasise that interpretations regarding shared processes with phenomena such as MTS remain speculative. Overall, the results are consistent with the idea that ASMR is associated with heightened sensitivity to observed and anticipated touch, particularly in socially meaningful contexts.

The most effective triggers seem to function as social grooming at a distance, engaging the listener in a soothing interpersonal exchange (albeit a virtual one). Consistent with this, ASMR responders also tend to have more positive attitudes toward social touch in everyday life; they report enjoying hugs, gentle touches, and even exhibit a higher prevalence of MTS. All of these observations point to ASMR's social essence - it is evoked most strongly when the stimuli simulate safe, caring interactions rather than just arbitrary auditory or visual stimuli in isolation.

1.3.2 Links to affective touch and grooming

Given this social emphasis, several theorists have proposed that ASMR taps into evolutionary pathways for affiliative touch and grooming. From an ethological perspective, ASMR might be understood as a kind of vestigial grooming response; an echo of the calming, trust-building mechanism that occurs during social grooming in primates (Lochte et al., 2018). In primates, being gently groomed by a companion has well-documented soothing effects: it lowers heart rate (Boccia et al., 1989; Aureli et al., 1999), reduces cortisol levels (Shutt et al., 2007), and increases beta-endorphin release (Keverne et al., 1989). Grooming also functions as social currency; monkeys trade grooming to reinforce alliances or receive tolerance from other members of their social group (Seyfarth, 1977), and the recipients of said grooming often appear relaxed and more prosocial afterward (Schino et al., 1988). ASMR's characteristic feelings of comfort and contentment parallel these grooming effects as many people describe ASMR tingles as feeling like someone is gently caressing their head or playing with their hair, or being elicited by viewing other people touch or play with their own or another's hair (Fredborg et al., 2017). The C-tactile (henceforth known as CT) afferent system is thought to underlie this pleasure of social touch in humans. CT nerve fibers (most commonly localised to hairy skin regions) respond optimally to slow, gentle stroking at velocities around 3-10 cm/s - essentially the speed of a tender caress. Activating CT fibers produces a uniquely pleasant, calming sensation rather than a tickle or itch, and researchers believe this system evolved to mediate affectionate touch between close companions (McGlone et al., 2014). Intriguingly, although ASMR is usually triggered through audiovisual cues without any physical contact, it seems to engage the brain's affective touch circuitry by implication. Many ASMR triggers (a whispered voice, the sound of brushing) can be thought of as auditory proxies for a gentle touch, carrying the prediction that a soothing tactile interaction is about to happen, or is already happening just out of sight. This might explain why ASMR often centers on scenarios of care (haircuts, spa treatments, personal attention) and why it induces relaxation; the brain is, in effect, conjuring the positive touch that normally accompanies those situations.

Empirical evidence supports this connection between ASMR and the affective touch system. A recent study by Liu and Kondo (2025) demonstrated that individuals who are more sensitive to pleasant touch (as measured by how much they enjoy a gentle stroking on the skin) also experience stronger ASMR tingles when watching trigger videos. In their experiment, participants first received soft brush strokes on the arm at CT-optimal speeds (around 3-5 cm/s) and rated how enjoyable those touches felt. Later, they watched ASMR videos while continuously reporting their tingle intensity. The results showed a clear positive

correlation, where those who found slow caresses especially pleasant tended to get more intense tingles from ASMR, and vice versa. In fact, affective touch sensitivity was a significant predictor of ASMR intensity, particularly for triggers that imply touch or social interaction (for example, videos of someone eating or whispering sounds, which can invoke an intimate, embodied context). This finding strongly suggests that ASMR is engaging the same neurophysiological pathways that respond to friendly physical touch. It echoes earlier work proposing that ASMR's tingles may arise from a kind of cross-activation or synesthesia between auditory and tactile brain regions (McGeoch & Rouw, 2020) meaning, certain sounds might be interpreted by the brain as gentle touches, activating CT-afferent pathways and the associated pleasant feelings.

Neuroimaging data lend further weight to the grooming hypothesis. Lochte et al. (2018) conducted one of the first ASMR fMRI studies and observed that during ASMR episodes, there was heightened activation in regions like the medial prefrontal cortex, anterior cingulate cortex, and insula, areas deeply involved in emotional regulation, social attachment, and interoception (monitoring the body's internal state). Notably, parts of the insular cortex (especially the posterior insula) are known to process affective touch signals from CT fibers, and activation there aligns with the idea that ASMR involves a bodily, touch-like sensation (Björnsdotter et al., 2009; McGlone et al., 2014). More recent neuroimaging work has examined how brain connectivity changes during ASMR. Lee et al. (2020) found that watching ASMR videos increased connectivity between the posterior cingulate cortex (PCC) and the superior temporal sulcus (STS), both key regions within the brain's social cognition network involved in mentalising and interpreting the intentions of others. Increased coupling was also observed between the anterior cingulate cortex and medial prefrontal cortex, areas implicated in empathy, social bonding, and self-referential thought. Crucially, the authors also highlighted activation of the posterior insula, a region that processes affective touch input from the aforementioned unmyelinated CT afferents, fibres known to signal slow, pleasant, socially relevant tactile stimulation. Lee et al. interpreted this constellation of changes as evidence that ASMR recruits a broad affiliative network encompassing affective touch, social cognition, and introspective processes. This suggests that ASMR does not merely elicit passive sensory pleasure, but may simulate comforting interpersonal scenarios that engage the same neural systems used during gentle touch, caregiving, and safe social connection. Future work should also consider the performer's voice, familiarity, and perceived warmth, as these may modulate listeners' affiliative priors and thereby influence neural and subjective responses. This view aligns with evidence that ASMR experiencers score higher on empathic and social-affective traits (Janik McErlean & Banissy, 2017), suggesting that sensitivity to interpersonal cues contributes to ASMR

proneness. It is telling that the physiological profile of ASMR aligns with relaxation: during ASMR, people show parasympathetic signs of calm (e.g. heart rate deceleration, increased vagal tone), rather than a fight-or-flight sympathetic surge (Poerio et al., 2018). This is exactly what one would expect from a grooming context, where being groomed or comforted by a trusted individual would trigger a contented, low arousal state.

While sociocognitive and affective touch accounts of ASMR are compelling, they highlight a need for more detailed mechanistic models. These accounts propose that a whisper in the ear works because it implies a physical caress or caring intent, but how does the brain perform this sensory translation? Why do some people's auditory pathways integrate with tactile/emotional circuits in this way, whereas others feel nothing special from the same stimuli? And why do ASMR tingles lead reliably to a relaxed, blissful feeling instead of stimulating excitement or alarm? These questions remain only partially answered. For instance, researchers have speculated that early developmental or attachment factors might modulate one's receptivity to ASMR; individuals who had affiliative, responsive caregiving in childhood may have stronger priors that associate soft sounds and close whispers with positive touch and safety, making them more likely to experience ASMR. Conversely, those with insecure or adverse attachment experiences might find the same triggers less effective or even unsettling. Preliminary support for this idea comes from the observation that ASMR-responders tend to score higher on traits like emotional empathy (McErlean & Banissy, 2017) and react more positively to social touch (Gillmeister et al., 2022), hinting that they may be predisposed to interpret ambiguous stimuli in a warm, affiliative light. However, a full process level explanation is still emerging. In summary, the literature increasingly links ASMR to the domain of affective social touch and proposes that it may originate in an evolutionary grooming mechanism, experienced through uniquely modern means (digital media triggers), bringing about a state of calm connection in those able to experience it.

1.3.3 Touching the Explanatory Gap

The literature convincingly places ASMR within a social-affiliative framework, linking its most effective triggers to simulated caregiving and touch. However, this body of work does not yet explain how social and tactile meaning is inferred from sound alone, or why such inference translates into a somatosensory experience (i.e., tingling), nor why this cascade terminates in vagal calm rather than sympathetic arousal. Specifically, current accounts do not spell out the neurocomputational sequence by which auditory proximity is interpreted as affiliative intent, transformed into a prediction of gentle touch, and ultimately resolved as a calming bodily state.

Chapter 2 responds to this need by proposing the Proximity Prediction Hypothesis (PPH) model, which bridges the gap between social context and bodily response. PPH explains how auditory signals rich in spatial intimacy activate the Peripersonal Space Network, along with affective touch priors, producing tingles as a somatosensory echo and shifting autonomic tone toward parasympathetic calm. In doing so, it formalises the social-affiliative interpretation of ASMR and connects it to testable neural and physiological mechanisms.

1.4 Neural Correlates and the Impact of Measurement Technique

Although neuroimaging studies have begun to reveal where ASMR related processes occur, the how and when of the experience remain underspecified. The present thesis addresses this issue by using EEG and MEG to examine ASMR's temporal dynamics and oscillatory profile, with particular attention to the biphasic cascade proposed in the Proximity Prediction Hypothesis. This section reviews existing spatial and temporal evidence and explains how Chapter 3 builds upon these foundations to test key predictions.

1.4.1 fMRI Findings: Strengths and Limitations

Functional magnetic resonance imaging (fMRI) has identified several candidate brain regions implicated in ASMR, especially those related to social-affiliative processing and reward. Lee et al. (2020), for instance, found increased functional connectivity during ASMR videos between regions such as the posterior cingulate cortex and superior temporal sulcus, areas involved in social cognition and self-referential processing. While this represents a valuable effort to investigate ASMR in real-time within the scanner, the study still relied on passive viewing and remains constrained by methodological limitations inherent to fMRI. The blood oxygen level dependent (BOLD) signal has relatively poor temporal resolution, on the order of several seconds, due to the sluggish haemodynamic response (Logothetis et al., 2001), making it difficult to resolve the rapid oscillatory and cross-modal dynamics characteristic of ASMR. Moreover, the high acoustic noise generated by gradient switching from the scanner itself (often exceeding 90 dB; Peelle et al., 2010) poses a major challenge for presenting the quiet, near-ear auditory cues that typically elicit ASMR, potentially masking or diminishing the effect.

More broadly, most fMRI research in this domain relies either on trait-level comparisons or passive exposure designs. Fredborg et al. (2021) conducted resting-state connectivity analyses comparing experiencers to non-experiencers, while Smith et al. (2017) examined differences in default mode and attentional networks. Such designs provide insight into baseline differences but may overestimate group-level distinctions, particularly without

concurrent in-scanner ASMR tracking. Moreover, they often assume fixed ASMR categories, without accounting for graded trigger sensitivity or the effects of unfamiliar stimulus delivery (e.g., via MRI-compatible audio systems).

Indeed, eliciting ASMR in the scanner poses logistical barriers. Acoustic interference from gradient switching, restricted supine posture, the requirement to stay still during the scan, and compromised sound immersion all reduce the likelihood of ecologically valid responses. The binaural, proximity-rich audio that typifies ASMR content is difficult to replicate using scanner-compatible earphones. Thus, while BOLD-based imaging has highlighted plausible affective and social brain networks, it remains ill-suited to capturing the fine grained temporal unfolding and sensory nuances of ASMR, underscoring the value of complementary time resolved methods.

1.4.2 EEG findings: Promise and Heterogeneity

Electroencephalography (EEG) studies complement fMRI by offering higher temporal resolution, allowing finer analysis of ASMR's unfolding sequence. Although the EEG literature is still heterogeneous in analytic methods and experimental paradigms, several findings broadly support the neural plausibility of the PPH model.

Fredborg et al. (2021) reported increases in the frequency bands alpha and gamma, and in sensorimotor rhythm (SMR) among ASMR experiencers during listening to auditory triggers. Lee et al. (2022) found similar spectral increases when comparing ASMR content to binaural beats, while Inagaki et al. (2022) demonstrated that ASMR videos helped restore alpha and high beta and low gamma activity following cognitive load, suggesting a rebalancing consistent with vagal engagement. Other studies show early reductions in alpha and increases in beta (Engelbregt et al., 2022), with spectral and topographical distinctions between ASMR and control stimuli over central, occipital, and temporal-parietal sites (Koo et al., 2021; Seifzadeh et al., 2021; Pedrini et al., 2021).

Despite this promise, current EEG work has limitations; few studies time-lock neural data to tingle onsets, most omit autonomic measures (e.g., high-frequency HRV or pupil diameter), and source modelling is rare, to localise the topographic regions involved in the brain. Consequently, existing EEG data hint at biphasic dynamics (e.g., early beta decreases, gamma increases, later alpha/SMR rises) but cannot always confirm their temporal profile or regional specificity.

1.4.3 Why Use EEG with MEG when measuring ASMR?

To address these gaps, Chapter 3 of this thesis applies both EEG and magnetoencephalography (MEG) in a cross-modal mirrored analysis paradigm, to test theoretically grounded predictions derived from the PPH. While source localisation in MEG will be undertaken in future work, frequency-specific sensor-space analysis in the present thesis offers high temporal resolution and internal validation through using the two different techniques in the same way, with the same stimuli. Unlike fMRI, MEG is silent and better suited to preserving the acoustic fidelity of near-ear stimuli, a critical requirement for ecologically valid ASMR elicitation. Participants can comfortably wear earphones or near-field transducers while seated upright or reclined, improving the likelihood of a genuine ASMR response.

This multimodal approach allows us to ask whether ASMR follows a biphasic sequence consistent with the PPH; early beta band (15-30 Hz) power decreases over posterior scalp sites, consistent with engagement of tactile simulation mechanisms, followed by gamma band (>30 Hz) increases potentially reflecting prediction updating. While precise cortical sources cannot be inferred from scalp data alone in EEG, the observed topographic patterns and timing dynamics provide a test of the sequence-level predictions made by the PPH model's parasympathetic cascade.

By integrating EEG time-frequency decomposition with MEG frequency-domain analyses, Chapter 3 of this thesis moves beyond the limitations of earlier fMRI and resting state work (e.g., Fredborg et al., 2021; Lee et al., 2020; Lochte et al., 2018) to investigate the neural cascade underlying ASMR in a theory-driven, temporally precise manner.

1.5 The Clinical Potential of ASMR

ASMR is no longer just an internet curiosity. Many listeners report using it in daily life to relax, sleep, and manage stress, and a small but growing empirical evidence base in the scientific literature points to measurable autonomic changes. At the same time, clinically oriented research remains sparse, especially for chronic pain, a condition with clear mechanistic links to arousal, interoception, sleep, and mood (Craig, 2002; Bushnell, Čeko, & Low, 2013). This subsection outlines what is known, what is missing, and why chronic pain provides a logical test-bed for translational approaches in Chapter 4.

1.5.1 What Has Been Shown Already

Large-scale surveys consistently show that people seek out ASMR primarily for relaxation, stress reduction, and sleep. For example, Barratt and Davis (2015) found that 98% of respondents use ASMR to relax, and 82% use it as a sleep aid; many viewers describe a

calm, soporific state accompanying the tingle experience. Laboratory studies support this parasympathetic profile. During self-reported ASMR moments, heart rate decreases and high-frequency heart rate variability (HF-HRV) increases, a pattern consistent with vagal engagement (Poerio et al., 2018). Interestingly, while heart rate slows, there may be a slight increase in skin conductance associated with the tingling effect, suggesting a complex interplay between relaxation and arousal (Poerio et al., 2018).

Beyond momentary effects, emerging evidence suggests short-term mood and sleep benefits. For example, Smejka and Wiggs (2022) report perceived improvements in relaxation and sleep among both good sleepers and individuals with insomnia. In a controlled experiment, Eid et al. (2022) found that ASMR viewing reduced state anxiety, but only in self-identified ASMR responders. Collectively, these findings suggest that ASMR can promote a vagally mediated, relaxed state and yield short-term affective benefits (e.g., calm, sleepiness, anxiety reduction) that align well with listeners' real-world therapeutic requirements.

1.5.2 What Is Needed

Despite these promising findings, structured and preregistered ASMR interventions are scarce. Most studies to date rely on cross-sectional surveys, single-session laboratory exposures, or retrospective self-reports. Few have applied longitudinal designs with standardised 'doses' of ASMR administered as an intervention, or incorporated a wider area of validated clinical outcome measures. In particular, no targeted, preregistered trial in chronic pain has yet been published, despite three converging justifications: (1) self-reports consistently indicate ASMR helps with anxiety, low mood, and sleep (Barratt & Davis, 2015); (2) chronic pain is highly comorbid with insomnia, anxiety, depression, and fatigue (Bair et al., 2003; Finan et al., 2013; Eccles & Davies, 2021); and (3) plausible neurophysiological mechanisms exist through which ASMR might influence these domains (see 1.5.3). This translational gap is notable given the progress of adjacent interventions, such as transcutaneous auricular vagus nerve stimulation (taVNS), which also modulates the LC-vagal axis in a mechanism that the PPH model would suggest is analogous to ASMR's underlying process, and taVNS has also shown improvements in sleep and anxiety across multi-week protocols (Wu et al., 2022; Zhang et al., 2024). To move beyond anecdotal and short-term lab findings, the field needs dose-controlled, clinically anchored, longitudinal intervention studies.

1.5.3 Why Chronic Pain?

Chronic pain presents a principled test case for ASMR's clinical potential because its symptom burden is intimately tied to arousal dysregulation, interoceptive sensitivity, and negative affect, all domains ASMR may influence. According to the PPH model, ASMR triggers initiate a biphasic sequence involving an initial orienting and alerting phase when attending to the stimuli, followed by vagal settling and parasympathetic calm due to the interplay between priors and predicted CT-touch cues when that specific stimulus is categorised as human-evoked, affiliative, and proximal. This mechanism parallels evidence that actual affective (CT-optimal) touch, slow, gentle stroking that activates unmyelinated CT afferents, can reduce both acute and chronic pain by down-regulating nociceptive processing within the insula and anterior cingulate cortex (Meijer et al., 2022). If ASMR mimics such CT-mediated soothing through auditory simulation, its predicted impact on vagal tone and affective regulation may have particular relevance for chronic pain, where physiological hypervigilance and affective load amplify discomfort (Eccleston & Crombez, 1999; Van Damme, Crombez, & Eccleston, 2004).

Chronic pain is linked to increased sympathetic tone, disrupted sleep, and reduced vagal flexibility, factors that exacerbate pain symptoms and worsen the overall experience of sufferers (Finan et al., 2013). If ASMR increases HF-HRV and lowers heart rate during tingles (Poerio et al., 2018), and reliably induces subjective calm (Barratt & Davis, 2015; Smejka & Wiggs, 2022), it may serve as a low-cost means to bias the autonomic state toward parasympathetic recovery and recalibrate interoceptive awareness. Even modest improvements in arousal balance could support better coping and pain interference outcomes.

Research on CT-optimal touch stimulation suggests it may influence pain via modulatory pathways that reduce affective pain signalling (Pawling et al., 2017). By acoustically simulating cues of proximity and care, ASMR may therefore partially engage similar regulatory circuits, promoting vagal activation and affective calm. This interpretation parallels non-invasive neuromodulation of the vagus nerve such as transcutaneous auricular vagus nerve stimulation (taVNS), which electrically stimulates the auricular branch of the vagus, enhancing parasympathetic output and influencing insula-limbic networks implicated in pain, mood and autonomic regulation (Badran et al., 2018; Farmer et al., 2021) and has yielded benefits in sleep and anxiety (Wu et al., 2022; Zhang et al., 2024). Although the modalities differ, both approaches converge on vagal, interoceptive regulation as a pathway toward reducing arousal and affective distress.

1.5.4 What does this thesis do to address the clinical gap?

In summary, existing research documents subjective and physiological indicators of ASMR's calming potential, but no structured or preregistered interventions have targeted chronic pain, a condition where mechanistic overlap is compelling. Chapter 4 addresses this translational gap via a preregistered, two-week longitudinal study with a chronic pain sample. Using consistent auditory ASMR triggers created by an ASMRtist and used in all of the thesis' studies, and validated outcome measures assessing sleep disturbance, fatigue, anxiety, depression, and pain, the study tests whether regular ASMR exposure can yield clinically meaningful change. It also explores whether trait ASMR susceptibility moderates outcomes, i.e., whether those who experience tingles benefit more from the intervention in any of those domains.

This empirical test is scaffolded by the mechanistic rationale developed in Chapters 2 and 3. If ASMR meaningfully shifts LC-vagal tone and modulates affective and interoceptive states as predicted, we expect improvements in mood, sleep, and perhaps pain interference in the ASMR group. Even in the potential absence of pain intensity change, gains in energy, restfulness, or emotional wellbeing would be meaningful for future translational approaches. More broadly, the chapter aims to bridge fundamental neuroscience with real-world application, using chronic pain as a means for evaluating ASMR's clinical potential.

1.6 Shared Methods and Design Rationale

The three empirical chapters in this thesis are intentionally connected through shared stimuli, coordinated measurement strategies, and a common theoretical framework. This coherence supports the accumulation of evidence across analytic levels: from the underlying mechanism (Chapter 2), to time-resolved neural dynamics (Chapter 3), to preliminary clinical translation (Chapter 4). Full methodological detail is provided in each chapter; here, the unifying logic across studies is summarised.

1.6.1 Common Stimuli and Design Logic

Across Chapters 2 to 4, the same curated set of experimental sound stimuli, slow, near-field cues such as soft tapping, paper handling, and gentle brushing sounds, were used in all three studies. These stimuli were produced by a professional ASMR content creator from a different project (Nader, 2023) using binaural dummy-head microphones, preserving realistic interaural time and level differences. It should be noted that delivery varied slightly across contexts, closed-back headphones in the EEG study, MEG-compatible earphones in the MEG study, and home listening (with headphones recommended) in the clinical protocol.

Standardising stimuli served two purposes. First, it enhanced construct validity by anchoring it to proximity-laden features that typify ASMR, such as high-frequency head-shadowing and lateralised auditory perception (Begault, 1994). Second, it enabled coherent cross-study inference: the same stimulus set underpins behavioural tests of mechanistic predictions from the PPH model in Chapter 2, the time-frequency analyses of EEG and MEG sensor-space data in Chapter 3, and the daily exposure in the longitudinal intervention study in Chapter 4.

1.6.2 Measurement Strategy

At some point in all experimental studies, participants indicated whether ASMR tingling sensations were felt in response to any of the stimuli, control or experimental (yes/no). Furthermore, there was survey data collected that related to the EEG study, which was used as illustrative data for the PPH model, in Chapter 2 rather than included in the larger EEG-MEG chapter, which focused on the time-frequency analyses from the EEG and MEG data itself rather than the accompanying survey that had been administered after the EEG session. In the illustrative survey data of Chapter 2, participants gave responses regarding the pleasantness of the stimuli - responses that were also given in the intervention study of Chapter 4 from the chronic pain sample. These trial-level data on pleasantness enabled mixed-effects models examining whether hedonic value, rather than mere sensory activation, predicts ASMR classification and intensity (Barratt & Davis, 2015; Poerio et al., 2018) and also provided another datapoint that could be assessed across the experimental and intervention studies, irrespective of differences in their paradigms.

Chapter 3 employed both EEG and MEG to track the temporal evolution of ASMR-related brain dynamics. EEG provided millisecond precision in tracking oscillatory changes; MEG offered complementary spatial sensitivity, though both analyses were restricted to sensor space for the purposes of the current project. The analysis focused on frequency-specific predictions from the PPH, namely, early beta-band (15-30 Hz) reductions as a marker of tactile simulation, followed by gamma-band (>30 Hz) increases reflecting prediction updating or salience. All neural analyses followed best-practice standards for EEG/MEG methodology (Gross et al., 2013; Keil et al., 2014) and adopted BIDS-compliant data organisation where applicable (Gorgolewski et al., 2016; Niso et al., 2018; Pernet et al., 2019).

The longitudinal intervention study of Chapter 4 utilised validated clinically meaningful measures: the Pain, Enjoyment, and General Activity (PEG) scale, Fatigue Severity Scale (FSS), Insomnia Severity Index (ISI), and Hospital Anxiety and Depression Scale (HADS), administered alongside standardised daily ASMR exposure. Preregistration was used to define primary outcomes and hypotheses in advance (Nosek et al., 2018).

1.6.3 Ethical and Data Practices

All studies received institutional ethical approval from the University of York Ethics Committee and York Neuroimaging Centre. Participants provided written informed consent, passed basic screening (e.g., hearing ability, neuroimaging safety guidelines and eligibility requirements for the MEG scanner sessions), and were free to withdraw at any time without penalty. Data handling complied with GDPR requirements, including anonymising data at source where possible, secure storage, and restricted access to identifiable information from any neuroimaging datasets.

Analyses were conducted using fully scripted, reproducible workflows implemented in Python and R. EEG and MEG data for each participant were organised in a BIDS-inspired directory structure, with separate folders for raw, preprocessed, and derived data, and accompanying JSON metadata files describing event codes, channel information, and acquisition parameters. This structure ensured that preprocessing, epoching, and FOOOF analyses could be applied systematically across participants using standardised scripts.

Version controlled code for all preprocessing, spectral analysis, statistical testing, and visualisation is hosted on GitHub (repository link provided in Appendix A). The repository includes a README file documenting software versions, dependencies, and execution order. Although raw EEG and MEG recordings cannot be shared publicly due to consent restrictions, derived summary data and all analysis scripts are openly available to support transparency and reproducibility. Where applicable, inferential statistics are reported with effect sizes and 95% confidence intervals, and preregistration records are cited within the relevant chapters.

1.7 Thesis structure

This introduction has situated ASMR in relation to neighbouring phenomena, distinguishing it from frisson (a brief, sympathetic rush culminating in piloerection) and misophonia (defensive arousal to personally aversive sound patterns). It reviewed sociocognitive and affective touch frameworks, which situate ASMR within affective, interpersonal contexts, and surveyed existing neuroimaging evidence implicating posterior temporal-parietal and interoceptive circuits. Despite these advances, critical explanatory gaps remain and were also highlighted in each section: the absence of a mechanistic account linking near-ear auditory cues to somatosensory tingles and parasympathetic settling; a lack of time-resolved evidence capturing sub-second dynamics; and minimal translation into structured, clinically

relevant interventions. In response, this thesis follows a sequential process of inquiry from theory to therapeutics.

Chapter 2 introduces the Proximity Prediction Hypothesis (PPH), a formal, testable account of ASMR in which near-ear auditory cues engage peripersonal space mechanisms and simulate CT-optimal affective touch. This generates a somatosensory tingle sensation and, when prediction error is minimised, a vagally mediated shift toward parasympathetic calm. The chapter synthesises evidence across predictive coding, affective touch, interoception, and autonomic regulation, deriving falsifiable predictions about temporal dynamics and candidate neural correlates for the phenomenon.

Chapter 3 tests predictions from the PPH using time-resolved EEG and MEG. Sensor-space analyses focus on oscillatory signatures (e.g., beta suppression, gamma enhancement) hypothesised to index early simulation and precision-weighted updating, and form a mirrored analysis pipeline across both modalities. This constitutes the first investigation of ASMR using the neuroimaging technique of magnetoencephalography in the current literature.

Chapter 4 translates this mechanistic account into a preregistered, two-week longitudinal study in individuals with chronic pain. Using the same near-field ASMR trigger set, it assesses changes in sleep, anxiety, depression, fatigue, and pain using validated clinically meaningful measures, thereby testing whether audio-based, remotely accessible exposure can shift LC-vagal balance in a therapeutically relevant direction.

Chapter 5, the general discussion chapter, integrates these strands, evaluating where the PPH is supported or challenged, identifying its boundary conditions (e.g., responder variability, stimulus features, how semantic considerations in the research area could be standardised), and outlining concrete directions for future mechanistic and translational research.

Chapter 2:

The Proximity Prediction Hypothesis: How predictive coding of CT-touch explains Autonomous Sensory Meridian Response and its therapeutic applications.

The following chapter has been peer reviewed and was published on October 24th 2025, in a special edition of the journal *Frontiers in Behavioral Neuroscience: Exploring ANS-CNS Communication: Implications for Mental and Physical Health*.

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2.1 Abstract

Autonomous Sensory Meridian Response (ASMR) is a pleasant tingling sensation felt across the scalp and neck, widely reported to reduce anxiety and improve sleep. The Proximity Prediction Hypothesis (PPH) is the first comprehensive predictive coding model explaining ASMR's underlying neural mechanism. PPH posits that near-field acoustic cues from common ASMR triggers (e.g., brushing sounds, whispered speech) engage the audio-tactile Peripersonal Space Network, generating a top-down prediction of gentle C-tactile (CT) touch on CT fibre-rich skin of the scalp and neck. This prediction suppresses locus coeruleus (LC) arousal and increases vagal output, offering a mechanistic explanation for the phenomenon's therapeutic benefits. In a subjective-experience survey (N = 64), ASMR-labelled trials were rated significantly more pleasant but only slightly more arousing than controls. Pleasantness predicted both the presence and intensity of tingles, supporting PPH's core claim that hedonic value, rather than sympathetic activation, drives the graded somatosensory response. PPH situates ASMR within the Neurovisceral Integration framework, predicting measurable Central Nervous System-Autonomic Nervous System (CNS-ANS) markers (beta-band desynchronisation in the posterior insula and proportional increases in high-frequency heart rate variability with tingle intensity). It further predicts reduced LC activity during ASMR, stronger effects in individuals with high interoceptive prediction error (e.g., anxiety, autism), and attenuation of tingles when spatial proximity cues are removed. By integrating auditory proximity, CT-touch anticipation, and autonomic regulation into a single predictive-coding account, PPH provides a unified, testable framework for explaining ASMR, offering a blueprint for translating this sensory phenomenon into targeted, evidence-based interventions for anxiety and sleep disorders.

2.2 Introduction

Autonomous Sensory Meridian Response (ASMR) is a sensory phenomenon characterised by a pleasant tingling sensation felt across the scalp and often moving down the back of the neck, elicited by very specific stimuli. The sensation is triggered by auditory and/or audiovisual cues. Sounds that induce ASMR are varied and broad ranging, but the most popular triggers are slow, whispered speech, rhythmic hair brushing and tapping sounds (Barratt and Davis, 2015; Fredborg et al., 2021; Poerio et al., 2018). Alongside this, ASMR is also elicited via videos on media sharing platforms like YouTube where content creators use objects or their own voices to produce sounds that trigger the response in listeners. This is done by placing the camera and microphone near to the performers' mouths or hands while they whisper or manipulate objects to make noises into the microphone. Over the past decade, ASMR content has transitioned from a niche phenomenon to a mainstream YouTube staple. As of 2022, there were approximately 500,000 ASMR-focused channels and an estimated 25 million ASMR videos on the platform, illustrating the breadth and scale of its cultural reach. Many ASMR videos fall into role-play genres that simulate close personal attention, including hairdresser visits, spa treatments, makeup application, doctor's appointments, and other interpersonal care scenarios.

This popularity is seemingly driven by perceived benefits from experiencing the ASMR phenomenon, which go beyond the initial pleasant sensation. Survey work with hundreds of viewers found that 98% reported using ASMR for relaxation, 82% to help fall asleep, and about 70% to reduce stress or anxiety (Barratt and Davis, 2015, N = 475). In laboratory follow-ups, participants who experience tingles report lower state-anxiety scores and improved mood up to thirty minutes after listening (Fredborg et al., 2021) suggesting the phenomenon provides more than just a pleasant distraction during the tingling experience itself and offers longer term affective benefits to those who enjoy it. These self-reports have also been scaffolded by physiological evidence. In a within subjects study that compared tingling to non-tingling segments of the same videos, Poerio et al. (2018) found a reliable

heart rate deceleration accompanied by an increase in high frequency heart rate variability (HF-HRV). HF-HRV is a widely accepted non-invasive index of parasympathetic nervous system activity, often associated with states of calm and relaxation. Specifically, greater HF-HRV reflects increased vagal influence on the heart, indicating a shift toward physiological rest and recovery.

Recent work by Hozaki et al. (2025) extends this evidence using finger photoplethysmography (PPG), which not only captures pulse rate but also pulse wave amplitude, a measure of peripheral blood flow and vascular tone. In their study, both ASMR and nature videos reduced pulse rate relative to baseline, but ASMR produced significantly greater reductions. Moreover, ASMR was associated with increased pulse wave amplitude, consistent with peripheral vasodilation. Because vasodilation reflects parasympathetic dominance over vascular tone, these PPG findings complement HR and HRV evidence by demonstrating that ASMR's autonomic effects extend beyond cardiac regulation to include vascular relaxation, supporting the interpretation of ASMR as inducing a coordinated parasympathetic shift. These parasympathetic-shift indicators are consistent with reduced sympathetic outflow, but the interpretation that ASMR down-regulates tonic locus coeruleus (LC) activity remains inferential. PPG cannot directly index LC firing, and the observed combination of bradycardia and vasodilation is best understood as a physiological profile compatible with reduced LC tone, rather than definitive evidence. Future work could test this pathway more directly. For example, pupillometry offers a non-invasive proxy for LC activity, with pupil diameter shown to covary with LC firing in humans (Murphy et al., 2014). Neuromelanin-sensitive MRI and LC-targeted fMRI approaches can provide in-vivo markers of LC integrity and activity (Betts et al., 2019; Trujillo et al., 2023), allowing individual differences in ASMR-related parasympathetic shifts to be linked with LC dynamics. Pharmacological modulation also provides a causal testbed: reducing LC output (e.g., with α_2 -agonists such as clonidine) should potentiate ASMR-related vagal indices, whereas elevating noradrenergic tone would be expected to blunt them (Wang et al., 2014). Together, such approaches would allow a more rigorous evaluation of whether the LC-vagus axis mediates the parasympathetic profile observed during ASMR.

Although most research on ASMR has focused on mood benefits, some survey studies have revealed that sleep improvement is also a strong motivation for listening in many people. In Barratt and Davis's (2015) 475 participant survey, 82% of responders reported using ASMR videos "often" or "always" to fall asleep faster. A later large-scale online study (Smejka and Wiggs, 2022; N = 1,037) found that ASMR viewing improved relaxation and mood across

participants who did and did not suffer from insomnia. Although improvements were strongest in those who experienced tingles, no significant differences emerged between insomniacs and other groups in their response magnitude.

A mechanistic account is needed to link three disparate elements of the ASMR phenomenon: the acoustic character of the triggers, the subjective percept of pleasant scalp tingles, and the body-wide calming represented by physiological correlates like HRV and PPG, as well as reported mood and sleep benefits. A natural starting point is the Neurovisceral Integration (NVI) Model (Thayer and Lane, 2000). NVI frames mental state regulation as an interaction between the cortical central-autonomic network (CAN) and subcortical autonomic nuclei. When this interaction is smooth, indexed by high vagal tone and HF-HRV, the organism is flexible and resilient; when it is disrupted, anxiety and rumination flourish. Within this hierarchy the locus coeruleus functions as a noradrenergic “gain knob”; meaning elevated tonic LC firing biases the body toward sympathetic readiness, whereas a drop in LC tone likely lifts inhibition over the dorsal-motor nucleus of the vagus (DMV) and permits parasympathetic dominance, and calm. In a way, the LC and the vagus operate a seesaw-like regulatory axis that modulates perception and bodily state between arousal and relaxation. Here, the term “arousal” is used in two related but distinct senses: (i) tonic vigilance, determined largely by baseline LC activity, and (ii) stimulus-specific activation, such as pupil dilation or SCR, reflecting transient orienting to an input. The PPH framework speculates that both occur in sequence during ASMR; a brief orienting arousal phase, followed by parasympathetic accommodation when the cue is integrated as affiliative. Existing ASMR findings, such as HRV increase during tingling and subjective experience reports, fit this framework, implying vagal activation and a downshift in LC tone. Yet no published stepwise neural model currently explains how auditory stimuli like whispers or brushing sounds could initiate this regulatory shift, let alone generate a tingling sensation across the scalp as a consequence.

Despite the range of auditory triggers that can elicit ASMR in listeners, one property which they arguably all have in common is that they can be categorised as proximal, near-ear stimuli, rich in spatial cues, illustrated by three key acoustic features shown across the literature. First, very large interaural level differences (ILDs) and sub-millisecond interaural time differences (ITDs) signal that the sound source is only a few centimetres from the listener’s head. ASMR YouTube video recordings are typically made with binaural “dummy-head” microphones whose fake pinnae and ear canals preserve these cues; playback over loudspeakers reduces them, but headphones, through which 90% of listeners choose to

experience ASMR (Barratt and Davis, 2015; N = 475), deliver them unchanged, recreating the illusion that a hand or brush is at the ear. Second, the spectrum is colour-shifted by head-shadowing, meaning high frequencies above 8 kHz roll off steeply in the contralateral ear, a cue which listeners tend to interpret as indicating close spatial proximity (Begault and Trejo, 2000). Third, ASMR content creators often favour slow amplitude envelopes and low overall sound pressure levels. This means that the loudness of the signal rises and falls gradually, over hundreds of milliseconds or more, rather than in sharp, percussive bursts. A whispered phrase, a brush stroke across a microphone, or a series of soft taps typically shows a smooth, rounded waveform without abrupt transients. In addition, keeping the overall sound pressure level low ensures the audio remains intimate and non-startling, helping listeners maintain a relaxed, parasympathetic state; louder levels would recruit the middle ear reflexes and risk activating the sympathetic “alerting” system, which would contradict the calming goal of ASMR.

These findings converge on an interesting idea, that ASMR stimuli may work to convince the auditory system that an object is virtually approaching or touching the ear or scalp, in the absence of any real physical contact. A mechanistic model must therefore account for the special spatial signature of these sounds, then explain how such proximity information could cascade into both the tingling percept and the parasympathetic shift measured in HRV and through reported improvements in mood and sleep. This paper proposes a Proximity Prediction Hypothesis (PPH) to integrate the audio-tactile features mentioned above, with the NVI framework, arguing that near-field sounds pre-activate the brain’s Peripersonal Space Network and prompt a top-down prediction of impending gentle CT-touch on the scalp.

Valtakari et al. (2019) observed that ASMR experiences are accompanied by pupil dilation, while Poerio et al. (2018) reported increased skin conductance responses (SCR) during tingling segments compared to control periods. Both pupil dilation and SCR are well-established markers of sympathetic nervous system activity, indicating that ASMR is not a purely parasympathetic phenomenon. This has caused some debate in the literature, given its reportedly calming profile. However, as McGeoch and Rouw (2020) note, the combination of heart rate deceleration and increased SCR suggests both sympathetic and parasympathetic involvement and, because eccrine sweat glands (underlying SCR) receive only sympathetic innervation, while the heart is dually innervated by both sympathetic and parasympathetic pathways, the net decrease in heart rate points to an overall shift toward increased vagal tone. This aligns with the PPH model, in which pupil dynamics in ASMR are

predicted to reflect a transition from orienting to affiliative calm, where near-ear cues initially engage the LC-noradrenaline system, producing a transient pupil dilation to enhance sensory gain. As peripersonal space and CT-afferent touch predictions converge, tonic LC activity is suppressed and parasympathetic output dominates in the model, leading to heart rate deceleration, increased HF-HRV, feelings of calm, and we predict, eventual pupil constriction, a hypothesis that is yet to be tested in future research. This biphasic pattern would accommodate both sympathetic (early attentional) and parasympathetic (later calming) components, supporting the interpretation of ASMR as a flow state (Peifer et al., 2014) of 'relaxed alertness' characteristic of safe, affiliative proximity.

This biphasic profile can also be interpreted as reflecting an initial mismatch between perception and reality; where the brain briefly treats the near-ear cue as if physical contact were imminent, engaging orienting and sympathetic resources. A subsequent 'accommodation' phase might follow, in which the system recognises the safety and affiliative value of the stimulus, allowing parasympathetic dominance to emerge. In this way, early sympathetic activation is not contradictory to ASMR's calming effects but may be a necessary precursor, sharpening sensory gain before the vagal system restores balance.

After explaining the theoretical background, current evidence in the area will be collated and assessed in the context of the PPH model. Then, we report original illustrative survey data from sixty-four listeners in an immersive ASMR listening study, demonstrating that hedonic valence drives the tingling experience and its intensity, thus providing empirical support for the PPH model. Clinical applications and the reported benefits to mental health and sleep in ASMR experiencers will be discussed with the PPH model and CNS-ANS integration in mind. Future research will be suggested to test the theory, with falsifiable predictions for findings across CNS-ANS research, encompassing heart rate variability, pupil-indexed LC dynamics, and beta band neural signatures, in behavioural, EEG, and MEG studies, if the model is to be supported.

2.3 Theoretical foundations

2.3.1 The interoceptive brain and predictive coding

According to Interoceptive Predictive Coding accounts (Critchley and Harrison, 2013; Barrett and Simmons, 2015), cortical areas generate continuous, probabilistic forecasts (or 'priors') about what the viscera, skin, and muscles should feel like. Incoming afferent data are compared with these priors and any difference found is the prediction error signal (Feldman and Friston, 2010). A close match is desirable; a mismatch registers as physiological surprise and, when sustained chronically, has been linked to heightened anxiety (Paulus and Stein, 2010). When the incoming signal and priors match (or the error is negligible), this implies that the sensory world is unfolding as expected. Most of this comparison takes place in areas such as the posterior and anterior insula, which influence autonomic nuclei in the brainstem. The posterior insula receives raw interoceptive input, constructs a sensory map of the body, and forwards that map to the anterior insula, where predictions and errors are integrated with the affective context (Critchley and Harrison, 2013). When the match between the prior and signal is close, and the prediction error is small to negligible, for example, if you feel the gentle pressure that you expected while holding a cup in your hand, the anterior insula sends an inhibitory signal to the locus coeruleus (LC), the brainstem hub for noradrenaline release. In simple terms, this inhibits the LC's usual role in promoting arousal and vigilance. As tonic LC firing drops, its noradrenergic brake on the dorsal-motor nucleus of the vagus (DMV) is lifted. The result is increased vagal output and a rise in high frequency heart rate variability (HF-HRV), the parasympathetic signature of calm suited for rest, digestion, and affective ease (Samuels and Szabadi, 2008).

This low precision gate explains everyday illusions like in the phantom phone buzzing phenomenon, where people report feeling their phone vibrate even when it is not; a strong learned prior ("my phone is about to vibrate") meets either minimal somatic noise or no detectable cutaneous input at all. Because any residual error is labelled as low precision, the posterior insula fills in the expected buzz with a somatosensory echo, a phantom vibration, the anterior insula reports "prediction fulfilled," and the LC-vagus axis remains calm (Lin et

al., 2013). Virtual reality touch has a similar mechanism where viewing a virtual stick stroking a forearm that you associate with your own body in virtual reality produces tingles in 89% of users despite zero skin input on their actual arm in real life, because the visual prior overwhelms the ill-defined cutaneous error (Pilacinski et al., 2023), it is more likely that you are being touched and it is light and not hugely noticeable, than that all other, more reliable, priors are wrong in anticipating that touch when your previous experience and the visual input suggests it is very likely. In both cases of touch illusions, the visual or contextual prior overwhelms the ambiguous tactile input. The cue is interpreted as consistent with expected gentle touch but not clear enough to generate high precision error, allowing the prior to dominate. Touch is considered ill-defined in these circumstances because the sensory evidence is either absent, ambiguous, or delivered through a channel (e.g., auditory or visual) that does not strongly engage tactile precision mechanisms. When this occurs, the brain is more likely to accept the predicted sensation and resolve the ambiguity in favour of the expected state.

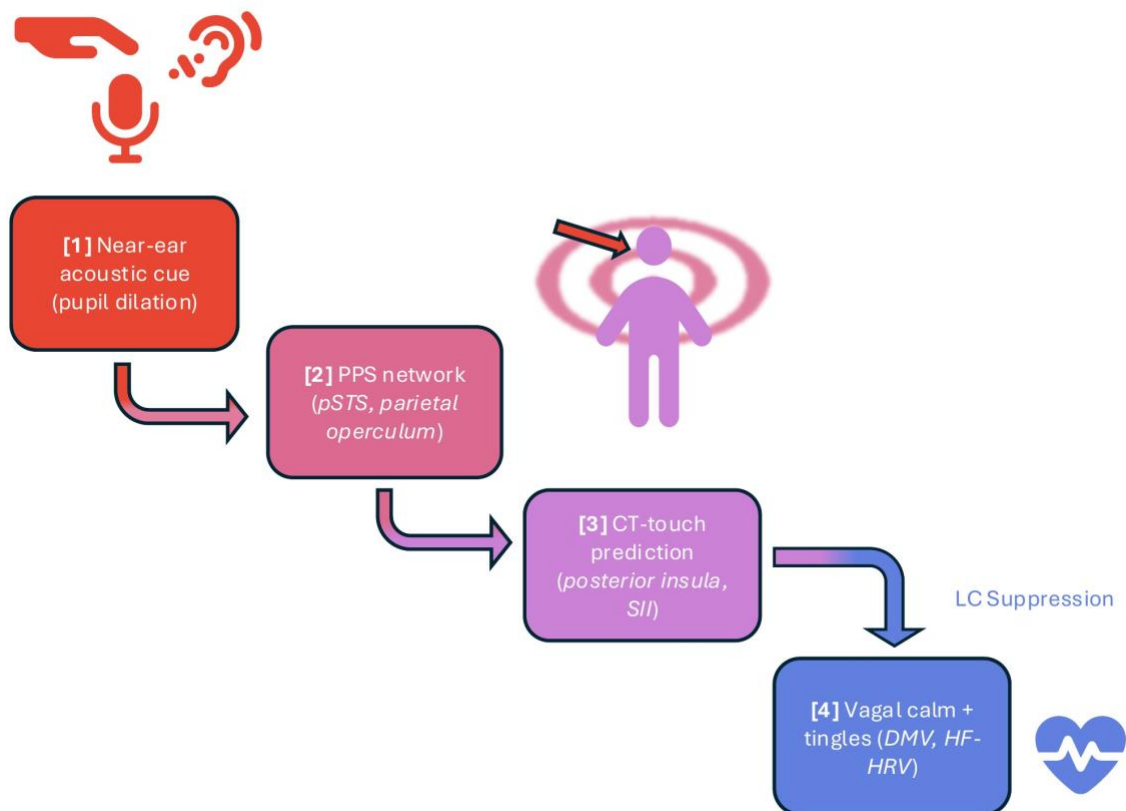
Crucially, “precision”, the brain’s estimate of sensory reliability, i.e., its confidence in the fidelity of a particular sensory channel, modulates how much any given error matters. High precision channels (e.g., retinal contrast, a pin-prick sensation) deliver errors that are hard to ignore; low precision channels however (faint rustling, diffuse light pressure) deliver errors that can be treated as background noise. Here we suggest that, when the brain issues a strong top-down prior like “I am about to feel a gentle stroke” and the incoming signal is fuzzy, delayed, or absent, the mismatch is labelled as low precision. In that case the posterior insula may simply fill in the expected sensation itself and send a “prediction fulfilled” message upstream. Because the error never gains salience, the anterior insula does not escalate to the LC, tonic LC firing falls, and the vagal brake is released even though no physical touch ever occurred.

It is important to note, however, that not all mismatches will be labelled low precision from the outset. When an ambiguous sensory cue first enters the system, for instance, a near-ear sound suggesting touch without any corresponding cutaneous input, the brain may briefly treat this as a salient error. In predictive coding terms, this transient up-weighting of error signals recruits the LC-noradrenaline system, manifesting as a short-lived sympathetic orienting phase (indexed by pupil dilation or SCR), evidence for this comes from several converging studies. Although much of the direct LC physiology comes from primate electrophysiology, these findings have been foundational for broader cross-species models of arousal. Aston-Jones and Cohen (2005) showed that phasic LC activity functions as an

orienting response to novel or behaviourally significant events, while Dayan and Yu (2006) framed phasic norepinephrine release as a neural interrupt signal marking unexpected uncertainty, i.e., prediction errors with high precision. Similarly, Sara and Bouret (2012) demonstrated that LC activity underlies rapid shifts in arousal when attention is reoriented to unexpected stimuli. Together, these accounts support the idea that the first stage of the proposed ASMR cascade may involve a sympathetic “alerting” phase driven by prediction error, before the system reclassifies the error as low precision and accommodates it. Once this occurs, the present theory suggests that the anterior insula inhibits tonic LC firing and parasympathetic dominance emerges, explaining the biphasic pattern of initial orienting followed by vagal calm. This series of predictive, neurophysiological events, from sensory prior to vagal activation, forms the basis of that theory, the Proximity Prediction Hypothesis (PPH) cascade, a stepwise model proposed to explain how the characteristic calm and tingling response of ASMR can arise from purely auditory cues. Each element of this cascade is explored in subsequent theoretical sections and visualised in Figure 2.1.

Figure 2.1

Proposed cascade of the Proximity Prediction Hypothesis (PPH).



Note: A near-ear sound activates peripersonal space (PPS) networks, which forwards a CT touch prediction to somatosensory and interoceptive regions; confirmation of that prediction suppresses locus coeruleus (LC) tone, disinhibits the vagal system, and generates parasympathetic calm and tingling sensations. [1] Near-ear acoustic cue → PPS detection: binaural whispers, tapping, and brushing sounds carry strong interaural time and level differences, interpreted by the posterior superior temporal sulcus (pSTS) and adjacent areas as proximal, human-origin sounds (Schürmann et al., 2006; Belin et al., 2000; Warren and Griffiths, 2003); at this early orienting stage, sympathetic attentional mechanisms such as pupil dilation are transiently recruited to enhance sensory gain (Valtakari et al., 2019). [2] PPS network → CT-touch prediction: pSTS and parietal operculum project to the posterior insula and secondary somatosensory cortex (SII), simulating tactile consequences of perceived social proximity, especially on CT-rich scalp/neck regions (Löken et al., 2009; Gazzola and Keysers, 2009). [3] Accurate prediction → LC suppression and vagal disinhibition: minimised prediction error reduces anterior insula drive to the LC, lowering tonic noradrenaline and lifting inhibitory control over the dorsal motor nucleus of the vagus (DMV), increasing parasympathetic tone and yielding cardiac deceleration and increased high-frequency HRV (Paulus and Stein, 2006; Samuels and Szabadi, 2008). [4] Conscious correlate; the tingles: pre-activation of insula/SII yields a synchronous, spatially diffuse cortical volley experienced as a tingling somatosensory echo of predicted contact. Icons (ear, hand, microphone, person, circular shapes, heart rate icon) from Font Awesome Free, licenced under CC BY 4.0; edited for size, colour, and orientation.

A similar process is proposed more generally in the Somatic Error Hypothesis (Khalsa and Feinstein, 2019), where the brain reduces prediction error by generating bodily sensations that match an expected state. While this mechanism is typically invoked to explain chronic symptoms in somatising disorders, here we extend its logic to a benign interoceptive illusion felt by those who experience ASMR.

2.3.2 The audio-tactile fabric of peripersonal space

Prediction in this case does not operate in isolation, it is shaped by multisensory maps of Peripersonal Space (PPS), which can be thought of as a region of 20-30 cm space surrounding the body where approaching objects are most likely to make contact. Importantly, PPS is not a simple distance gradient; it behaves like a biological boundary. Stimuli presented just inside the bubble elicit abrupt neural and behavioural changes, whereas equally small decrements in distance once the stimulus is outside the peripersonal space have little effect (Làdavas and Serino, 2008; Serino et al., 2015). A substantial body of multisensory work shows that the brain treats a near-ear sound as a potential touch event.

Early single-unit electrophysiology in macaque monkeys revealed a class of multisensory neurons in ventral premotor and parietal regions, including the ventral intraparietal area (VIP), that integrate tactile, visual, and auditory signals relevant to peripersonal space. Auditory cues alone can activate neurons in peripersonal space-sensitive regions, including the VIP, for instance, Graziano et al. (1999) reported that broadband noise sources moving toward the head, from 70 cm to 10 cm, caused multisensory neurons in VIP to fire more vigorously than when those same stimuli moved within far space. This indicates that approaching sounds, even in the absence of visual input, can signal potential contact and recruit defensive spatial coding. Moreover, Avillac et al. (2007) demonstrated that VIP neurons integrate visual and tactile input when sensory events are spatially and temporally aligned. These neurons often integrate tactile and auditory information, reinforcing the idea that auditory proximity cues are biologically relevant indicators of incoming contact.

Importantly, VIP neurons respond to stimuli that occur both on the body (i.e., within a neuron's tactile receptive field) and just beyond it, typically within a few tens of centimetres. This alignment of visual and somatosensory receptive fields reflects a body-centred coding of nearby space, a neural basis for anticipating contact (Colby et al., 1993; Rizzolatti et al., 1997).

Magnetoencephalography supports the idea that ASMR-like stimuli can activate such somatosensory regions. Schürmann et al. (2006) played realistic sounds resembling haircut and water-dripping scenarios and found beta-band desynchronisation in the secondary somatosensory cortex (S2). This effect is supported across broader studies. Canzoneri et al. (2012) found that sounds approaching the hand significantly accelerated tactile responses once perceived within peripersonal space. A meta-analysis by Holmes et al. (2020) confirmed a modest (15 ms) reduction in tactile reaction times when sounds occurred near the body versus farther away, although they noted variability and small effect sizes.

Additionally, Taffou and Viaud-Delmon (2014) demonstrated that looming “rough” sounds, those with threat-like acoustic properties, expanded the effective PPS boundary, triggering tactile facilitation at greater distances than smoother sounds. Together, these findings support the notion that sound proximity is a potent modulator of sensory integration and may help explain how ASMR content elicits embodied responses despite being purely auditory.

These findings demonstrate that the posterior STS, inferior parietal cortex, and the parietal operculum behave like proximity detectors, amplifying their response when an auditory object crosses the PPS boundary, and is therefore likely to make physical contact. Within this framework, PPS responses could be generating a transient orienting mismatch, when a stimulus is detected inside the boundary without accompanying tactile confirmation. This mismatch could recruit sympathetic arousal to heighten vigilance, but once sensory prediction resolves in favour of a safe, affiliative source, parasympathetic accommodation then follows. ASMR may therefore harness this sequential PPS dynamic, beginning with an alerting phase and culminating in vagal release. Such proximity-sensitive firing is proposed to represent the first node in the PPH cascade, the moment when the brain interprets near-ear sounds as predictive of imminent affective touch, triggering downstream autonomic changes detailed in the next sections (see Figure 2.1).

2.3.3 C-tactile afferents and the mechanism behind affective touch

If, as PPH suggests, ASMR is occurring through prediction of affective touch, it is important to consider what exactly the brain is predicting and how that links to the reported ASMR experience. When contact does occur on the skin, it is detected by at least two tactile channels. Fast, myelinated A- β fibres handle discriminative features, conveying facts about the touch, like location, texture, and force, whereas C-tactile (CT) afferents are slow, unmyelinated fibres that overwhelmingly tend to innervate hairy skin regions. Microneurography shows that CT afferents respond optimally to gentle stroking at 1-10 cm s⁻¹, with a firing peak at around 3 cm s⁻¹, which is exactly the velocity of social grooming strokes in primates (Löken et al., 2009; Ackerley et al., 2014). Their firing rate predicts subjective pleasantness and drives oxytocin release, posterior-insula activation and a parasympathetic drop in heart rate (Ackerley et al., 2014; Pawling et al., 2017).

Human CT afferents have been recorded in scalp, face, forearm, abdomen and thigh areas (McGlone et al., 2014) and show a clear preference for hairy skin. While detailed follicle density maps are scarce, regions such as the scalp midline, nape, and upper back are widely associated with social grooming in primates and are plausible candidates for dense CT innervation (McGlone et al., 2014). These zones are therefore likely to be particularly

well populated by CT-touch fibres. They are also prime cortical targets for affective touch, where the brain predicts a gentle, grooming-like sensation to land. Crucially, this is indeed where the ASMR tingling sensation is reported to be localised: the scalp, face, neck, and upper back (Barratt and Davis, 2015; Poerio et al., 2018; Lochte et al., 2018).

Behavioural data echo the physiology; in barbary macaques, bouts of allogrooming (a prosocial behaviour where animals of the same species groom one another) lower basal cortisol and heart rate within minutes (Shutt et al., 2007). In humans, five minutes of scalp massage at CT-optimal velocity produces a significant HF-HRV increase and self-reported anxiety reduction in Spielberger state-anxiety test scores (Diego and Field, 2009).

Consistent with this, a 45 min relaxation massage before bed has been shown to enhance sleep efficiency in individuals with insomnia (Ntoumas et al., 2025). Moreover, meta-analytic evidence indicates that interventions involving head touch specifically, such as face or scalp massage, may confer particularly strong physical and mental health benefits (Packheiser et al., 2024), reinforcing the potential relevance of affective touch to ASMR-related somatosensory modulation. As the second stage of the cascade, CT-touch predictions anchor the brain's expectation of safety and interpersonal care. Taken together, these findings establish CT-touch as a hedonic, anxiolytic, and sleep-promoting modality, and identify the scalp and neck as privileged substrates for such contact, exactly the locations where ASMR listeners report feeling their tingles.

One complementary, structural account of ASMR has been offered by McGeoch and Rouw (2020), who propose that ASMR may involve synesthetic cross-activation between the primary auditory cortex (A1) and affective-touch maps in the dorsal posterior insula (dplns). Earlier functional evidence by Lochte et al. (2018) supports this coupling: in ASMR experiencers, moments of tingling elicited elevated BOLD activation not only in auditory and somatosensory regions but also in the nucleus accumbens and mPFC, implicating reward and affiliative circuitry in the perceptual experience. This suggests that ASMR may recruit not just tactile-sensory prediction routes but also reward/bonding networks. Under proximal, interpersonal conditions, near-ear sounds may recruit such regional cross-activation to simulate gentle social touch, triggering posterior-insula activity, activating reward/affiliative circuits, and promoting vagal engagement. Unlike the PPH, however, these accounts do not address the state-dependent gating, peripersonal space integration, or temporal autonomic cascade that determine when and how this cross-activation occurs. The two perspectives can therefore be viewed as complementary, with cross-activation describing the same plausible neural route (i.e., A1 to dplns, and then on to reward/affiliative circuits) and the PPH specifying the predictive coding logic and dynamic conditions under which that route is

engaged in the ASMR phenomenon. Furthermore, while McGeoch and Rouw's hypothesis and Lochte's findings link auditory input to affective touch and reward areas, they do not specify the computational mechanism by which tingles emerge, nor how such activation alone would produce the distinct, wave-like somatosensory echo characteristic of ASMR. The PPH extends this by proposing the predictive coding process and time-resolved neural signature capable of transforming such cross-activation into the tingling percept itself. To our knowledge, the PPH draws upon and extends these key models but represents the first explicit attempt in the literature to explain the ASMR tingling percept via a predictive coding account, linking sensory priors, insular prediction errors, and downstream autonomic responses.

2.3.4 The social neurocognitive context of ASMR

If ASMR indeed reflects a prediction of affiliative touch, then understanding the social and cognitive conditions that shape those priors becomes crucial. ASMR triggers overwhelmingly reflect socially salient acts like whispering, soft-spoken instruction, and gentle, attentive behaviours, many of which imply close interpersonal proximity. These cues may be sufficient to evoke predictions of touch-like feedback, particularly in individuals predisposed to interpret such signals as affiliative or comforting.

Recent empirical studies have identified five principal ASMR trigger categories, all of which share a perceptual association with human interaction: (1) viewing individuals interact with objects, (2) watching socially intimate acts, (3) hearing soft repetitive sounds, (4) simulated social interaction, and (5) whispering or chewing (Smith et al., 2020; Fredborg et al., 2018). Even seemingly nonsocial triggers, such as tapping or crinkling, often co-occur with goal-directed behaviours that implicitly suggest a human source (McErlean and Banissy, 2017). This convergence supports the idea that ASMR is scaffolded by social perceptual priors, often concerning caregiving or affiliative intent.

Recent work by Poerio et al. (2023) developed the ASMR Trigger Checklist (ATC), a validated tool for systematically identifying and categorising common ASMR triggers, to assess how individuals respond to a wide range of sounds. They found considerable variability in which triggers reliably induced tingles across participants. Critically, the most potent triggers tended to be those that implied gentle, interpersonal interaction or close physical proximity, such as whispering or soft tapping. This variability is consistent with the precision-weighting mechanism proposed by the PPH; individuals may assign higher predictive value to particular sensory cues based on their internal priors about social intent, interpersonal closeness, or expected affective touch. The ATC therefore offers a structured

way to assess which auditory signals carry predictive weight in different individuals, and why the same cue may trigger ASMR in one person but not another. These findings reinforce the notion that ASMR emerges from a socially grounded predictive model shaped by prior experience, attachment tendencies, and interoceptive sensitivity.

Most significantly for the PPH, Poerio et al. (2023) study also showed that physical touch itself, rather than sound or visual cues, was not only the most commonly endorsed ASMR trigger reported to elicit a tingling sensation in participants (98%) but also the most intense, with minimal variation across individuals; the ATC subset of tactile and interpersonal triggers gave examples like “close-up movements directed at you” and “light touch on your face, e.g., make-up application”. This highlights that touch itself, whether anticipated or actively experienced is a core trigger for the ASMR tingling sensation, making the idea of a somatosensory echo even more plausible as it is clear that the tingling sensation is a ground truth for the phenomenon, not an abstract, novel response the brain is predicting. This emphasises that the tingles are less of a bodily illusion, as some may argue, and more of a plausible sensory prediction based on what it does actually feel like when people are really being touched.

Importantly, Poerio and colleagues argue that online ASMR content should be seen as a simulation of real-world interpersonal encounters rather than as distinct from them, and that trait ASMR may be meaningfully defined by a person’s sensitivity to touch-related triggers. In this way, their work empirically supports the idea that ASMR operates through predictive interoceptive mechanisms shaped by tactile expectation and affiliative social context. Consistent with this view, Gillmeister et al. (2022) demonstrated that ASMR-experiencers reported greater sensitivity to positive social touch in daily life and a higher likelihood of feeling mirror touch, than non-experiencers, reinforcing the role of trait-dependent priors for affiliative interaction in driving the ASMR response. This final phase of the cascade, the culmination of proximity, touch prediction, and arousal regulation, is therefore likely shaped by an individual’s social priors, attachment style, and interoceptive sensitivity.

Neuroimaging studies further reinforce the social grounding of ASMR. Lee et al. (2020) found that during ASMR experiences, participants showed activation in brain regions implicated in social cognition and mental state simulation, including the posterior cingulate cortex, superior and middle temporal gyri, and the lingual gyrus. These areas are key components of the brain’s social mentalizing network, suggesting that ASMR may engage the same systems we use to interpret and internalise others’ intentions; particularly when those intentions are perceived as caring, attentive, or intimate.

Earlier work by Lochte et al. (2018) proposed that ASMR may function as a vestigial grooming response, with Lochte going on to suggest that ASMR may be a polymorphic trait, a term used in evolutionary biology to describe a characteristic that is present in some individuals of a species but not all, due to genetic or developmental variability. Common examples include wisdom teeth or lactose tolerance, traits that were once adaptive, but are now only expressed in certain subsets of the population. If ASMR is indeed a polymorphic vestige of an ancestral grooming response, this could explain why only some individuals report experiencing tingles in response to specific stimuli. Again, aligning with the PPH model's suggestion that ASMR emerges only when an individual's internal predictive model assigns high precision to interpersonal proximity cues, a tendency that may itself vary across individuals based on neurocognitive, social, or interoceptive traits. These accounts offer an ethological framework for why ASMR stimuli elicit pleasure and calm in a specific subset of individuals.

That subset may be defined, in part, by individual differences in trait empathy and sensory-emotional inhibition. McErlean and Banissy (2017) reported that ASMR experiencers tend to score higher on "Empathetic Concern", suggesting a heightened sensitivity to social-affective cues. Others have found that ASMR is associated with reduced functional connectivity in the prefrontal cortex and default mode network (Smith et al., 2017; Fredborg et al., 2021), implying diminished top-down inhibition of incoming sensory-affective stimuli. In predictive coding terms, such individuals may assign greater precision to exteroceptive social cues while allowing these predictions to unfold with minimal suppression, creating fertile ground for ASMR to emerge.

2.3.5 Individual differences in ASMR

A consistent theme across the ASMR literature is the striking individual variability in both susceptibility and trigger potency. Not everyone experiences tingles, and those who do, often have different personal preferences for effective triggers. Using the ASMR Trigger Checklist (ATC), Poerio et al. (2022) showed that responses to different triggers are relatively stable within individuals but highly idiosyncratic across the population; whispering and soft tapping were amongst the most reliable elicitors, while other sounds such as chewing or eating were inconsistent and could even be aversive. This heterogeneity is echoed in misophonia, an intolerance for specific sounds (often human oral/nasal sounds like chewing or breathing) that reliably evoke strong negative emotional reactions (e.g., anger, disgust) and autonomic arousal in many people (Edelstein et al., 2013). Notably, some misophonia triggers overlap with ASMR triggers (e.g., chewing and other mouth sounds), therefore, the same cue can be reported as intensely aversive by some listeners

yet induce pleasant tingling in others. McGeoch and Rouw (2020) argued that ASMR and misophonia can be seen as opposing outcomes of auditory-affective processing, with one yielding affiliative calm, the other defensive aversion depending on the preferences of the listener.

The PPH naturally accommodates such variability within a predictive coding framework. In ASMR experiencers, near-ear cues are weighted as affiliative priors, reducing insular prediction errors and downregulating LC-noradrenaline tone. In others, the same cues may be assigned negative priors, heightening error and sympathetic arousal, as in misophonia. This provides a mechanistic explanation for why identical auditory inputs can generate diametrically opposed affective outcomes.

Individual differences in social-emotional traits further moderate this process. Those who report experiencing strong ASMR tend to score higher on empathic concern (McErlean and Banissy, 2017) and exhibit reduced prefrontal and default-mode network connectivity (Smith et al., 2017; Fredborg et al., 2021), suggesting that greater sensory-affective permeability may support ASMR proneness. Conversely, individuals with atypical interoception or altered affective empathy, such as those with autism or anxiety, may experience either enhanced benefits or blunted responses, depending on how their predictive models weigh affiliative cues - a topic that will be explored further in section 6.2 of this paper. Attachment style may also play a role: early caregiving experiences calibrate priors about the reliability and comfort of close interpersonal contact (Mikulincer and Shaver, 2010). Securely attached individuals may be more likely to interpret ASMR cues as soothing and affiliative, whereas those with avoidant or anxious attachment might assign lower precision or even aversive value to the same signals.

Taken together, ASMR should be understood not as a uniform response but as a polymorphic trait (Lochte et al., 2018), expressed in some individuals but not others, shaped by differences in priors, attachment style, interoceptive processing, and sensory-emotional inhibition. Recognising this variability is essential both for theory, by preventing overgeneralisation, and for clinical translation, where personalisation will be necessary to ensure that interventions based on ASMR do not inadvertently provoke discomfort or aversion.

2.4 The role of pleasantness in ASMR

If a near-ear whisper is effective because it forecasts a slow, pleasant interpersonal contact, then the strength of the ASMR response should depend on how rewarding that predicted

contact feels, not on its sheer acoustic energy. Within the PPH framework, pleasantness (valence) is expected to determine two outcomes: whether a listener classifies a segment as ASMR at all, and how intense the tingles feel during the ASMR experience. This mirrors genuine affective touch, where CT firing rates track subjective pleasantness (Löken et al., 2009) and hedonic ratings predict downstream effects on pain perception (Pawling et al., 2017). By analogy, ASMR tingles should scale with pleasantness because the posterior insula propagates stronger predictions of affective touch when the hedonic prior is stronger.

This valence-first logic is also illustrated by the content ecology of ASMR. The most watched videos on YouTube are spa, hairdresser, and make-up roleplays in which creators whisper reassurances, move brushes and scissors centimetres from the microphone, and enact a caretaking script. Such clips are maximising both near-field spatial cues and a social-grooming context, forming a strong hedonic prediction with minimal arousal load. Because the CT-touch prediction is intrinsically hedonic, a dominance of pleasantness over arousal would mirror the physiology of real affective touch. Microneurography shows that the firing rate of CT afferents rises monotonically as stroking speed approaches the 3 cm s^{-1} optimum and that subjective pleasantness ratings track this firing curve with an almost unit slope (Löken et al., 2009). Follow-up psychophysics demonstrated a similar scaling for behavioural impact; in Pawling et al. (2017) each one-point increase on a 10-point pleasantness scale produced an additional 0.9-point decrease in pain rating during concurrent heat stimulation, confirming that the more pleasant the predicted stroke, the stronger its sensory-affective consequence. In other words, CT-touch intensity is modulated by valence in exactly the way PPH predicts ASMR might be.

This account is further supported by recent behavioural data from Gillmeister et al. (2022), who found that ASMR responders report more frequent and intense vicarious tactile sensations when observing touch, compared to non-responders. There was also some evidence that individuals with stronger ASMR traits experienced more pronounced vicarious touch, although this relationship was not consistently supported across statistical approaches. In addition, ASMR responders reported greater reactivity to positive, but not negative, interpersonal touch. Together, these findings suggest that ASMR is associated with heightened sensitivity to socially relevant and affectively positive tactile cues, even when these are only observed rather than directly experienced. While the study does not directly test underlying mechanisms, the pattern of results is consistent with the PPH framework, in which predicted or inferred social touch plays a role in shaping the intensity of the ASMR experience.

At this point, it is useful to clarify how “arousal” can be defined; in some contexts, arousal refers to a general state of vigilance or sympathetic readiness (baseline tonic LC activity), while in others it denotes stimulus-specific activation, i.e., the subjective energetic quality evoked by a cue (Aston-Jones and Cohen, 2005; Sara and Bouret, 2012; Dayan and Yu, 2006). The PPH highlights that ASMR appears to involve both: a brief orienting arousal response [as seen during pupil dilation by Valtakari et al. (2019)] during the initial prediction error phase, followed by a lower-intensity, stimulus-specific activation that co-occurs with pleasant tingling and parasympathetic calm (reflected in self-reports and both heart rate deceleration and HF-HRV increase; Poerio et al., 2018).

Importantly, this does not mean that arousal is irrelevant to ASMR. Some triggers may increase both pleasantness and arousal, and the role of arousal remains equivocal. What PPH predicts, however, is that pleasantness will be the primary driver of whether a sound crosses the tingle threshold and of how strong those tingles become.

The next section tests this prediction directly, using trial-level behavioural data on pleasantness, arousal, and ASMR reports from an original survey dataset by the authors.

2.5 Illustrative behavioural evidence

2.5.1 Method

2.5.1.1 Participants

Undergraduate students (N = 64) from the University of York took part. Recruitment did not require prior experience of ASMR, to avoid expectation bias while still allowing inclusion of those who had previously engaged with ASMR content. All participants were over 18 in age, gave informed consent, and none reported adverse reactions to ASMR sounds. It should be noted that this sample was restricted to undergraduate students, which may limit the generalisability of findings to other age groups or clinical populations.

2.5.1.2 Stimuli

The auditory stimuli were drawn from a larger experiment in which these same participants had taken part. The present section focuses solely on the behavioural survey data.

The stimuli comprised of 18 sound clips (Appendix C): 13 experimental sounds intended to plausibly elicit ASMR (e.g., paper folding, tapping, stroking, brushing) created by a professional ASMR content creator (Nader, 2023), plus 5 control sounds (ambient traffic noise) presented via Sennheiser HD280 Pro Dynamic Hi-Fi Stereo headphones, as 5 s

sound clips embedded within the online survey. Participants completed the survey within a sound-attenuated room to minimise distraction. Mouth sounds were deliberately excluded to avoid inadvertently triggering misophonia, though this reduces ecological validity given that chewing and whispering are both major triggers for many ASMR viewers.

2.5.1.3 Procedure

Participants listened to 5 s clips of the 13 ASMRtist-created experimental sounds and the 5 control sounds mentioned above, during a digital questionnaire assessing their subjective responses to the experimental stimuli. For each sound, participants were asked whether they believed they experienced ASMR (“Yes” or “No”). The participants were informed of the definition of ASMR in the information sheet provided, and there was no requirement to have been familiar with ASMR or know if you could experience it, to sign up for the study. If “Yes” was selected to suggest ASMR had been experienced for any sound, participants were prompted to provide a retrospective estimate of tingle intensity on a 0–10 scale, if they could recall the sensation. All participants also used on-screen sliders to rate the pleasantness and arousal associated with each sound on continuous scales from –250 (extremely unpleasant or calming) to +250 (extremely pleasant or arousing) whether they experienced ASMR for that sound or not. The survey was completed immediately after a separate EEG experiment where the participants had listened to longer versions of all sound clips, while participants remained in the sound-proof testing room environment, to minimise memory decay and distraction, and using the same headphones for sound clip delivery. Not all participants provided tingle intensity ratings for each sound, as this question was optional and conditional on an ASMR report as well as their memory of it.

This retrospective design was chosen to avoid interrupting the listening session itself and has precedent in accepted foundational ASMR studies, where both survey (Barratt and Davis, 2015; Smejka and Wiggs, 2022) and laboratory work (Poerio et al., 2018) have relied on post-exposure reports to capture ASMR experiences. While immediate post-exposure

ratings mitigate memory bias, they remain vulnerable to under- or over-estimation compared with real-time capture so this limitation should still be considered when interpreting the results.

2.5.1.4 Statistical analysis

To investigate what drives whether a sound elicits ASMR, and the strength of the associated tingling sensation, mixed-effects regression models were implemented in R (version 4.4.0) using the lme4 (Bates et al., 2015), lmerTest (Kuznetsova et al., 2017), and broom.mixed (Bolker et al., 2022) packages. Predictors were z-scored to aid interpretation and comparability. A logistic mixed-effects model was used to predict ASMR classification (either Yes or No) from pleasantness and arousal ratings, with random intercepts for participant and sound. A subsequent model tested whether the effect of pleasantness on reported ASMR experience was moderated by arousal using an interaction term.

For trials where participants reported experiencing ASMR and rated its intensity, a linear mixed-effects model was used to predict tingle strength from pleasantness and arousal, again including an interaction term in a follow-up model. Visualisations were created using ggplot2 (Wickham, 2016) and ggeffects (Lüdtke, 2018), with predicted probability heatmaps and scatter plots depicting the effects of predictors across trials and sound clips.

The scripts used to perform these analyses are accessible via a Github repository found in Appendix A of this thesis.

2.5.2 Results

2.5.2.1 What drives the ASMR decision?

A mixed-effects logistic regression model was fit with ASMR classification (Yes or No) as the outcome and z-scored pleasantness and arousal as fixed effects, with random intercepts for participant and sound.

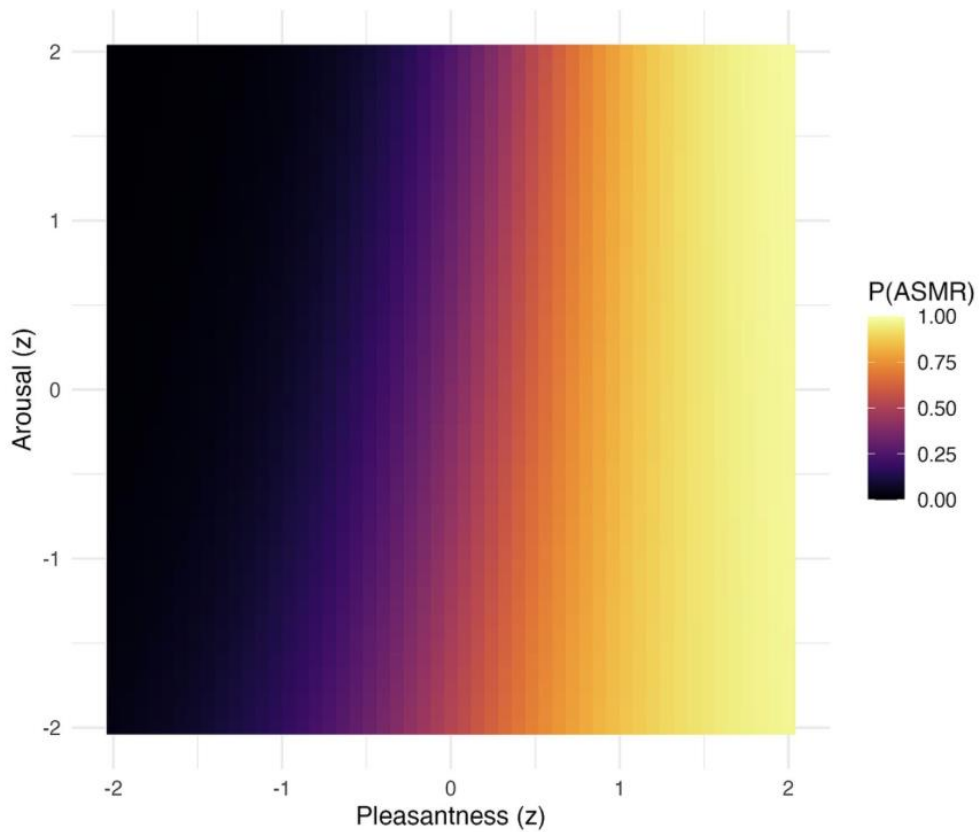
Pleasantness emerged as a strong positive predictor of ASMR reports ($\beta = 2.07 \pm 0.23$, $z = 8.86$, $p < 0.001$), corresponding to an odds ratio (OR) = 7.90 with a 95% CI = [5.00, 12.45], i.e., each 1 SD increase in pleasantness increased the odds of reporting ASMR by ~8×; while arousal showed a non-significant negative trend ($\beta = -0.29 \pm 0.17$, $p = 0.092$; OR = 0.75, 95% CI = [0.53, 1.05]). This suggests that hedonic valence, rather than activation level, primarily drives the ASMR decision.

An interaction term between pleasantness and arousal was also tested to assess whether arousal modulated the effect of pleasantness. However, the interaction was not statistically significant ($\beta = 0.20 \pm 0.15$, $p = 0.194$; OR = 1.22, 95% CI = [0.91, 1.64]), and did not improve model fit (likelihood ratio test: $\chi^2(1) = 1.68$, $p = 0.195$). Therefore, the probability of classifying a sound as ASMR was strongly driven by pleasantness across the full arousal range. Model performance indices were: AIC = 552.71, BIC = 579.55, R^2 (marginal) = 0.279, R^2 (conditional) = 0.767, ICC = 0.677, indicating substantial between-participant/sound clustering with a sizeable fixed effects contribution.

Figure 2.2 visualises the predicted probability of reporting ASMR as a function of z-scored pleasantness and arousal. The near-vertical gradient in predicted probabilities underscores the dominance of hedonic valence in the ASMR decision; increases in pleasantness robustly predict ASMR reports across the full arousal range, while arousal adds minimal predictive power.

Figure 2.2

Predicted probability of ASMR classification as a function of z-scored pleasantness (x-axis) and arousal (y-axis).



Note: Colours show predicted probabilities from a logistic mixed-effects model with random intercepts for participant and sound (N = 64). Near-vertical contour lines indicate pleasantness as the dominant predictor, with minimal modulation by arousal.

Table 2.1 presents the fixed effect estimates from the full logistic regression model, including the interaction term.

Table 2.1

Fixed-effect estimates from the logistic mixed-effects model predicting ASMR classification from z-scored pleasantness and arousal (including their interaction), with random intercepts for participant and sound.

Term	Estimate	SE	Z	p	95% CI (b)	OR	95% CI (OR)
(Intercept)	-0.499	0.424	-1.18	0.239	[-1.330, 0.332]	0.607	[0.264, 1.39]
Pleasantness (p_z)	2.070	0.233	8.86	0.000	[1.610, 2.520]	7.90	[5.00, 12.45]
Arousal (a_z)	-0.293	0.173	-1.69	0.091	[-0.633, 0.047]	0.746	[0.531, 1.048]
Pleasantness x Arousal (p_a:a_z)	0.196	0.151	1.30	0.194	[-0.100, 0.492]	1.22	[0.905, 1.636]

Note: Odds ratios (OR) with 95% CIs are shown. Model fit: AIC = 552.71, BIC = 579.55; $R^2(\text{marginal}) = .279$, $R^2(\text{conditional}) = .767$; ICC = .677.

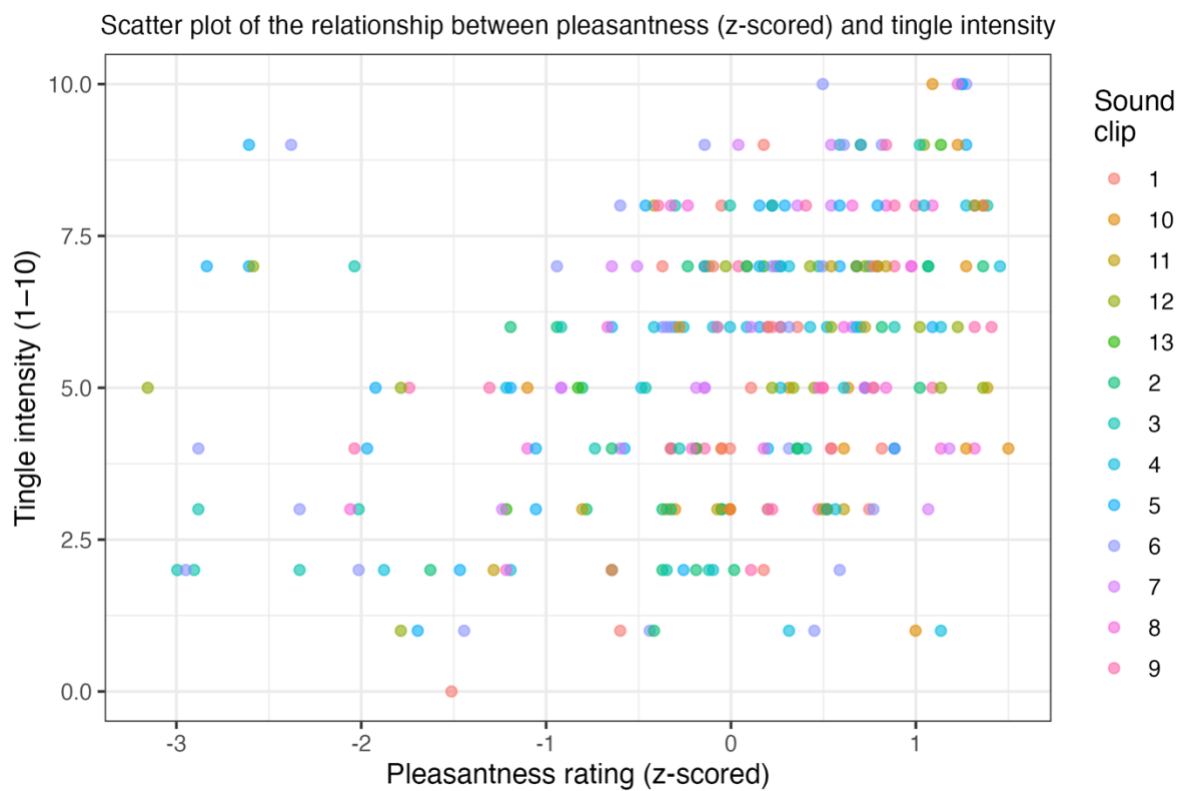
2.5.2.2 Tingle intensity

On 136 ASMR-positive trials with self-rated intensity scores, tingle intensity was positively associated with pleasantness ($\beta = 1.06 \pm 0.22$, $t = 4.71$, $p < .001$). Arousal did not show a significant main effect in this initial model ($\beta = -0.19 \pm 0.16$, $t = -1.15$, $p = .25$), suggesting that its relationship with intensity may depend on other variables. Figure 2.3 provides a

visualisation of the relationship between pleasantness and tingle intensity across trials, colour-coded by sound clip.

Figure 2.3

Scatter plot of the relationship between pleasantness (z-scored) and tingle intensity across 136 ASMR-positive trials.



Note: Each point represents one trial; colours indicate different sound clips. The scatter plot (Figure 2.3) illustrates a positive association between pleasantness (z-scored) and tingle intensity across trials, with higher pleasantness ratings generally corresponding to higher reported intensity. Considerable variability is evident across sound clips and participants.

To examine whether the relationship between pleasantness and intensity depended on arousal, an interaction model was fitted. Pleasantness was a strong positive predictor ($\beta = 1.22 \pm 0.23$, $t = 5.25$, $p < .001$), while arousal showed a modest negative association ($\beta =$

-0.48 ± 0.20 , $t = -2.39$, $p = .018$). The Pleasantness \times Arousal interaction was also significant ($\beta = 0.40 \pm 0.18$, $t = 2.22$, $p = .028$; see Table 2.2).

Table 2.2

Linear mixed-effects model of tingle intensity ratings (1-10 scale) on ASMR labelled trials

Predictor	β (Estimate)	SE	df	t	p	95% CI (lower, upper)	Partial R^2	95% CI (Partial R^2)	Std. Beta (β^*)
Intercept	4.51	0.31	24.8	14.33	<.001	[3.86, 5.16]	–	–	–
Pleasantness (z)	1.22	0.23	126.0	5.25	<.001	[0.76, 1.68]	0.145	[0.055, 0.262]	0.536
Arousal (z)	-0.48	0.20	119.3	-2.39	.018	[-0.88, -0.08]	0.032	[0.001, 0.112]	-0.211
Pleasantness \times Arousal	0.41	0.18	114.3	2.22	.028	[0.04, 0.77]	0.027	[0.000, 0.103]	0.177

Note: Predictors were z-scored pleasantness and arousal ratings from the affect grid, and their interaction. Random intercepts were included for participant and sound. Pleasantness was a strong positive predictor of tingle intensity, arousal showed a weak negative association, and the Pleasantness \times Arousal interaction was significant. Fixed-effect estimates (β), SEs, dfs, t, p, 95% CIs, and partial R^2 (95% CIs) are reported. Model fit: AIC = 589.59; BIC = 609.97; R^2 (marginal) = .146; R^2 (conditional) = .529; ICC = .448; RMSE = 1.40. Std. Beta (β^*) from a refit with z-scored outcome.

The significant Pleasantness \times Arousal interaction indicates that the association between pleasantness and tingle intensity varied as a function of arousal. Specifically, the positive

relationship between pleasantness and intensity was somewhat stronger at higher levels of arousal. Interpreted on the original 1-10 scale, a 1 SD increase in pleasantness corresponded to an average +1.22 point increase in intensity. Effect sizes indicated that pleasantness accounted for the largest proportion of variance (partial $R^2 = 0.145$), with smaller contributions from arousal (partial $R^2 = 0.032$) and the interaction term (partial $R^2 = 0.027$). Overall, these results suggest that pleasantness is the primary predictor of tingle intensity, with arousal modestly modulating this relationship.

2.5.3 Discussion in relation to the Proximity Prediction Hypothesis

These behavioural data align closely with key predictions of the PPH, which views ASMR as a vagal cascade triggered by a predicted social-touch event.

2.5.3.1 Valence dominance

ASMR moments are defined by a large hedonic boost and only a minor arousal increase, this dissociation is exactly what would be expected if a slow-stroking CT-touch prediction drives the cascade while sympathetic output is actively suppressed, as the PPH predicts.

2.5.3.2 ASMR experience

The logistic mixed-effects model revealed that pleasantness significantly predicted whether a trial was classified as ASMR, whereas arousal did not. The interaction between pleasantness and arousal was not significant, and model fit was not improved by its inclusion. These findings suggest that the ASMR classification decision relies primarily on the perceived reward value of the sound, a direct prediction of the PPH, and is largely unaffected by concurrent arousal levels.

2.5.3.3 Intensity gradient

Within trials when ASMR was reportedly experienced in response to the sound, the intensity of tingles increased with pleasantness. Furthermore, an interaction emerged where pleasantness was an even stronger predictor of tingle strength when arousal was high. Under the PPH, this fits the notion that tingle intensity is a graded posterior-insula simulation of predicted touch value, with arousal acting as a gain control mechanism. That is, when arousal is elevated, the system may amplify the hedonic signal, but only when that signal is already strong. These data align with recent behavioural evidence from Gillmeister et al. (2022), who found that tingle intensity during ASMR closely tracked pleasantness and was further amplified by interpersonal touch. This could suggest that affective valence is central

to the ASMR simulation, while arousal may act as a gain control mechanism, steepening the link between high pleasantness cues and tingling under certain conditions.

2.5.3.4 Conclusion

Taken together, the data suggest that hedonic valence is the primary driver of both ASMR occurrence and intensity. Arousal shows a more equivocal role, sometimes enhancing the pleasantness-tingle gradient, but otherwise exerting weak or inconsistent effects. This ambiguity fits with the PPH view that arousal may reflect both proximity-based alerting and vagally mediated suppression, depending on the listener and context. Nevertheless, the data must be interpreted cautiously. First, the retrospective survey design may not capture tingles with the precision of real-time reports. Second, the undergraduate sample limits generalisability, and the exclusion of mouth sounds reduces ecological validity. Finally, while EEG data were collected in the broader study, no neural analyses are reported here; instead, this behavioural dataset is intended to provide illustrative, hypothesis-testing support for the PPH, with complementary EEG and MEG findings by the authors to be addressed in an upcoming study. The remaining discussion in this paper will cover the clinical applications and other proposed future tests of the PPH.

2.6 Current EEG evidence in relation to the PPH

While the present paper focuses on illustrative behavioural data, as the PPH is a predictive coding account that posits specific cortical dynamics, it is also important to situate it within the context of existing EEG findings on ASMR. Although the literature is still limited and heterogeneous in methods, several converging results speak to the neural plausibility of the PPH cascade, and can be assessed with regards to conventional frequency bands: alpha \approx 8–12 Hz; sensorimotor rhythm, SMR \approx 12–15 Hz; beta \approx 15–30 Hz; gamma \geq 30 Hz (Schomer and Lopes da Silva, 2018). The PPH anticipates that when a near-ear cue engages peripersonal space and the system begins to simulate CT-optimal touch in posterior insula and secondary somatosensory cortex, beta activity over somatosensory/posterior insular regions would decrease as an index of active sensory processing. If the prediction is then accepted and integrated as a result of the individual's priors, a rise in gamma band power may reflect precision-weighted updating of the interoceptive state. As the system settles into parasympathetic calm, alpha and SMR could

increase, consistent with sensorimotor quieting and relaxed alertness. Transient alpha reductions at the outset would also be compatible with early sensory analysis, so an assessment of the response's temporal profile is critical for future research.

Viewed through this lens, the heterogeneous EEG literature becomes more interpretable without being committed to a single outcome. Several reports align with the hypothesised updating-and-settling biphasic PPH cascade. Fredborg et al. (2021) found increases in alpha, gamma, and SMR in ASMR experiencers relative to non-experiencers during auditory triggers, patterns that could reflect precision updating (gamma) followed by sensorimotor quieting (alpha/SMR). Lee et al. (2020) similarly observed increases in SMR, alpha, and gamma for ASMR compared with binaural beats, suggesting a shift beyond simple drowsiness. Ohta and Inagaki (2021) reported that when cognitive load suppressed alpha and elevated high-beta/gamma, exposure to ASMR stimuli moved alpha and gamma/high-beta back toward resting levels, which may indicate re-balancing and parasympathetic calm once the prediction is accepted.

Other findings appear more consistent with earlier stages of processing. Engelbregt et al. (2022) reported reductions in alpha and theta with elevated beta, including alpha decreases over temporal-parietal sites, which could reflect early sensory analysis and the initial orienting phase in posterior temporal-parietal regions involved in audio-tactile integration. Seifzadeh et al. (2021) likewise observed alpha reductions during ASMR video viewing, again consistent with an initial engagement phase when first alerting to the stimuli, rather than the later parasympathetic settling phase. Regionally, Koo et al. (2021) showed that ASMR and control videos diverge in gamma modulation over occipital and central sites, compatible with cross-modal recruitment of sensory networks. Pedrini et al. (2021) identified distinct spectral signatures across baseline, relaxed, and ASMR states, implying large-scale network shifts that might be expected when the system transitions from orienting into affiliative calm.

A recent EEG study by Swart et al. (2022) provides an important complementary perspective on ASMR-related oscillatory dynamics. Using both sensor and source level analyses, the authors reported increases in low- to mid-frequency power (particularly alpha) alongside reductions in higher frequency activity (beta/gamma) during self-reported ASMR states relative to baseline and relaxed conditions. They further observed that high frequency reductions persisted into post-ASMR periods, accompanied by sustained alpha enhancement, which they interpret as reflecting a prolonged relaxation response. These findings appear to contrast with reports of increased gamma activity during ASMR (e.g., Fredborg et al., 2021; Lee et al., 2020). However, an important distinction lies in the

temporal framing of the analyses. Whereas Swart et al. (2022) emphasise sustained state changes associated with relaxation and post-ASMR decay, other studies may capture earlier or transient stages of the response. Within the PPH framework, this divergence may reflect different phases of the proposed cascade, with early prediction-related updating potentially involving gamma enhancement, followed by a later shift toward lower-frequency dominance as the system settles into parasympathetic calm. As such, variability across studies may reflect differences in temporal resolution, analysis windows, and the extent to which ASMR onset is time-locked, rather than fundamentally incompatible neural mechanisms.

Taken together, current EEG findings do not yet provide a single, time-resolved demonstration of the full sequence of events involved in the proposed PPH cascade. Nevertheless, recurrent reports of gamma and alpha changes, and posterior temporal-parietal involvement are compatible with key stages suggested by the PPH model. A decisive test now calls for time-locking analyses to reported tingle onsets, source-localised EEG or MEG focusing on posterior insula/OP1-S2 and posterior STS regions, and concurrent autonomic indices such as measures of high-frequency HRV and pupil diameter. If the PPH is a plausible explanation for the mechanism behind the ASMR phenomenon, future work should observe a stepwise pattern in which early beta reductions in somatosensory/post-insular regions are followed by a gamma increase in the posterior insula and, subsequently, an alpha/SMR up-shift consistent with sensorimotor quieting, with the magnitude of these changes covarying with vagal markers. Critically, this sequence is falsifiable: a failure to observe the predicted timing, regional specificity, or coupling with autonomic measures would argue against the PPH account. Suggestions for this kind of future research are discussed in Section 7.4 of this paper.

2.7 Integrating PPH with CNS-ANS communication and clinical angles

The Proximity Prediction Hypothesis (PPH) describes how a near-ear sound can initiate a cascade that ends in tingles and calm. The present section places that mechanism within wider brain–body communication processes, details the existing physiological clues that suggest the proposed PPH chain is real, and explains why the same mechanism could become a cheaper, accessible alternative to treatments like vagus nerve stimulation (VNS), particularly valuable for anxious and autistic populations as well as those suffering from sleep issues. VNS refers to the implanted, pulse generator therapy in which electrodes are wrapped around the cervical vagus to deliver periodic electrical bursts, a treatment approved for drug-resistant epilepsy and difficult to treat depression already.

2.7.1 Parallels with transcutaneous auricular vagus nerve stimulation and its clinical benefits

Electrical transcutaneous auricular vagus nerve stimulation (taVNS) is a wearable version of implanted VNS. Instead of placing electrodes on the cervical vagus, two small clip electrodes are positioned on the cymba conchae, this is the upper hollow of the outer ear where the auricular branch of the vagus nerve (ABVN) terminates in the skin. A battery-powered stimulator then delivers painless, low frequency pulses (typically 25 Hz, 200–300 μ s) for about 15 min. Because the ABVN projects directly to the nucleus tractus solitarius (NTS) in the brainstem, the current accesses central vagal pathways without passing through major muscle or bone tissue, unlike cervical VNS. From the NTS the signal ascends to the LC and parabrachial complex and descends to the dorsal motor nucleus of the vagus (DMV), shifting the LC to DMV balance toward parasympathetic dominance. The immediate physiological signature (heart rate deceleration and a rise in high frequency HRV) has been reported to appear within five minutes of stimulation (Borges et al., 2021) and mirrors the pattern ASMR listeners report during tingles.

The current model proposes that taVNS offers a useful clinical precedent for what the PPH suggests ASMR may achieve through sensory prediction. However, important constraints must be acknowledged. Unlike taVNS, ASMR cannot guarantee stimulation of the auricular branch of the vagus, and responsiveness to ASMR varies considerably across individuals. Thus, while the analogy might be compelling, its translational potential should be understood as conditional on ASMR susceptibility.

Controlled trials have demonstrated that nightly sessions of taVNS significantly enhance sleep quality, reduce insomnia severity, and increase total sleep duration in individuals with chronic insomnia (Zhang et al., 2024; Wu et al., 2022). Similarly, heart rate deceleration of 3–5 bpm and a 5–8% HF-HRV gain has been found during reported ASMR tingling episodes (Poerio et al., 2018) and around 80% of habitual listeners use ASMR to fall asleep (Barratt and Davis, 2015).

Recent studies have demonstrated that brief taVNS courses translate the vagal tone shift into clinically meaningful anxiety relief. In a double-blind, randomised controlled trial, Ferreira et al. (2024) found that a brief taVNS protocol significantly reduced anxiety symptoms in university students, as measured by the Beck Anxiety Inventory, with effects persisting up to 2 weeks after stimulation. A recent randomised clinical trial by Zhang et al. (2024) found that 8 weeks of taVNS significantly reduced anxiety and depression symptoms, as measured by the Hamilton Anxiety Scale (HAMA) and Hamilton Depression Scale (HAMD) alongside

significantly improved Pittsburgh Sleep Quality Index (PSQI) scores. These studies confirm that taVNS can pivot the LC-DMV axis from sympathetic vigilance toward parasympathetic calm and that standard clinical measures of sleep, anxiety, and depression offer realistic indices of this shift. Eid et al. (2022) found that ASMR-experiencers, who began with higher baseline state anxiety, experienced a significant reduction in State–Trait Anxiety Inventory–State subscale (STAI-S) scores after viewing an ASMR video, while non-experiencers did not.

Taken together, both taVNS and ASMR appear to converge on a common LC-DMV pathway, but by different routes: taVNS through exogenous current, and ASMR through a sensory prior that “pleasant CT-touch is imminent”. The PPH therefore predicts that the magnitude of an individual’s ASMR-induced HF-HRV burst should correlate with sleep and anxiety improvements, but such effects will depend on whether the person is an ASMR responder.

Future work can evaluate this prediction with single night polysomnography and standard anxiety inventories such as STAI-S. Currently, taVNS is being trialled as an intervention for treatment-resistant depression, PTSD and insomnia, yet it requires specialised hardware and clinical monitoring. ASMR could offer a headphone based, low cost, surrogate for taVNS, potentially expanding vagal tone interventions to populations who lack access to medical hardware, if listeners do experience meaningful levels of ASMR from the chosen stimuli, potentially providing similar clinical benefits that anyone with headphones could utilise.

2.7.2 Why anxious and autistic listeners might benefit most from ASMR-based interventions

A growing evidence base confirms that listeners do not seek out ASMR videos merely for curiosity or entertainment but because the experience delivers measurable relief from anxiety and sleeplessness, ASMR is therefore ripe with potential clinical applications.

How can the predictive coding mechanism underpinning PPH further hone ASMR’s clinical applications to specific populations? Both anxiety disorders and autism spectrum conditions are thought to be characterised by fundamentally over-precise interoceptive prediction errors (Paulus and Stein, 2006; Pellicano and Burr, 2012). In functional terms the insula “cries wolf,” keeping LC tone elevated and vagal tone low. A parallel Bayesian account proposes that autistic brains under-weight priors and over-weight sensory evidence, forcing even mundane events to register as surprising and arousal-worthy (Pellicano and Burr, 2012;

Lawson et al., 2014). Both scenarios keep the insula-LC loop chronically engaged. From this perspective, PPH generates the hypothesis that individuals with higher baseline LC tone (such as those with anxiety or autistic traits) may show a larger dynamic range for LC suppression during ASMR, and thus greater HF-HRV gains and stronger subjective relief. This remains to be tested; future studies could compare autonomic responses and symptom reductions in anxious, autistic, and neurotypical groups during ASMR exposure, using metrics such as HF-HRV, pupil dynamics, and validated anxiety scales.

Reported experiencers of ASMR have been shown to score higher on neuroticism and anxiety than non-experiencers, suggesting they may have more to gain from a parasympathetic tilt in general (McErlean and Banissy, 2017). Supporting this, Poerio et al. (2022) demonstrated that ASMR experiencers exhibit heightened sensory sensitivity across multiple modalities, including increased bodily awareness and interoceptive sensitivity. Autistic individuals often display atypical interoception; atypical emotional clarity, alexithymia, and interoceptive confusion (Bonete et al., 2023). Sensory processing in autism is also frequently atypical, with both hyper- and hypo-responsiveness across modalities (Elwin et al., 2013), possibly accompanied by somatosensory amplification. Interestingly, some autistic adults report heightened bodily awareness despite reduced interoceptive accuracy, indicating a mismatch between subjective and objective interoceptive states (Garfinkel et al., 2016). This convergence suggests that ASMR may be especially impactful for individuals with enhanced sensory and emotional responsiveness, although responses will likely vary depending on how the social and affiliative meaning of ASMR stimuli is interpreted. For some autistic individuals, the social cues embedded in whispers or gaze may not carry the same affiliative value, which could reduce ASMR efficacy. Future studies should therefore stratify participants by both sensory sensitivity and social priors.

While ASMR proneness also correlates with trait empathic concern (McErlean and Banissy, 2017), this does not preclude its relevance for autistic individuals, who may differ in “cognitive empathy”, i.e., imagining another person’s mental state, but not necessarily “affective empathy”, the capacity to emotionally resonate with affiliative or caring cues (Dziobek et al., 2008). These findings map onto the PPH cascade: a powerful, but non-intrusive, prior, silences insular error signals, drops LC tone, and brings the body into a parasympathetic state that many autistic and anxious individuals may otherwise struggle to access. If near-ear audio can normalise the LC-DMV balance in these populations, it may again serve as a low-cost alternative to taVNS, especially for children or adults who are needle-averse or have restricted access to neurostimulation clinics, but its clinical utility will depend on individual responsiveness and the interpretation of the sensory cues.

Evidence from tactile research reinforces the clinical logic. Even a single session of massage, can produce immediate reductions in state anxiety, along with decreases in blood pressure and heart rate (Moyer et al., 2004). Scalp massage specifically, in office workers, significantly reduced cortisol, blood pressure, heart rate, and self-reported stress (Kim et al., 2016), suggesting that even brief, localised tactile input can rapidly shift the autonomic balance toward parasympathetic dominance. Yet CT-touch is not always socially available or desired by people with heightened sensory sensitivities. ASMR supplies a predictive, contact-free analogue that can be self-administered with nothing more than headphones for people in anxious or autistic populations who may be otherwise touch-avoidant.

Crucially, autistic individuals often show altered tactile sensitivity and hedonic perception to stimuli targeting CT-innervated regions (Cascio et al., 2008), with neuroimaging further indicating that CT-evoked responses in social brain regions such as the orbitofrontal cortex and superior temporal sulcus are diminished in individuals with higher autistic traits (Voos et al., 2013), while EEG work shows that neural potentials to CT-targeted touch scale negatively with autistic trait load (Haggarty et al., 2020). Moreover, the coupling between subjective reports of pleasantness and central neural representations of touch has previously been found to be weaker in samples of adolescents with autism, suggesting a disconnect between afferent input and hedonic experience (Perini et al., 2021). Taken together, these findings imply that while CT afferents may be intact, their central processing and translation into pleasant affect is atypical in autism. This supports the novel possibility that auditory ASMR cues, which deliver the prediction of affiliative contact without relying on CT-fibre stimulation, could bypass these atypical responses and more effectively evoke pleasantness and parasympathetic calming. Although this remains to be tested directly, it highlights a potential route by which ASMR might provide sensory-affective benefits to autistic individuals even where CT-touch itself is less effective.

Notably, not all individuals with anxiety or autism may benefit equally from ASMR cues that mimic CT-optimal light stroking. For those with atypical CT processing, auditory ASMR may bypass tactile deficits and still evoke affiliative priors, as argued above. However, other evidence suggests that some anxious or autistic individuals instead find deep pressure touch more calming than light touch, with studies of weighted blankets and squeeze devices showing reductions in arousal, anxiety, and insomnia in certain responders (Grandin, 1992; Edelson et al., 1999; Ekholm et al., 2020; Fava et al., 2021). Within the PPH framework, this raises a distinct, testable hypothesis: for individuals less responsive to CT-mimetic ASMR, auditory cues that mimic the sensory qualities of deep pressure, such as low-frequency, steady, broadband sounds, may better initiate a vagal release and lead to parasympathetic

calm. This refinement does not imply that all ASMR works via multiple routes, but rather that individual differences in tactile preference may determine which acoustic simulations are likely to be effective given individual differences. Future work can therefore stratify participants by CT sensitivity and deep-pressure preference to identify which subgroups might benefit most from which classes of ASMR stimuli.

2.7.2.1 Exploratory clinical protocol

A logical next step is to evaluate ASMR in structured clinical trials using designs comparable to those employed in taVNS research. A preliminary protocol could involve nightly exposure to a curated ASMR playlist, delivered via headphones, for 15–20 min before sleep over a period of 4–8 weeks. These parameters deliberately mirror the taVNS insomnia trials mentioned earlier in this report, which used sessions of up to 30 min, within multi-week courses; preserving the pre-sleep timing and cumulative dosing window (Wu et al., 2022; Zhang et al., 2024). Participants would complete validated measures of anxiety (e.g., STAI-S), depression (HAMD), and sleep quality (PSQI), alongside autonomic monitoring (HF-HRV, pupillometry) in a subset of sessions. Long-term follow-up (e.g., 1–3 months later) could test the durability of effects using the same clinically meaningful measures, while stratifying participants by ASMR susceptibility would identify which subgroups (e.g., anxious, autistic, or neurotypical) derive the greatest benefit. Such a design would provide a concrete test of ASMR’s translational potential, clarifying both its efficacy and its boundary conditions.

2.7.3 Refining new CAN biomarkers

The PPH model lends itself to proposing two straightforward improvements to the resting HF-HRV score as a biomarker of CAN health, which dominates the current literature. Firstly, instead of looking at HRV in a long, resting baseline, how it changes from trial to trial could be observed while someone is listening to ASMR. A mixed-effects regression of HF-HRV gain and reported tingle intensity will provide a slope for each person. A steep positive slope should indicate that the person’s vagus nerve immediately answers the brain’s “this is pleasant and safe” signal; a flat slope means it does not. If the PPH model is correct, the individual differences in responsiveness are possibly more informative about anxiety risk or sleep quality for that individual than a single resting HF-HRV snapshot. Furthermore, if the PPH is supported, then combining biomarkers like beta band power decreases in the posterior insula (suggesting the system in PPH is predicting a gentle CT-touch is about to happen), along with HF-HRV gain, would provide a mechanistically coherent biomarker that directly indexes the hypothesised cascade from cortical prediction to autonomic change.

Such a multimodal index could predict who will report feeling less anxious or who might fall asleep faster, more reliably than either brain or heart signal could when taken on its own.

In short, the PPH model suggests that ASMR tingling could be used as a convenient stress-test of the CAN loop across individuals, one that can be quantified in real time and may add to the current diagnostic toolkit for anxiety, insomnia, and related conditions. Further experimental paradigms that could be used to test the PPH model are discussed in the next section.

2.8 Future tests of the PPH model

The PPH makes concrete, falsifiable claims about where in the sensory chain the ASMR cascade begins and how it propagates through the insula-LC-vagus axis. Below, a series of experimental predictions, ranging from psychophysics to source-localised MEG, are outlined, to suggest what results would support these claims in future research.

2.8.1 Distance manipulation predictions

2.8.1.1 Binaural morphing of approach cues

The PPH model suggests that the ASMR cascade is gated by perceived proximity, such that a continuous morphing of binaural cues from far (>1 m) to near field (<30 cm) should show a non-linear inflection point in ASMR reports, with tingle likelihood, pleasantness, and vagal markers (e.g., HF-HRV gain) rising sharply as the sound enters the peri-aural space. This would reflect the transition into the brain's peripersonal comfort zone, aligning with prior PPS boundaries observed in audio-tactile studies (Ferri et al., 2015; Serino et al., 2009). Given that pupil dilation has been observed during ASMR listening, likely reflecting heightened attentional engagement with the sound, the PPH further predicts that a delayed pupil constriction should follow as parasympathetic dominance increases during the latter stages of the response. This later-phase constriction has not yet been empirically tested but would be expected if the LC-vagus balance shifts toward sustained calm. This could be tested using interaural time/level difference manipulations of a typically ASMR-inducing stimulus, such as "realistic haircut sounds" (Schürmann et al., 2006).

2.8.1.2 Disrupting spatial coherence across ears

If spatial proximity is integrated across both ears to determine whether the stimulus is near or far, then presenting conflicting distance cues across ears (e.g., one ear hears a close

whisper; the other a far-filtered version) should reduce ASMR responses and vagal activity, relative to conditions with coherent near-field input in both ears. This would support the view that the brain uses spatial coherence as a gating signal for engaging the insula-LC-vagus cascade and disrupting it should reduce the probability of experiencing tingles.

2.8.2 Combining real CT-touch with near-ear audio predictions

Recent findings by Gillmeister et al. (2022) indicate that ASMR responders not only exhibit a higher incidence of mirror-touch synaesthesia but also report greater positive emotional reactions to social touch, especially those with stronger ASMR traits. While this supports the notion that affective touch and ASMR share common hedonic mechanisms, the next step is to test whether these effects reflect underlying prediction-based neural dynamics. If the PPH is correct, combining real CT-touch with auditory cues should produce distinct physiological and neurophysiological signatures that reflect audio-tactile congruence and temporal precision.

2.8.3 Audio-tactile congruence

Stroking of the listener's scalp at CT-optimal velocity (3 cm s^{-1}) while presenting either a near-ear brushing sound (congruent) or an identical sound filtered to far-space (incongruent) should boost posterior-insula β -ERD and HF-HRV if the PPH is to be supported. In contrast, incongruence between auditory and tactile stimuli should dilute both markers, because the prediction error becomes more precise when the auditory prior and tactile evidence disagree (Ellingsen et al., 2016).

2.8.4 Expectation modulation and proximity cue predictions

Ellingsen et al. (2013) devised an elegant "placebo-hedonia" protocol; an inert nasal spray presented as a "pleasure enhancer", followed by slow brush strokes on the forearm during fMRI. The placebo increased subjective pleasantness ratings by around 25%, with enhanced BOLD activity in S1, S2, and the posterior insula, and elevated functional coupling between the pregenual ACC (pgACC) and periaqueductal gray areas, supporting a top-down prediction-based modulation of somatosensory gain. In predictive coding terms, the positive label increased the precision of the "this will feel good" prior, allowing top-down signals to dominate and turn up the gain on the incoming CT volley.

Building on Ellingsen's finding that positive expectancy amplifies CT-touch processing, if the PPH is correct in asserting that tingle cascades result from precision-weighted predictions of CT-optimal touch, then positively framing a binaural track (e.g., labelling it as a "clinically

validated tingle inducer”) should increase posterior-insula β -band desynchronization, enhance vagal tone (HF-HRV), lead to a constriction in tonic pupil diameter, and raise subjective ratings of tingle intensity and pleasantness, provided the track contains proximal, near-ear spatial cues. Furthermore, if spatial proximity is a prerequisite for CT-touch predictions, then far-filtered versions of the same track should fail to elicit ASMR responses, even under positive expectancy conditions. That is, labelling alone will not boost tingles or parasympathetic markers when the sensory input lacks coherent proximity information. This prediction sharply distinguishes the PPH from a purely cognitive account: both sensory proximity and cognitive framing must converge to silence prediction error and initiate ASMR.

2.9 Predicted EEG and MEG signatures of the PPH cascade

If the PPH is correct, ASMR should elicit a specific neural-autonomic sequence reflecting affective touch simulation and vagal modulation. Empirically, EEG studies show that ASMR triggers produce increased alpha, gamma, and modulations in sensorimotor rhythms (Fredborg et al., 2021) and reduced theta coupled with elevated beta (Engelbregt et al., 2022), along with immediate pupil dilation (that the PPH model would suggest relates to the proposed initial orienting stage) during strong ASMR episodes (Pedrini et al., 2021). Building on this, PPH predicts a time-locked cascade in the EEG: an initial beta-band suppression over centroparietal sites reflecting S2/posterior-insula activation for CT touch, followed by a transient gamma enhancement indexing precision-weighted updating. Later increases in beta reported in some studies may correspond to regulatory or arousal-related processes rather than the initial sensory stage. Time-resolved EEG and source-localised MEG are therefore crucial to test whether early beta decreases and later gamma increases can be distinguished in real ASMR episodes. MEG, with better spatial resolution, should localise this beta-gamma sequence to the posterior insula and OP1/S2, with earlier beta suppression in pSTS marking peripersonal space detection, and elevated beta-band coherence between the posterior insula and pgACC/vmPFC regions during the tingling window (reflecting precision-weighted prediction). Crucially, stronger posterior-insula beta suppression should correlate with larger increases in high frequency heart rate variability (HF-HRV), supporting the proposed insula-LC-DMV coupling underlying vagal gain in ASMR. These neural-autonomic patterns should be absent or markedly reduced in control trials without reported tingles, or when identical stimuli are presented with far-field spatial filtering, providing a decisive test of PPH.

It is important to note that not all EEG findings align with this predicted beta-gamma profile. For example, Swart et al. (2022) reported reductions in high-frequency activity alongside increases in alpha during ASMR states, consistent with a relaxation-dominant interpretation.

Within the PPH framework, such findings may reflect later stages of the response or sustained post-tingling states, rather than the initial prediction and updating processes that are the primary focus of the present hypotheses.

If this mechanism is supported in future work, ASMR videos could evolve from quirky bedtime rituals into evidence-based, widely accessible therapeutic interventions for anxiety reduction and sleep promotion. This is especially salient for populations such as those with autism, where prediction error is chronically elevated and conventional relaxation techniques often fail. Moreover, the proposed neural-autonomic markers of beta suppression in the posterior insula, HF-HRV gain, and pupil constriction, could serve as future biomarkers for personalised treatment selection and efficacy tracking. Each paradigm offered in the future research section isolates a different link in the proposed chain; proximity detection, CT-touch prediction, LC suppression, and vagal release. Convergent success across distance manipulation, expectancy modulation, and longitudinal outcome trials would transform PPH from a heuristic into a mechanistically validated account of ASMR and, by extension, into a blueprint for audio-based vagal therapies in mental health. By explicitly integrating predictive coding principles with the neurophysiology of interoception, PPH also offers a broader contribution to our understanding of how the brain regulates the body in response to socially salient sensory cues.

2.10 Conclusion

The Proximity Prediction Hypothesis does more than explain an unusual, pleasant tingling sensation; it places the ASMR phenomenon within the LC-vagus system that modern affective neuroscience regards as influential to various physiological and neurological functions, including emotional regulation, stress responses, and even cognitive abilities. Near-ear sounds appear capable of fooling the brain, leading to emotional modulation, where a sensory cue suppresses the noradrenergic accelerator (the LC), allowing disinhibition of the vagal brake, and ushers both the brain and body into a restful state. The PPH therefore provides a predictive-coding framework specifying when and how the plausible neural routes proposed by previous structural accounts, such as McGeoch and Rouw's (2020) cross-activation model, might be engaged, and how the characteristic tingling experience could be generated as a result.

The illustrative data reported here offer behavioural support for this framework. Across trials, ASMR experiences were strongly predicted by hedonic valence (pleasantness), not by

physiological arousal, and tingle intensity scaled with pleasantness, in a manner that was modestly amplified by arousal. These patterns are consistent with the PPH account of ASMR as a reward-based simulation of safe affective proximity, rather than a state of heightened energetic activation.

In summary, the Proximity Prediction Hypothesis situates ASMR within predictive coding accounts of interoception, offering a mechanistic framework that links acoustic cues, tingling sensations, and parasympathetic calming. The behavioural data reported here are consistent with this account, though they remain preliminary. Rather than providing definitive empirical validation, the present study illustrates how PPH can integrate existing autonomic and behavioural findings into a coherent model, and points toward future work needed to directly test its neural predictions.

Chapter 3:

The Predictive Neural Oscillations behind Autonomous Sensory Meridian Response: A combined EEG-MEG study.

3.1 Abstract

Autonomous Sensory Meridian Response (ASMR) is a sensory-affective phenomenon in which specific auditory stimuli elicit pleasant tingling sensations, felt across the scalp and neck, and widely reported subsequent therapeutic benefits to anxiety, sleep disturbance, and even chronic pain, from listening alone. Despite its burgeoning global popularity, ASMR's neural mechanism remains relatively underexplored. The oscillatory predictions of the Proximity Prediction Hypothesis (PPH), were tested in this cross-modal EEG and MEG investigation (N = 64 in the EEG session, and a partially overlapping N = 30 in the MEG session). The PPH posits that in ASMR near-field auditory cues associated with social priors of affective touch recruit predictive coding mechanisms that result in somatosensory simulations manifesting as a tingling response, and subsequent parasympathetic calm. Participants listened to the same 18 audio clips (13 experimental stimuli meant to induce ASMR, and 5 naturalistic controls) in both experiments and indicated by button press if they experienced ASMR tingling in response to any of the stimuli. Oscillatory activity was examined using FOOOF-based spectral parameterisation and time-frequency analysis, with nonparametric cluster-based permutation tests. In the EEG experiment, trials in which participants specifically reported tingling sensations were marked by both significant beta desynchronisation and gamma enhancement, relative to control trials. This oscillatory profile was mirrored in MEG sensor-space data, which revealed robust beta reductions and widespread gamma-band enhancement for ASMR stimuli relative to controls. These convergent findings support the PPH prediction that ASMR engages predictive mechanisms in sensorimotor and multisensory networks. Specifically, beta desynchronisation reflects the activation of top-down predictions of affiliative touch, while gamma-band enhancement signals cross-modal prediction errors when expected tactile input fails to arrive. The resulting somatosensory echo, felt as the tingling percept, may emerge as the brain's resolution for this sensory mismatch, providing novel evidence for hierarchical predictive coding in ASMR.

3.2 Introduction

Autonomous Sensory Meridian Response (ASMR) is a sensory-affective phenomenon in which specific auditory or audiovisual cues, such as whispering, soft speaking, brushing, or tapping sounds, elicit pleasant tingling sensations in listeners, often beginning on the scalp and spreading down the neck and spine (Barratt & Davis, 2015; Fredborg et al., 2021). Millions of individuals worldwide report using ASMR media to relax, fall asleep, and manage anxiety or chronic pain; over 80% of individuals who seek out ASMR have reported doing so to facilitate sleep, and around 70% for stress reduction (Barratt & Davis, 2015). These subjective reports are supported by physiological markers of parasympathetic activation, such as heart rate deceleration and elevated high-frequency heart rate variability during ASMR (Poerio et al., 2018), suggesting that ASMR may offer a promising candidate for therapeutic applications in anxiety and sleep disorders.

Despite its popularity and potential translational relevance, the neural mechanisms underlying ASMR remain elusive. Several EEG studies have begun to characterise its oscillatory dynamics, revealing altered power in multiple frequency bands. Some findings report increased alpha and gamma power during ASMR (Fredborg et al., 2021), whereas others note reductions in alpha and theta alongside increases in beta (Engelbregt et al., 2022; Seifzadeh et al., 2021). This variability may reflect different phases or intensities of the ASMR experience, ranging from orienting to sensory settling.

To address this ambiguity, the recently proposed Proximity Prediction Hypothesis (PPH; Flockton et al., 2025) offers a mechanistic account of ASMR. The PPH posits that near-field auditory cues, such as whispering or tapping sounds which appear to be stemming from a source in close proximity to the listener, activate the brain's Peripersonal Space network and lead to predictions of affective touch, via top-down predictive mechanisms. These predictions are proposed to recruit interoceptive and somatosensory regions (e.g., the posterior insula and secondary somatosensory cortex), resulting in a 'somatosensory echo', felt across the scalp, in the absence of actual physical contact. This somatosensory echo reflects the brain's internal generation of a socially-relevant tactile state based on probabilistic sensory inference. The PPH model proposes a biphasic neural response: (1) early beta-band desynchronisation reflecting sensory disinhibition and tactile prediction, and (2) gamma-band enhancement reflecting precision-weighted multisensory integration.

EEG and MEG studies consistently show that beta-band desynchronisation, i.e., reductions in beta power, is a reliable neural signature of affective or socially meaningful touch. This has been particularly evident during C-tactile (CT) optimal touch, which refers to slow, gentle stroking at approximately 3 cm/s, known to activate unmyelinated CT afferents and induce pleasant emotional responses. For instance, von Mohr et al. (2018) applied CT-optimal versus non-optimal touch and found significant attenuation of alpha and beta power, especially over parietal sites, with greater beta suppression during affective (CT-optimal) touch. Importantly, even the observation of affective touch has been shown to produce similar neural effects; Pereira et al. (2025) reported significantly lower beta-band power when participants viewed videos of CT-optimal stroking compared to fast or static touch, and implicated the somatosensory cortex in the vicarious experience of social touch. These findings suggest that beta desynchronisation, often interpreted as reflecting sensorimotor disinhibition or increased cortical excitability, is sensitive not only to actual physical contact but also to the expectation or simulation of affective touch. This fits within a predictive coding framework, in which beta-band activity is thought to mediate top-down predictions from higher order brain areas (Bastos et al., 2012). Supporting this view, van Pelt et al. (2016) found that beta coherence increased during predictable versus unpredictable action sequences, consistent with beta rhythms encoding expected sensory outcomes. This aligns with the PPH, which posits that near-field auditory cues can evoke predicted touch states through expectation alone, leading to endogenous beta suppression in somatosensory regions like the posterior insula and somatosensory cortex - consistent with both empirical data and predictive coding models of sensory inference.

In contrast to beta suppression, gamma-band power (30-100 Hz) often increases during conscious multisensory integration. Numerous EEG studies show that gamma synchrony is enhanced when sensory cues are semantically congruent or temporally aligned, supporting the role of gamma oscillations in perceptual binding. For example, Schneider et al. (2008) used a cross-modal priming task and found early gamma-band increases (40-50 Hz) for congruent audiovisual pairings. Later, Schneider et al. (2011) extended these findings to touch-auditory interactions; when participants touched an object and then heard a matching or mismatching sound, gamma activity was significantly greater for semantically congruent touch-sound pairings, peaking around 250-350 ms post-stimulus. These effects are thought to reflect the brain's attempt to unify related sensory events into coherent percepts. Other studies reinforce the role of gamma synchrony in cross-modal coherence. Yuval-Greenberg and Deouell (2007) reported stronger induced gamma activity when auditory and visual inputs were congruent compared to incongruent, suggesting gamma supports semantic

integration across modalities. Likewise, Kanayama et al. (2009), in a visuo-tactile rubber hand illusion paradigm, showed that gamma-band synchrony in the parietal cortex scaled with the strength of the illusory percept, linking gamma to the conscious experience of bodily self-location through multisensory integration. Senkowski et al. showed a wide body of evidence that consistently links gamma oscillations to multisensory integration and feature binding, in their review paper (2007), particularly when stimuli converge spatially and temporally. Applied to ASMR, this suggests that whispering, hair brushing, or otherwise proximal auditory stimuli may simulate touch near the ear, and when this input is cross-modally congruent (i.e. sounds that would plausibly be elicited by forms of impending, affective touch) gamma synchrony may increase to support the binding of these auditory cues with their predicted tactile experiences. This gamma-band enhancement, therefore, could reflect the culmination of multisensory integration processes that give rise to the ASMR tingling sensation, consistent with the Proximity Prediction Hypothesis.

While gamma activity is often interpreted as a signature of successful multisensory binding, converging evidence also suggests that it may reflect prediction error signals when top-down expectations are violated. For instance, gamma-band power increases in response to omitted stimuli (Todorovic et al., 2011) or cross-modal mismatches (Arnal et al., 2011), suggesting that gamma synchrony may also index sensory surprise. This view complements traditional accounts by framing gamma as a marker of prediction evaluation, not just integration. Within ASMR, gamma enhancement may therefore emerge not only from cross-modal coherence, but also from the brain's attempt to reconcile predicted affective touch with its absence, a novel idea explored further in this study.

To properly test the neural dynamics predicted by the PPH, a method with high temporal resolution and minimal acoustic interference is required. Functional MRI, while spatially precise, is too temporally sluggish and acoustically noisy to accurately capture the neural dynamics elicited by ASMR triggers in real time. EEG and MEG, by contrast, provide millisecond-level temporal resolution and are well-suited to detecting the real-time oscillatory shifts involved in ASMR. MEG additionally offers improved spatial precision and reduced signal distortion from skull conductivity, making it a valuable complement to EEG.

The current study provides this cross-modal test, in a combined EEG and MEG investigation into ASMR. Participants listened to 18 auditory stimuli (13 sound clips meant to elicit ASMR and 5 naturalistic controls), while reporting tingling sensations they experienced in response to any of the sounds, via button press. Comparisons between control trials and all experimental ASMR stimuli trials will be made along with comparisons of control trials

compared against button-press tingling trials specifically. This refers to the ASMR stimuli trials that induced tingling sensations for each listener, as there are individual differences in trigger preferences and not all experimental stimuli designed to elicit ASMR would definitely induce ASMR for all listeners, so the button presses provided self-reports of which experimental ASMR stimuli actually did elicit the tingling sensation for each listener, on a trial by trial basis. This distinction is necessary because ASMR exhibits substantial individual variability in trigger selectivity, with different stimulus types eliciting tingles in different people (Poerio et al., 2018). Spectral parameterisation (FOOOF), with cluster-based permutation tests, will be used in a mirrored EEG-MEG analysis pipeline across both datasets. It is predicted that ASMR stimuli will evoke beta-band desynchronisation and gamma-band enhancement, consistent with the PPH's proposed mechanism involving predicting coding of affective touch. Because no prior MEG studies have examined ASMR, this cross-modal approach offers the first cross-modal characterisation of its sensor-level oscillatory dynamics using this method alongside EEG.

3.3 Methods

3.3.1 Participants

Sixty-four participants (38 female; mean age \pm SD = 22.7 \pm 4.1 years) completed the EEG experiment, and a partially overlapping sample (N = 24 participants also took part in the EEG study), of thirty participants (19 female; 23.4 \pm 4.8 years) took part in the MEG study. All participants reported normal hearing and no history of neurological or psychiatric conditions. Ethical approval was granted by the University of York Department of Psychology Ethics Committee and York Neuroimaging Centre Ethics Committee, and all participants provided written informed consent.

3.3.2 Experimental Design and Procedure

The experiment followed a within-subjects design in which participants listened to 18 auditory stimuli: 13 ASMR sounds and 5 Control sounds (Appendix C). Each clip lasted 30 seconds and was followed by a 5-second silent interval. To ensure comfort and immersion, participants lay in a supine position on a standard massage table in a sound-attenuated room. Auditory stimuli were presented via Sennheiser HD280 Pro padded dynamic stereo headphones connected to a Cambridge Research Systems AudioFile stimulus generator, which delivered the sounds with zero latency triggers to synchronise recording.

The 13 ASMR stimuli were created by a professional ASMR content creator (“ASMRtist”) and featured widely recognised ASMR triggers, including, hair brushing, and soft tapping (Nader, 2023). The creator provided explicit permission for the use of these sounds in this study. The 5 control stimuli were field recordings of ambient traffic noise (e.g., recorded from a moving bus). These control sounds were not formally matched to the ASMR stimuli on acoustic parameters (e.g., RMS amplitude or spectral profile), but were selected to be naturalistic, non-speech, and devoid of rhythmic, proximal, or affectively soothing features typical of ASMR triggers.

Participants were instructed to keep their eyes closed during each sound and to press and hold a response button whenever they experienced tingling sensations or a deep feeling of relaxation characteristic of ASMR. These time-locked button press periods enabled us to analyse both all-trial activity and tingling-specific windows of interest.

Stimuli were presented in pseudorandom order during a single continuous block in the EEG experiment. In the MEG experiment, the same set of stimuli was presented in pseudorandom order but distributed across three blocks to acquire more data, and allow for periodic head position tracking and breaks between runs.

3.3.3 EEG Data Acquisition and Preprocessing

EEG was recorded using a 64-channel ANT Neuro WaveGuard cap with electrodes arranged according to the international 10-20 system. Conductive gel was applied to each electrode to achieve impedance levels below 10 k Ω . Signals were referenced online to the common mode sense and sampled at 1,024 Hz. Data were processed using MNE-Python (v1.7). A 1- 48 Hz band-pass filter and a 50 Hz notch filter were applied. Electrooculogram (HEOG, VEOG) and mastoid (M1, M2) channels were excluded, to identify and remove blink and muscle artefacts, the standard 10-20 montage was applied (with `on_missing='ignore'`), and signals were re-referenced to the common average. Continuous data were epoched from -2 to 30 s relative to stimulus onset. The -2 to 0 s period served as a pre-stimulus inspection window but was not used for baseline correction, in line with frequency-domain analyses. Trials exceeding ± 100 μ V were rejected. Event codes 1 - 13 indexed ASMR stimuli and 14 - 18 indexed Control stimuli.

3.3.4 MEG Data Acquisition and Preprocessing

MEG recordings were acquired in a magnetically shielded room using a 4D Neuroimaging Magnes 3600 whole-head system (248 axial magnetometers and 23 reference sensors). Head position was digitised prior to each of the three stimulus blocks using a Polhemus

FASTRAK system. Data were sampled at 678 Hz. Preprocessing included static signal-space separation (Maxwell filtering; correlation limit = 0.98), followed by a 1- 48 Hz band-pass filter and notch filters at 50 and 100 Hz. Data were epoched from - 2 to 30 s relative to stimulus onset, with the - 2 to 0 s baseline period marked but not subtracted.

3.3.5 Spectral Parameterisation Analysis

To examine oscillatory activity in EEG and MEG sensor space, power spectral densities (PSDs) were computed using Welch's method (2s Hanning windows, 50% overlap) across the 1- 48 Hz range. For each sensor and condition, average PSDs were parameterised using the FOOOF algorithm (specparam implementation), which separates the aperiodic 1/f-like background from superimposed periodic (oscillatory) peaks.

Model parameters were: `peak_width_limits = [0.25, 6]`, `max_n_peaks = 6`, and `aperiodic_mode = 'fixed'`. Aperiodic-adjusted peak amplitudes were extracted for canonical frequency bands: theta (4- 8 Hz), alpha (8- 12 Hz), beta (15- 30 Hz), and gamma (30- 48 Hz).

Group-level comparisons between ASMR and Control conditions were performed separately for EEG and MEG data using cluster-based permutation testing (Maris & Oostenveld, 2007) with 2,000 permutations and a two-tailed alpha threshold of 0.05. For MEG, cluster formation used spatial adjacency based on magnetometer sensor layout; p-values were Bonferroni-corrected across frequency bands to control family-wise error.

For each band, topographic maps illustrated the mean difference in peak amplitude (ASMR - Control), with statistically significant clusters highlighted. The MEG analysis included only magnetometer channels.

3.3.5.1 EEG Button Press Analysis

In addition to full-trial comparisons, a separate EEG analysis examined neural activity time-locked to the subjective onset of ASMR tingling, as indexed by button-press events. Due to technical limitations with MEG button-press recordings, this analysis was restricted to the EEG dataset.

Button-press onset times were extracted from Biopac logs and aligned to EEG event markers corresponding to ASMR stimuli (codes 1- 13). EEG data were epoched from -2 s to +6 s relative to each button-press onset. The -2 to 0s period reflects neural activity preceding the reported onset of tingling, though the sensory experience itself may have begun earlier. This window therefore captures anticipatory and early perceptual processes

leading up to the decision to press the button, rather than the absolute onset of tingling. All valid press onset epochs were averaged within participants to create an ASMR tingle condition, which was compared to duration matched Control epochs from non-ASMR trials.

Power spectral densities (PSDs; 1- 48 Hz, Welch's method, 2 s Hanning windows, 50 % overlap) were parameterised with the FOOOF algorithm (peak_width_limits = [0.25, 6]; max_n_peaks = 6) to separate periodic and aperiodic components. Aperiodic-adjusted peak amplitudes were compared between conditions using cluster-based permutation testing (1,000 permutations, two-tailed $\alpha = 0.05$) with spatial adjacency defined across EEG sensors. Bonferroni correction across frequency bands (θ , α , β , γ) controlled for multiple comparisons.

3.3.6 EEG Time-Frequency Decomposition

To examine the temporal evolution of oscillatory activity during ASMR stimuli trials, EEG data were subjected to time-frequency decomposition using complex Morlet wavelets. Frequencies ranged from 2 to 48 Hz, with a constant cycle length of five cycles across frequencies and a temporal resolution of 50 ms steps. Power estimates were baseline-corrected by converting to decibel (dB) change relative to the - 2 to 0 s pre-stimulus interval. Time-frequency representations (TFRs) were averaged across trials for each participant and condition. Grand-average TFRs were computed for ASMR and Control stimuli. Exploratory cluster-based permutation tests (2,000 permutations, two-tailed $\alpha = 0.05$) were applied across time, frequency, and sensors to identify significant temporal- spectral differences between conditions.

3.3.7 EEG Event-Related Potential Analysis (Button-Press Onset)

In a complementary analysis, event-related potentials (ERPs) were computed to examine time-locked cortical dynamics surrounding the subjective onset of ASMR sensations. Button-press events were recorded whenever participants reported tingling sensations during ASMR trials. For each participant, EEG data were epoched from -1 s to +1 s relative to the onset of each button press (time = 0 s).

This temporal window does not assume that tingling begins precisely at the moment of the button press. Rather, the button press is interpreted as the point at which participants become consciously aware of, or decide to report, the sensation. The pre-press interval (-1 to 0 s) therefore captures neural activity leading up to this report, which may include perceptual, interoceptive, and decision-related processes, as well as motor preparation. This approach is consistent with previous work examining neural activity preceding subjective awareness in perceptual and interoceptive paradigms (e.g., Del Cul et al., 2007; Hauck et al., 2007; Park & Blanke, 2019).

Continuous EEG data were preprocessed prior to epoching. Data were re-referenced to the average reference, and non-scalp channels (HEOG, VEOG, M1, M2) were removed where present. A standard 10-20 montage was applied. Line noise was attenuated using a 50 Hz notch filter, followed by band-pass filtering between 0.1 and 30 Hz using a zero-phase finite impulse response (FIR) filter.

Button-press onset times were extracted from externally recorded CSV files and aligned to the EEG recordings. Where necessary, timing values were converted to seconds based on recording duration. Only events that permitted the full epoch window (-1 to +1 s) within the continuous recording were retained.

Epochs were baseline-corrected using the pre-press interval (-0.5 to 0 s). Linear detrending was applied, and epochs containing large artefacts were rejected using an amplitude threshold of $\pm 150 \mu\text{V}$. Additionally, epochs overlapping with annotated bad segments were excluded.

For each participant, ERPs were computed by averaging across all valid button-press epochs. Grand-average ERPs were then calculated across all included participants. The resulting grand-average waveform was visualised using MNE-Python's `plot_joint` function, displaying both voltage-over-time traces and scalp topographies at representative time points (-300 ms, 0 ms, +200 ms, and +500 ms). Additional scalp topography sequences and butterfly plots were generated to characterise the spatial and temporal structure of the response.

The final number of participants included in the grand average ($n = 46$) reflects those with sufficient valid epochs after artefact rejection.

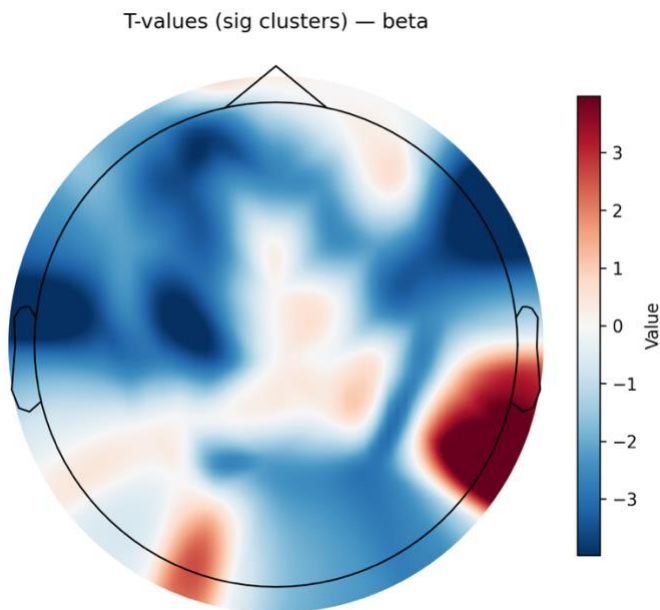
3.4 EEG Results

3.4.1 EEG Spectral Parameterisation: ASMR vs Control

Cluster-based permutation analysis on FOOOF-derived oscillatory peak amplitudes revealed a significant negative cluster in the beta-band (15-30 Hz), indicating lower beta power during ASMR relative to Control trials ($p < .05$, Bonferroni-corrected). This beta desynchronisation cluster was most pronounced over right-central and parietal electrodes (Figure 3.1), consistent with decreased sensorimotor synchrony during ASMR listening. No significant clusters were observed in the theta (4-8 Hz), alpha (8-12 Hz), or gamma (30-48 Hz) bands.

Figure 3.1

Topographic distribution of significant t-values for the ASMR - Control contrast in the beta band (15-30 Hz), based on FOOOF-derived peak amplitudes.



Note: Beta-band cluster-corrected T-map for the contrast ASMR – Control.

Values are masked to significant spatial clusters obtained via cluster-based permutation testing ($p < .05$). Colour intensity reflects the magnitude of the T-statistic within electrodes belonging to the significant spatial cluster identified by cluster-based permutation testing. Warmer colours indicate larger T-values within the significant cluster. The colours therefore only reflect the distribution of significance not the direction of power changes in the beta band.

3.4.2 EEG Spectral Parameterisation: ASMR Button-Press Onset

To assess oscillatory activity time-locked to the onset of reported ASMR tingling, EEG data were epoched from -2 s to +6 s relative to each button press onset, capturing neural activity immediately preceding and during the consciously reported tingling experience. Power spectra from these epochs were parameterised using the FOOOF algorithm, and a within subject cluster-based 1 sample permutation test compared aperiodic-adjusted peak amplitudes between ASMR button press and duration-matched Control epochs.

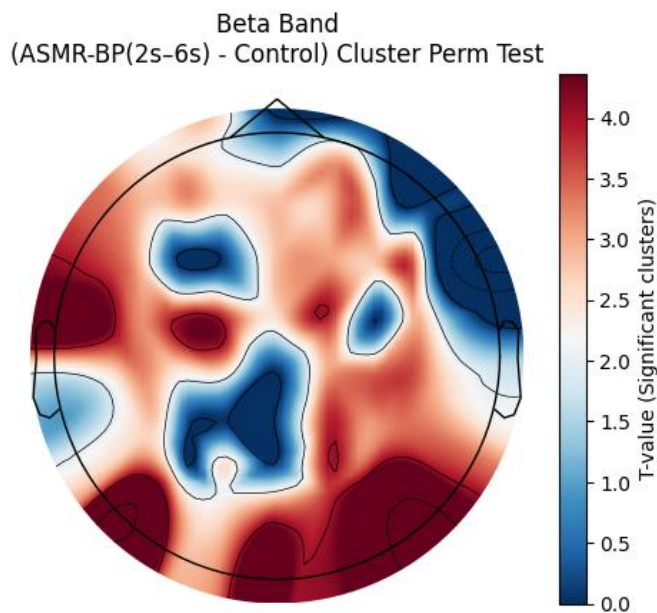
Significant frequency-specific differences were observed in the beta and gamma bands. In the beta band (15-30 Hz), a significant negative t-statistic distribution emerged over parietal and posterior electrodes, the figures shown demonstrate the cluster location not the direction of power change but reduced beta-band power was seen during ASMR button-press trials relative to Control (Figure 3.2). This beta desynchronisation is consistent with a release of top-down inhibitory control and enhanced sensory receptivity characteristic of affective and anticipatory touch.

In the gamma band (30-48 Hz), a significant positive cluster was identified over midline and anterior scalp regions (Figure 3.3), reflecting increased high-frequency synchronisation during ASMR tingling episodes that reached statistical significance. This enhancement may index multisensory integration and interoceptive-somatosensory binding processes accompanying the ASMR percept.

Together, these results demonstrate that the subjective onset of ASMR sensations is accompanied by a coordinated shift in oscillatory dynamics, beta suppression coupled with gamma enhancement, supporting the Proximity Prediction Hypothesis that ASMR engages predictive-affiliative neural mechanisms integrating auditory, somatosensory, and interoceptive signals. Notably, this pattern differs from that reported by Swart et al. (2022), who observed reductions in high-frequency activity during ASMR states. One possible explanation for this divergence is the time-locking of the present analysis to the onset of self-reported tingling, which may preferentially capture transient prediction-related dynamics. In contrast, Swart et al. (2022) analysed broader state transitions and post-ASMR periods, which may be more sensitive to sustained relaxation-related processes. This distinction is consistent with the PPH account, which predicts an initial phase of precision-weighted updating (indexed by gamma enhancement) followed by a later shift toward lower-frequency dominance as the system stabilises.

Figure 3.2

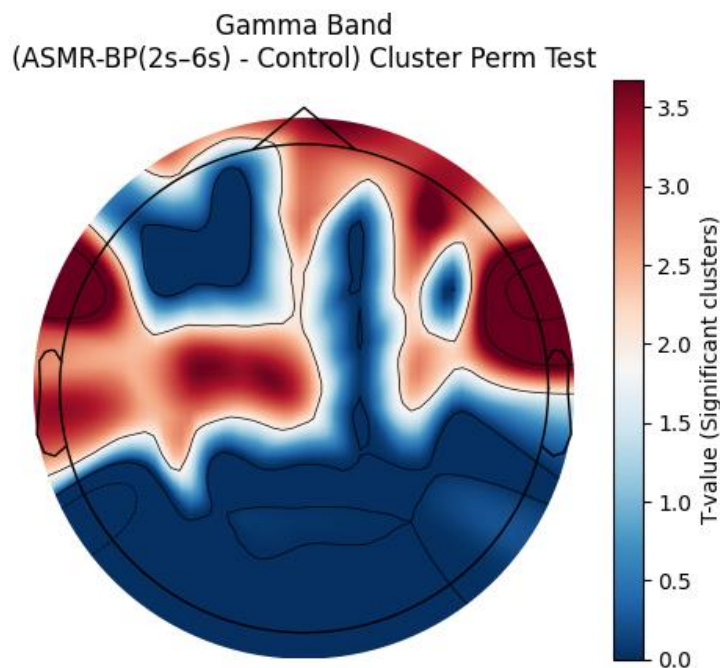
Cluster-corrected topography of beta-band peak amplitude differences (15-30 Hz) for ASMR button-press vs. Control trials.



Note: This map shows T-values masked to the electrodes forming the significant spatial cluster identified by the cluster-based permutation test ($p < .05$). Colour intensity reflects the magnitude of the T-statistic within electrodes belonging to the significant spatial cluster identified by cluster-based permutation testing. Warmer colours indicate larger T-values within the significant cluster. The colours therefore only reflect the distribution of significance not the direction of power changes in the beta band.

Figure 3.3

Cluster-corrected topography of gamma-band peak amplitude differences (30-48 Hz) for ASMR button-press vs. Control trials.



Note: This map shows T-values masked to the electrodes forming the significant spatial cluster identified by the cluster-based permutation test ($p < .05$). Colour intensity reflects the magnitude of the T-statistic within electrodes belonging to the significant spatial cluster identified by cluster-based permutation testing. Warmer colours indicate larger T-values within the significant cluster i.e. in this case the cluster encompasses significant increases in power for gamma, however it would still be red if there were decreases because the colours on this map only reflect the distribution of clusters that reached significance, not whether the power changes in the gamma band that were significant in this cluster were increasing or decreasing.

3.4.3 EEG Time-frequency analysis: ASMR vs Control

To characterise the temporal evolution of oscillatory activity during ASMR, EEG data were decomposed using complex Morlet wavelets (2-48 Hz, 50 ms steps, 5-cycle width). Figure 3.4 A shows the grand-mean time-frequency representation of baseline-corrected power differences between ASMR and Control trials, averaged across all electrodes. Overall power changes were small and spatially diffuse, with no sustained or sharply localised frequency-specific effects across the 30s stimulus period.

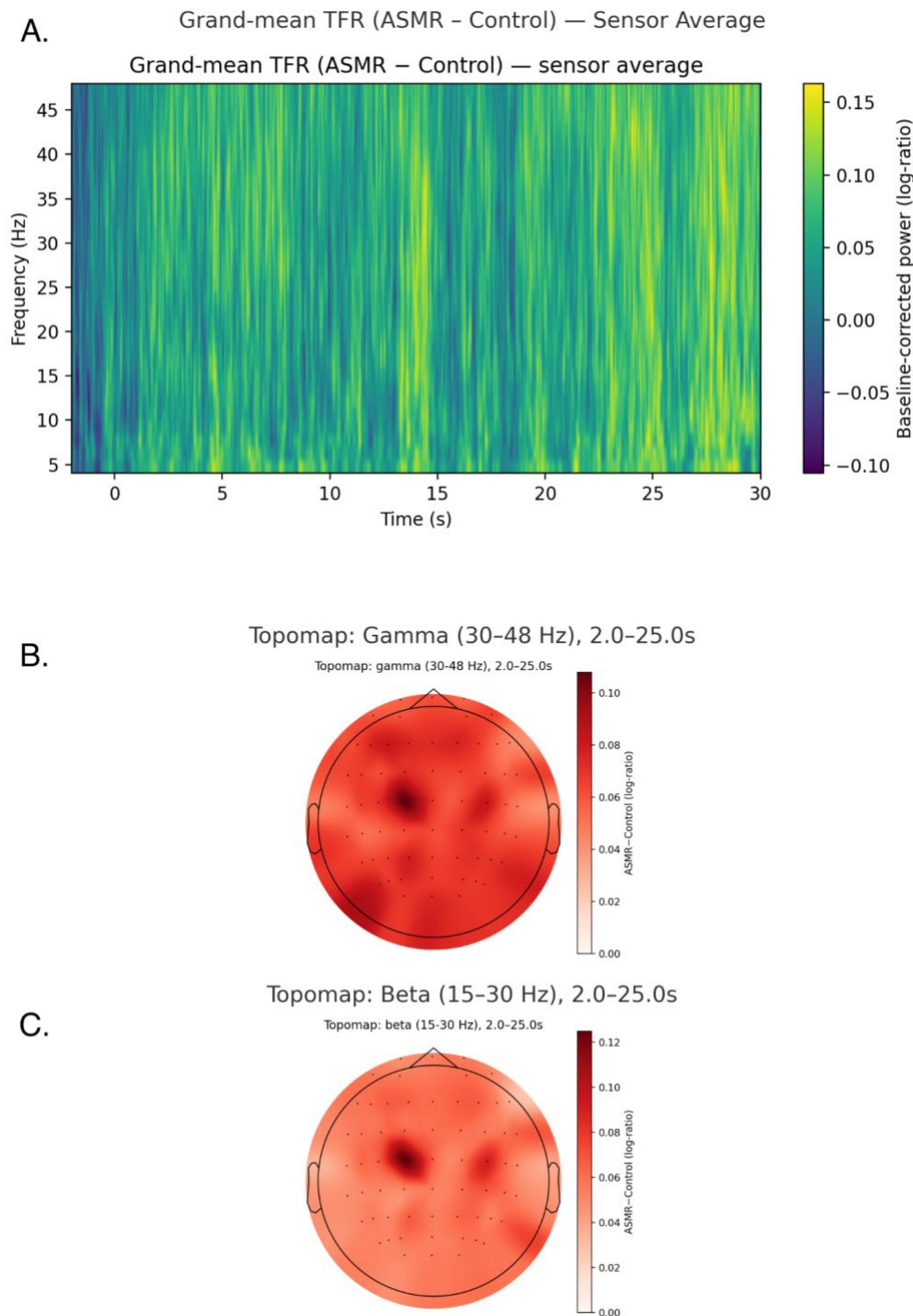
Topographic averages of band-limited power over the 2-25s window indicated slightly higher uncorrected power for ASMR relative to Control in both the gamma (30-48 Hz; Figure 3.4B) and beta (15-30 Hz; Figure 3.4C) ranges. These unthresholded maps illustrate the direction of the effect, with warm colours denoting regions where ASMR power exceeded Control power. The differences were of low magnitude and broadly distributed, suggesting weak and variable increases in high-frequency activity rather than consistent spatially specific modulation.

Exploratory cluster-based permutation tests applied across time, frequency, and channels did not yield statistically significant clusters after correction. The lack of robust group-level effects likely reflects inter-individual variability in the timing of tingling responses, combined with the normalisation imposed by baseline correction. Although the TFR analysis did not replicate the beta-band suppression observed in the FOOOF spectral results, both analyses converge in showing greater gamma-band engagement during ASMR, consistent with enhanced high-frequency cortical synchronisation during the experience.

The absence of robust TFR effects may also reflect variability in the temporal alignment of ASMR experiences across participants. Studies examining broader state-level changes, such as Swart et al. (2022), may be better positioned to capture sustained low-frequency shifts associated with relaxation, whereas the present approach prioritises transient, event-related dynamics.

Figure 3.4

(A) Grand-mean time-frequency representation (ASMR - Control) averaged across all EEG sensors, baseline-corrected relative to -2 to 0s. **(B)** Topographic distribution of mean gamma-band (30-48 Hz) power difference over 2-25s. **(C)** Topographic distribution of mean beta-band (15-30 Hz) power difference over 2-25 s.



Note: Warm colours represent regions where ASMR power exceeded Control power (unthresholded). No effects reached significance after correction.

3.4.4 EEG Event-Related Potentials: Button-Press Onset

To examine transient neural dynamics surrounding the subjective onset of ASMR tingling, event-related potentials (ERPs) were computed time-locked to the onset of each button press (0 s). The grand-average ERP across participants (Figure 3.5; N = 46) showed modest voltage fluctuations around the time of the button press, without a sharply defined or high-amplitude canonical ERP component.

Inspection of the waveform suggests gradual changes in activity both preceding and following the button press, rather than a discrete, time-locked peak. The pre-press interval (-300 to 0 ms) shows subtle variation across channels, while post-press activity (0-500 ms) is characterised by low-amplitude, spatially distributed responses.

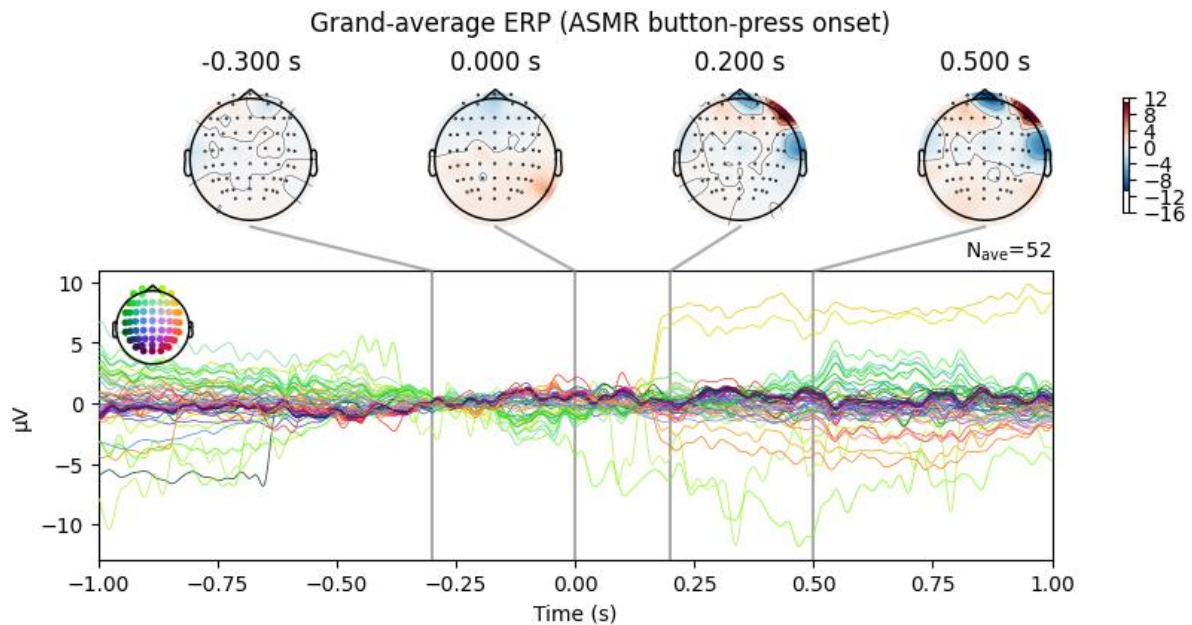
Topographic maps at representative time points (-300 ms, 0 ms, 200 ms, and 500 ms) indicate that activity is broadly distributed across frontal, central, and posterior regions, with no clear contralateral or focal motor pattern. Around button press onset, a relative frontal negativity with posterior positivity is observable, which persists to some extent into the post-press interval. However, these effects are modest in magnitude and do not form a sharply localised or stereotypical ERP component.

Although a direct motor-matched control condition was not available, the spatial distribution of the observed activity does not conform to a purely lateralised motor potential. Instead, the broadly distributed topography suggests that the response may reflect a combination of processes, including motor execution, perceptual awareness, and interoceptive or affective components associated with the subjective experience of ASMR tingling.

Given the absence of formal statistical testing and the relatively low amplitude of the observed effects, these findings should be interpreted cautiously and primarily as descriptive. Nonetheless, the ERP results are consistent with the broader pattern observed in the spectral analyses, suggesting that the neural response associated with ASMR onset is distributed and involves multiple interacting processes rather than being reducible to a simple motor artefact.

Figure 3.5

Grand-average event-related potential (ERP) time-locked to button press onset during ASMR trials (N = 52).



Note: EEG waveforms represent the grand-average voltage (μV) across electrodes from -1 s to +1 s relative to button-press onset (0 s), illustrating neural activity surrounding the subjective report of ASMR tingling. The waveform shows modest voltage fluctuations before and after the button press, without a sharply defined or high-amplitude ERP component. Topographic maps display scalp voltage distributions at representative latencies (-300 ms, 0 ms, +200 ms, +500 ms), indicating broadly distributed activity across frontal, central, and posterior regions rather than a focal, lateralised motor pattern. Warm colours indicate relative positive voltages and cool colours indicate relative negative voltages.

3.5 MEG Results

3.5.1 MEG Spectral Parameterisation: ASMR vs Control

MEG sensor-space analysis revealed significant frequency-specific differences in oscillatory activity between ASMR and Control conditions, corroborating and extending the EEG results. A robust gamma-band increase (30-48 Hz) was observed during ASMR relative to Control ($p < 0.001$, Bonferroni-corrected), spanning 243 (of 248) magnetometer sensors.

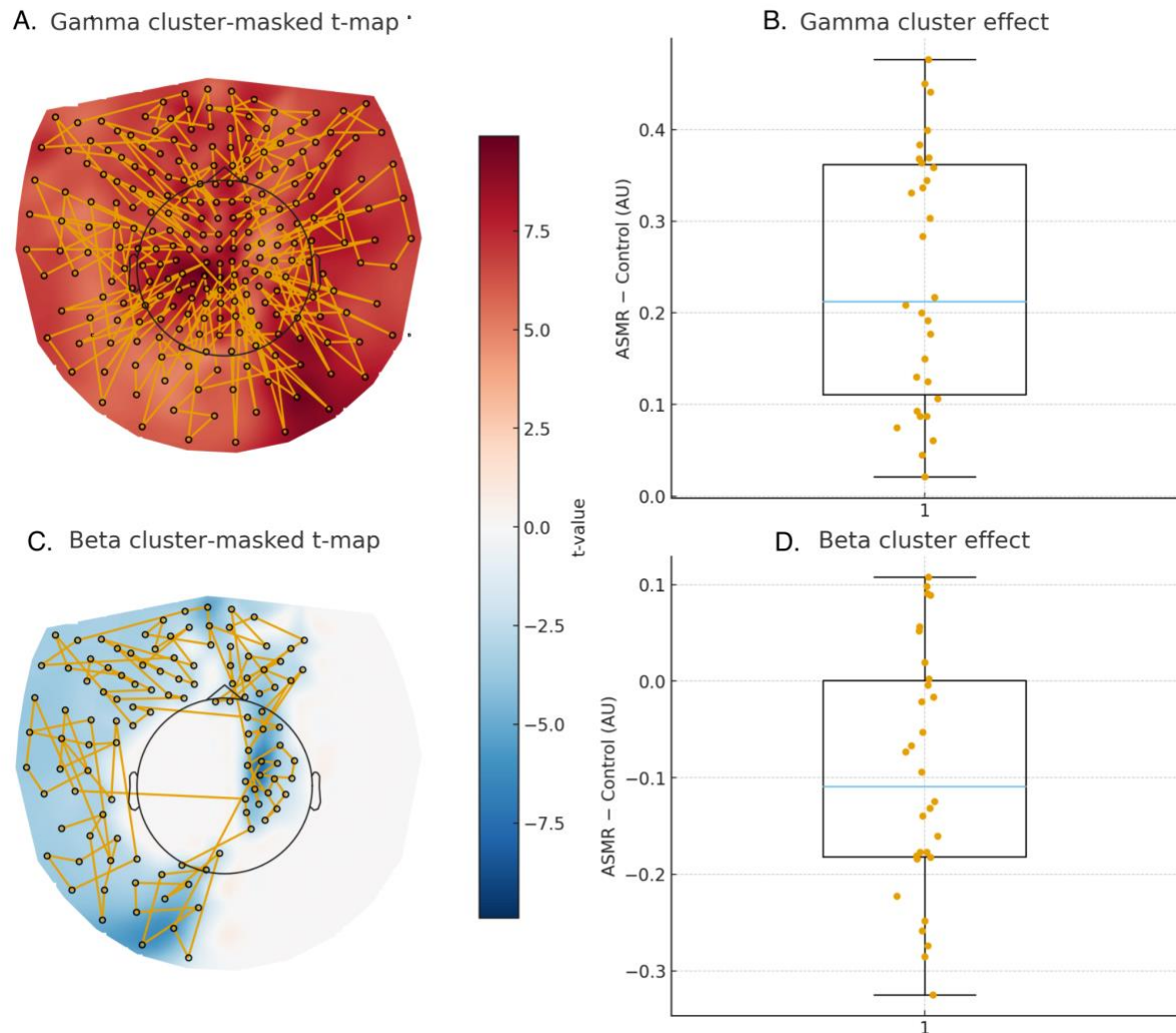
This enhancement was strongest over bilateral temporal-parietal sensors, with a mean amplitude difference of $\Delta = +0.239$ a.u. (95 % CI [0.188, 0.291]; $t(29) = 9.46$, $d_z = 1.73$).

In contrast, the beta band (15- 30 Hz) showed a significant decrease in power for ASMR compared to Control ($p = 0.007$, Bonferroni-corrected), localised over left-central sensors (116 channels), with a mean difference of $\Delta = - 0.096$ a.u. (95 % CI [- 0.144, - 0.049]; $t(29) = - 4.13$, $d_z = - 0.75$). No significant effects were found for theta (4- 8 Hz) or alpha (8- 12 Hz) bands after multiple comparisons correction.

The MEG topographies mirrored the power change profiles observed in the EEG: beta suppression and gamma enhancement (Figure 3.6). This convergence across modalities supports the conclusion that ASMR likely engages a sensorimotor-interoceptive network, characterised by low-frequency desynchronisation and high-frequency enhancement during ASMR experiences.

Figure 3.6

Sensor-space MEG effects of ASMR vs. Control on FOOOF-derived oscillatory peak amplitude.



Note: Cluster-based permutation testing was applied to aperiodic-adjusted spectral peak amplitudes (ASMR - Control) across magnetometer sensors. **(A)** Gamma-band (30- 48 Hz) t-map; colour scale denotes t-values. Sensors contributing to the significant cluster are outlined ($p < .001$; 243 of 243 sensors). **(B)** Participant-level gamma effect within the cluster: mean = 0.239 AU, 95 % CI [0.188, 0.291], $t(29) = 9.46$, $p = 2.28 \times 10^{-10}$, $d(z) = 1.73$. **(C)** Beta-band (15- 30 Hz) t-map with significant clusters highlighted ($p = .007$; 116 sensors). **(D)** Participant-level beta effect: mean = -0.096 AU, 95 % CI [-0.144, -0.049], $t(29) = -4.13$, $p = 2.77 \times 10^{-4}$, $d(z) = -0.75$. Positive values reflect stronger peak amplitudes in ASMR vs. Control; AU = arbitrary units. Tests used 2,000 permutations (two-sided $\alpha = 0.05$) with

adjacency defined across magnetometers. Bonferroni correction for frequency bands (θ , α , β , γ) is reported in the main text.

3.6 Discussion

The present EEG and MEG findings reveal a reproducible oscillatory signature of the Autonomous Sensory Meridian Response (ASMR), across two modalities. ASMR was characterised by a dual pattern of beta-band desynchronisation and gamma-band enhancement, consistent with coordinated shifts in predictive sensorimotor control and multisensory integration. These effects were robust at the group level, converging across independent datasets and complementary analytic approaches. Together, they provide the first cross-modal evidence for a distinctive oscillatory state underlying the ASMR experience.

3.6.1 Reconciling FOOOF, ERP, and TFR Findings

Although the time-frequency decomposition did not yield statistically significant clusters after correction, these TFR results remain informative when interpreted alongside the FOOOF and ERP analyses. The relative flatness of the grand-average TFR likely reflects the temporal and spatial variability of tingling episodes across participants. Indeed, the corresponding behavioural data for this EEG paradigm was presented in Chapter 2, demonstrating the individual variability in which triggers elicited ASMR across all participants, and the striking heterogeneity in the pleasantness attributed to each (Flockton et al., 2025); suggesting tingling episodes' reported occurrence, intensity, and length of duration were pursuant to the affective priors individuals held about each sound, rather than sensitivity to acoustic properties of the stimuli. Therefore, TFR analyses are limited to this heterogeneity, predicted by the PPH model, and illustrated in the self-report data from the present EEG study, assessed in Chapter 2. Unlike the FOOOF analysis, which integrates spectral power over an extended (30s) window, time-frequency methods are sensitive to trial-to-trial fluctuations in phase and amplitude. Given that ASMR tingling onsets and duration are idiosyncratic, genuine oscillatory shifts in beta and gamma activity may have been temporally misaligned and thus attenuated when averaged across individuals.

The ERP findings further support this interpretation of temporal variability. When time-locked to button-press onset, the grand-average ERP showed only modest and broadly distributed voltage fluctuations, without a sharply defined or high-amplitude component. This suggests that the neural processes underlying ASMR onset are not tightly phase-locked to the

moment of behavioural report, but instead unfold gradually and variably across trials and participants. Importantly, the absence of a strong, focal motor-related ERP (e.g., a contralateral readiness potential) also indicates that the observed signal is not dominated by the motor act of pressing the button. Rather, the ERP appears to reflect a mixture of perceptual, interoceptive, and decision-related processes that are temporally smeared when aligned to subjective report.

Furthermore, the use of baseline-corrected log-ratio power (-2 to 0s) may have normalised subtle tonic differences between ASMR and Control conditions, reducing apparent contrasts in the TFR plots. By contrast, FOOOF's parameterisation of the power spectrum into periodic (oscillatory) and aperiodic ($1/f$) components isolates condition-dependent changes in oscillatory strength more robustly. In this light, the FOOOF-derived beta and gamma modulations are likely to represent more sustained and stable shifts in neural state during ASMR engagement. However, this interpretation should be considered alongside Swart et al. (2022), who reported a different oscillatory profile, with increased low- to mid-frequency activity and reduced high frequency activity during ASMR. Their findings suggest that ASMR may also involve a sustained relaxation-related state, particularly when analyses capture broader state transitions and post-ASMR periods rather than activity tightly linked to reported tingling onset. This distinction may help explain why the present FOOOF and button-press analyses identified beta suppression and gamma enhancement, whereas Swart et al. observed high-frequency reductions.

3.6.2 Convergent Oscillatory Correlates of ASMR and Predictive Coding

In both EEG and MEG, ASMR stimuli elicited reliable reductions in beta power relative to non-tingling control sounds. This desynchronisation was broadly distributed across central-parietal scalp electrodes in EEG and left-lateralised in MEG sensor space, consistent with decreased top-down inhibition within the sensorimotor system. Beta-band desynchronisation is classically interpreted as a release of the motor system's 'status quo' state, facilitating sensory updating and adaptive processing (Engel & Fries, 2010). That is, beta-band oscillations reflect the maintenance of internal models, and their suppression signals readiness for new sensory input. Consistent with this, affective touch research has shown that gentle CT-optimal stroking leads to alpha and beta suppression over somatosensory regions (Singh et al., 2014), while von Mohr et al. (2018) reported significantly lower beta power in response to CT-optimal versus non-affective touch, localised over parietal electrodes. Similarly, Pereira et al. (2025) found that even the observation alone of slow, affective touch resulted in reduced beta power relative to watching videos of faster or static touch, suggesting that vicarious

tactile processing engages similar mechanisms. Analogously, ASMR auditory triggers, such as tapping or brushing sounds, may acoustically simulate a proximal, socially affiliative contact, thereby eliciting cortical disinhibition through inferred tactile proximity. Within the framework of the Proximity Prediction Hypothesis (PPH), this beta suppression can be interpreted as a neural correlate of updating tactile-interoceptive predictions in response to near-ear acoustic cues. According to the PPH, ASMR triggers signal the inferred approach of gentle social touch, prompting the brain to downregulate the status quo prediction embodied in beta activity. The resulting decrease in beta power therefore reflects a shift toward a sensory state optimised for detecting socially meaningful, low intensity contact. Therefore beta desynchronisation here likely reflects a temporary relaxation of top-down inhibitory control, enhancing receptivity to subtle, socially meaningful cues.

During subjective tingling episodes, indexed by button presses, this beta suppression was accompanied by gamma-band amplification over midline and anterior sensors. Gamma-band enhancement is widely regarded as a marker of multisensory binding and precision-weighted prediction updating (Bastos et al., 2012). Its emergence during tingling suggests transient coupling between auditory, somatosensory, and interoceptive representations as the ASMR percept unfolds. The co-occurrence of beta suppression and gamma enhancement during ASMR may reflect the hierarchical dynamics of predictive processing: beta desynchronisation signals the suspension of ongoing sensorimotor predictions (preparing for the inferred affiliative touch), while gamma enhancement marks bottom-up prediction errors elicited by the absence of that expected tactile input. The resultant interoceptive sensation, the ASMR tingle, may therefore represent a somatosensory echo generated to resolve cross-modal prediction conflict, as predicted by the PPH model (Flockton et al., 2025). This interpretation contrasts with Swart et al. (2022), who found reductions in high-frequency activity during ASMR and ASMR-decay periods. Rather than treating these findings as incompatible, they may reflect different temporal phases of the ASMR response. In the present study, gamma enhancement was most evident during button-press-defined tingling episodes, which may capture transient prediction error or multisensory integration processes. By contrast, the high frequency reductions reported by Swart et al. may reflect the later relaxation-dominant phase of ASMR, after the initial sensory-affective response has stabilised.

A key conclusion to be drawn from the current study is therefore that the emergence of gamma-band enhancement during ASMR tingling episodes may index a cross-modal

prediction error rather than just reflecting stimulus repetition or low-level auditory features. While ASMR stimuli (e.g., brushing, tapping) are repetitive in temporal structure, there were aspects of both repetition and surprise in the control stimuli recordings (e.g., traffic hum, bus engine noise), yet gamma enhancement occurred exclusively during ASMR trials, particularly at the point of subjective tingling; indeed, no control trials were reported to elicit this tingling sensation in the EEG study by participants (Flockton et al., 2025). This selective engagement suggests that gamma enhancement in this context reflects neither acoustic regularity nor exogenous novelty, but rather a multisensory mismatch between inferred tactile predictions and absent somatic input, specifically in relation to the types of proximal, socially relevant ASMR condition stimuli. Within predictive coding accounts, gamma-band activity has been proposed to carry prediction error signals, i.e. indexing the mismatch between top-down expectations and incoming sensory input, effectively acting as a comparator signal in cortical hierarchies (Arnal et al., 2011). This role has been supported across paradigms; violations of auditory expectations, as in mismatch negativity (MMN), consistently evoke increases in gamma activity and topographic reconfiguration (Haenschel et al., 2000; Nicol et al., 2012; von Stein et al., 2000). This encompasses prediction error processing across sensory modalities, such as audiovisual incongruence, which have been shown to modulate gamma power (Arnal et al., 2011). Critically, studies show that fulfilled expectations reduce evoked gamma, while unexpected omissions or mismatches amplify it (Todorovic et al., 2011; Bendixen et al., 2009; Fujioka et al., 2012; Iversen et al., 2009). In the present context, the rich social, affiliative cues in ASMR stimuli (e.g., hair brushing sounds) may implicitly generate predictions of affiliative, gentle touch, as described by the Proximity Prediction Hypothesis (Flockton et al, 2025), and when no tactile input arrives, this cross-modal mismatch triggers gamma enhancement, reflecting the brain's attempt to resolve the discrepancy. These types of effects are already thought to reflect the brain's attempt to unify related sensory events into coherent percepts, even in the absence of consistent input, as demonstrated in paradigms like the rubber hand illusion where gamma synchrony scales with the strength of the illusory tactile experience (Kanayama et al., 2009). The subjective tingling percept may thus emerge as a somatosensory echo, another kind of illusory tactile experience; a perceptual inference that minimizes tactile prediction error in the absence of expected contact.

These oscillatory signatures provide empirical support for the PPH's proposition that ASMR involves both the disinhibition of sensorimotor circuits and the integration of multisensory predictive coding associated with affective touch. This framework also

offers a novel reinterpretation of the ASMR tingle, not as a passive sensory reaction, but as an active inferential process that resolves cross-modal mismatches through interoceptive forecasting.

In the present data, the gamma-band cluster was widespread across bilateral temporal and parietal MEG sensors, while beta suppression appeared over left-central sensor regions (Figure 4.3). Although these sensor-space distributions cannot determine the precise cortical generators of sources in the brain, they appear spatially consistent with the hypothesised engagement of a sensorimotor-interoceptive network however, as this is sensor-space data, no activity can be resolved to those regions, without further source-space localisation. The prediction that would align with the overarching theoretical explanations of this sensors-level data in future research would be that gamma enhancement may arise from regions such as the secondary somatosensory cortex (S2), the posterior insula, or the posterior cingulate cortex, areas implicated in integrating interoceptive and exteroceptive cues. Similarly, the beta-band desynchronisation may reflect sensorimotor disinhibition within circuits underlying predictive tactile engagement.

Importantly, the MEG replication provides convergent evidence for these oscillatory dynamics, with denser sensor coverage and greater sensitivity to tangential and deeper cortical sources.

3.6.3 Methodological Considerations

A key methodological strength of this work is its crossmodal replication. Using identical stimuli and preprocessing across EEG and MEG datasets enabled cross-validation of oscillatory findings with complementary sensitivities. This reduces the likelihood that the effects are artefactual or modality-specific. The use of spectral parameterisation (FOOOF/specparam) further strengthened interpretability by disentangling oscillatory peaks from broadband $1/f$ components, revealing true periodic shifts that traditional power estimates might obscure. Together, these approaches provide a rigorous foundation for interpreting ASMR-related neural dynamics.

Although the control sounds were not acoustically matched to ASMR stimuli in a formal signal-processing sense (e.g., matched for RMS power, spectral tilt, or temporal envelope), this was an intentional design choice. At present, there are no established standardised control stimuli in ASMR research, and prior studies have used a diverse range of non-ASMR comparisons (e.g., silence, or so-called 'non-ASMR speech' (Fredborg et al., 2021),

however, such controls may still contain acoustic features (e.g., mouth sounds, pitch, speech cadence) known to trigger ASMR in some individuals (Barratt et al., 2017). To avoid this ambiguity, we opted for naturalistic environmental recordings, such as ambient bus or traffic sounds, that were plausibly immersive but explicitly lacked the near-field, soft, and repetitive characteristics typical of ASMR triggers in this study. Crucially, none of the participants reported experiencing tingling sensations in response to these control sounds: across all EEG and MEG sessions, no button presses were recorded during control trials, even though participants were explicitly instructed to indicate any tingling responses regardless of whether they would classify the stimulus type as an ASMR style sound or not. This complete absence of reported ASMR responses suggests that the chosen control stimuli were effective in capturing a naturalistic, non-affective baseline condition, against which ASMR-specific neural activity could be meaningfully contrasted.

While the present findings establish a clear oscillatory signature of ASMR, certain limitations warrant consideration. Firstly, all analyses were only conducted at the sensor level, meaning directly resolving activity seen to specific brain regions that are predicted to be involved by the PPH model was not possible. Secondly, the 64-channel EEG montage provided limited spatial resolution, and the self-report button-press measure of tingling may have introduced temporal imprecision. Discrete responses capture the onset of conscious awareness but lag behind underlying neural changes by several hundred milliseconds (Schurger, Sitt, & Dehaene, 2012), potentially blurring fine-grained oscillatory dynamics. Future work could incorporate continuous self-report tools (e.g., affective sliders) and concurrent physiological indices such as pupil dilation, skin conductance, and heart-rate variability to map the evolving trajectory of ASMR more precisely. Pupil diameter offers a millisecond level proxy for LC-noradrenergic activity (Joshi & Gold, 2020), while HRV and electrodermal activity provide complementary measures of parasympathetic and sympathetic balance (Laborde, Mosley, & Thayer, 2017; Critchley & Harrison, 2013). Simultaneous acquisition of these indices would enable direct testing of the proposed biphasic transition from orienting arousal to parasympathetic restoration. Furthermore, because participants only pressed the button during tingling, the current data cannot fully separate motor-related potentials from perceptual or interoceptive components. Future experiments should include control button presses during non-tingling periods to isolate purely perceptual effects.

Finally, exploring multimodal ASMR triggers (e.g., videos of whispering or brushing sounds) could reveal broader cortical engagement across auditory, visual, and somatosensory pathways, potentially enhancing the affective and parasympathetic components of the

response more than this study's limited auditory only, non-mouth sounds, stimuli sample could.

3.6.4 Conclusion

The EEG and MEG results presented here converge on a consistent oscillatory signature of ASMR, defined by beta-band desynchronisation and gamma-band enhancement. These findings offer empirical support for the Proximity Prediction Hypothesis, suggesting that ASMR engages a multisensory predictive coding network that simulates affiliative touch and integrates exteroceptive and interoceptive cues.

Critically, the co-occurrence of beta suppression and gamma enhancement, particularly during subjective tingling episodes, compatible with a mechanistic account of ASMR as a hierarchical inference process. Beta desynchronisation likely reflects the suspension of sensorimotor predictions, while gamma synchrony indexes cross-modal prediction errors triggered by the absence of expected tactile input. This mismatch may result in a compensatory somatosensory echo, giving rise to the characteristic ASMR tingling sensation. In this framework, ASMR emerges as a perceptual inference: a cross-modal hallucination of affiliative contact, generated to resolve prediction conflict in the absence of actual touch.

This predictive coding interpretation deepens our understanding of ASMR as more than passive sensory processing. Instead, it suggests that ASMR actively recruits hierarchical generative models of social touch and body state, dynamically updated via oscillatory precision-weighting. The convergence of beta suppression and gamma enhancement provides a testable neural signature for such embodied simulations and inferred tactile representations, that give rise to ASMR's unique phenomenology. Future source-localisation and autonomic-coupling analyses (e.g., heart rate variability, skin conductance) will help map these dynamics onto specific cortical and physiological systems.

Chapter 4:**Autonomous Sensory Meridian Response Reduces Fatigue in Chronic Pain: Evidence for Affective-Autonomic Modulation.**

The following study is being prepared for peer review and its preregistration can be found on the Open Science Framework online preregistration repository:

<https://doi.org/10.17605/OSF.IO/PV5WS>.

4.1 Abstract

Autonomous Sensory Meridian Response (ASMR) is a pleasant tingling sensation felt across the scalp and neck, elicited by auditory or visual triggers. While anecdotal reports suggest ASMR may alleviate symptoms of chronic pain and its comorbidities, no existing research has specifically examined its effects in this population. This preregistered study evaluated whether daily ASMR sound exposure over two weeks would improve pain, sleep, fatigue, anxiety, and depression among individuals with chronic pain. 120 adults with chronic pain were randomized to daily ASMR sound exposure or a delayed-access control group for two weeks. 64 participants (ASMR = 33, Control = 31) completed all study phases and were included in the final analyses. Standardized clinically relevant outcome measures: the Pain, Enjoyment, and General Activity (PEG) scale, Fatigue Severity Scale (FSS), Insomnia Severity Index (ISI), and Hospital Anxiety and Depression Scale (HADS), were completed at baseline, Week 1, and Week 2. Linear mixed-effects models assessed change from baseline. The ASMR group showed significantly greater reductions in fatigue severity relative to controls, whereas no significant group differences were observed for pain, sleep, anxiety, or depression. Exploratory analyses indicated that greater self-reported enjoyment of ASMR listening correlated with improvements in pain interference. The fatigue reduction is consistent with models linking affective-autonomic regulation to energy restoration. Notably, this improvement emerged despite variable listening adherence, suggesting that ASMR's effects may be robust and achievable under naturalistic conditions. This implies that ASMR sound exposure may serve as a scalable, self-directed intervention for reducing fatigue in chronic pain populations.

4.2 Introduction

Chronic pain is highly prevalent and disabling, affecting approximately one in five adults in large population surveys across Europe and the United States (Breivik et al., 2006; Dahlhamer et al., 2018). Beyond the persistent sensory experience of pain, comorbid anxiety, depression, sleep disturbance, and fatigue are common and inter-related, together contributing to marked reductions in quality of life and daily functioning (Finan, Goodin, & Smith, 2013; Meeus et al., 2013). These comorbidities often prove resistant to conventional treatment, and pharmacological approaches, while sometimes beneficial, carry significant risks, including tolerance, dependence, sedation, and falls, particularly in longer term or older-adult use (Kaye et al., 2019). Consequently, there is growing interest in scalable, non-pharmacological interventions capable of improving multiple symptom domains simultaneously.

Contemporary models of chronic pain emphasise that distress and disability often arise from impaired cognitive-emotional modulation rather than the magnitude of nociceptive input itself (Bushnell, Čeko, & Low, 2013; Eccleston & Crombez, 1999). Consequently, interventions that restore affective-autonomic regulation, for example by reducing arousal, improving sleep, or enhancing positive emotion, may yield clinically meaningful benefits even when sensory pain remains unchanged. Despite theoretical advances in understanding affective-autonomic dysregulation in chronic pain, few studies have examined interventions designed to directly target these mechanisms in daily life.

Autonomous Sensory Meridian Response (ASMR) is a perceptual phenomenon characterized by pleasant form of paresthesia (tingling sensations not elicited by direct physical contact), often beginning on the scalp or neck and spreading down the spine, in response to specific auditory or visual triggers such as whispering, tapping, or personal attention (Fredborg et al., 2017). Beyond paresthesia, ASMR is widely reported to induce relaxation, improve sleep, and reduce stress (Barratt & Davis, 2015). Laboratory studies show that ASMR exposure can decrease heart rate and promote parasympathetic calm (Poerio et al., 2018), consistent with its use as a tool for mood regulation. Importantly, these outcomes map closely onto the comorbidities most problematic in chronic pain, suggesting that ASMR could be clinically valuable in this population. Despite growing interest in its relaxing effects, surprisingly little research has explored ASMR in relation to pain itself. One study to examine ASMR in relation to pain perception (Janik McErlean, Ellis, & Walsh, 2022) reported that individuals who experience ASMR showed greater pain sensitivity than non-experiencers, although pain tolerance did not significantly differ between groups. Given the study's small sample size (N = 19), these findings should be interpreted cautiously. The

authors suggested that ASMR may influence the affective and cognitive appraisal of pain rather than directly modulating nociceptive input, highlighting the need to investigate its potential within clinical contexts where sensory hypersensitivity and affective dysregulation co-occur, such as in chronic pain (Bushnell, Čeko, & Low, 2013; Eccleston & Crombez, 1999).

Physiological and affective evidence supports the therapeutic potential of ASMR. For example, Poerio et al. (2018) found that individuals who experience ASMR reported significantly increased pleasant affect and reduced heart rate after viewing ASMR videos. Barratt and Davis (2015) further demonstrated that around 80% of ASMR experiencers reported a temporary reduction in depressive mood while engaging with ASMR content, with the strongest but most short-lived improvements observed among those with higher baseline depressive symptoms. McErlean and Osborne-Ford (2020) extended this work by showing that ASMR-prone individuals exhibit heightened absorption (focused immersion) during ASMR stimuli, which may underpin its mood-modulating effects. Additional findings suggest that brief ASMR exposure can improve sleep initiation and quality in student populations (Hardian et al., 2020) and may reduce depressive feelings in ASMR-sensitive individuals (Engelbregt et al., 2022). Notably, even participants who do not report paresthesia show physiological changes such as increased pupil diameter (Valtakari et al., 2019), suggesting benefits may extend beyond subjective awareness of the response. This body of evidence indicates that ASMR can modulate arousal and affective states in ways relevant to the comorbidities (e.g., sleep disturbance, fatigue, mood) commonly observed among chronic pain sufferers.

In addition to its mood-regulating properties, ASMR has been widely adopted by listeners to aid sleep. Self-report studies consistently show that falling asleep is one of the most common motivations for ASMR use (Barratt & Davis, 2015; McErlean & Osborne-Ford, 2021). While empirical research on sleep outcomes remains limited, there is emerging evidence that ASMR may facilitate sleep initiation through cognitive and physiological relaxation mechanisms. Hardian et al. (2020) conducted a randomized controlled trial in which participants viewed a 20 minute ASMR video each night at 9 pm, before going to bed, over a two week period; the intervention group exhibited significantly decreased Pittsburgh Sleep Quality Index (PSQI) scores, indicating improved sleep quality compared to controls.

Sleep problems are closely intertwined with chronic pain and meaningfully worsen patients' lives. Across studies, individuals with chronic pain who also report sleep disturbance tend to experience greater pain severity, longer pain duration, higher levels of disability, and reduced physical activity, along with elevated rates of depression, anxiety, catastrophizing,

and suicidal ideation compared with those without sleep problems, underscoring the clinical burden of this comorbidity (Husak & Bair, 2020). In a wider meta-analysis of 37 polysomnography studies, adults with chronic pain showed longer sleep-onset latency and more frequent awakenings, lower sleep efficiency, and greater time awake after sleep onset relative to healthy controls, as well as smaller but still significant impairments in total sleep time, stage shifts, respiratory events, and periodic limb movements (Mathias, Cant, & Burke, 2018). The pooled prevalence of diagnosed sleep disorders in chronic pain was 44%, with insomnia (72%), restless legs syndrome (32%), and obstructive sleep apnoea (32%) being the most common (Mathias et al., 2018). Alongside more recent longitudinal evidence demonstrating that poor sleep often predicts next-day pain intensity and vice versa (Finan, Goodin, & Smith, 2013; Haack & Simpson, 2020), these findings highlight the necessity of assessing and targeting sleep disturbances in chronic pain interventions. Using a validated measure of sleep quality, such as the Insomnia Severity Index (ISI), therefore represents a clinically meaningful outcome for this population.

ASMR has also been proposed to alleviate fatigue through autonomic modulation, although few studies have directly assessed this effect using validated fatigue scales. It is important to distinguish between sleep disturbance, characterized by difficulty falling or staying asleep, as measured by the Insomnia Severity Index (ISI; Bastien et al., 2001), and fatigue, which reflects a persistent sense of physical or mental exhaustion not necessarily resolved by sleep, as captured by the Fatigue Severity Scale (FSS; Krupp et al., 1989), both of which will be used in the present study. In chronic pain populations, both are common and debilitating but reflect distinct phenomena (Meeus et al., 2013; Finan et al., 2013). Fatigue is among the most commonly reported and disabling secondary symptoms of chronic pain, affecting up to 70-80% of patients and contributing substantially to functional impairment, reduced quality of life, and emotional distress (Nielson & Jensen, 2004; Häuser et al., 2015). While sleep disturbance can exacerbate fatigue through disrupted restorative processes, fatigue often persists independently due to autonomic dysregulation, inflammatory cytokine activity, altered hypothalamic-pituitary-adrenal (HPA) axis function, and psychological distress (Zielinski, Systrom, & Rose, 2019; Meeus et al., 2013). Given the interplay among these factors, ASMR may offer multidimensional benefits by simultaneously reducing sleep-related arousal and fatigue-related exhaustion, thereby improving the overall well-being of chronic pain sufferers.

While most ASMR research has focused on relaxation and sleep, understanding its potential relevance to underlying pain mechanisms is equally important. McErlean and colleagues (2022) proposed that ASMR might act as a buffer for heightened sensory sensitivity rather

than a direct analgesic to target pain itself, an interpretation consistent with chronic pain models emphasising central sensitisation and descending pain modulation, i.e., altered affective 'gating' (Latremoliere & Woolf, 2009; Bushnell, Čeko, & Low, 2013). The present study does not directly test this buffering mechanism but examines its potential downstream manifestations. In other words, whether repeated ASMR exposure can alleviate symptom clusters linked to affective-autonomic dysregulation, such as fatigue, sleep disturbance, and pain interference, that are otherwise difficult to address pharmacologically.

Although ASMR has not yet been systematically evaluated as an intervention for pain, its mechanisms show overlap with neuromodulatory treatments already used in clinical settings. For instance, transcutaneous vagus nerve stimulation (tVNS) has demonstrated benefits for mood, sleep, and pain by increasing vagal tone and reducing sympathetic activity (Costa et al., 2024; Wu et al., 2022; Bretherton et al., 2019). Given reports that ASMR similarly reduces heart rate and promotes vagal-like states (Poerio et al., 2018), it is plausible that ASMR provides a naturalistic analogue of vagal stimulation. This aligns with the recently described Proximity Prediction Hypothesis (PPH), which proposes that ASMR arises when near-field auditory cues are processed as predictions of affiliative touch, triggering parasympathetic shifts towards a state of calm (Flockton et al. 2025).

The Pain, Enjoyment, and General Activity (PEG) scale (Krebs et al., 2009) was selected in the present study as a concise yet validated measure of pain severity and its functional impact. Unlike longer pain inventories, the PEG captures core dimensions of the chronic pain experience, average pain intensity, interference with enjoyment of life, and interference with daily activity, making it particularly relevant for evaluating ASMR's potential benefits. Given that ASMR is widely used to promote relaxation, pleasure, and rest, it may plausibly influence both pain perception and the affective and behavioural consequences of pain, as indexed by the enjoyment and activity items of the PEG scale.

This investigation aimed to evaluate whether daily exposure to ASMR sounds over a two week period could improve key clinically relevant outcome measures in a sample of individuals with chronic pain. Specifically, changes in pain intensity, sleep disturbance, fatigue severity, anxiety, and depression were investigated using the validated self-report questionnaires mentioned above. Participants were randomly assigned to either an ASMR sound exposure condition where they were asked to listen every day to a selection of sound stimuli designed to elicit ASMR in some people, or a delayed-access control group who were not provided with the sounds until after the two week period. It was hypothesized that the daily listening ASMR group would demonstrate significantly greater improvements across all outcome domains compared to the delayed-access controls. This preregistered, longitudinal

design represents the first controlled study of ASMR in a chronic pain population, with potential to inform novel home-based interventions for long-term health conditions.

4.3 Methods

4.3.1 Participants

An initial sample of 120 participants enrolled. Eligibility required self-reported chronic pain; a formal diagnosis was not required due to recent findings indicating that a need for official diagnoses could be a barrier to facilitating research within that population (Hansford et al., 2024) no additional exclusion criteria were applied. Participants were recruited via the University of York online participant pool (course credit) and the University of the Third Age (U3A; voluntary, no compensation). Three surveys were scheduled across two weeks. Inclusion in the primary analyses required completion of at least two surveys; responses that could not be reliably linked across timepoints (missing or mismatched unique codes) were excluded, and duplicate submissions at a given timepoint were resolved by retaining the earliest or most complete response. After cleaning, the analytic sample comprised 64 participants (Experimental $n = 33$; Control $n = 31$) This sample ranged in age from 18 to 74 years ($M = 22.1$, $Mdn = 19$) and included 55 females. Because change scores were analysed, the number of non-missing observations varied by outcome and timepoint (see Table 1 for degrees of freedom).

4.3.2 Materials

The primary materials for this study were a series of validated clinical outcome questionnaires, administered via Qualtrics. These included the Pain, Enjoyment of life, and General activity (PEG) scale (Krebs et al., 2009) for pain intensity, the Insomnia Severity Index (ISI; Bastien et al., 2001), the Fatigue Severity Scale (FSS; Krupp et al., 1989), and the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983).

Pain, Enjoyment of life, and General activity (PEG).

Pain interference/intensity was assessed with the 3-item PEG scale (Krebs et al., 2009), adapted from the Brief Pain Inventory. Items are rated 0-10 ("no pain/interference" to "worst imaginable"), and scores were averaged to yield a total from 0-10, with higher scores indicating greater pain-related interference/severity. A representative item is: "What number best describes your pain on average in the past week?" In prior validation work, the PEG has demonstrated good internal consistency (Cronbach's $\alpha \approx .73-.89$) and strong convergent validity with longer pain inventories (Krebs et al., 2009).

Insomnia Severity Index (ISI).

Insomnia symptoms over the past two weeks were measured with the 7-item ISI (Bastien et al., 2001). Items use 0-4 response options and are summed to a total of 0-28 (higher = worse insomnia). Conventional interpretive bands are: 0-7 (no clinically significant insomnia), 8-14 (subthreshold), 15-21 (moderate), and 22-28 (severe). An example item is: "Difficulty falling asleep." The ISI has demonstrated acceptable to good internal consistency in previous research (Cronbach's $\alpha \approx .74-.91$), along with good test-retest reliability and sensitivity to change (Bastien et al., 2001).

Fatigue Severity Scale (FSS).

Daytime fatigue was assessed with the 9-item FSS (Krupp et al., 1989). Items are rated 1-7 ("strongly disagree" to "strongly agree") and averaged to produce a mean score from 1-7, with higher values indicating greater fatigue severity. A representative item is: "Fatigue interferes with my physical functioning." Validation work has shown excellent internal consistency ($\alpha \approx .89$) and good test-retest reliability ($\approx .84$) across medical populations.

Hospital Anxiety and Depression Scale (HADS).

Mood symptoms were measured with the HADS (Zigmond & Snaith, 1983), comprising 7 Anxiety and 7 Depression items (0-3 each), summed to subscales ranging 0-21. Standard interpretive bands are: 0-7 (normal), 8-10 (borderline), and 11-21 (clinical). Example items include: "I feel tense or 'wound-up'" (Anxiety) and "I still enjoy the things I used to enjoy" (Depression; reverse scored). Typical internal consistency is good ($\alpha \approx .80$ for each subscale), and the HADS has extensive evidence for construct validity in medical settings.

Engagement with ASMR (experimental group only).

In Surveys 2 and 3, participants in the experimental arm reported listening frequency (e.g., "More frequently than once a day," "Once every day this week," "Less frequently than once a day," "I have not been able to listen at all this week") and enjoyment of the provided sounds ("Yes, all of them"; "Yes, some of them"; "No, none of them"). These items were used only in exploratory analyses.

4.3.3 Stimuli

The auditory stimuli consisted of 18 30-second audio clips (e.g., paper folding, tapping, stroking, brushing) designed to plausibly elicit tingling or relaxation responses. All clips were created and professionally recorded by an established ASMR content creator (Nader, 2023; Appendix C) to ensure ecological validity and high audio quality. Participants in the daily listening group were provided with a Google Drive link granting access to the stimuli throughout the two-week intervention period, with instructions to listen once per day at a time

of their choosing. Participants in the delayed listening (control) group received access to the same Google Drive folder only after completing the two-week study period, thereby controlling for expectancy and exposure effects.

4.3.4 Design

The study employed a mixed, repeated measures design. The between-subjects factor was group assignment, with participants randomly allocated to either the experimental or control group following the completion of the baseline survey (T1). Participants in the experimental group were instructed to listen to ASMR sounds from an online repository at least once per day for two weeks. For all participants surveys were administered at three timepoints: baseline (T1), end of week one (T2), and end of week two (T3). Participants in the control group did not receive any ASMR sound exposure during the study period but were told they would be granted access to the sounds after completing the final survey. The control group served as a delayed-access comparison condition. All participants followed the same timeline and survey structure. The design was online, to avoid the need for participants to travel, given previous research highlighting this as a factor impacting the experience of chronic pain sufferers when asked to participate in research (Hansford et al., 2024).

4.3.5 Procedure

Participants first completed Survey 1 (T1), which served as the baseline assessment. Following completion of this survey, participants were randomly assigned via Qualtrics' built-in randomization function to either the experimental or control group. Randomization occurred at the individual participant level and after inputting their email addresses at the end of the first survey, pre-written follow-up emails were triggered to be sent to those email addresses from Qualtrics via an embedded data system.

Participants assigned to the experimental group were informed via the automated follow-up email, that they had been placed in the 'Daily Sound Group' and were provided with access to an online repository of sounds that could elicit ASMR. They were instructed to listen to at least one sound per day for the duration of the two week study. In Surveys 2 and 3, administered at the end of each subsequent week (7 and 14 days from T1, respectively), participants in the experimental group were asked additional questions about their ASMR listening frequency and enjoyment.

Participants assigned to the control group were informed via the other automated follow-up email that they had been placed in the 'Delayed Sound Group'. They were told they would not have access to the experimental sounds (created to plausibly induce ASMR) during the

study period but would be provided with the sound repository upon completion of the final survey. Surveys 2 and 3 for the control group were identical to those completed by the experimental group, except that they did not include any sound-related questions.

All participants were asked to enter the same unique code at each timepoint to allow their responses to be matched across the three surveys. Links to Surveys 2 and 3 for the experimental group and the alternative survey 2 and survey 3 for the control group were sent to the respective participants via the same Qualtrics trigger system of pre-written, automated emails.

4.3.6 Analysis

The main analyses were preregistered (<https://doi.org/10.17605/OSF.IO/PV5WS>) and conducted using linear mixed effects models (LMMs) to account for the repeated measures structure of the study. The scripts used to perform these analyses are accessible via a Github repository found in Appendix A of this thesis. The primary dependent variables were difference scores, which were calculated to reflect changes from baseline to the end of Week 1 (T2 - T1) and from baseline to the end of Week 2 (T3 - T1). Analyses were conducted in R (4.4.3) using lme4, lmerTest, emmeans, and the tidyverse. Ninety-three participants contributed data; because of missing data across outcomes and timepoints, the number of usable observations (and therefore degrees of freedom) varied by model. No imputation was performed; all models used all available cases under the LMM missing-at-random assumption.

For each outcome (pain intensity, sleep disturbance, fatigue severity, anxiety, and depression), an LMM was fitted with fixed effects of Group (Experimental vs. Control) as a between-subjects factor, Timepoint (W2 vs. W1) as a within-subjects factor, and their Group × Timepoint interaction, and a random intercept for Participant to account for the non-independence of repeated observations. Sum-to-zero (effect) coding was used for fixed factors so that the intercept corresponds to the Control group at W1, and so that the main effects are marginal (i.e., averaged over the other factor). Satterthwaite degrees of freedom were obtained via lmerTest. Two-tailed tests used $\alpha = .05$.

The preregistered primary hypothesis concerned the main effect of Group (Experimental vs. Control) on change scores averaged over timepoints. Secondary, exploratory tests examined the main effect of Timepoint (W2 vs. W1) and the Group × Timepoint interaction (differential W2-W1 change by group), with no specific predictions. Type-III tests for Group, Timepoint, and Group × Timepoint are reported in Table 1. Fixed-effect estimates (β),

standard errors (SE), df, t, p, and 95% confidence intervals for all terms (intercept, Group, Timepoint, Group × Timepoint) are reported in Table 2. Where helpful for interpretation, estimated marginal means and planned contrasts were obtained with emmeans; when post-hoc pairwise tests were conducted, Holm-Bonferroni correction was applied.

Model assumptions were evaluated using residual Q-Q plots, residual-versus-fitted plots, and Shapiro-Wilk tests; no remedial transformations were applied. To contextualize the null findings on mood outcomes, descriptive analyses summarised baseline symptom distributions (means, standard deviations, and clinical cutoffs) and visualised change score distributions with histograms; the proportion of participants showing any improvement was also reported. All descriptive analyses were conducted using the tidyverse and ggplot2 packages in R (4.4.3).

Finally, exploratory correlational analyses were conducted within the ASMR group to test whether self-reported listening frequency and enjoyment ratings (collected at T2 and T3) were associated with the magnitude of any clinically relevant improvements. Pearson correlations were used, and Holm-Bonferroni correction was applied to control for multiple comparisons. These analyses were not preregistered as confirmatory tests and are reported as exploratory.

4.4 Results

4.4.1 Primary Analyses

Primary analyses used linear mixed-effects models (LMMs) on change-from-baseline scores (change = post - baseline). For each outcome (PEG, ISI, FSS, HADS anxiety, and HADS depression measures), an LMM with fixed effects of Group (Experimental vs Control), Timepoint (Week 1 [T2 - T1] vs Week 2 [T3 - T1]), and their Group × Timepoint interaction, was fitted, along with a random intercept for Participant. Sum-to-zero (effect) contrasts and Satterthwaite degrees of freedom were used. By construction, negative change indicates improvement on the clinically meaningful measures used. Ninety-three participants contributed data; usable observations varied by outcome/timepoint (see degrees of freedom). Type-III tests are summarized in Table 1, and fixed-effect estimates (β , SE, df, t, p, 95% CI) are shown in Table 2. Internal consistency at baseline was acceptable to good across measures in the present sample: PEG ($\alpha = .84$), ISI ($\alpha = .79$), FSS ($\alpha = .88$), HADS Anxiety ($\alpha = .83$), and HADS Depression ($\alpha = .73$).

Table 1. Type-III tests from linear mixed-effects models of change-from-baseline.

Outcome	Effect	df (num, den)	F	p
PEG	Group	1, 103.55	3.64	.059
PEG	Group × Timepoint	1, 89.52	2.39	.126
PEG	Timepoint	1, 89.52	0.02	.894
FSS	Group	1, 133.98	4.96	.028
FSS	Group × Timepoint	1, 87.64	0.09	.767
FSS	Timepoint	1, 87.64	1.21	.275
ISI	Group	1, 92.02	1.30	.257
ISI	Group × Timepoint	1, 90.06	1.13	.290
ISI	Timepoint	1, 90.06	0.86	.356
Anxiety	Group	1, 88.44	0.36	.550
Anxiety	Group × Timepoint	1, 130.16	0.51	.474
Anxiety	Timepoint	1, 130.16	0.12	.734
Depression	Group	1, 87.78	0.27	.606
Depression	Group × Timepoint	1, 126.40	1.82	.180
Depression	Timepoint	1, 126.40	0.37	.543

Note. Each outcome (PEG, ISI, FSS, Anxiety, Depression) was analysed with an LMM predicting change scores ($W1 = T2-T1$; $W2 = T3-T1$) from Group (Experimental vs. Control), Timepoint ($W1, W2$), and their interaction, with a random intercept for participant. Sum-to-zero contrasts were used; degrees of freedom were Satterthwaite-approximated. The F statistics, numerator/denominator dfs, and p values are reported; negative change indicates improvement. Significant effects ($p < .05$) are interpreted in the text and highlighted in pink in the table.

Table 2. Fixed-effect estimates from linear mixed-effects models of change from baseline.

Outcome	Term	β	SE	df	t	p	95% CI
PEG	Intercept (Grand mean change)	-0.45	0.22	60.66	-1.99	0.051	[-0.89, -0.01]
PEG	Group	0.37	0.19	103.55	1.91	0.059	[-0.01, 0.75]
PEG	Timepoint (W2 vs W1)	0.01	0.1	89.52	0.13	0.894	[-0.18, 0.21]
PEG	Group x Timepoint	-0.15	0.1	89.52	-1.54	0.126	[-0.35, 0.04]
FSS	Intercept (Grand mean change)	0.3	0.18	59.43	1.7	0.094	[-0.05, 0.65]
FSS	Group	0.3	0.14	133.98	2.23	0.028	[0.04, 0.57]
FSS	Timepoint (W2 vs W1)	-0.06	0.05	87.64	-1.1	0.275	[-0.17, 0.05]
FSS	Group x Timepoint	-0.02	0.05	87.64	-0.3	0.767	[-0.12, 0.09]
ISI	Intercept (Grand mean change)	-0.47	0.44	60.59	-1.08	0.283	[-1.33, 0.38]
ISI	Group	0.45	0.39	92.02	1.14	0.257	[-0.32, 1.22]

ISI	Timepoint (W2 vs W1)	0.21	0.23	90.06	0.93	0.356	[-0.24, 0.67]
ISI	Group x Timepoint	-0.25	0.23	90.06	-1.06	0.29	[-0.70, 0.21]
Anxiety	Intercept (Grand mean change)	-0.1	0.49	68.28	-0.21	0.836	[-1.07, 0.87]
Anxiety	Group	0.28	0.47	88.44	0.6	0.55	[-0.64, 1.20]
Anxiety	Timepoint (W2 vs W1)	0.11	0.32	130.16	0.34	0.734	[-0.52, 0.73]
Anxiety	Group x Timepoint	-0.23	0.32	130.16	-0.72	0.474	[-0.85, 0.40]
Depression	Intercept (Grand mean change)	0.12	0.31	65.5	0.37	0.715	[-0.50, 0.73]
Depression	Group	0.15	0.29	87.78	0.52	0.606	[-0.42, 0.73]
Depression	Timepoint (W2 vs W1)	-0.12	0.19	126.4	-0.61	0.543	[-0.49, 0.25]
Depression	Group x Timepoint	-0.25	0.19	126.4	-1.35	0.18	[-0.62, 0.12]

Note. Change = post - baseline; W1 = T2 minus T1; W2 = T3 minus T1. Each model included fixed effects of Group (Experimental vs Control), Timepoint (W2 vs W1), and their Group by Timepoint interaction, with a random intercept for Participant. Sum-to-zero (effect) contrasts and Satterthwaite degrees of freedom were used. The Intercept is the grand mean change across groups and timepoints (under effect coding). Group is the main effect of

Experimental versus Control averaged over timepoints (Experimental minus Control). Timepoint is the main effect of W2 versus W1 averaged over groups. Group by Timepoint is the differential W2-minus-W1 change between groups (i.e., the difference-in-differences). Significant effects ($p < .05$) are interpreted in the text and highlighted in pink in the table. Negative coefficients indicate improvement (more negative change). Type-III tests corresponding to these terms are reported in Table 1.

Pain, Enjoyment, and General Activity (PEG)

The main effect of Group approached significance, $F(1, 103.55) = 3.64, p = .059$, suggesting a potential non significant trend toward greater improvements in the experimental group. No significant main effect of timepoint, $F(1, 89.52) = 0.02, p = .894$, or interaction, $F(1, 89.52) = 2.39, p = .126$, was observed. Baseline PEG scores were available for 77 participants. The mean PEG score at baseline was 4.72 (SD = 1.62), indicating moderate pain-related interference on average. Although the PEG is primarily intended to track change over time rather than define fixed clinical thresholds (Krebs et al., 2009), scores can be descriptively contextualised using conventional 0-10 pain severity bands (0-3 mild, 4-6 moderate, 7-10 severe). The majority of participants fell within the moderate range.

Fatigue Severity Scale (FSS)

A significant main effect of Group was observed, $F(1, 133.98) = 4.96, p = .028$. Estimated group difference (averaged over W1 and W2; Exp - Ctrl) was -0.60 (SE = 0.27), $t(134.25) = -2.20, 95\% \text{ CI } [-1.14, -0.06], p = .030$. Because change scores were computed as post - baseline, negative values index improvement; thus, the ASMR group showed greater reductions in fatigue than controls. There were no significant effects of Timepoint, $F(1, 87.64) = 1.21, p = .275$, nor Group \times Timepoint, $F(1, 87.64) = 0.09, p = .767$ (Table 1).

Insomnia Severity Index (ISI)

No significant effects were found. The main effect of Group, $F(1, 92.02) = 1.30, p = .257$; timepoint, $F(1, 90.06) = 0.86, p = .356$; and group \times timepoint interaction, $F(1, 90.06) = 1.13, p = .290$, were all non-significant.

Anxiety

No significant effects were observed. The main effect of Group, $F(1, 88.44) = 0.36, p = .550$; timepoint, $F(1, 130.16) = 0.12, p = .734$; and the interaction, $F(1, 130.16) = 0.51, p = .474$, were all non-significant.

Depression

Similarly, no significant effects were found. The main effect of Group, $F(1, 87.78) = 0.27, p = .606$; timepoint, $F(1, 126.40) = 0.37, p = .543$; and the group \times timepoint interaction, $F(1, 126.40) = 1.82, p = .180$, were non-significant.

4.4.2 Residual Diagnostics

Residual diagnostics (Q-Q plots and residual-versus-fitted plots) indicated acceptable fit for PEG and ISI: residuals were approximately normal (Shapiro-Wilk, PEG $W = 0.987, p = .166$; ISI $W = 0.994, p = .725$) with roughly constant variance. FSS showed mild departures (Shapiro-Wilk $W = 0.981, p = .038$) and slight fanning in residuals. HADS Anxiety ($W = 0.971, p < .001$) and HADS Depression ($W = 0.956, p < .001$) exhibited clear non-normality and some heteroscedasticity, consistent with the right-skew/floor effects noted for baseline mood symptoms. Given that LMM fixed-effect tests are generally robust to modest deviations from normality, results for outcomes with non-ideal diagnostics are interpreted with appropriate caution (Figure S2 in Appendix B).

4.4.3 Exploratory Analyses

Exploratory correlational analyses were conducted within the experimental group to assess whether self-reported ASMR listening frequency and enjoyment by the end of the two week intervention (measured at T3) were associated with clinical outcome changes from baseline (T3 - T1). Greater enjoyment at T3 was associated with greater improvement in PEG (T1 - T3), $r(28) = -.61, p < .001, 95\% \text{ CI } [-.80, -.32], \text{ Holm-Bonferroni } p = .004$. No other correlations reached significance ($r \leq .20, \text{ all } p \geq .23; \text{ all } p_{(\text{Holm})} = 1.000$). All other correlations between engagement (i.e. measures of reported enjoyment or listening frequency) and any improvement in the other outcome measures, of fatigue, insomnia, anxiety, or depression, were small in magnitude ($r \leq .20$), had confidence intervals that included zero, and did not survive correction for multiple tests; full results are provided in Table S2 of the Supplementary Material.

4.4.4 Self-reported Listening Frequency

Within the ASMR listening group ($n = 33$), self-reported engagement indicated variable adherence to the daily listening protocol. During Week 1, all respondents reported at least some listening that week, with the majority (79%) engaging once or more per day in the week. By Week 2, adherence declined: 45% reported daily listening, while 21% indicated that they did not listen at all that week. Across the two-week intervention period, approximately half of participants ($\approx 45\%$) maintained daily or more frequent listening on average. These patterns contextualize the exploratory findings, suggesting that while enjoyment was reliably associated with clinical improvement, listening frequency itself diminished over time. The reduced engagement in Week 2 may partly explain the absence of further improvement between T2 and T3.

4.4.5 Baseline Mood Symptoms and Change Scores

To further contextualize these null findings, descriptive statistics for baseline mood symptoms and change scores were explored. At T1, anxiety scores were moderate on average and depression scores were low. Based on HADS cut-off points, 58% of participants met the clinical threshold for anxiety (≥ 11), 22% were borderline (8-10), and 20% were normal; for depression, 70% were normal, 27% borderline, and 3% clinical. This pattern (especially the concentration at the low end for depression) suggests possible floor effects that may limit sensitivity to detect change. Full descriptive summaries (means, SDs, ranges) and histograms of change scores are provided in Table S1 and Figure S1 (Supplementary Material in Appendix B of this thesis), where negative change denoted improvement.

4.5 Discussion

This preregistered, longitudinal design represents the first controlled study of ASMR applied as a therapeutic intervention in a chronic pain population, with potential to inform novel home-based interventions. By examining changes across multiple symptom domains, the study aimed to determine whether ASMR's proposed affective-autonomic modulation extends beyond subjective relaxation to hypothesized significantly greater improvements across validated measures of pain, insomnia, fatigue, anxiety, and depression.

The primary analysis revealed that the experimental group experienced a significant improvement in fatigue severity compared to the control group, as indicated by the main effect of Group on the Fatigue Severity Scale (FSS). This effect was consistent across both post-baseline timepoints, suggesting a stable reduction in self-reported fatigue following

daily exposure to ASMR sounds. This finding aligns with previous reports that ASMR can promote relaxation and may contribute to reductions in fatigue by supporting rest and recovery (Barratt & Davis, 2015). Notably, the present pattern of results echoes the preliminary findings of McErlean, Ellis, and Walsh (2022), who explored ASMR's effects on experimentally induced pain. In their small sample study, ASMR experiencers showed greater pain sensitivity than controls, with no reliable difference in pain tolerance, leading the authors to propose that ASMR's influence operates primarily through affective reappraisal or autonomic modulation rather than direct nociceptive suppression. The current intervention is partly consistent with this, finding a robust reduction in fatigue, but also, notably, a near significant trend toward reduced pain interference ($F(1, 103.55) = 3.64, p = .059$). While preliminary, this emerging pattern suggests that ASMR may not only engage restorative and interoceptive mechanisms supporting energy regulation and affective calm, but may also begin to alleviate the functional burden of pain through affective-autonomic recalibration.

This reduction in fatigue among participants exposed to ASMR sounds may therefore reflect the capacity of ASMR to downregulate physiological arousal and facilitate parasympathetic activation. Prior research has linked ASMR experiences with reductions in heart rate (Poerio et al., 2018) and increased skin conductance (Fredborg et al., 2018), consistent with a shift toward vagal dominance. Given that chronic fatigue is frequently associated with dysregulation of the autonomic nervous system (Meeus et al., 2013), it is plausible that ASMR acts to normalize arousal levels, promoting bodily rest and improved energy regulation. From a theoretical standpoint, these effects may be understood through the lens of the Proximity Prediction Hypothesis (PPH), which posits that ASMR is a perceptual state triggered by multisensory predictions of safe proximity and affiliative touch (Flockton et al., 2025). According to PPH, such predictions downregulate noradrenergic tone and increase vagal gain, thereby supporting recovery-related processes through reduced fatigue and improved sleep quality. The fact that fatigue symptoms improved significantly from ASMR exposure, while pain and mood symptoms did not, may reflect the sensitivity of fatigue to subtle shifts in autonomic balance and interoceptive prediction, as proposed by the PPH model.

Chronic pain and fatigue commonly co-occur and likely share central mechanisms (e.g., altered nociceptive processing and autonomic/HPA-axis imbalance), which makes fatigue a legitimate and clinically important treatment target in chronic pain rather than a secondary by-product of pain change (Kindler et al., 2011). The present finding of reduced fatigue within the ASMR exposure group is therefore consistent with accounts that emphasise autonomic down-regulation and recovery processes and

aligns with recommendations to assess and treat fatigue as a primary outcome in chronic pain care (Eccles & Davies, 2021).

The finding that ASMR significantly reduced fatigue, but not insomnia (which was non-significant), may reflect important differences in how these constructs are experienced and measured. While both are associated with energy depletion, the FSS captures subjective exhaustion during waking hours, whereas the Insomnia Severity Index (ISI) specifically measures difficulty initiating or maintaining sleep. ASMR's effects may primarily influence perceived restfulness and daytime recovery through downregulation of arousal, rather than through direct modification of sleep onset or architecture. This is consistent with the PPH, which proposes that ASMR reduces noradrenergic tone and increases vagal activity; mechanisms more likely to impact bodily relaxation and energy conservation than entrenched cognitive processes like rumination or circadian misalignment that often underlie clinical insomnia. Moreover, prior studies linking ASMR to sleep improvements are typically based on self-selected users engaging with ASMR acutely before bed (e.g., Poerio et al., 2022), rather than over a sustained intervention period in a general chronic pain population. Therefore, while ASMR may support sleep in real-world usage contexts, its measurable impact on insomnia symptoms may require more personalized application, consistent listening at bedtime, or targeting individuals with high sleep reactivity at baseline. Importantly in the present study, listening time was neither prescribed nor standardised, and bedtime exposure was not ensured. Consequently, null effects on insomnia could plausibly reflect suboptimal timing (i.e., participants not listening before bed), rather than an absence of efficacy. Future trials should schedule or verify bedtime listening (e.g., via app timestamps or daily logs) to test sleep-specific effects.

Importantly, pain related suffering can lessen even when nociceptive transmission remains stable, because affective and cognitive reappraisal mechanisms determine how pain is experienced and integrated into behaviour (Bushnell et al., 2013). This distinction is central to contemporary chronic pain models that view restoration of descending inhibitory and emotional-regulation pathways as therapeutic in itself. Within this framework, ASMR may influence the meaning and emotional colouring of pain, dampening its interference, without directly altering intensity.

For the Pain, Enjoyment, and General Activity (PEG) scale, the main effect of Group approached significance, suggesting a potential trend toward improved pain-related functioning in the experimental group. The trend is interesting and may reflect a modest benefit of ASMR exposure on pain-related functioning but should be interpreted cautiously as it did not reach significance. Notably however, the direction of the estimate is consistent

with the exploratory finding that greater enjoyment of the ASMR sounds was associated with larger improvements on PEG within the experimental group. This convergence suggests that any ASMR-related benefit for pain interference may depend on engagement and responsiveness.

Prior work has demonstrated that the PEG is a brief yet sensitive tool for detecting clinically meaningful change in chronic pain populations (Reed et al., 2024; Krebs et al., 2009). From a mechanistic perspective, the PPH model posits that ASMR operates by downregulating noradrenergic arousal and increasing vagal gain in response to sensory cues of affiliative proximity; this process may attenuate the affective component of pain, reflected in measures like the PEG, even in the absence of changes in sensory intensity. In line with this account, within the ASMR group, higher enjoyment ratings at T3 were associated with larger PEG improvements from T1 to T3, suggesting that pleasantness and engagement may index the extent to which ASMR engages the proposed mechanism. As such, these results may point to an analgesic potential of ASMR that deserves further investigation in larger or more symptom-targeted samples. This interpretation again echoes McErlean et al. (2022), who argued that ASMR's potential analgesic properties may emerge indirectly, through changes in emotional appraisal and autonomic tone, rather than by increasing pain thresholds directly. Their observation that ASMR-prone individuals are more sensitive to pain but also more emotionally responsive suggests that ASMR may modulate how pain is experienced rather than how it is detected. The modest, engagement-linked improvements in PEG scores observed in the current study are therefore consistent with the notion of affective gating, where pleasant sensory cues reduce the unpleasantness or interference of ongoing pain without necessarily lowering its intensity.

No significant effects were observed for anxiety and depression outcomes, indicating that daily ASMR exposure did not produce measurable improvements in these outcomes within this chronic pain sample over the two week intervention period. This result could be understood in light of several contextual considerations. First, baseline mood score distributions suggested limited room for improvement, particularly for depression, raising the possibility of reduced sensitivity; however, this cannot be separated from the alternative explanation, that effects were genuinely null. Second, the study was not powered to detect small mood effects, and engagement timing was not standardised or recorded, which may further attenuate detectable change. Third, in chronic pain samples, anxiety and depression are often closely coupled to pain interference; given that between-group differences on PEG did not reach conventional significance, large secondary mood improvements would not be expected. Residual diagnostics indicated some non-normality and heteroscedasticity for the

mood models, which can reduce power, but mixed-model fixed-effect tests are reasonably robust, so strong claims either way are not warranted. Future work that recruits participants with elevated baseline mood symptoms, increases power, and tests whether mood change is mediated by pain-interference change, would better adjudicate between reduced sensitivity and truly null effects. The current findings diverge from earlier studies that observed broader benefits of ASMR on pain and mood. However, those studies often involved participants actively seeking ASMR for emotional regulation or sleep, which may have amplified the intervention's effects, due to elevated individual baseline levels in these domains. ASMR responses vary considerably across individuals, and population-level surveys suggest that the greatest benefits are found among those with high baseline anxiety, trait neuroticism, or explicit interest in ASMR (Smith et al., 2017; Barratt & Davis, 2015).

In contrast, the fatigue model showed only minimal violations of statistical assumptions, lending greater confidence to the group difference observed in this outcome. Fatigue may be particularly responsive to ASMR because of its association with mental relaxation, decreased somatic arousal, and improved sleep onset; mechanisms often reported anecdotally by ASMR listeners (Barratt et al., 2017). As Eccles & Davies (2021) note, mood symptoms in chronic pain are multifactorial and may not shift over short interventions unless the relevant drivers (e.g., sleep disturbance, autonomic arousal, activity patterns) are directly targeted. This may help explain why mood outcomes did not change in parallel with fatigue in the current study.

Although not preregistered, exploratory analyses suggested that the extent to which participants engaged with and enjoyed the ASMR intervention may have played a meaningful role in its therapeutic impact. Participants who reported enjoying the ASMR sounds showed notably greater reductions in pain-related interference, as measured by PEG scores. These results, while preliminary, support the hypothesis that ASMR's effectiveness may partially depend on individual responsiveness or preference, in line with previous literature highlighting variability in ASMR susceptibility (Fredborg et al., 2017; Poerio et al., 2018). The lack of substantial associations for depression may also reflect the previously discussed floor effect, as most participants began the study with HADS depression scores in the normal or borderline range, limiting the potential for measurable improvement. For insomnia, it is possible that the timing of ASMR listening influenced its effectiveness. Prior studies investigating ASMR's effects on sleep typically involve listening before bedtime (e.g., Hardian et al., 2020), whereas the present study did not control for or instruct when participants should engage with the sounds. As such, future research might benefit from examining whether listening time moderates the relationship between ASMR exposure and

improvements in sleep quality. These results support the hypothesis that ASMR's effectiveness may partially depend on individual responsiveness and contextual factors, offering useful directions for future personalised or time-targeted interventions.

These exploratory findings also provide tentative support for the PPH model, which posits that ASMR elicits a parasympathetic, homeostatic response by simulating the sensory features of close, affiliative contact. Specifically because this study found that greater enjoyment of ASMR sounds was modestly associated with greater reductions in anxiety symptoms. This aligns with the idea that perceived pleasantness may reflect successful matching between predicted and received sensory input in socially safe contexts, resulting in decreased arousal and improved regulation of internal states. This interpretation is consistent with neuroaffective models proposing that affective touch (e.g., slow, gentle stroking that activates CT afferents) plays a crucial role in social interoception and homeostasis - the brain's ongoing effort to maintain internal equilibrium (von Mohr & Fotopoulou, 2018). Affective touch is thought to contribute not only to soothing and pain relief but also to emotion regulation by facilitating predictions of safety and social support (Crucianelli et al., 2016). By mimicking such sensory characteristics, ASMR may offer listeners a socially relevant cue that recalibrates emotional and physiological responses, particularly when the sounds are rated as enjoyable, as seen in the present investigation's exploratory analyses.

Mechanistically, ASMR may alleviate aspects of the chronic pain experience not by directly suppressing nociceptive transmission, but by restoring balance within the affective-autonomic systems that regulate pain perception. Chronic pain is increasingly recognised as a disorder of dysregulated descending control and emotional modulation, in which heightened sympathetic arousal and impaired prefrontal-limbic gating amplify pain salience, fatigue, and affective distress (Bushnell, Čeko, & Low, 2013; Eccleston & Crombez, 1999). Within this framework, interventions that enhance parasympathetic tone or re-engage affective safety cues can meaningfully improve well-being even when sensory pain remains unchanged. The present pattern, a significant reduction in fatigue and a trend toward reduced pain interference, tentatively supports this view and may indicate that ASMR's effects on pain are somewhat stronger than previously reported in smaller laboratory studies. While McErlean, Ellis, and Walsh (2022) found no reliable change in pain tolerance in their limited sample, the current data hint that prolonged or repeated ASMR exposure might influence pain perception more meaningfully, perhaps through cumulative modulation of affective-autonomic tone. Both sets of findings converge on the same mechanistic pathway;

ASMR appears to alter the emotional appraisal and interoceptive meaning of pain rather than its direct sensory magnitude.

4.5.1 Limitations and Future Directions

Limitations in this study should be noted. Firstly, adherence to the ASMR listening protocol was self-reported rather than objectively monitored, which limits the precision with which the engagement and response relationship can be inferred. Nevertheless, approximately 45% of participants in the experimental group reported listening to the ASMR sounds at least once per day across the two week intervention, while a smaller subset listened less frequently than once every day across the week, or not at all, during the second week. The observation that significant reductions in fatigue emerged despite this variation in listening frequency suggests that ASMR exposure may exert meaningful effects even under conditions of partial adherence. This resilience to engagement variability is encouraging for the ecological validity and potential scalability of ASMR as a self-directed intervention. However, future work should measure exposure more rigorously, using some kind of digital app tracking, timestamps, or embedded listening verification techniques for at home protocols, or monitored use in clinical protocols using this method, to determine whether higher listening consistency further enhances clinical benefit.

Secondly, residual non-normality in the anxiety and depression models suggests that alternative analytic strategies, such as generalised mixed models, variable transformation, or non-parametric tests, may improve sensitivity in future trials. Third, the generalizability of findings may be limited, as participants were not selected based on ASMR responsiveness or elevated mood or insomniac symptoms at baseline.

While exploratory, the correlation results between enjoyment, frequency of listening, and the clinically relevant outcome measures mentioned above suggest that individual differences in perceived enjoyment may index the degree to which ASMR stimuli engage the neurophysiological mechanisms proposed by the PPH. However, these associations do not rule out nonspecific explanations, including expectancy or placebo effects, demand characteristics, and general relaxation from pleasant audio. More conclusive tests could use an expectancy-matched active control consisting of non-ASMR relaxing audio with matched duration and volume, standardized and balanced briefing scripts delivered verbatim to both groups, and explicit measurement of treatment credibility and expectancy; for example, using the Credibility-Expectancy Questionnaire (Borkovec & Nau, 1972). A preregistered mediation analysis could then contrast ASMR-specific mediators such as self-reported paresthetic tingling or enjoyment, with expectancy. Concurrent physiological recordings

during exposure, such as heart-rate variability and skin conductance, showing effects that remain after adjustment for expectancy, would further strengthen a mechanistic interpretation. It should be noted however that these additional metrics could also necessitate further effort for the participant that may lead to exacerbated fatigue after the experiment - a factor that has been reported in recent work highlighting the barriers in chronic pain research engagement from the perspective of sufferers themselves (Hansford et al., 2024).

Longer intervention periods, stratification by ASMR responsiveness, and targeted recruitment of individuals with clinically significant anxiety or depression may also improve outcome sensitivity to clinically meaningful results in chronic pain sufferers. Also, incorporating manipulation checks and monitoring adherence to the intervention protocol, as mentioned above, would enhance internal validity and aid in interpreting null findings. Future research might assess whether ASMR modulates specific components of pain (e.g., interference, emotional burden) more reliably than intensity, and whether these effects emerge more strongly in individuals with high ASMR sensitivity or emotional reactivity, and with those who listen more frequently.

Although the preregistered hypotheses predicted improvements across all clinical outcomes, including pain interference, the selective improvement in fatigue observed in the current results aligns with contemporary pain theory. Cognitive-affective models of chronic pain emphasise that distress and disability often stem from dysregulation of emotional and autonomic control systems rather than heightened nociceptive transmission alone (Bushnell, Čeko, & Low, 2013; Eccleston & Crombez, 1999). Within this framework, ASMR may act primarily on affective-autonomic processes, reducing arousal, restoring interoceptive balance, and improving energy regulation, which can translate into meaningful gains in wellbeing even when sensory pain ratings are not significantly reduced. Future trials might therefore treat fatigue, sleep disturbance, and mood regulation as primary mechanistic outcomes rather than merely peripheral comorbidities, with pain interference conceptualised as a downstream indicator that may require longer or more intensive exposure to change.

In terms of clinical implications, improving fatigue is clinically meaningful in its own right for people living with chronic pain. Prospective work in sick-listed chronic low back pain shows that patients with substantial fatigue report greater disability over 3-12 months, even after accounting for pain and depressive symptoms, underscoring fatigue's independent contribution to functional limitations (Snekkevik et al., 2014). The significant reduction in fatigue observed here is consistent with models that suggest the maintenance of chronic pain-fatigue constellations is a result of central nervous system dysregulation, including

altered HPA-axis activity, aberrant pain processing, and autonomic imbalance. In their review, Clauw and Chrousos (1997) argued that these central mechanisms can generate overlapping symptom profiles across fibromyalgia and chronic fatigue-type presentations. Framed this way, an intervention that reliably reduces arousal and shifts the autonomic state toward parasympathetic dominance, as ASMR is proposed to do, would be expected to preferentially impact fatigue, even when effects on pain intensity or mood are less evident.

Recent patient-led evidence also emphasizes why fatigue is a practical barrier to engagement for people living with chronic pain. In a mixed-methods study on research participation, Hansford and colleagues found that beyond issues of trust and access, a major barrier cluster reflected ongoing symptoms and comorbidities, with fatigue singled out by patients as a recurrent concern. Participants described how even ostensibly low-demand activities (travel, scheduling, questionnaires) could trigger or worsen fatigue and pain flares, making day-to-day functioning, and optional commitments like research, harder to sustain (Hansford et al., 2024). These patient reports align with the current rationale to treat fatigue as a primary clinical burden in chronic pain and to evaluate it explicitly (alongside sleep) as an outcome that matters to patients, as well as something to consider to improve research protocols in future studies, and ensure participation results in minimal negative effects for the chronic pain sample themselves.

4.5.2 Conclusion

In this preregistered study, daily access to ASMR sounds produced a significant reduction in fatigue severity relative to a no-intervention control, while no reliable effects were observed for pain interference, insomnia, anxiety, or depression. The fatigue finding indicates that ASMR may have value as a non-pharmacological adjunct for energy-related symptoms in chronic pain. Exploratory analyses suggested that greater enjoyment of the sounds was associated with larger improvements in pain-related interference. Notably, this improvement in fatigue emerged despite variable adherence to the listening protocol, suggesting that ASMR's benefits may be robust even under less-than-daily use.

In summary, the pattern is consistent with accounts in which ASMR facilitates autonomic down-regulation, leading most proximally to improved perceived energy (ameliorating fatigue). Any mechanistic interpretation (including the Proximity Prediction Hypothesis) remains tentative and secondary to the present empirical finding: fatigue significantly improved for chronic pain sufferers who listened to ASMR trigger stimuli daily, for two weeks, while pain interference only showed a non-significant trend towards improvement. This may reflect ASMR's primary impact being in affective-autonomic domains rather than on

nociceptive transmission itself, consistent with McErlean et al. (2022). This distinction supports a growing view that chronic pain interventions need not directly suppress pain signals to be clinically valuable; modulating associated states such as fatigue, sleep, and affective load can meaningfully improve quality of life and overall experience of chronic pain (Bushnell, Čeko, & Low, 2013; Eccleston & Crombez, 1999).

Chapter 5:

General Discussion

5.1 Overview and Integration

The present thesis aimed to develop and test a mechanistic account of Autonomous Sensory Meridian Response (ASMR) that could unify its perceptual, neural, and clinical features. Across three complementary chapters, the work progressed from formulating a mechanistic model to explain the phenomenon, to an empirical, cross-modal exploration of its oscillatory dynamics, and finally ended on a longitudinal assessment of its therapeutic potential as a clinical intervention tool. Chapter 2 introduced the Proximity Prediction Hypothesis (PPH), a predictive coding model proposing that near-ear auditory cues simulate the expectation of gentle, affective touch in ASMR. This sensory prior, it argued, triggers a cascade in which simulated tactile processing in the posterior insula and secondary somatosensory cortex leads to beta-band desynchronisation, as well as gamma-band enhancement. This indexes precision-weighted updating, and leads to the subsequent engagement of parasympathetic efferents to produce the characteristic tingling sensation and subsequent calm induced by ASMR.

Chapters 3 and 4 translated this framework into empirical tests spanning neural and behavioural levels. The EEG-MEG study in Chapter 3 provided convergent evidence for the predicted oscillatory dynamics: ASMR triggers did indeed elicit robust beta-band desynchronisation and gamma-band enhancement across both modalities, supporting the proposal that ASMR reflects an active inferential process rather than passive sensory reactivity. Chapter 4 then extended these mechanistic insights to a preregistered, longitudinal intervention in individuals with chronic pain, showing that daily exposure to ASMR sounds significantly reduced fatigue and produced near-significant improvements in pain interference, consistent with the model's predictions around affective-autonomic modulation.

Together, the findings advance ASMR from a descriptive curiosity to a tractable model of predictive interoceptive regulation, illustrating how the brain can use externally generated sensory cues to recalibrate internal bodily states. At the methodological level, this thesis provides the first cross-modal neurophysiological replication of ASMR's oscillatory signature to ever use MEG. It also constitutes the first preregistered clinical evaluation of its therapeutic potential in a chronic pain sample. The sections that follow in this chapter integrate these strands to consider their theoretical implications, mechanistic interpretation, and clinical significance, within both this body of work and future research.

5.2 Theoretical Implications: ASMR as a Predictive-Interoceptive Simulation of Affective Touch

The results reported across chapters converge on the view that ASMR represents a distinctive form of predictive-interoceptive simulation. The Proximity Prediction Hypothesis (PPH) situates ASMR within the broader framework of hierarchical predictive coding, proposing that near-field auditory cues, like whispers, brushing, and tapping sounds, engage Peripersonal Space (PPS) mechanisms involved in the anticipation of affective touch. When these cues are appraised as safe and proximal, the brain's generative model predicts the imminent arrival of gentle, likely CT, contact. This prediction transiently releases sensorimotor inhibition (manifesting as beta-band desynchronisation) and increases the gain on prediction error channels (manifesting as gamma-band enhancement). The ensuing percept, the characteristic tingle, constitutes a somatosensory echo, i.e. a low precision interoceptive illusion that resolves cross-modal prediction conflict in the absence of real tactile input.

Numerous studies converge on the idea that beta-band desynchronisation reflects a release of sensorimotor inhibition during affective or socially meaningful contact. Von Mohr et al. (2018) and Pereira et al. (2025) showed that both real and observed CT-optimal touch lead to pronounced beta suppression over parietal sites, supporting its role in tactile prediction and embodied simulation. Likewise, van Pelt et al. (2016) demonstrated that beta coherence increases with predictable sensory sequences, implicating beta oscillations in the maintenance of top-down priors. Within this framework, ASMR can be viewed as a special case in which auditory cues alone suspend these priors, eliciting tactile anticipation and perceptual disinhibition consistent with a predictive coding account.

This interpretation extends existing predictive coding models of emotion and interoception (Barrett & Simmons, 2015) by demonstrating that top-down expectations about social proximity can elicit bodily sensations normally bound to exteroceptive touch. ASMR thus

provides a naturalistic instance of cross-modal predictive inference, in which an auditory prior is sufficient to generate an interoceptive experience and a concomitant parasympathetic shift. The EEG-MEG results support the first two stages of the PPH cascade, beta suppression and gamma enhancement, offering empirical evidence for a model that was previously theoretical. Crucially, this oscillatory sequence is falsifiable; the absence of a temporally ordered beta-gamma progression, or its decoupling from autonomic indices such as high-frequency heart-rate variability (HF-HRV), would directly contradict the model in future work.

5.2.1 The Biphasic Cascade

The PPH further proposes that ASMR unfolds as a biphasic temporal process, reconciling apparently paradoxical evidence in the literature of both sympathetic and parasympathetic activity associated with ASMR. Near-ear cues, the theory argues, likely first evoke a transient orienting stage, indexed by initial pupil dilation and increased skin conductance (Poerio et al., 2018; Valtakari et al., 2019), reflecting phasic locus coeruleus (LC) activation and heightened sensory precision (Aston-Jones & Cohen, 2005; Dayan & Yu, 2006; Murphy et al., 2014). This momentary alerting state enhances signal gain within the Peripersonal Space network, preparing the listener for potential contact. Once the auditory cue is labelled as affective rather than threatening, prediction error is reclassified as low precision, tonic LC activity subsides, and the vagal system is disinhibited, allowing parasympathetic dominance to emerge (Flockton et al., 2025). The ASMR state therefore exemplifies what might be termed ‘relaxed alertness’, a coordinated sequence between orienting arousal and restorative calm.

This biphasic trajectory aligns closely with the Neurovisceral Integration Framework (Thayer & Lane, 2000), in which the LC and vagus operate as a regulatory see-saw, controlling the organism’s position along the arousal to calm continuum. By capturing both the phasic LC response to proximal stimuli and the ensuing vagal rebound, ASMR potentially offers a rare, empirical example of dynamic CNS-ANS coupling in real time.

5.2.2 From Predictive Coding to Somatosensory Simulation

Conceptually, ASMR diverges from related affective phenomena such as frisson or aesthetic chills, which have been thought to derive their hedonic impact from brief violations of musical expectancy that evoke sympathetic arousal (Grewe et al., 2007; Salimpoor et al., 2011; Huron, 2006; Harrison & Loui, 2014). ASMR, by contrast, involves a transient prediction error, from the absence of expected tactile input, but resolves it through a benign inference

of affective, social contact. The resulting somatosensory echo satisfies top-down predictions of safety, allowing the system to settle into parasympathetic calm. In this sense, the pleasure of ASMR arises not from ongoing uncertainty, but from the successful resolution of prediction error, a shift from local surprise to global confirmation of safety and social contact.

In addition to their distinct phenomenology, ASMR and frisson diverge markedly in their neurophysiological profiles. Neuroimaging work on frisson consistently links chills to heightened sympathetic arousal, expressed neurally as phasic increases in beta and gamma power associated with heightened attentional and reward system engagement (Salimpoor et al., 2011; Mori & Iwanaga, 2017). EEG studies of musical chills frequently report transient gamma bursts over fronto-central regions during peak emotional moments (Mori & Iwanaga, 2017), alongside increased skin conductance and piloerection, indexing arousal-driven salience. MEG work likewise demonstrates strong dopaminergic reward network engagement during frisson, peaking in the NAcc and caudate (Salimpoor et al., 2011), reflecting anticipation-reward cycling.

By contrast, the present cross-modal EEG-MEG study demonstrates that ASMR is characterised by a biphasic, parasympathetically biased oscillatory sequence. During ASMR button press onsets, we observed beta-band desynchronisation in somatosensory and posterior sensor regions, and posterior gamma enhancement, a pattern consistent with sensory prediction and subsequent precision weighted updating, rather than arousal driven surprise. Importantly, this gamma enhancement in ASMR occurs alongside heart rate deceleration and parasympathetic markers (Poerio et al., 2018), whereas gamma bursts in frisson co-occur with sympathetic activation like piloerection, and heightened arousal. Thus, while both ASMR and frisson evoke high frequency activity, their meaning and physiological context diverge: frisson reflects arousal amplifying surprise, whereas ASMR reflects the resolution of a near-ear predictive mismatch into a state of affiliative safety and vagal calm. The presence of this parasympathetic settling phase, evident in both our EEG-MEG results and concurrent behavioural literature, provides strong evidence that ASMR constitutes a distinct affective, sensory process rather than a variant of aesthetic chills.

Although both ASMR and frisson are rooted in sensory-affective dynamics, the argument that they differ fundamentally in the trajectory of those dynamics, made in the introduction to this thesis, has therefore been supported by the neuroimaging results in Chapter 3. In frisson, research links the hedonic rush and goosebumps to sudden musical surprises (crescendos, harmonic shifts) that elicit sympathetic arousal (Mori & Iwanaga, 2017; Harrison & Loui, 2014). ASMR, by contrast, is proposed to begin with a transient local mismatch, where near-field auditory cues predict gentle touch, yet tactile input is absent

(Flockton et al., 2025). This sensory incongruence evokes a brief orienting response, expressed neurally as gamma-band enhancement and phenomenologically as tingling. Crucially, the system then resolves this mismatch through the inference of affective social contact and safety. The resulting state, indexed by beta desynchronisation and parasympathetic engagement, represents successful integration of the missing tactile input into a coherent, safe predictive model, according to the PPH framework. In this sense, the pleasure of ASMR arises not from sustained surprise, but from the resolution of prediction error, transforming initial uncertainty into embodied reassurance.

The PPH also situates ASMR within a social neurocognitive context. Most effective triggers, whispering, brushing, slow rhythmic movements, simulate interpersonal care. These cues are proposed to be processed within brain systems dedicated to social cognition and PPS representation, like the posterior superior temporal sulcus, parietal operculum, and posterior cingulate (Allison et al., 2000; Serino et al., 2009; Björnsdotter et al., 2010). ASMR therefore constitutes a socially grounded interoceptive simulation, in which the brain predicts and internally generates the sensory consequences of affiliative proximity. The tingling percept marks the point at which a social prior ('someone close is gently attending to me') is accepted as fact by the interoceptive hierarchy.

This account aligns with established evidence that PPS representations dynamically integrate auditory and tactile information to anticipate contact (Graziano & Cooke, 2006; Serino, 2019). Near-field cues such as whispering or brushing sounds naturally fall within this proximity coding system, activating sensorimotor networks that prepare for social touch. ASMR may therefore represent a perceptual reuse of the PPS mechanism of multisensory integration, but for affective touch simulation, translating near-ear acoustics into embodied expectations of social contact despite a lack of actual touch ever occurring.

5.2.3 Interoceptive Precision and Individual Variability

Finally, the PPH explains why differences in reports of which individuals experience ASMR in response to specific stimuli. In predictive coding terms, these listeners may assign high precision to affiliative auditory cues and low precision to the absence of confirming tactile input, allowing the top-down prediction to dominate. Others may assign opposite precision weightings, experiencing the same cues as intrusive or aversive, a pattern characteristic of misophonia.

The behavioural findings from Chapter 2 deepen the contrast between ASMR and misophonia, two phenomena that can occasionally be triggered by acoustically similar stimuli

yet produce diametrically opposed affective and autonomic outcomes. Misophonia is defined by disproportionately strong negative emotional reactions, typically anger, disgust, or anxiety, to specific human-generated sounds such as chewing, breathing, or lip-smacking (Schröder, Vulink & Denys, 2013; Rouw & Erfanian, 2018). Physiologically, these triggers evoke heightened skin conductance and defensive sympathetic arousal (Edelstein et al., 2013), and neuroimaging work suggests hyper-reactivity of the anterior insula and salience network to the offending sounds (Kumar et al., 2021). Importantly, some of these cues overlap with common ASMR triggers, a point highlighted by McGeoch and Rouw (2020), who note that the same auditory input can elicit pleasant tingles in some listeners yet strong aversion in others.

The behavioural data presented in Chapter 2 illustrate this kind of divergence in individual responses. Even though mouth sounds were intentionally excluded to avoid provoking misophonia, participants nevertheless showed striking heterogeneity in pleasantness ratings to the same ASMR triggers, and only a subset reported tingles being elicited, to specific triggers. Mixed-effects analyses revealed that pleasantness was the dominant predictor of ASMR reports, each one standard deviation increase in pleasantness increased the odds of reporting ASMR by almost eightfold, whereas arousal contributed minimally. This pattern suggests that listeners differ not in their sensitivity to the acoustic properties of the stimuli, but in the affective value they assign to near-ear cues. In ASMR prone individuals, these cues appear to be interpreted as affiliative and soothing, producing a hedonic boost that facilitates tingles. In others, the same cues elicit neutral or mildly negative affect, consistent with the wider literature showing that responses to ASMR triggers are highly idiosyncratic (Poerio et al., 2022).

Building on this divergence, the PPH reconciles the two phenomena computationally, ASMR and misophonia may reflect opposite outcomes of precision weighting along a social-affective predictive axis. In ASMR, near-ear cues are assigned high precision as indicators of gentle, affective contact, leading to reduced interoceptive prediction error, pleasant tingles, and parasympathetic settling. In misophonia, the same categories of cues may instead be assigned high precision as signals of social threat or contamination, amplifying prediction error and driving sympathetic arousal. While this interpretation extends beyond the claims of existing misophonia models, the present behavioural data, showing large individual differences in pleasantness and in whether tingles occur at all, provide initial support for the idea that divergent priors over identical inputs may underpin the opposing affective outcomes seen across listeners. Considerations around such individual differences

are discussed further, in section 5.5 of this chapter, regarding potential limitations in this thesis.

5.3 Neural and Mechanistic Insights

The combined EEG and MEG analyses revealed a reproducible oscillatory signature of ASMR: central-parietal beta-band desynchronisation co-occurring with midline and posterior gamma-band enhancement across EEG scalp and MEG magnetometer topographies. These dynamics support the notion that ASMR recruits hierarchical predictive mechanisms spanning sensory, motor, and interoceptive systems. Within this framework, beta desynchronisation reflects the relaxation of established sensorimotor predictions, analogous to the release from status quo phenomenon observed during affective touch (Engel & Fries, 2010; Singh et al., 2014). Gamma enhancement is proposed here to mark precision-weighted integration of cross-modal signals as the interoceptive model is updated (Herrmann et al., 2004; Arnal et al., 2011; Todorovic et al., 2011; Kanayama et al., 2009). The joint modulation of these rhythms corresponds closely to proposals that beta and gamma mediate complementary roles in predictive hierarchies with top-down expectation versus bottom-up error signalling (Arnal & Giraud, 2012; Bastos et al., 2020).

The gamma enhancement observed here also fits a broader literature linking gamma synchrony to perceptual binding and multisensory coherence. Early EEG work by Schneider et al. (2008, 2011) showed that congruent audiovisual and audio-tactile pairings elicit gamma increases between 40-60 Hz, reflecting the brain's attempt to unify temporally aligned sensory streams. Similarly, Yuval-Greenberg and Deouell (2007) and Senkowski et al. (2007) reported stronger induced gamma for cross-modal congruence, while Kanayama et al. (2009) demonstrated that gamma synchrony in the parietal cortex scales with the strength of illusory body ownership. The present findings therefore extend this well-characterised binding mechanism to the auditory domain of ASMR, in which proximal, socially meaningful sounds appear to trigger a comparable cross-modal integration process.

Importantly, while gamma synchrony often accompanies successful multisensory binding, converging evidence also indicates that it can index prediction error when expectations are violated (Arnal et al., 2011; Todorovic et al., 2011). Within ASMR, gamma enhancement may therefore mark the system's attempt to reconcile the predicted sensation of affiliative touch with its physical absence, yielding a somatosensory echo that resolves cross-modal discrepancy through a simulated perceptual inference.

Spatially, the observed topographies may implicate regions including the secondary somatosensory cortex (S2/OP1), posterior insula, and posterior cingulate, nodes known to integrate exteroceptive and interoceptive input (Eickhoff et al., 2010; Craig, 2002; Critchley & Harrison, 2013). These regions align with the cortical targets predicted by the PPH cascade: S2 as the generator of tactile simulation (Mazzola et al., 2012), the posterior insula as the interoceptive comparator (Craig, 2002), and cingulate areas as autonomic mediators (Vogt, 2005; Leech & Sharp, 2014). While such inferences are based on topographical correspondence in sensor-space data and conceptualisation of the PPH model, in this thesis, as source localisation was not performed, activity cannot be definitively resolved to specific brain area yet, but the convergent EEG-MEG results in sensor-space serve to delineate the temporal architecture of the ASMR cascade with high precision.

5.3.1 From Cortical Oscillations to Autonomic Control

Mechanistically, the observed beta-gamma dynamics can be interpreted as cortical antecedents of the parasympathetic experience reported behaviourally during and after ASMR (Poerio et al., 2018; Valtakari et al., 2019; Barratt & Davis, 2015). Beta desynchronisation has been associated with the suspension of top-down drive from motor and premotor cortices, consistent with reduced tonic activity of the LC-noradrenergic system (Engel & Fries, 2010; Aston-Jones & Cohen, 2005). Whereas, gamma enhancement may reflect re-entrainment of insula-brainstem pathways within the central autonomic network that facilitate vagal output (Critchley & Harrison, 2013; Beissner et al., 2013; Chang et al., 2013). Together these oscillations exemplify a cortical, autonomic loop linking predictive fulfilment to bodily calm (Thayer & Lane, 2009), such that the ASMR tingle sensation represents both a perceptual and a regulatory event, the sensory correlate of a shift toward parasympathetic dominance.

5.3.2 Interoceptive Illusion and Neural Economy

The concept of a somatosensory echo offers a novel way to interpret these oscillatory signatures. In ordinary touch, posterior-insular activation tracks actual cutaneous input, particularly signals carried by CT afferents associated with affective touch (Olausson et al., 2002; Craig, 2002; Bjornsdotter et al., 2010). In ASMR, the PPH model argues that similar activation may arise to represent the simulation of expected touch. By generating a low-precision interoceptive illusion rather than seeking new sensory evidence, the brain achieves energetic efficiency, minimising prediction error without requiring motor action.

Comparable top-down modulation of the internal state has been observed in other contexts where interoceptive predictions are voluntarily altered, such as mindfulness and meditative attention (Farb et al., 2013; Lutz et al., 2008), or expectation-driven placebo analgesia (Büchel et al., 2014). ASMR therefore potentially demonstrates that internal bodily states can be recalibrated purely through predictive inference, extending predictive coding principles into the domain of affective self-regulation, suggesting that sensory priors can actively scaffold homeostatic balance. This also suggests that expectation in the intervention study of Chapter 4 may have lent itself to increased analgesic benefits in the same way as expectation-driven analgesia can be seen to do (Büchel et al., 2014), particularly given the predictive coding mechanism that the PPH proposes is foundational to the emergence of ASMR. Recruiting predictive processing necessitated during ASMR exposure could prove to be a strength when applied in a clinical setting, enhancing the likelihood of analgesic results.

Methodologically, the use of spectral parameterisation (FOOOF) in the EEG-MEG analysis pipeline, a rigorous method to isolate periodic from aperiodic power changes, strengthens confidence that these dynamics reflect genuine oscillatory shifts rather than broadband artefacts. The resulting identification of beta-gamma co-occurrence as a reproducible neural signature provides a candidate biomarker for future mechanistic and translational studies linking cortical prediction to autonomic tone.

5.4 Translational and Clinical Significance

The intervention findings in Chapter 4 extend the theoretical and neural evidence of this thesis to an applied context, showing that daily ASMR exposure can yield measurable improvements in wellbeing within a chronic pain population. Participants who listened to ASMR sounds for two weeks showed a significant reduction in fatigue severity relative to controls, accompanied by a trend toward reduced pain-related interference. These outcomes are congruent with the PPH prediction that repeated engagement of social, affective auditory cues promotes parasympathetic recalibration via the LC-vagus axis.

From a mechanistic standpoint, the observed fatigue reduction aligns with down-regulation of noradrenergic tone and up-regulation of vagal activity, restoring flexibility within the central-autonomic network. Chronic fatigue and pain both entail dysregulated arousal systems; by reinstating vagal dominance, ASMR may re-establish homeostatic balance. The pattern also underscores the centrality of pleasantness as the driver of reported benefit. In the behavioural study presented in Chapter 2, where a survey was undertaken by the participants who were involved in the EEG part of the EEG-MEG paradigm of Chapter 3, subjective reports of pleasantness strongly predicted both the likelihood and intensity of

tingles, suggesting that hedonic prediction is the key determinant of physiological gain. The same mechanism may underpin the therapeutic effect observed here: the more rewarding the predicted contact, the stronger the vagal modulation and the greater the relief from fatigue or discomfort.

These findings also position ASMR within the emerging field of affective-autonomic intervention science, alongside mindfulness, slow-breathing, and vagus-nerve-stimulation protocols (Tang, Hölzel, & Posner, 2015; Zaccaro et al., 2018; Gerges et al., 2024). Unlike these techniques, ASMR operates through sensory priors, offering a passive yet targeted route to autonomic regulation. Repeated exposure could potentially train the LC-vagus system to re-engage predictive safety cues, providing a self-directed mechanism for down-regulating tonic arousal.

Chronic pain syndromes are now understood less as purely nociceptive phenomena and more as disorders of affective-autonomic dysregulation as well (Bushnell et al., 2013; Eccleston & Crombez, 1999). From this perspective, interventions that recalibrate arousal, sleep, and emotion can deliver genuine clinical benefit even without significantly altering pain intensity directly. The present fatigue reduction therefore represents not a secondary outcome but a mechanistic validation of ASMR's proposed therapeutic applications to chronic pain in particular, supporting calls for scalable non-pharmacological treatments that restore affective-autonomic balance.

The pattern of reduced fatigue and the trend toward improved pain interference mirrors effects reported for transcutaneous auricular vagus nerve stimulation (taVNS), which increases parasympathetic tone and reduces sympathetic outflow (Clancy et al., 2014; Burger et al., 2020) and engages insula pathways (Frangos et al., 2015). ASMR may thus represent a non-invasive, sensory analogue of vagal stimulation, leveraging perceptual prediction instead of electrical input to access the same brainstem-autonomic circuits. Framed this way, ASMR offers an inexpensive, self-administered route to affective-autonomic recalibration, consistent with observed reductions in heart rate during ASMR exposure in previous literature (Poerio et al., 2018).

Clinically, this has several implications. First, ASMR is a feasible, low cost adjunct to conventional pain and fatigue management, deliverable remotely via headphones. Second, it highlights fatigue, a symptom arguably both understudied in experimental chronic pain research and neglected in terms of its effects on patients' lived experience during participation in such research (Hansford et al., 2024), and a sensitive index of parasympathetic engagement. Third, the link between pleasantness, engagement, and

outcome invites personalised intervention design, where auditory profiles are matched to an individual's sensory, social priors to maximise benefit. More broadly, the results support the proposition that modulating interoceptive prediction and vagal tone can produce clinically meaningful improvements even without altering nociceptive transmission directly through medication, reframing pain relief as being possible through predictive recalibration rather than just through a sensory blockade, in future research.

5.5 Conceptual and Methodological Strengths and Emergent Considerations

Several broader issues emerge from this work:

5.5.1 Individual Variability and Predictive Weighting

Not all participants experienced tingling or relaxation, underscoring that ASMR depends on individual priors and the precision weighting of interoceptive predictions. Considerable inter-individual variability in both susceptibility and trigger potency has been well documented. Some people reliably experience tingles from whispering or soft tapping, while others remain unresponsive or even find such cues aversive; using their ASMR Trigger Checklist, Poerio et al. (2022) demonstrated that trigger preferences are highly idiosyncratic across individuals but stable within them, suggesting enduring differences in how auditory-affective cues are internally modelled. This heterogeneity parallels findings in misophonia, a condition in which similar, often human oral or nasal sounds in particular (e.g., chewing, breathing), evoke strong negative affect and sympathetic arousal (Edelstein et al., 2013). Notably, several ASMR and misophonia triggers overlap, implying that identical acoustic inputs can elicit affiliative calm or defensive aversion depending on the listener's predictive priors. McGeoch and Rouw (2020) previously proposed that ASMR and misophonia may represent opposite poles of auditory-affective processing, with one yielding parasympathetic soothing, and the other sympathetic alarm.

Within the PPH framework, such variability reflects individual differences in social and affective precision weighting, i.e. how confidently the brain interprets near-ear cues as signals of social contact and safety. Those with heightened empathic concern or more secure attachment styles may more readily infer affiliative intent, reinforcing pleasant prediction fulfilment (Janik McErlean & Banissy, 2017; Mikulincer & Shaver, 2007). Conversely, individuals with anxious or avoidant attachment, or with atypical interoception, may assign lower precision or even negative valence to the same stimuli (Flockton et al., 2025). In this way, the PPH accommodates both ASMR and misophonia as divergent outcomes of predictive inference in the social and interoceptive domains. Studying these

contrasting responses provides a unique naturalistic testbed for predictive coding models of emotional perception and the construction of safety versus threat in future work,

5.5.2 Measurement Precision and Multimodal Alignment

In the EEG study, button press markers of subjective tingling introduced temporal uncertainty, potentially obscuring fine grained oscillatory sequences. Although discrete responses capture the onset of conscious awareness, they lag behind the underlying neural changes by several hundred milliseconds, limiting temporal precision for dynamic spectral analyses (Schurger, Sitt, & Dehaene, 2012). Future paradigms combining continuous self-report methods (e.g., affective sliders or dial-based ratings) with concurrent physiological monitoring could capture the evolving intensity of ASMR experiences more accurately. Integrating pupillometry, skin conductance, and heart-rate variability (HRV) would help delineate the temporal coupling between cortical oscillations and the LC-vagal axis of arousal regulation. Pupil diameter provides a sensitive, millisecond-level index of LC-noradrenergic activity (Joshi & Gold, 2020), while HRV and electrodermal activity track parasympathetic and sympathetic fluctuations, respectively (Laborde, Mosley, & Thayer, 2017; Critchley & Harrison, 2013). Simultaneous acquisition of these measures would allow direct testing of the proposed biphasic trajectory from orienting arousal to parasympathetic restoration in ASMR in future research.

Similarly, adherence monitoring in the clinical intervention study relied on self-report, which constrains both internal validity and interpretability of the relationship between the ASMR intervention and subsequent responses. Employing app-based timestamp verification, passive sensing (e.g., smartphone audio detection or wearable technology logging listening duration), or ecological momentary assessment (EMA) could substantially improve engagement tracking and contextual accuracy (Shiffman, Stone, & Hufford, 2008). Such multimodal, time-synchronised methods would strengthen causal inferences between sensory exposure, autonomic modulation, and clinical outcomes in future ASMR interventions.

5.5.3 Stimulus and Definitional Heterogeneity

In the research field, a lack of consensus about what constitutes an ASMR stimulus continues to impede replication and comparability across studies. The current thesis addressed this by using naturalistic control sounds that successfully avoided tingling reports; however, future work should develop a systematic acoustic taxonomy that quantifies the relevant physical features of ASMR triggers, including interaural disparities, amplitude

envelopes, and spectral gradients. Such objective descriptors will help clarify whether ASMR depends on specific acoustic regularities or higher-order perceptual interpretations, or both.

Equally critical is the issue of participant classification; the common division between 'experiencers' and 'non-experiencers' oversimplifies what is likely a continuous spectrum of sensitivity. Trigger responses vary idiosyncratically across individuals (Poerio et al., 2022), and many supposed non-experiencers may simply not have encountered their effective trigger, or may require particular emotional or attentional contexts for ASMR to emerge. Rather than a categorical trait, ASMR sensitivity may reflect individual differences in affective prediction and interoceptive precision weighting, implying that the capacity for ASMR experience is widely distributed even if its expression is context dependent. Within this thesis, participants were not categorised as being definitively able to experience ASMR or not, and were not selected based on any prior experience of the phenomenon; there were no such eligibility requirements across the studies, though demographic data collection included questions on whether participants were familiar with ASMR or had ever experienced it before and all studies involved a mix of participants who had heard of the phenomenon before and who had not, perhaps providing a sample more representative of the general population.

Finally, expectancy-matched instructions should be implemented to disentangle genuine predictive mechanisms from generic relaxation or placebo effects. Current paradigms might describe ASMR stimuli as stimuli that induce tingles, while presenting controls as neutral sounds, inadvertently biasing expectations. Providing equivalent framing across conditions, for instance, describing all stimuli as potentially relaxing without mentioning tingles, would control for suggestibility and ensure that observed effects reflect the specific predictive structure of the auditory cues, not differential expectancy or demand characteristics. In the current research, tingles were described but stimuli were carefully outlined as eliciting them in some people, and a lack of any such experience was highlighted as being normal and equally important to report, to help avoid expectation bias as much as possible.

5.5.4 Theoretical Synthesis and Methodological Rigour

Finally, preregistration in the intervention study, transparent reporting across studies, open access to all analysis pipeline scripts, and cross-modal replication in the EEG-MEG study, represent methodological benchmarks for consolidating ASMR research as a rigorous experimental domain. Beyond ASMR, these practices contribute to the maturation of affective neuroscience by linking subjective phenomenology, neural dynamics, and autonomic physiology within a single predictive framework.

5.6 Limitations and Boundary Conditions

Although the present body of work provides convergent evidence for the Proximity Prediction Hypothesis (PPH) across neural and behavioural domains, several conceptual and methodological constraints delimit its scope and lend themselves to ideas for future refinement.

5.6.1 Sensor Space Constraints

At the neural level, analyses in this thesis were restricted to sensor space. While the topographies of beta desynchronisation and gamma enhancement are consistent with activity in posterior-insular and somatosensory regions. The 64 channel EEG montage offers limited spatial resolution for disentangling cortical from subcortical contributors; future high-density EEG or source-reconstructed MEG data will therefore be essential for verifying whether the oscillatory cascade truly originates from S2/OP1 and posterior-insular regions and whether its magnitude covaries with brainstem indices of parasympathetic engagement. Such multimodal verification would decisively test the LC-vagus model that underpins the PPH.

Another methodological boundary concerns temporal granularity. The EEG and MEG analyses averaged spectral power across 30s epochs to capture stable frequency patterns, but this inevitably blurs rapid transitions predicted by the biphasic orienting to accommodation sequence of the PPH model. The early sympathetic orienting phase when the listener first attends to the sounds, potentially marked by brief gamma bursts and pupil dilation, may have been temporally compressed within these averages. Time-resolved approaches combining short window spectral estimation with simultaneous pupillometry or cardiac monitoring could clarify how cortical oscillations couple to the phasic-tonic dynamics of LC firing and vagal upshift that are proposed in the PPH.

5.6.2 Dependence on Spatial Acoustics and Sensory Precision

A further limitation lies in the ecological and technical constraints of ASMR stimuli themselves. The PPH explicitly predicts that spatial proximity cues, such as large interaural level and time differences (ILD and ITD), slow amplitude envelopes, and high frequency roll-off of auditory triggers (Flockton et al., 2025), are key to triggering the peripersonal space system and initiating the predictive cascade. Any degradation of these cues, for example through mono playback (which collapses binaural information into a single channel) or excessive reverberation, should attenuate or abolish the effect. This aligns with auditory spatial research showing that near-field perception depends critically on ILD, ITD, and

spectral distance cues (Kopčo & Shinn-Cunningham, 2011; Begault, 1994). The present experiments used high-fidelity headphones in the EEG study to preserve these properties, yet individual variation in hearing acuity, headphone fit, or ambient noise could alter perceived proximity. In the clinical intervention study, participants were asked to use headphones at home to listen to the sounds in the experimental group, every day, yet there was no direct way to confirm that they did do this or what types of headphones they were using. It should be highlighted that this did mean the clinical intervention study reflected a naturalistic setting akin to how most listeners choose to listen to ASMR in their daily lives, encompassing individual variability in delivery methods and personal preferences. Systematic manipulation of these spatial parameters will still be necessary to delineate the acoustic boundary conditions under which ASMR engages predictive touch priors in future research.

The reliance on low precision sensory channels is also a theoretical constraint. The PPH proposes that ASMR arises only when tactile evidence is ambiguous or absent; if the tactile channel becomes high precision, through actual touch or conflicting somatosensory feedback for example, the illusion should collapse. Although direct empirical evidence is lacking, analogous findings from multisensory illusion research support this logic. In the rubber hand illusion, for example, asynchronous or incongruent tactile feedback abolishes the sense of ownership (Botvinick & Cohen, 1998; Shimada et al., 2009), while in virtual reality touch paradigms, mismatched haptic cues reduce embodiment (Slater et al., 2009). Similarly, phantom vibration experiences occur under conditions of low sensory precision and disappear once unambiguous tactile input is provided (Rothberg et al., 2010). These parallels suggest that ASMR may likewise depend on the maintenance of sensory uncertainty; it thrives in predictive ambiguity but dissipates under unequivocal evidence.

5.6.3 Clinical and Ecological Limits

At the clinical level, the two week intervention provided only a preliminary test of ASMR's translational potential. Participants were not selected for elevated anxiety or insomnia, conditions that the PPH predicts would show the greatest benefit due to chronic sympathetic dominance. Prior work indicates that ASMR's therapeutic effects are most pronounced in individuals with pre-existing symptoms. Eid et al. (2022) found that high trait anxiety participants showed a significant reduction in state anxiety after ASMR exposure, with no such benefit in low anxiety individuals. Survey evidence similarly suggests that habitual ASMR users frequently rely on it for anxiety relief and sleep aid, particularly when these problems are longstanding (Barratt & Davis, 2015; Fredborg et al., 2018). Existing evidence suggests that clinical or high symptom populations may experience greater and more

reliable benefits from ASMR exposure. The null effects found for mood and sleep measures therefore do not necessarily represent evidence against ASMR's therapeutic efficacy in these domains; longer interventions with clinical subgroups may reveal stronger and more durable improvements once cumulative parasympathetic engagement is achieved.

Moreover, the intervention relied on self-selected listening contexts. Environmental factors such as lighting, posture, and concurrent cognitive load likely modulated outcomes. If ASMR indeed operates through predictive simulation of affiliative safety, the surrounding context must support this interpretation; background distractions or impersonal settings may weaken the likelihood of experiencing the somatosensory echo. Embedding ASMR exposure within intentionally calming, socially framed environments, such as therapeutic relaxation sessions or guided imagery protocols, could optimise its efficacy in clinical settings.

5.6.4 Conceptual Boundaries of the Proximity Prediction Hypothesis

Finally, several conceptual caveats qualify the generality of the PPH. The model was derived from auditory-driven ASMR, yet visual or tactile variants (e.g., slow hand movements, gentle stroking) may engage overlapping but not identical circuitry. Whether the same proposed LC-vagus sequence could be generalised across sensory modalities remains to be empirically tested. Likewise, the PPH explains ASMR as a benign interoceptive illusion of touch; it does not preclude additional contributions from reward circuitry or oxytocinergic systems that reinforce affiliative contexts. Integration of these motivational components may eventually broaden the model into a more complete account of predictive social soothing, encompassing ASMR, affective touch, and certain meditative states.

Unifying these limitations outlines a realistic perimeter for interpretation. ASMR emerges most robustly when near-ear cues are spatially intact, socially framed as affective, and processed by individuals whose predictive hierarchies permit low precision integration of interoceptive signals. Outside of these conditions, when spatial cues degrade, social context is ambiguous, or arousal systems are dysregulated, the predictive cascade may fail to initiate or may resolve along defensive rather than affiliative lines (as in misophonia). Understanding these boundary conditions is crucial not only for theoretical accuracy but also for designing interventions that harness ASMR's therapeutic potential while respecting its dependence on safety, proximity, and personal meaning.

5.7 Future Directions

The work presented in this thesis establishes a coherent mechanistic framework for understanding ASMR as a predictive, interoceptive phenomenon linking cortical inference to autonomic regulation. The next phase of research should extend this foundation along four converging axes: mechanistic elaboration, experimental refinement, clinical expansion, and technological innovation.

5.7.1 Mechanistic Elaboration: Mapping the LC-vagus cascade

Future research should explicitly test the physiological cascade proposed by the PPH, linking cortical oscillatory dynamics to locus coeruleus (LC) suppression and vagal activation. Several approaches offer complementary paradigms in this area:

Concurrent CNS-ANS Measurement

Simultaneous EEG-MEG, pupillometry, and cardiac monitoring could resolve the predicted biphasic sequence, linking beta-gamma oscillatory dynamics with pupil dilation (phasic LC activation), heart rate deceleration, and increased HF-HRV (vagal rebound). This multimodal design would allow direct assessment of how cortical precision-weighting translates into autonomic change.

LC Imaging

Advances in neuromelanin-sensitive MRI and LC-targeted fMRI (Betts et al., 2019; Trujillo et al., 2023) now permit in-vivo visualisation of LC structure and function. Individual differences in LC integrity could be correlated with ASMR-related vagal indices to test whether reduced LC excitability predicts stronger parasympathetic shifts after experiencing ASMR.

Causal Manipulations

Pharmacological modulation of noradrenergic tone or non-invasive brain stimulation of insular targets could test directionality proposed by the PPH. Enhancing vagal tone through transcutaneous auricular vagus nerve stimulation (taVNS) should potentiate ASMR responses, whereas elevating noradrenergic drive should attenuate them. Such studies would move the field from correlational inference to mechanistic validation.

Source Localised Neural Oscillations

As an extension of the MEG study reported in chapter 3 we have collected additional MRI data to allow for source localisation to be conducted, resolving the reported oscillatory dynamics to particular regions of interest in the brain. Predictions for these results have already been pre-registered (<https://doi.org/10.17605/OSF.IO/2URXK>) and will identify

whether beta suppression and gamma enhancement originate from posterior-insular and S2/OP1 generators in the brain, and whether their coherence predicts parasympathetic indices. Cross-frequency coupling analyses could further examine whether gamma bursts nest within beta desynchronisation, reflecting hierarchical prediction updating in real time.

5.7.2 Experimental Refinement: Probing the computational logic

A major challenge for future experiments is to dissect the computational architecture of the PPH cascade, how sensory priors, precision weighting, and contextual safety interact to yield the tingling percept. Potential future experiments to test these considerations could include:

Parametric Manipulation of Proximity Cues

Systematically varying interaural level differences, amplitude-envelope speed, and spectral roll-off of sound stimuli, will reveal how each acoustic feature contributes to PPS activation. Distance-morphing paradigms could quantify the threshold at which near-ear cues transition from neutral to affective in ASMR elicitation.

Audio-Tactile Congruence Paradigms

Presenting congruent and incongruent combinations of sound and gentle touch would test whether ASMR arises specifically from cross-modal mismatch and whether restoring congruence suppresses gamma-indexed prediction errors.

Expectancy Modulation

Explicitly manipulating listener expectations, through instruction, deception, or predictive cues, could test whether the tingling percept scales with the strength of prior beliefs. This would provide a direct behavioural index of predictive coding in an affective domain.

Temporal Precision Analyses

High resolution time-frequency methods could track rapid transitions between the orienting and accommodation phases, resolving how cortical oscillations orchestrate the LC-vagus dynamics on a subsecond scale.

5.7.3 Clinical Expansion: From mechanism to intervention

The translational potential of ASMR extends beyond chronic pain to a broader class of disorders characterised by dysregulated arousal and interoceptive imbalance, which could be explored clinically in the following ways:

Longer and Monitored Intervention Protocols

Extending ASMR exposure to four to eight weeks would align with established timeframes in affective-autonomic interventions such as mindfulness training, HRV biofeedback, and taVNS protocols, where reliable improvements in HRV and mood have been shown to emerge within this intervention window (Lindsay & Creswell, 2017; Laborde, Mosley, & Thayer, 2017; Badran et al., 2018). Incorporating automated playback logs and physiological monitoring could establish relationships between exposure duration, HRV change, and symptom improvement and enable tailoring of stimuli to individuals based on their subjective and physiological responses.

Trait-Based Stratification

Baseline measures of attachment style, interoceptive accuracy, empathy, and LC integrity should be incorporated in future research, to predict responsiveness. ASMR experiencers consistently show higher empathic concern and absorption (McErlean & Banissy, 2017; Fredborg et al., 2017) and exhibit reduced default mode connectivity consistent with greater sensory-affective permeability (Smith et al., 2017). ASMR responders have already been shown to display lower interoceptive accuracy but an increased tendency towards sensation seeking than non-responders, particularly for tactile, olfactory, and gustatory modalities (Poerio et al., 2023); with Poerio and colleagues suggesting that this could indicate a compensatory mechanism in trait ASMR. Furthermore, individual differences in sensitivity to interoceptive cues have been shown to correlate with experiencing 'spontaneous' tingling sensations more generally (Michael et al., 2015; Tihanyi & Köteles, 2017), therefore assessing interoceptive accuracy should be a useful benchmark for predictions directly relating to whether the characteristic manifestation of ASMR is likely to be felt, as this tingling is a conscious interoceptive mechanism (Tihanyi et al., 2018) and ASMR research could therefore extend our understanding of interoception and sensory processing as a whole. Identifying phenotypes with high precision social priors or enhanced interoceptive sensitivity could also guide personalised ASMR protocols and clarify for whom such interventions are most effective (Mikulincer & Shaver, 2007), and further delineate how to categorise whether there are discrete ASMR experiencers and non-experiencers.

Comparative Valence

Juxtaposing ASMR with misophonia stimuli can test the PPH's prediction that opposite affective outcomes (pleasure versus aversion) arise from differential precision weighting of identical sensory cues. Such contrasts would illuminate how predictive systems assign hedonic valence to proximal sounds in future work.

5.7.4 Technological and Applied Innovation

The same features that make ASMR an ideal research model, naturalistic stimuli, low cost, and digital accessibility, also make it a promising target for applied innovation and digital healthcare, in the following ways:

Closed-Loop Digital Delivery

Smartphone or VR applications could integrate real-time biosensing (HRV, pupil diameter) to adapt playback dynamically, maintaining the listener within the optimal arousal window for parasympathetic engagement.

Machine Learning Personalisation

Adaptive algorithms could learn an individual's acoustic preference profile, favouring frequencies, timbres, or tempos that maximise vagal gain, and generate customised ASMR stimuli. Such systems could serve as self-administered bio-neurofeedback tools.

Integration with Clinical Practice

Embedding ASMR intervention protocols within pain management or sleep rehabilitation programmes could complement mindfulness and relaxation training, offering a sensory-driven route to autonomic recalibration. Pilot trials in digital health contexts like mobile applications, could evaluate feasibility and adherence at scale.

5.8 Overall Conclusion

Across theoretical, neurophysiological, and clinical levels, this thesis demonstrates that Autonomous Sensory Meridian Response (ASMR) is far more than a cultural or internet phenomenon. It represents a reproducible sensory, affective state grounded in the principles of predictive and interoceptive neuroscience. Through the integrative model of the Proximity Prediction Hypothesis, the work presented here unites three domains that have rarely been addressed together: the perceptual construction of social proximity, the neural dynamics of predictive inference, and the physiological regulation of affective state, particularly in relation to chronic pain.

Methodologically, this thesis provides the first cross-modal replication of ASMR's oscillatory signature in EEG and MEG, the first application of FOOOF spectral parameterisation to dissociate periodic from aperiodic power in this context, and the first preregistered longitudinal test of ASMR's clinical potential in chronic pain. Conceptually, it advances the field by embedding ASMR within an explicit computational framework, pioneering a novel theoretical model that links sensory prediction, bodily regulation, and affective experience. Together, these studies trace a coherent mechanistic chain linking subjective

tingling to reproducible cortical dynamics and measurable improvements in clinically meaningful domains.

The theoretical contribution extends beyond ASMR itself. It offers a proof of principle that predictive coding can be empirically demonstrated in the interoceptive domain, that parasympathetic calm can emerge from the resolution of sensory prediction error, and that subjective sensory 'pleasantness' may be understood through the lens of successful prediction in the brain.

The PPH model's current scope in this body of work is restricted to auditory driven ASMR and sensor-space oscillatory correlates. The precise cortical and subcortical circuitry, the relative contribution of reward and oxytocinergic systems, and the generalisation to visual or tactile variants remain to be explored. Yet the boundaries delineated in this thesis are productive ones, they clarify where prediction-based models are supported already, and where additional motivational or contextual factors may intervene.

Future work should therefore pursue the directions outlined in Section 5.7: source-localised mapping of ASMR's neural underpinnings, concurrent LC and vagal measurements, parametric manipulations of proximity cues, and longer clinical trials targeting anxiety, fatigue, and autonomic imbalance. By revealing that simple, naturalistic sounds can entrain neural and autonomic systems through the logic of predictive simulation, the work contributes to a broader understanding of how the human brain maintains homeostasis through perception. It also points to an optimistic translational horizon, where personalised, non-pharmaceutical interventions derived from predictive neuroscience and auditory stimuli, could help alleviate fatigue, anxiety, and pain.

ASMR thus emerges not as just an eccentric sensory quirk but as a paradigmatic instance of embodied prediction; a demonstration that affective touch can be *heard*, that it can be felt without contact, through the brain's own inferential mechanisms. The phenomenon also offers a potential therapeutic bridge, between certain sensory issues and the sounds that can silence them.

Appendix A: Analysis Code Repository

All analysis scripts used in this thesis are hosted in a publicly accessible GitHub repository:

<https://github.com/Josephine162/Thesis-Analysis-Scripts->

The repository contains:

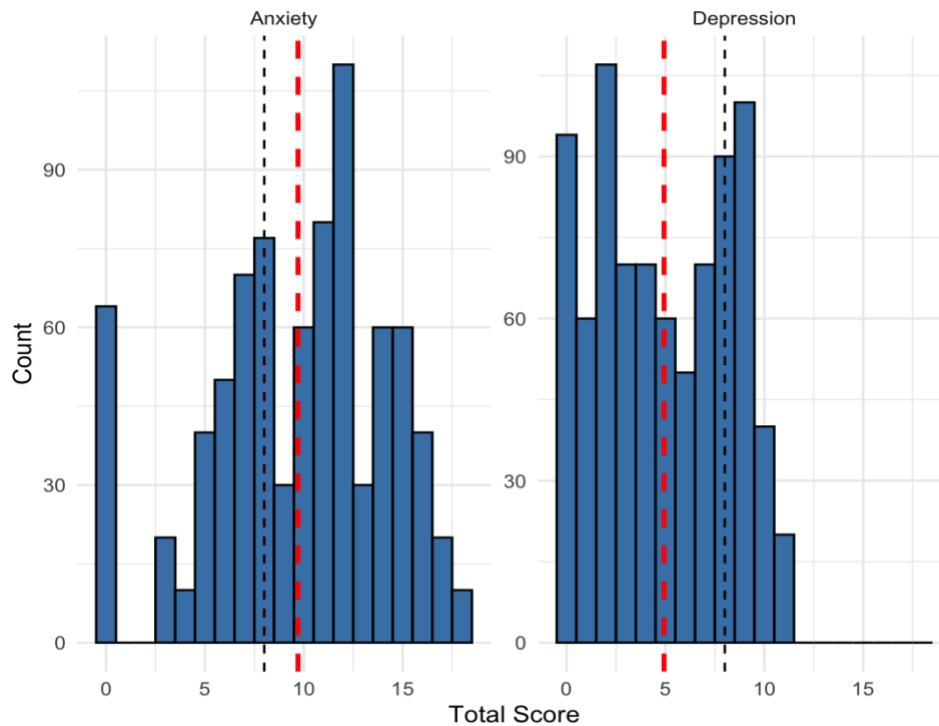
- EEG and MEG sensor-space analysis scripts
(preprocessing, artefact handling, epoching, spectral analysis, FOOOF spectral parameterisation, time-frequency representations (TFR), ERPs, and cluster-based permutation testing)
- MEG preprocessing and analysis workflows
(where applicable, including steps for data conversion, filtering, event handling, and sensor-level group analyses)
- Behavioural data analysis scripts in R
(self-report analyses for the EEG study, linear mixed-effects models for the intervention study, and all descriptive and inferential statistics)
- Figure generation code
(Python and R scripts used to produce the plots included in the thesis)

This repository will remain publicly available to support transparency, reproducibility, and future extensions of this research.

Appendix B: Supplementary Material from Chapter 4.

Fig S1

Distribution of baseline HADS anxiety and depression scores (Week 1).



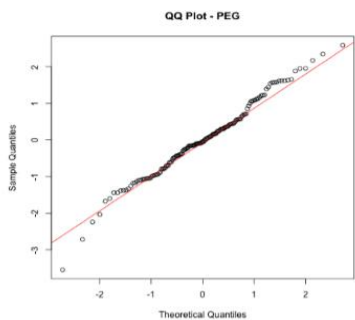
Note: Black vertical dashed lines mark the clinical threshold (score = 8) and red vertical dashed lines mark the group mean scores (Anxiety: 8.70; Depression: 4.41). The depression distribution shows a right-skew, indicating potential floor effects.

Figure S2

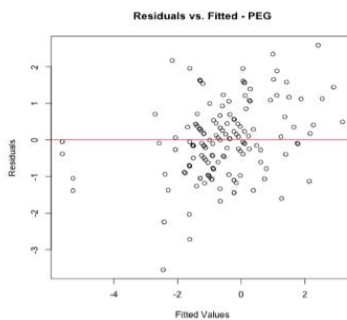
Residual diagnostics for preregistered linear mixed-effects models. Each row corresponds to one outcome (PEG, ISI, FSS, HADS-Anxiety, HADS-Depression).

Figure S2. Residual diagnostics for preregistered LMMs
(Left: QQ plots; Right: Residuals vs Fitted)

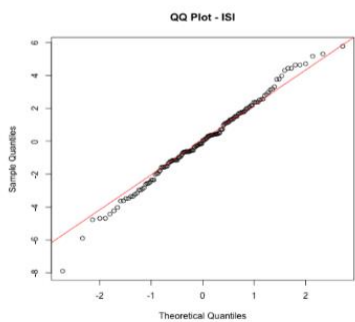
PEG - QQ plot



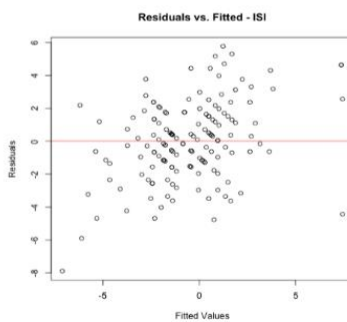
PEG - Residuals vs Fitted



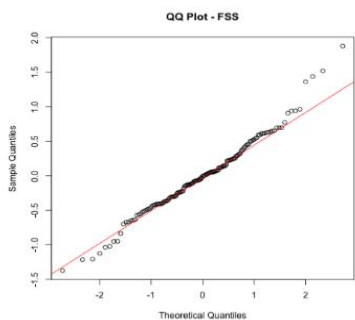
ISI - QQ plot



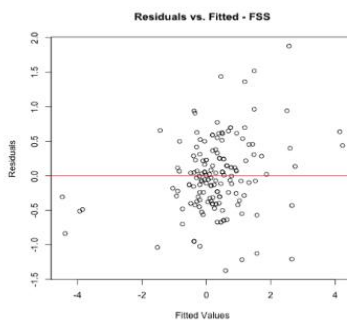
ISI - Residuals vs Fitted



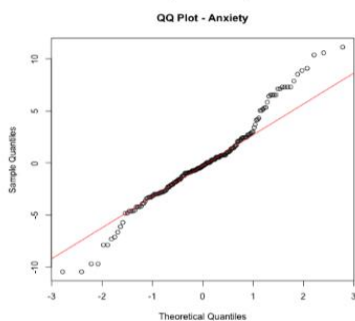
FSS - QQ plot



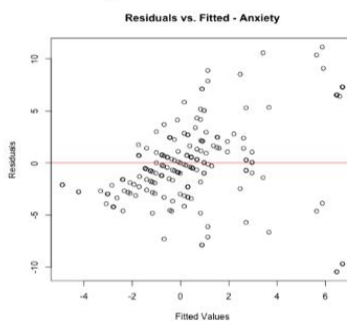
FSS - Residuals vs Fitted



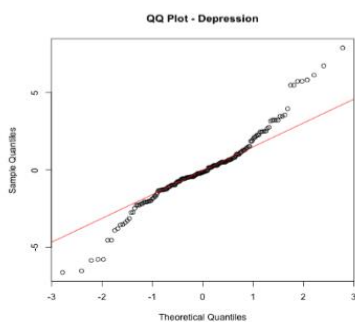
Anxiety - QQ plot



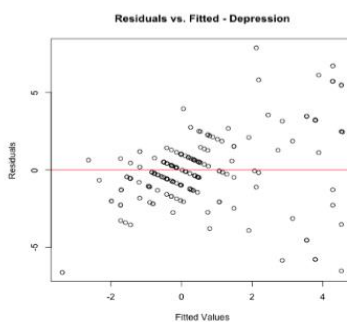
Anxiety - Residuals vs Fitted



Depression - QQ plot



Depression - Residuals vs Fitted

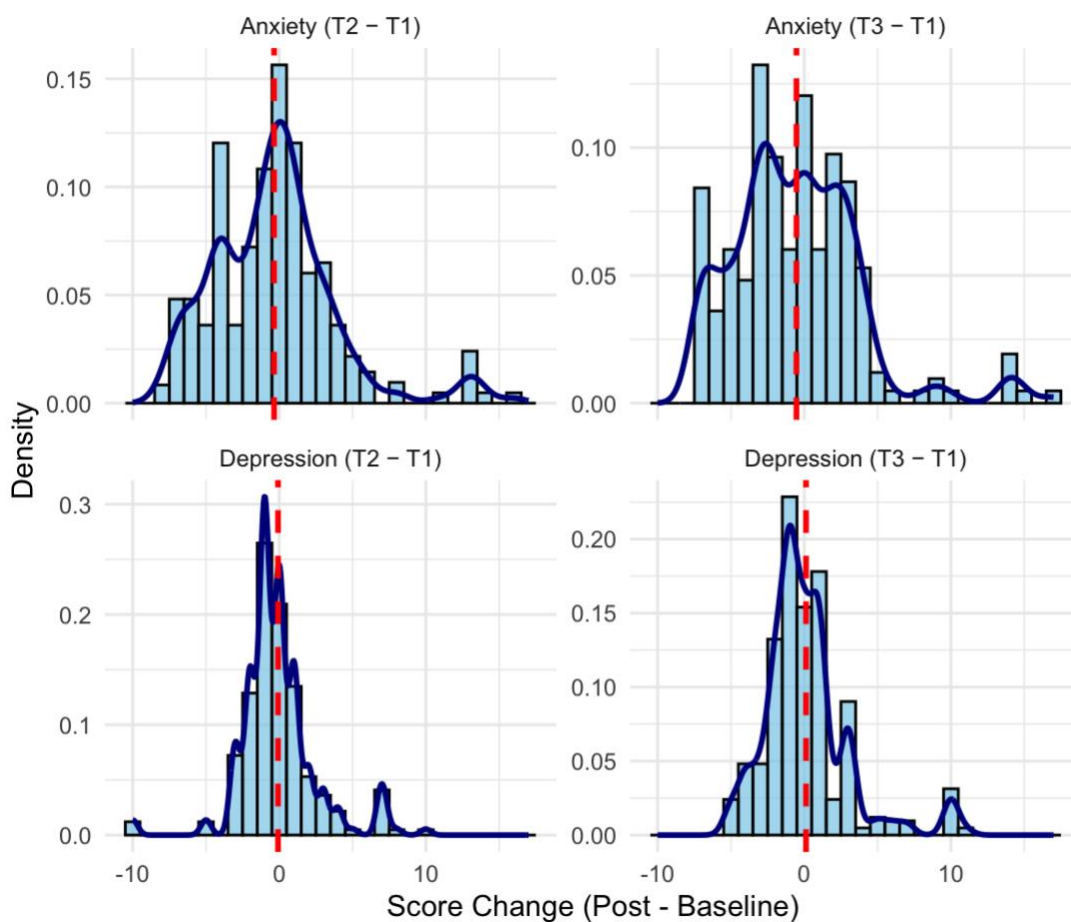


Note: Left panels show normal Q–Q plots of standardized residuals; right panels show residuals versus fitted values. Points close to the 45° line indicate approximate normality; a random cloud around zero without funneling indicates homoscedasticity. Visual inspection suggests PEG and ISI residuals are approximately normal with near-constant variance; FSS shows minor deviations and slight fanning; HADS-Anxiety and HADS-Depression exhibit heavier tails and some heteroscedasticity, consistent with baseline floor effects. See Results text for complementary Shapiro–Wilk tests.

Despite non-significant LMM results, histograms of individual change scores showed modest improvements and interindividual variability. From T1 to T3, 51.7% of participants improved on anxiety, and 48.1% on depression. From T1 to T2, 47.8% improved on anxiety, and 49% on depression. Figure S3 displays the change score distributions and Table S2 provides associated descriptive statistics.

Fig S3

Distribution of HADS anxiety and depression change scores from T1 to T2 and T3.



Note: Red vertical dashed lines indicate group mean change. Negative scores represent reductions in symptoms.

Table S1

Means (M) and standard deviations (SD) for HADS anxiety and depression subscales at baseline (T1) and for change scores from T1 to T2 and T1 to T3.

Measure	M	SD
HADS Anxiety (T1)	8.7	5.17
HADS Depression (T1)	4.41	3.47
Anxiety Change (T2 - T1)	-0.35	4.25
Anxiety Change (T3 - T1)	0.46	5.33
Depression Change (T2 - T1)	-0.09	2.62
Depression Change (T3 - T1)	0.69	3.45

Table S2

Correlations between ASMR engagement at T3 and improvement in outcomes (Improvement = T1 - T3; positive values indicate symptom reduction).

Outcome (Improvement)	Predictor (T3)	n	r	95% CI	p	P (Holm)
Anxiety	Enjoyment	36	-0.03	[-0.36, 0.30]	.858	1.000
Anxiety	Listening Frequency	43	0.07	[-0.23, 0.37]	.639	1.000
Depression	Enjoyment	36	-0.02	[-0.34, 0.31]	.925	1.000
Depression	Listening Frequency	43	-0.05	[-0.34, 0.26]	.758	1.000
FSS	Enjoyment	30	0.18	[-0.19, 0.51]	.334	1.000
FSS	Listening Frequency	37	0.20	[-0.13, 0.49]	.234	1.000
ISI	Enjoyment	30	0.05	[-0.32, 0.40]	.800	1.000
ISI	Listening Frequency	37	-0.15	[-0.45, 0.18]	.380	1.000
PEG	Enjoyment	30	-0.61	[-0.80, -0.32]	<.001	.004
PEG	Listening Frequency	37	-0.18	[-0.48, 0.15]	.282	1.000

Note: Only the association between enjoyment and PEG improvement was statistically significant after Holm-Bonferroni correction ($r = -.61$, 95% CI [-.80, -.32], $p < .001$, $p_{Holm} = .004$).

Appendix C: Experimental and Control Stimuli

Please find the link to the auditory stimuli google drive repository used in this thesis below. The first 13 sounds labelled ASMR 1-13 are the experimental stimuli, and the last 5 sounds labelled CONTROL 14 - 18 are the naturalistic traffic noise control stimuli. Across all empirical studies in this thesis, the auditory stimuli were always presented in a random order. This includes the ASMR stimuli given to the experimental group in the longitudinal study of Chapter 4 - the same experimental sounds, along with the five control sounds, were used in the EEG-MEG cross-modal neuroimaging protocols of Chapter 3, as well as the self-report survey paradigm that had occurred after the same EEG experiment, analysed as illustrative data in Chapter 2.

https://drive.google.com/drive/folders/1NokJhY-keYGVWeFcj_8pq4kX0ehoaJUZ?usp=sharing

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