# Correlation of approach-avoidance behaviour and negative affect with cortical alpha oscillations in context of pain experience

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Kipu on epämiellyttävä sensorinen ja emotionaalinen kokemus, joka viestii usein kehoa uhkaavasta vaarasta. Samaan aikaan kipu on merkittävä haitta yksilön elämälle ja yhteiskunnalle. Neuraalisesti kipuärsyke kulkeutuu keskushermostossa alkaen kipureseptoreista jatkaen selkäytimen kautta aivojen sisempiin osiin ja sieltä aivokuorelle. Aivojen eri osat aktivoituvat riippuen kivun vaiheesta. Sen sijaan, varsinainen kokemus kivusta on aina yksilöllinen ja useat taustatekijät vaikuttavat siihen.

Tämän pro gradu tutkielman tarkoituksena on selvittää, miten yksilölliset taipumukset ja erityisesti temperamenttipiirre negatiivinen affektiivisuus ja välttämis- ja lähestymiskäyttäytyminen korreloivat aivojen lepoaktiivisuuden kanssa ennen ja jälkeen kipukokemuksen. Tutkimukseen osallistui 18 naistutkittavaa (iän vaihteluväli: 18–42 vuotta) ja heidän kipukynnyksensä määritettiin ennen tutkimuksen varsinaista aloittamista. Aivojen sähköistä aktiivisuutta mitattiin magnetoenkefalografian (MEG) avulla. MEG mittauksen aikana kipu tuotettiin lyhyenä sähköisenä kipuärsykkeenä tutkittavien sormiin. Kipuärsykesarjoja ennen ja jälkeen mitattiin aivojen lepoaktiivisuutta ja sen muutosta alfarytmin taajuudessa. Kipuärsyke stimulaation lisäksi tutkittavat täyttivät temperamenttikyselyn (ATQ-SF) sekä välttämis- ja lähestymiskäyttäytymistä mittaavan kyselylomakkeen (BIS-BAS 20), joista poimimme tutkimukseemme negatiivisen affektiivisuuden sekä BIS-BAS taipumuskokonaisuuden.

Kahden lepomittauksen välillä ei tapahtunut merkittävää muutosta alfarytmin taajuudessa. Sen sijaan alfa taajuus ennen kipukokemusta oli merkittävästi yhteydessä BAS palkintoherkkyyteen. Lisäksi negatiivinen affektiivisuus pelko alaluokan ja kipukokemuksen jälkeen mitatun alfa taajuuden väliltä löydettiin merkittävä yhteys. Tutkimuksen tulokset viittaavat siihen, että yksilölliset taipumukset ovat yhteydessä ihmisaivojen rytmiseen lepotilaan ennen kipukokemusta ja sen jälkeen.

Avainsanat: kipu, temperamentti, negatiivinen affektiivisuus, välttämis- ja lähestymiskäyttäytyminen, maksimialfan taajuus, magnetoenekfalografia (MEG)

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Pain is an unpleasant sensory and emotional experience that often signals a danger to the human body. Concurrently pain is a significant disadvantage to an individual's life and for society. Neurally, the pain stimulus travels in the central nervous system, starting from nociceptive receptors and continuing through the spinal cord to the limbic parts of the brain and from there to the cortex. Various regions of the brain are activated depending on the stage of the pain. Instead, the experience of pain is different for every individual and it is influenced by several background factors.

The purpose of this master's thesis is to investigate how individual tendencies, especially temperament trait negative affect and avoidance and approach behaviour correlate with resting activity of the brain before and after the pain experience. The study had 18 female subjects (age range: 18–42 years) and their pain thresholds were determined before the actual measurements. The electrical activity of the brain was measured by magnetoencephalography (MEG). During the MEG measurement, pain was produced by brief electrical pain stimulus on the fingers of the subjects. Before and after pain stimuli, the brain resting activity and its change was measured by focusing on frequency of alpha rhythm. In addition to pain stimulation, subjects completed a temperament questionnaire (ATQ-SF) and a questionnaire measuring avoidance and approach behaviour (BIS-BAS 20). From these behavioural variables, we extracted the negative affectivity and the BIS-BAS predispositions for our study.

There was no significant change in alpha frequency between the two resting state measurements. Instead, the alpha frequency before pain experience was significantly associated with BAS reward responsiveness. Furthermore, a significant association was found between the negative affect subscale fear and the alpha frequency after pain experience. The results of our study suggest that individual tendencies are related to the resting state of human brain before and after pain experience.

Keywords: pain, temperament, negative affect, behavioural inhibition and activation system, peak alpha frequency (PAF), magnetoencephalography (MEG)

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# 1. INTRODUCTION

Pain is a vital sense for humans to survive, since it signals danger and malfunction of the human body (Nelson, 2013). Concurrently pain is a significant disadvantage to individuals and for society. It consumes one's resources, impairs ability to function and affects peoples' quality of life (Haanpää & Vainio, 2018; Hadjistavropoulos & Craig, 2004). Frequently pain causes stress and overtime it may impact one's mental health, causing fear of pain, depression and anxiety. Pain can also increase family members' stress and consume their resources (Asmundson, Vlaeyen & Crombez, 2004; Dueñas, Ojeda, Salazar, Mico & Failde, 2016). For society pain generates various economic burdens due from the challenging pain diagnostics, expensive treatments and the expenses caused by absenteeism and incapacity to work (Haanpää & Vainio, 2018). The department of health and human services of Finland stated that in 2013, back pain alone generated 469 million euros in costs. Furthermore, pain patients' prospective need for assistance in everyday matters may create a major burden to the healthcare system (Haanpää & Vainio, 2018; Hadjistavropoulos & Craig, 2004).

Pain changes the state of body and mind. This change is due from the automatic and learned reactions (Nelson, 2013) regulated by the central nervous system, where brain and its structures play a key leading role (Purves, 2012). During pain, several areas of the brain are activated (Chen, 2001), and it appears that rhythmic activity of brain may impact on how one copes with the pain (Furman et al., 2020). In general, pain can be cured with pharmacological and psychological interventions. Yet several individuals are still suffering from pain without a successful cure and thus new approaches are needed. Often the focus is on pain perception and its sensory qualities, though it is well known that pain is also a subjective process where one's emotions and behaviour take a part. (Asmundson et al., 2004; Nelson, 2013.) Indeed, some research results refer that the negative consequences of pain could mediate through behavioural characteristics such as temperament and approach-avoidance system (Serrano-Ibáñez, López-Martínez, Ramírez-Maestre, Esteve & Jensen, 2018a). These behavioural tendencies are known to have a relatively strong biological and cerebral background (Carver & White, 1994; Haslam, 2007; Keltinkangas-Järvinen, 2015). The aim of this thesis is to study pain from the point of view that focuses on behavioural tendencies and brain function during resting state.

## 1.1 Pain

According to the International Association for the study of Pain (IASP) pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage (IASP, 1994). In literature pain is usually divided into chronic and acute forms (Nelson, 2013). However, pain can be also described as current, recurrent, temporary or intermittent (Chen, 2001). In this study, we concentrate on brain activity before and after recurrent temporary pain stimulus in the so-called resting state.

The pain sensory pathway is well-established and the sense of the pain, nonciception, arises from the free nerve endings that are divided into A delta and C fibers. A delta fibers generate sensation of immediate sharp pain. In turn, C fibers are responsible for slower pain output and they produce most of the human pain sensations. Both, A delta and C fibers conduct the pain stimuli in the central nervous system (CNS) along with the spinal cord, thalamus and cortex. (Nelson, 2013; Purves, 2012.) Multiple brain imaging methods have been utilized to reveal the brain structures activated during pain stimulus (Aprakian, Bushnell, Treede & Zubieta, 2005). Studies using magnetoencephalography (MEG) have discovered that anterior cingulate cortex (ACC) (Ploner, Gross, Timmermann and Schnitzler, 2002), primary (S1) and secondary somatosensory cortex (S2) (Kitamura et al., 1995; Ploner et al., 2002; Timmermann et al., 2001) activate during pain perception. The S1 and S2 areas are known to locate in the parietal region of the brain (Johnsen & Agerskov, 2009). Electroencephalography (EEG) pain studies have similar results, these studies have revealed that additionally to somatosensory cortex areas (Tarkka & Treede, 1993) and ACC (Downman & Schell, 1999; Tarkka & Treede, 1993), the area of vertex (Kitamura et al., 1995), supplementary motor cortex (SMA) (Downman & Schell, 1999) and insula cortex (IC) (Schulman, Zonenshayn, Ramirez, Ribary and Llinas, 2005) are perceived to be activated during pain.

Besides being a sense, pain is an emotion that is usually described in terms of discomfort and suffering (Bushnell, Ceko & Low, 2013; Haanpää & Vainio, 2018; Nelson 2013). Moreover, pain itself is influenced by emotions (Bushnell et al., 2013). For instance, Villemure and Bushnell (2009) discovered with functional magnetic resonance imaging (fMRI) that negative emotions increase the pain experience while positive emotions decrease it. In turn, Yoshino and colleagues (2012) revealed with MEG that more specific emotions, such as sadness, intensify the pain experience. The emotional nature of pain is also discovered in brain research and it appears that during the emotional phase of pain orbitofrontal cortex (OFC), periaqueductal gray nucleus (PAG) (Villemure & Bushnell, 2009), ACC (Rainville, Duncan, Price, Carrier & Bushnell, 1997; Villemure & Bushnell, 2009), insula

(Bushnell et al., 2013) and amygdala (Neugebauer, 2016; Simons et al., 2014) become active. These brain structures are known to take a part in motivational behaviour, creating memories and emotions such as fear and threat (Krabbe, Gründemann & Lüthi, 2018; Rolls, 2004; Roxo, Franceschini, Zubaran, Kleber & Sander, 2011).

Frequently pain stimulus causes automatic withdrawal reflex and draws attention to its location (Garcia-Larrea & Jackson, 2016; Nelson, 2013). Therefore, one can hypothesize that the human brain reacts to pain before it takes place, especially when cue from pain is involved (Nelson, 2013). Indeed, different pain processing stages induce activity in various structures of the brain. When concerning the brain state before pain experience, Carlsson and others (2006) found with fMRI that pain anticipation and predictable pain stimulus activated posterior insula, S1 and S2 areas. In turn, when pain stimulus was unexpected anterior insula and OFC were perceived to be activated (Carlsson et al., 2006). Seifert and colleagues (2013) discovered with fMRI that pain anticipation activated similar areas (e.g IC, ACC and S2) than the actual pain stimulus. The brain state after pain experience has been less studied. However, one can say pain initiates cognitive modulation process and it appears that during this state S2 (Nakamura, Paur, Zimmermann & Bromm, 2002; Ploner et al., 2002) and ACC (Ploner et al., 2002) are perceived to be activated.

Although the pathway of pain sensation is relatively universal, the experience of pain can vary widely among people (Bushnell et al., 2013; Nelson, 2013). Diverse cultural backgrounds, gender, pain history, learned beliefs and actions modify the experience of pain (Nelson, 2013). As a result, two individuals can sense the similar sensory pain input with different intensity and interpretation (Bushnell et al., 2013; Nelson, 2013). For instance, Baetz and Bowen (2008) discovered religious individuals undergo less pain than the nonreligious ones. Gender as well seems to have a great effect to pain experience and similar pain is estimated less intense among men than in women (Fillingim, King, Ribeiro-Dasilva, Rahim-Williams & Riley, 2009). Moreover, individual previous painful events and learning modify the pain experience and it appears that severe previous pain events and higher pain expectations increase the pain intensity (Fields, 2018; Jepma, Koban, Doorn, Jones & Wager, 2018; Paquet, Plansont, Labrunie, Malauzat & Girard, 2017).

## 1.2 Individual trait characteristics and pain

### 1.2.1 Temperament and pain

As discussed above, experiencing pain is subjective and the reaction to pain is unique to every person. The biologic and psychologic makeup of an individual has been recognized as a significant factor modulating the perception of pain and the response to it. Thus, this composition contributes to the differences between and within individuals (Ranger & Campbell-Yeo, 2008.) Generally, pain experiences have been linked together with negative emotionality and neuroticism-related temperament traits (Asmundson et al., 2004; Martucci, 2017).

Temperament is a set of innate tendencies, traits and abilities, that influence the way we behave and interact with others (Haslam, 2007; Keltinkangas-Järvinen, 2015). It has been related to the differences in one's fundamental constitution (Rothbart, 1989). Temperament refers to inherent individual differences in reactivity and self-regulation, in the realm of affect, activity and attention (Rothbart & Bates, 2006; Rothbart & Sheese, 2007; Rothbart & Gartstein, 2008). Rothbart and Derryberry (1981) describe *reactivity* as a response to stimulation and change in the external and the internal environments (e.g., hunger, exhaustions and pain). The range of responses is extensive, including negative affect, fear etcetera. *Self-regulation* concerns processes that adjust reactivity, such as inhibition and behavioural approach (Rothbart & Gartstein, 2008). These distinctive tendencies are relatively stable: they appear early in life and are rather consistent across time and situations (Haslam, 2007; Rothbart & Gartstein, 2008). Furthermore, this diversity in the tendencies to react to environments and regulate emotions reflect the dissimilarities in the activity of the central nervous system and neural regulation system (Keltinkangas-Järvinen, 2015).

Since temperament emerges early in development, most of the research has focused on infancy and early childhood (Rothbart & Sheese, 2007). Later on, Rothbart and Bates (2005) formed a basic classification of temperament to adulthood. Adult Temperament Questionnaire (ATQ) by Rothbart measures the self-regulatory processes and individual reactivity of adults using four traits: negative affect, extraversion/surgency, effortful control and orienting sensitivity (Wiltink, Vogelsang & Beutel, 2006). Negative affect measures heightened sensitivity to extensive range of negative stimuli and it includes four subscales: fear (unpleasant affect associated with anticipation of pain and anxiety), sadness (unpleasant affect associated with disappointment, the loss of a person or an object), discomfort (unpleasant affect resulting from sensory qualities of stimulation) and frustration

(unpleasant affect associated with interruption of task and behaviour or with the obstruction of a coveted objective). (Evans & Rothbart, 2007.)

Studies, that have utilized Rothbart's temperament questionnaires and his theory, have found a connection between trait negative affect and pain across age groups. Former studies have shown that subscale fear from the Early Adolescent Temperament Questionnaire (EATQ-R) had significant positive correlation with pain catastrophizing which refers to the cognitive tendency to focus on pain and exaggerate the seriousness of the pain sensations (Muris et al., 2007; Sullivan et al.,2001). Klein, Gaspardo, Martinez, Grunau and Linhares (2009) explored how toddlers' pain reactivity and recovery during painful medical procedures predicted their temperament. Results suggested, that the higher the heart rate was during needle insertion, higher the scores were on the subscales fear and sadness in the Early Childhood Behaviour Questionnaire (ECBQ) (Klein et al., 2009). Additionally, temperament trait negative affect from Child Behaviour Questionnaire (CBQ) was associated with more intense postoperative pain behaviour (e.g. higher pain levels) in children (Uhl et al., 2019).

### 1.2.2 Approach-avoidance behaviour and pain

Behavioural motivational tendencies such as approach and avoidance behaviour affect how one reacts to pain (Serrano-Ibáñez et al., 2018a; Serrano-Ibáñez et al., 2018b). Behavioural inhibition system and behavioural activation system (BIS-BAS) are the two general motivational systems that regulate behaviour and affect (Carver & White, 1994). These systems are thought to function independently, yet in the pain process they seem to work partly interactively, BIS affecting BAS and vice versa (Serrano-Ibáñez et al., 2018a). Moreover, both BIS and BAS are influenced by learning experiences and operate automatically (Jensen, Ehde & Day, 2016). The theory of the BIS-BAS systems is developed by Jeffrey Gray and the base of his model is on Hans Eysenck's personality theory. In Eysenck's theory personality has a neural basis and it is drawn by internal and environmental cues. In BIS-BAS theory Gray replaced Eysenk's dimensions extraversion and neuroticism to impulsivity and to anxiety traits and from internal and environmental cues, he underlined the motivational factors of the environment. (Carver & White, 1994; Haslam, 2007.)

Frequently pain is experienced as a punishment or as an unapproachable stimulus and therefore researchers suggest that pain is more related to higher activation of the behavioural inhibition system (BIS) (Jensen et al., 2016; Serrano-Ibáñez et al., 2018a). The BIS system refers to avoidance behaviour and it is responsive to punishment, nonreward and novelty signals (Carver & White, 1994; Haslam, 2007). The neural basis of the BIS system is in the septohippocampal system's

monoaminergic afferents, which start from the brainstem and project into the frontal lobe (Carver & White, 1994). In general BIS system restrains a person's behaviour to avoid situations that can have negative or painful consequences (Carver & White, 1994; Haslam, 2007). Furthermore, Gray includes to the BIS system negative emotions such as fear, anxiety, frustration and sadness and some of these emotions operate as a subscale for the avoidance system in the BIS-BAS questionnaire. (Carver & White, 1994.)

In former studies, pain has been associated with a lower function of the behavioural activation system (BAS) and negative emotions. (Asmundson et al., 2004; Serrano-Ibáñez et al, 2018a). The BAS system refers to approach behaviour and it is responsive to reward and nonpunishment signals (Carver & White, 1994; Haslam, 2007). The neural basis of the BAS system is more incoherent than in the BIS system. However, researchers suggest that the BAS system operates through dopaminergic pathways (Carver & White, 1994) including the ventral striatum and ventral segmental area and their connections to the prefrontal cortex (PFC) (Depue & Collins, 1999; Kennis, Rademaker, Geuze, 2013). The BAS system activity supports one's behaviour to approach goals and positive outcomes. According to Gray, in contrast to the BIS system the BAS includes positive emotions such as hope, joy and happiness. (Carver & White, 1994; Haslam, 2007.)

### 1.3 MEG, alpha oscillations and pain

In the past two decades, few pain studies have implemented magnetoencephalography (MEG) as a brain imaging method. The MEG is a non-invasive brain imaging method that measures magnetic fields generated by electrical currents in the brain. Especially postsynaptic currents, generated mostly in the pyramidal cells, create prolonged magnetic fields that MEG measures. (Hansen, Kringelbach & Salmelin, 2010.) MEG has high temporal and spatial resolution, and it is ideal for studying electrical activity in certain regions of the brain in experimental settings (Singh, 2014). Moreover, unlike majority of brain imaging methods, MEG detects directly the electrical activity of the brain and therefore illustrates the transmission of input and output in the brain (Hall, Robson, Morris & Brookes, 2014).

MEG uses hundreds of sensors to identify and locate the source of the rhythmic fluctuations of neural field signals, brain oscillations (Ploner, Sorg & Gross, 2017). These oscillations in the brain are created by coordinated electrophysiological activity, where excitation and inhibition of populations of neurons interplay dynamically (Jensen, Spaak & Zumer, 2019; Ploner et al., 2017).

Brain oscillations are associated with a wide range of cognitive, behavioural and perceptual functions and oscillations in different frequencies indicate these different functions (Ploner et al., 2017).

This spontaneous phenomenon, where neurons fire together in synchrony can occur at any frequency, but it is most prominent between 1-100 Hz (Buzsaki & Draguhn, 2004). Alpha oscillations are neural oscillations in the frequency range of 8-13 Hz, that are involved in multiple cognitive processes, such as memory (Basar, Basar-Erogly, Karakas & Schurmann, 2001; Jensen, Gelfans, Kounios & Lisman, 2002) and inhibition functions (Haegens, Cousijn, Wallis, Harrison & Nobre, 2014). Inhibition helps people to control the engagement and disengagement of sensory areas depending on task demands and studies have shown that alpha oscillations play an active role in these attentional mechanisms (Klimesch, 2012; Mathewson et al., 2011). Furthermore, alpha oscillations are connected with "idling" (rested alertness without engagement in perceptual and cognitive activities) state of the brain: it is the dominant oscillation present in somatosensory cortices during resting state measurements (Furman et al., 2020; Hauck, Lorenz & Engei, 2008; Pfurtscheller & Lopes da Silva, 1999)

The specific frequency of alpha oscillation, that has maximal power is called the peak alpha frequency (PAF) (Furman et al., 2018). PAF varies between individuals and it changes throughout the years, gradually slowing with age (Hashlem et al., 2016; Li, Sun & Jiao, 1996). Fast PAF has been considered as a marker for cognitive preparedness and cognitive performance (Rathee, Bhatia, Punia & Singh, 2020). Interestingly, the peak frequency of alpha activity has been proposed as an alleged biological marker for the individual differences in the experience of pain (Bazanova and Vernon, 2014; Furman et al., 2018, 2020). For example, people with slower PAF during pain-free state are more sensitive to pro-longed pain, since they reported higher pain intensity compared to people with greater PAF (Furman et al., 2018, 2020). Nir, Sinai, Raz, Sprecher and Yarnitsky (2010) discovered using continuous EEG that the resting state PAF increased after acute pain in healthy individuals whilst in chronic pain patients' PAF was slower (Furman et al., 2018). Overall, Furman and others (2019) concluded, that PAF presents important information about the entire process of pain.

### 1.4 Aim of this study and research questions

Aim of this study is to investigate how brain resting state before and after pain stimulation (i.e. pain approach and pain recovery) appears in alpha oscillations, particularly in PAF. Moreover, the connection of negative affect, BIS and BAS to PAF during resting state before and after the pain stimulation is examined. The research questions are:

1. Does peak alpha frequency change between resting states before and after pain stimulation?

   *H1: Resting state peak alpha frequency will increase after pain stimulation.*

2. Do negative affect, behavioural inhibition system and behavioural activation system correlate with peak alpha frequency before and after pain stimulation?

   *H2: Negative affect and behavioural inhibition system have association with the resting states of the pain stimulation.*

3. Do these behavioural tendencies associate with the change of peak alpha frequency between before and after measurement?

   *H3: Behavioural inhibition system and negative affect have also association with the change of peak alpha frequency.*

Generally, alpha oscillations are related to relaxation and the resting brain activity (Hauck, et al., 2008). PAF is associated with pain and it is believed to provide information about the entire pain process (Furman et al., 2020, 2019, 2018; Nir et al., 2010). In line with Nir and colleagues' (2010) previous findings, our hypothesis is that resting state PAF will increase after the pain stimulation. Former studies have shown that BIS and negative emotions have been related to pain more than BAS and positive emotions (Jensen et al., 2016; Martucci, 2017; Serrano-Ibáñez et al., 2018a). Therefore, we hypothesize that negative affect and BIS have association with the resting states of the pain stimulation. Particularly we conclude that the resting state PAF before pain is connected with BIS and negative affect, as alpha associates with the inhibition functions (Haegens et al., 2014; Klimesch, 2012; Mathewson et al., 2011) and PAF is linked with cognitive preparedness (Rathee et al., 2020).

Since BIS and negative affect have associated with pain, one could hypothesize that BIS and negative affect have also association with the change of PAF. (Jensen et al., 2016.) All in all, this topic, concerning PAF, negative affect, BIS, BAS and the resting states before and after pain in healthy participants has never been explored before. Moreover, though there are some dozens of pain studies done with MEG, most of the existing pain studies have been done with fMRI or EEG and therefore the need for MEG pain studies is still present.

## 2. METHODS

This thesis is part of the Pain Brain study, which is made in collaboration with Norwegian University of Science and Technology. Our study took place at the end of 2019 and it was executed in the Centre of Interdisciplinary Brain Research (CIBR) unit in the University of Jyväskylä's Department of Psychology. The analyses were done at the end of 2019 and in the beginning of 2020.

### 2.1 Participants

The participants were recruited from the research register of Jyväskylä Centre for Interdisciplinary Brain Research (CIBR) and by advertising the study in the student email lists and in the general notice boards of the university.

This study recruited 18 females ranging between 18-42 years of age (mean=26.0 years, standard deviation=6.7 years). To participate, subjects had to be healthy adults (18-45 years of age), right-handed and in the normal weight range according to BMI (body mass index, normal range: 18.5-24.9). Individuals with any of the following exclusion criteria: 1) somatic, neurological or psychiatric diseases 2) injuries to the head 3) disorders associated/causing with chronic pain 4) metal in the body 5) prescription drugs (excluding contraceptive pills) 6) pregnancy or nursing 7) rash, cuts or scars on the skin of forearm, hand, wrist or fingers, could not partake in this study. Furthermore, participants were asked not to consume alcohol 24 hours before the MEG measurements and abstain from nicotine and caffeine three hours before the data acquisition.

### 2.2 Adult Temperament Questionnaire Short Form (ATQ-SF)

Finnish version of Adult Temperament Questionnaire (ATQ-SF) short form (translated by Räikkonen-Talvitie*)* was used to estimate the temperament of the participants. The ATQ-SF contains 77 items which are self-rated using a 7-point scale, that varies from 1 (extremely untrue) to 7 (extremely true). The ATQ-SF has an additional option to answer, "not suitable for me". The questions are divided to four scales: effortful control, negative affect, extraversion/surgency, and orienting sensitivity. The negative affect scale includes 26 items and these items are divided into four subscales: fear (e.g. item 1: "I get scared easily", item 51: "Sometimes I panic or feel horror with no apparent reason"), sadness (e.g., item 20: "I rarely feel sad while watching a sad movie", item 56: "I often feel sad"), discomfort (e.g., item 4: "In my opinion loud noises are irritating", item 42: "Bright colours bother me sometimes") and frustration (e.g., item 6: "Waiting in slowly progressing que does not make me usually nervous", item 48: "I get annoyed and frustrated over quite little things"). (Evans & Rothbart, 2007.)

### 2.3 Behavioural Inhibition and Behavioural Activation System Scale (BIS-BAS 20)

Finnish version of Behavioural Inhibition (BIS) and Behavioural Activation (BAS) System Scale without the additional questions (BIS-BAS 20) was used to estimate the two of the motivational systems that regulate behaviour and affect of the participants. The BIS-BAS 20 questionnaire contains 20 items which are self-rated using a 4-point scale that varies from 1 (strong agreement) to 4 (strong disagreement). Twenty questions are divided into their own BIS and BAS scales. The BIS scale includes seven items (e.g., item 1: "If I think something unpleasant is going to happen, I usually get pretty "worked up"", item 9: "Criticism or scolding hurts me quite a bit", item 19: "I feel worried when I think I have done poorly at something important"). All in all, the BAS scale includes 13 items, and these questions are divided into three subscales: drive, fun seeking and the reward responsiveness. The BAS drive scale includes four items (e.g., item 3: "When I want something, I usually go all-out to get it", item 11: "If I see a chance to get something, I want I move on it right away"). The BAS fun seeking scale includes four items (e.g., item 4: "I will often do things for no other reason than that they might be fun", item 16: "I often act on the spur of the moment") and the rest five items are included in the reward responsiveness subscale (e.g., item 2: "When I get something I want, I feel

excited and energized", item 10: "When good things happen to me, it affects me strongly", item 14: "It would excite me to win a contest"). (Carver & White, 1994.)

### 2.4 MEG data acquisition

MEG device used in the study was Elekta Neuromag TRIUX system (Elekta AB, Stockholm, Sweden) with 306 channels and 102 sensor units, each including one magnetometer and two gradiometers. MEG recordings were conducted in magnetically shielded room. To localize the participants' head during measurements head position coils (HPI) were attached to participants head before measurements. The study was executed in two research appointments. The MEG measurements, pain stimulation and questionnaires were done at the first appointment. During the MEG participants' breathing was measured by breathing belt and the heart rate was measured using photoplethysmography (PPG) in the left-hand middle finger. MEG measurement took a maximum one and a half hours. Pain stimulation was produced by needle electrode to three locations on the participants' right hand: intermediate phalanges of the middle finger, and proximal phalanges of the middle and ring finger. The produced pain was brief electric stimulation, which can be compared to pinch-like sensation. The pain threshold was identified individually for every participant with digimeter and intensity calibrated to tolerable, but still unpleasant stimuli and the pain had to be 6/10 from the pain scale of 1-10.

Figure 1. displays the research design. The study was distributed to three different sections: medical ointment, alternative ointment and without ointment. The order of the three sections varied throughout the study for each participant. Each of these sections started without any medical or alternative ointment. There were 150 pain stimuli in one section and during every three subsections the fingers of the participants received 50 pain stimuli. Preceding the stimuli participants received brief tone stimulus as a clue for coming pain stimuli. Additionally, research included four-minute resting state measurements before and after the sections. Moreover, after first pain stimuli serie and before the second stimuli serie started, came two-minute resting state measurement. During every resting state measurement, participants were asked to close their eyes and keep still. In this study, we focus on the four-minute resting state sections before and after pain stimulations.

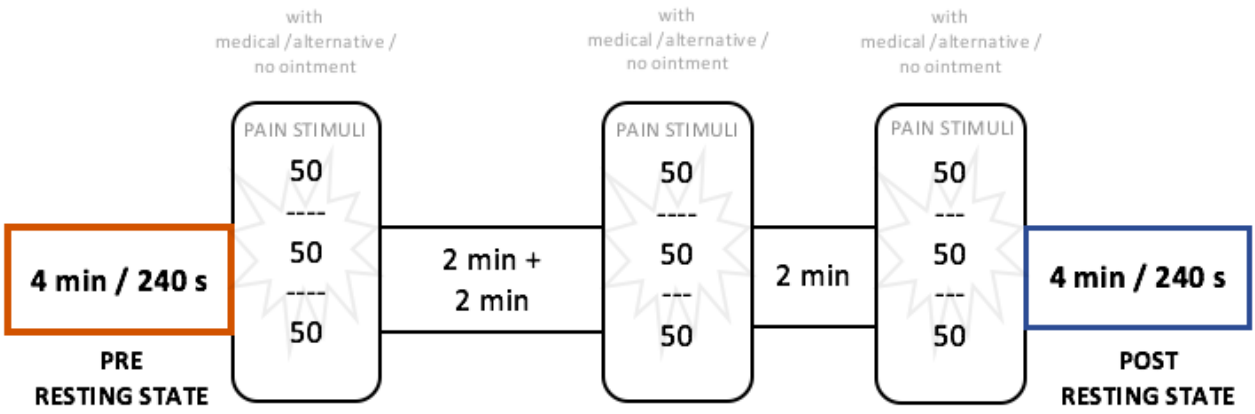


FIGURE 1. Illustration of the research setting used in MEG measurement.

### 2.5 MEG data analysis

This study's MEG analysis focused on the PAF. MEG data was filtered with Maxfilter 2.2 software (Elekta AB, Stockholm, Sweden) to remove the noise caused by external distractions such as head movements. Meggie was used for further processing and artefacts caused by eye movements and heartbeat were removed with Independent Component Analysis (ICA). Assumed pain related brain regions were ensured with time-frequency representation (TFR) analysis. TFR revealed the location of the PAF occurring during the resting states of pain stimulation measurements. According to TFR and previous assumptions the left and right parietal cortex and the vertex were selected for further analysis.

The MEG signals were low pass filtered 1 to 40 Hz (length of the window 1024, overlap 512). The final resting state time periods of MEG were selected from broader data with time adjustment that limited recordings to first the 240 seconds in the beginning of the measurements and to last the 240 seconds of the measurements. The first and the last ten seconds were not included in analysis and the final resting state time period length was from 10 to 230 seconds.

### 2.6 Statistical analysis

Microsoft Excel was used to determine the resting state PAF for each selected area (right and left parietal, vertex) in two situations (before and after pain stimulation). PAF was computed by finding the peak amplitude within the range of 8–13 Hz in each recording and marking the corresponding frequency. Additionally, the possible change of PAF between pre- (before pain stimulus) and post-measurement (after pain stimulus) was illustrated in Excel with various graphs. Rest of the statistical analyses were performed with the SPSS Statistics 26 program. A repeated measures analysis of variance (ANOVA) was performed to compare PAF between pre- and post- measurements statistically. We used time of measurement (pre and post) and the hemisphere (left and right) as within-subject factors in repeated measures ANOVA, when analysing parietal areas of the brain. We conducted separate repeated measures ANOVA for vertex, where time of measurement (pre and post) was the only within-subject factor. Furthermore, Spearman correlation was used to reveal the associations between ATQ Negative affect, BIS, BAS, PAF in parietal left, right and vertex and change of the PAF.

## 3. RESULTS

### 3.1 The change of PAF between resting states of pain stimulation

Figure 2. depicts how the PAF in all three areas (parietal left, parietal right and vertex) averaged among all participants in pre- and post-measurements. The following figures refer to possible minor change between the pre-and post-measurements.

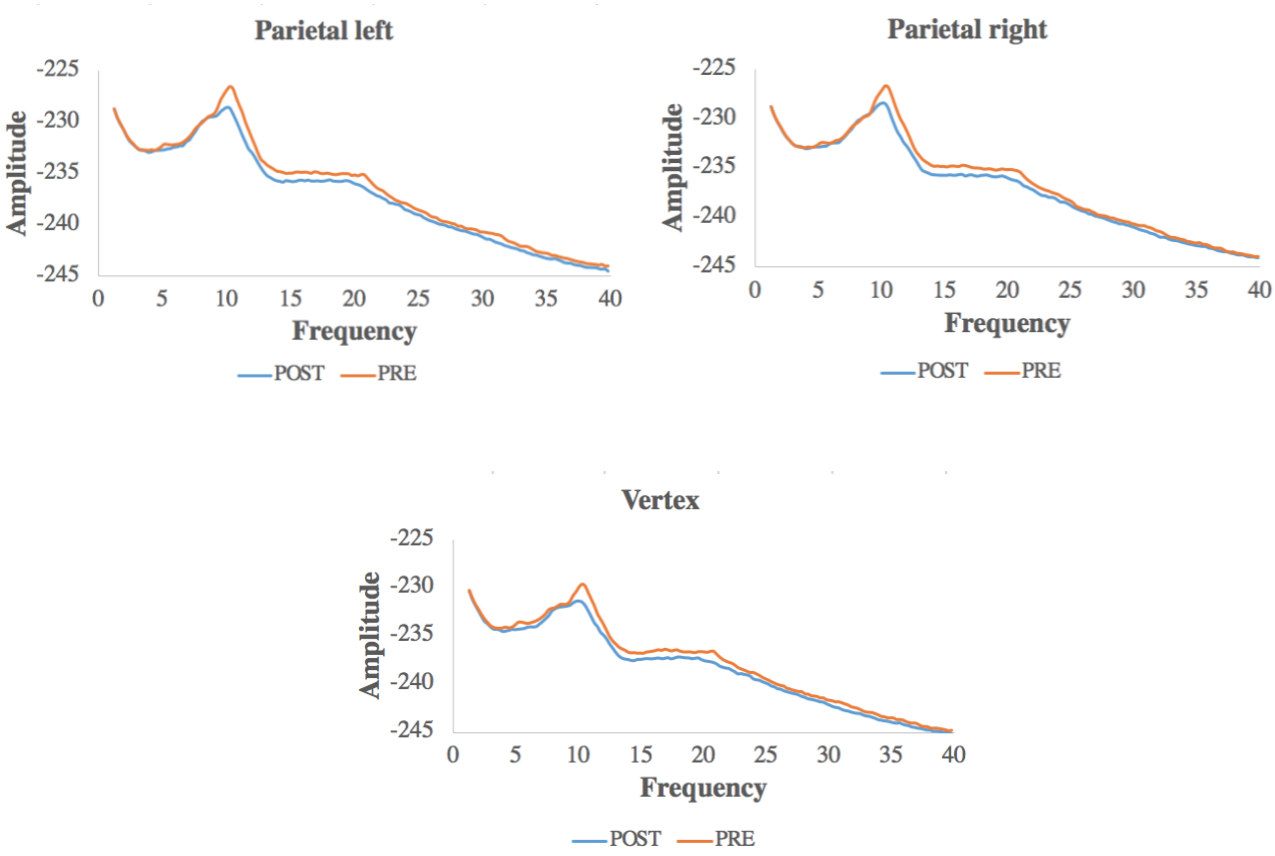


FIGURE 2. Line charts of the PAF in the vertex, the left and the right side of parietal lobe.

Repeated measures ANOVA further revealed that there was no significant change in PAF between the two times the resting state measurement was performed and figure 3. supports these results. As illustrated, the average PAF was not significantly different in pre- measurement compared to post-measurement.

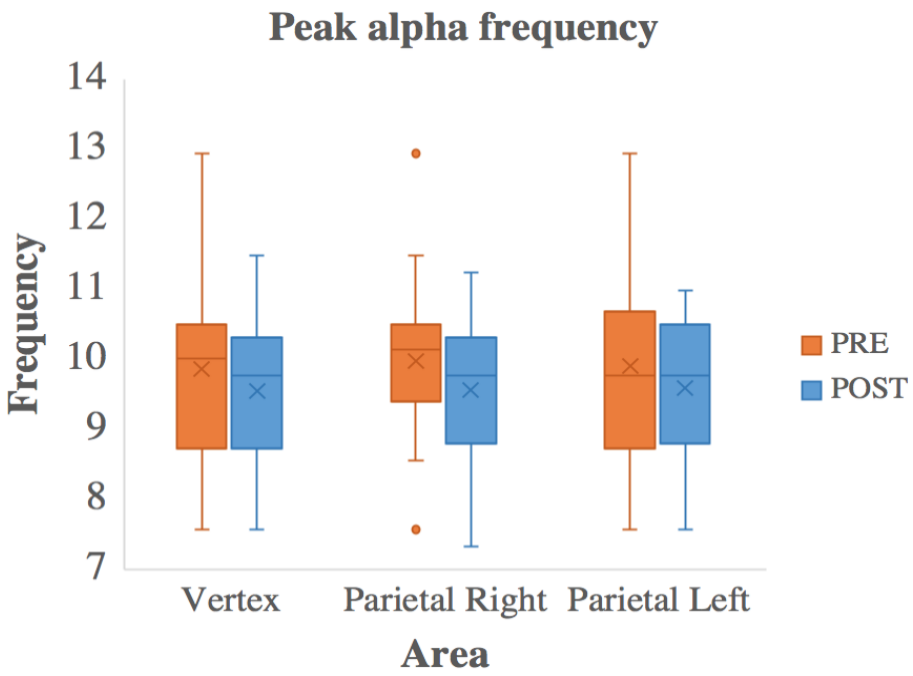


FIGURE 3. Boxplots of PAF during pre-and post- measurement for vertex, the right side and left side of parietal lobe. The average PAF in all three areas before pain stimulus was 9.9 Hz (vertex = 9.9 Hz, parietal right = 9.9 Hz, parietal left = 10.0 Hz) and after pain stimulus was 9.5 Hz (vertex = 9.5 Hz, parietal right = 9.6 Hz, parietal left = 9.5 Hz).

### 3.2 The correlation of approach-avoidance system and negative affect with PAF

Table 1. depicts the correlations. Negative affect correlated significantly with the PAF in the vertex ($r = .469$, $p = .049$) and in the left ($r = .470$, $p = .049$) side of the parietal area during the post-measurements. Discomfort correlated significantly with the PAF in the right ($r = .482$, $p = .043$) and in the left side ($r = .505$, $p = .032$) of the parietal lobe during the pre-measurement. Fear correlated significantly with the PAF in the vertex ($r = .616$, $p = .006$) and in the left ($r = .586$, $p = .011$) and in the right ($r = .493$, $p = .038$) side of the parietal area during the post-measurements. Sadness correlated significantly with the PAF in the vertex ($r = .470$, $p = .049$) during the post-measurements. BAS

reward responsiveness correlated significantly with the PAF in the vertex (r = -.588, p = .01) and in the right (r = -.557, p = .016) and left (r = -.605, p =.0089) side of the parietal lobe during the pre-measurement. BIS, BAS fun seeking, BAS drive and ATQ negative affect subscale frustration did not correlate significantly with the resting states.

Due to high number of correlations, we additionally evaluated the results with a threshold criterion for significance of correlations. When we only accepted correlations with significance level $p \leq 0.01$ and correlation coefficient greater than -/+ 0.5, the following correlations were left: BAS reward responsiveness with PAF in the vertex during pre-measurement (r = -.588, p = .01), BAS reward responsiveness with PAF in the left side of parietal lobe during pre-measurements (r = -.605, p =.0089) and ATQ subscale fear with the PAF in the vertex during post-measurement (r = .616, p = .006). Figure 4 displays the scatter plots of the significant correlations after correction and the scatter plots of results that did not pass the threshold criterion is found from Appendix.

| | **PRE** | | | **POST** | | |
|---|---|---|---|---|---|---|
| | **Vertex** | **Parietal (right)** | **Parietal (left)** | **Vertex** | **Parietal (right)** | **Parietal (left)** |
| **BIS** | .086 | .091 | .039 | .425 | .385 | .428 |
| **BAS reward responsiveness** | **-.588**** | **-.557*** | **-.605**** | -.393 | -.349 | -.333 |
| **BAS drive** | .70 | .187 | .030 | -.026 | .129 | .052 |
| **BAS fun seeking** | -.010 | .032 | -.084 | -.332 | -.194 | -.289 |
| **Negative Affect** | .322 | .335 | .364 | **.469*** | .389 | **.470*** |
| **Fear** | .305 | .328 | .323 | **.616**** | **.493*** | **.586*** |
| **Sadness** | .051 | .022 | .095 | **.470*** | .278 | .369 |
| **Frustration** | .002 | .061 | .035 | .294 | .276 | .361 |
| **Discomfort** | .452 | **.482*** | **.505*** | .181 | .268 | .265 |

**= $p \leq .01$ *=$p \leq .05$

TABLE 1. Spearman correlations of temperament scales (BIS, BAS, ATQ negative affect) and peak alpha frequencies during pre- and post-measurement for vertex, the right and the left side of parietal area of the brain.

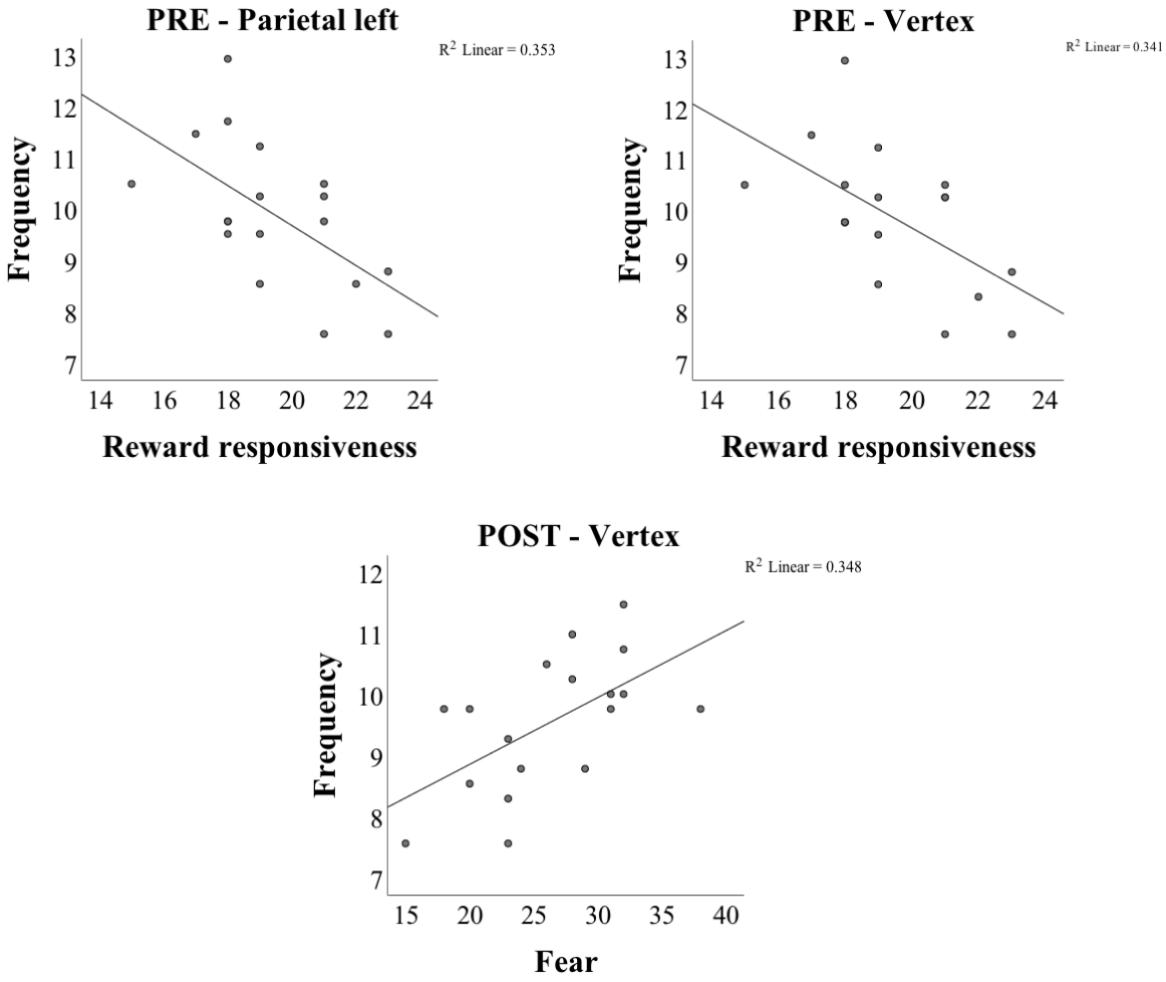


FIGURE 4. Scatter plots of the significant correlations, that passed the significance threshold criterion ($p \leq .01$; $r \geq 0.5$; $r \leq -0.5$).

## 4. DISCUSSION

The aim of this thesis was to investigate the relationship between pain, PAF and behavioural characteristics. Our study examined the change of PAF between resting states, that were measured before and after pain stimulation. Furthermore, we researched how temperament trait negative affect and behavioural approach-avoidance system appear in this process. We hypothesized that PAF would increase from the resting state measured before to the resting state measured after the pain stimuli. It was assumed that negative affect and avoidance system (BIS) would have association with the resting state PAF of pain experience and with the change of PAF.

The results of this study showed that there was no significant change in PAF between the two times of measurement: resting state PAF before pain experience did not differ significantly from the resting state PAF after the pain experience. In turn, there was a significant association between BAS reward responsiveness and PAF of the first resting state measurement in vertex and left parietal lobe. The PAF of the resting state measurement conducted after the pain stimuli was significantly related to ATQ subscale fear. Additionally, there was weaker significant correlation ($p \leq .05$) with the resting state PAF before pain stimuli and ATQ subscale discomfort. Likewise, the resting state PAF after pain stimuli had similar association ($p \leq .05$) with negative affect and ATQ subscale sadness.

## 4.1 Change of PAF

In our study, there was no significant change in resting brain activity: the PAF did not increase significantly from the resting state recording measured after the pain stimuli compared to the one measured before the pain sensation. This outcome suggests that forthcoming or past pain experience do not alter the brain resting state. These findings might be affected by the sample size, and the gender and age distribution of this research.

The sample size used was relatively small with only 18 participants. Pain research might not attract as many volunteers as other studies, thus recruiting participants can be strenuous. Perhaps individuals who view pain more undesirable than others, may not gravitate towards these kinds of studies in contrast to people who are more curious about painful experiences. Moreover, all the 18 participants were women, and it might have an impact on the results. Multiple studies have shown that there are differences in pain sensitivity and pain reaction between women and men (Bartley & Fillingmin. 2013; Fillingim & Maixner. 1995; Fillingim et al., 2009; Wiesenfeld-Hallin, 2005). According to few research outcomes, women tend to express higher pain sensitivity, decreased pain inhibition and undergo more severe clinical pain compared to men (Bartley & Fillingim, 2013). Furthermore, PAF and gender are correlated: according to Zappasodi and others (2006) PAF among females is higher in comparison to males. Since females report more clinical conditions (Ruau, Liu, Clark, Angst & Butte, 2012) and have a greater risk of developing prevalent chronic pain (Feijó et al., 2018; Mogil, 2012), it is crucial to have more research on how women could strengthen their resilience to pain and prevent the consequences of it. As previously noted, PAF decreases along the years (Hashlem et al., 2016; Li et al., 1996). The participants of this study were between 18-42 years old with the average

age being 26 years. One can say the participants in this sample are quite young which means that the PAF would be faster within this group. The results might vary substantially, if the sample would be more heterogeneous or if the average age of the sample would be higher.

Previous findings have revealed a relationship between alpha oscillation and pain, but especially PAF has been presented as a significant factor indicating differences in pain experience between individuals (Bazanova and Vernon, 2014; Furman et al., 2018, 2020). Furman and others (2020) found that the slower the PAF is, the more sensitive the person is to pain. In other words, these results suggest, that higher PAF indicates higher resilience to pain. Multiple studies that have proven the role of PAF in pain process, have utilized EEG (Bazanova and Vernon, 2014; Furman et al., 2018, 2019, 2020; Nir et al., 2010). Since MEG provides greater spatial resolution in comparison to EEG (Singh, 2014), it is crucial to continue the research on the relationship between PAF and pain using MEG as a brain imaging method. Moreover, in the future PAF and pain should be explored with more versatile and greater samples.

## 4.2 Individual trait characteristics

### 4.2.1 Approach-avoidance behaviour

Since there was no change in the PAF between resting states of pain stimulation, there was no association between the change of PAF and behavioural motivational systems. In turn, contrary to what we hypothesized the approach behavioural tendency (BAS) was associated with the resting brain state before pain stimulation. Particularly BAS reward responsiveness was significantly associated with the PAF during before pain resting state measurement in left parietal lobe and vertex. These results propose that the higher scores are in BAS reward responsiveness, the lower the PAF is before the pain experience. As mentioned earlier, lower PAF has been related to greater pain sensitivity and vulnerability to experience prolonged pain (Furman et al., 2020). Hence it could be concluded that higher BAS reward responsiveness or higher approach behavioural tendency would be related to greater pain sensitivity. Researches focused on pain, PAF and BAS reward responsiveness has not been yet studied. However, there are few studies concerning association between BAS reward responsiveness and pain, approach behaviour style and neural reward system, pain and resting brain activity.

To begin, Elvemo, Landrø, Borchgrevink and Håberg (2015) revealed that chronic pain was associated significantly with decreased BAS reward responsiveness and activity in reward related brain regions. These findings suggest brain reward system may impact generally how sensitive one is for prolonged pain. Furthermore, these outcomes underlie the connection between approach behavioural system and reward circuit (Elvemo et., 2015). Also, pain and especially pain relief, has been connected with the reward system (Navratilova et al., 2016) and Navratilova and Porreca (2014) revealed that pain relief increased activity of the neural reward circuit. Thus, one could conclude that BAS reward responsiveness or approach behavioural tendency would relate to the after pain resting state, the "relief" state, when pain is not present. Despite this effect was not visible in our study and the approach behavioural tendency was related to before pain resting state, the knowledge of the neural reward circuit could be utilized in the development of pain management.

As aforementioned approach behaviour system is frequently linked with positive emotions (Carver & White, 1994), while positive emotions are associated with decreased pain experience (Villemure & Bushnell, 2009). Therefore, one could conclude higher tendency to approach style is related to lower pain experience. However, the connection between BAS and positive emotions is challenged. For instance, Serrano-Ibáñez and others (2018a) found association between BAS and negative emotions. Moreover, when concerning the link between pain and BAS, there are more contradictory results such as the connection between pain intensity and BAS. Day, Matthew, Newman, Mattingley & Jensen (2019) found strong association between pain intensity and BAS, while other identical researches have not found similar outcomes (Jensen, Tan & Chua, 2015; Sanson, Hach, Moran & Mason, 2020). These findings underlie the need for research focusing on approach behaviour and pain.

Research concerning BAS and resting brain activity (alpha) have mainly focused on frontal area of the brain. These studies have discovered that frequently approach behavioural tendency is associated with alpha activity in left frontal area (Balconi, Vanutelli & Grippa, 2017; De Pascalis, Schneider et al., 2016; Sommer & Scacchia, 2018). In turn, BAS, pain and alpha association has only been studied once to our knowledge and in this particular study the emphasis was again on frontal lobe (Day et al., 2019). However, contrary to other BAS and resting brain activity studies, Day and colleagues (2019) did not find significant connection between frontal area and approach behaviour system. Since former studies and our results have proven the role of parietal and vertex brain regions in pain process, it is important to focus more on these areas in future research of BIS-BAS and pain.

Our results together with Furman and others (2020) findings propose that high approach behavioural tendency would increase pain sensitivity and risk for chronic pain. The former researches underlie both positive and negative connection between pain and approach behaviour system and this

contradiction verifies the need for BIS-BAS, PAF and pain studies. These studies could provide more information about the quality of the association between approach-avoidance behavioural tendencies and pain. Further investigations may even lead to consider whether activation of the avoidance or approach system could be utilized in the pain management interventions. There are already some studies concerning the factors that increase approach-avoidance system activation. For instance, one approach to increase avoidance or approach system activation could be mind-body training (MBT) (Jung, Lee, Jang & Kang, 2016).

### 4.2.2 Negative affect

Considering that, PAF did not change significantly between the resting state measurements, temperament trait negative affect did not correlate significantly with the change of PAF. The PAF in vertex during the resting state measurement after the pain stimuli was significantly correlated with negative affect subscale fear. In addition, few less significant correlations were established between PAF and temperament trait negative affect. ATQ factor scale negative affect and subscale sadness had connection with the PAF of the resting state measurement performed after the pain experience while the resting state PAF measured before pain stimuli, was significantly linked with subscale discomfort. Our results imply, that the higher the scores are in the negative affect subscale fear, the greater the resting state PAF is after experiencing the pain stimuli. Other outcomes propose, that PAF is faster after the pain stimuli in the participants who scored higher in the ATQ factor scale negative affect and subscales sadness and fear.

Rothbart and Evans (2007) describe fear as an unpleasant affect that is related to anticipation of pain and anxiety. Indeed, fear is an essential part of the pain process (Asmundson et al., 2004) and our findings support this idea since tendency to sense fear was significantly linked with the resting state PAF. Interestingly, the link was detected only during resting state after the pain stimuli which does not match with Rothbart and Evans' (2007) definition of fear. Former researches have established this considerable relationship between pain and fear; however, this observation was done amongst toddlers and young adolescents (Klein et al., 2009; Muris et al., 2007). Furthermore, according to previous fear studies, fear has been associated with anxiety related traits (Asmundson et al., 2004; Sep, Steenmeijer & Kennis, 2019) and avoidance behaviour (Asmundson et al., 2004; Boersma, 2017; Vlaeyen & Linton; 2000). BIS scale measures avoidance behaviour in our study and commonly high BIS and high negative affect scores correlate with one another (Hundt et al., 2013). For that reason, one could hypothesize, that since negative affect was significantly correlated with

the after pain resting state PAF, BIS scale would also have an association with the same resting state measurement in our study. Hence, our results do not endorse these earlier findings and question the connection between BIS and negative affect.

Overall, all the findings on negative affect are consistent with the general perception of pain: pain is an unpleasant stimuli and feelings of discomfort before, during or after painful event are common in most of the individuals (Bushnell et al., 2013; Haanpää & Vainio, 2018; Nelson 2013). On the basis of this study, individuals, who are more disposed to the unpleasant feelings developing from sensory qualities of stimulation (discomfort), may have stronger reaction to anticipation of pain than people who score lower in negative affect subscale discomfort. Pain can induce fear and sadness, or individuals who are more prone to unpleasant feelings following from discomfort, disappointment and anticipation of agony, may take longer to recover from uncomfortable experiences. Our results support the earlier findings on how innate and long-lasting individual tendencies affect the way one reacts to the anticipation, encounter and recovery of noxious stimulus (Bushnell et al., 2013; Fillingim, 2005; Nelson, 2013; Ranger & Campbell-Yeo, 2008).

Villemure and Bushnell (2009) describe how positive emotions relate to less severe pain experience. In concordance Basten-Günter, Peters and Lautenbacher (2018) concluded that optimism is associated with lower amount of pain encounters and it seems to protect individuals from chronic pain. Although previous findings propose positive emotions as a protective factor for pain, our outcomes suggest the opposite: higher levels of fear, sadness, negative affect and discomfort are positively correlated with faster PAF. As previously mentioned, faster PAF indicates higher resilience to pain (Furman et al., 2020). This discovery is in contrary with the theories, where temperament trait negative affect has been associated with exaggeration of pain sensations and with more intense pain experiences (Klein et al., 2009; Muris et al., 2007; Sullivan et al., 2001; Uhl et al., 2019). The role of negative affectivity (trait and emotions) in pain perception may not be as obvious as former literature implies. Temperament does influence the way individuals perceive pain and therefore further research on this subject is required. The demand is present, especially amongst adults, since there is notable absence of pain studies focusing on adult temperament, negative affect and PAF.

## 4.3 Limitations and strengths

When interpreting results there are some limitations that should be taken into account. As mentioned earlier, the sample size was small in this study. However, if one acknowledges the fact that pain studies are thought to be unpleasant for participants, our sample size is to some extent adequate. Moreover, previously adduced gender and age distribution needs to be considered since all the participants were women and the age dispersion was insufficient (mean of age=26 years, standard deviation= 6.7 years).

During the analysis of MEG data there were few difficulties with ICA artefact removal with three participants because of their weak heart artefact rate and strong alpha activity, which made it difficult to find heart artefact. For two participant, evoked responses were not calculated because of heart artefact contamination and ICA problems. These problems may have affected the findings of this study. Furthermore, Spearman's correlation does not disclose the causal connection between two variables. For instance, people who have higher tendency to BAS reward responsiveness may have in general slower PAF or pain experience can be the reason for this phenomenon. Therefore, it would be valuable to continue the analysis of the causality to understand which factor is causing the other.

Additionally, the pain stimulus in this study was produced in experimental settings. Thus, pain was known to happen several days before and the proper baseline to compare the results was absent. The real-life acute pain situations usually strike suddenly and gather attention in different way than the predictable situation. In turn, this qualitative difference in pain situation may activate different neural processes and areas. However still in practical terms the real-life acute pain events are currently impossible to research with brain imaging methods such as MEG due to its operational conditions.

## 4.4 Future studies

Our thesis and the lack of studies in this topic, underlie the need for research that focus on brain activity during, before and after pain. The further exploration should investigate how one's behavioural tendencies such as temperament and approach-avoidance behaviour impact to individual pain sensitivity and how one copes with pain afterwards. For instance, the tentative results concerning the association between PAF, BAS and pain are controversial in comparison to former studies concerning BAS and pain.

Additionally, most of the studies on temperament and pain focus on children and chronic pain. Since majority of workforce consist of adults, there should be more pain research focusing on 18 to 65 years old healthy individuals. Taking account, the significant correlation between fear and PAF after experiencing pain, it would be valuable to concentrate on the impact that fear has in the process of pain, possibly using the fear of pain questionnaire (FPQ-III). All in all, the awareness about one's temperament might help health care professionals to learn about individual's pain behaviour and understand how they experience pain. Therefore, temperament assessment could be beneficial when customizing the pain management and treatment plans for patients.

### 4.5 Conclusions

Although the association between change of PAF during pain resting states and its connection to approach-avoidance behaviour and negative affect remains unsolved, our study discovered new information with the MEG as a brain imaging method. This study not only revealed the connection between approach system, pain and resting brain activity but furthermore discovered the link between fear and pain. All results considered, this study questioned the role of negative affectivity in pain and reinforced the need for further analysis for the relationship between pain and approach-avoidance behaviour.

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**REFERENCES**


Apkarian, A. V., Bushnell, M. C., Treede, R. D., & Zubieta, J. K. (2005). Human brain mechanisms of pain perception and regulation in health and disease. *European journal of pain (London, England)*, *9*(4), 463–484. https://doi.org/10.1016/j.ejpain.2004.11.001

Asmundson, G. J., Vlaeyen, J. W. S., Vlaeyen, J. W., & Crombez, G. (Eds.). (2004). *Understanding and treating fear of pain*. Oxford University Press, USA. Retrieved from https://jyu.finna.fi/Record/jykdok.1412047

Baetz, M., & Bowen, R. (2008). Chronic pain and fatigue: Associations with religion and spirituality. *Pain research & management*, *13*(5), 383–388. https://doi.org/10.1155/2008/263751

Balconi, M., Vanutelli, M. E., & Grippa, E. (2017). Resting state and personality component (BIS/BAS) predict the brain activity (EEG and fNIRS measure) in response to emotional cues. *Brain and behavior*, *7*(5), e00686. https://doi.org/10.1002/brb3.686

Bartley, E. J., & Fillingim, R. B. (2013). Sex differences in pain: a brief review of clinical and experimental findings. *British journal of anaesthesia*, *111*(1), 52–58. https://doi.org/10.1093/bja/aet127

Başar, E., Başar-Eroglu, C., Karakaş, S., & Schürmann, M. (2001). Gamma, alpha, delta, and theta oscillations govern cognitive processes. *International journal of psychophysiology : official journal of the International Organization of Psychophysiology*, *39*(2-3), 241–248. https://doi.org/10.1016/s0167-8760(00)00145-8

Basten-Günther, J., Peters, M., & Lautenbacher, S. (2019). Optimism and the Experience of Pain: A Systematic Review. *Behavioral medicine (Washington, D.C.)*, *45*(4), 323–339. https://doi.org/10.1080/08964289.2018.1517242

Bazanova, O. M., & Vernon, D. (2014). Interpreting EEG alpha activity. *Neuroscience and biobehavioral reviews*, *44*, 94–110. https://doi.org/10.1016/j.neubiorev.2013.05.007

Boersma K. (2017). Is the search for a "pain personality" of added value to the Fear-Avoidance-Model (FAM) of chronic pain?. *Scandinavian journal of pain*, *17*, 226–227. https://doi.org/10.1016/j.sjpain.2017.08.019

Bushnell, M. C., Ceko, M., & Low, L. A. (2013). Cognitive and emotional control of pain and its disruption in chronic pain. *Nature reviews. Neuroscience*, *14*(7), 502–511. https://doi.org/10.1038/nrn3516

Buzsáki, G., & Draguhn, A. (2004). Neuronal oscillations in cortical networks. *Science (New York, N.Y.)*, *304*(5679), 1926–1929. https://doi.org/10.1126/science.1099745

Carlsson, K., Andersson, J., Petrovic, P., Petersson, K. M., Ohman, A., & Ingvar, M. (2006). Predictability modulates the affective and sensory-discriminative neural processing of pain. *NeuroImage*, *32*(4), 1804–1814.
https://doi.org/10.1016/j.neuroimage.2006.05.027

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology*, *67*(2), 319–333.
https://doi.org/10.1037/0022-3514.67.2.319

Chen A. C. (2001). New perspectives in EEG/MEG brain mapping and PET/fMRI neuroimaging of human pain. *International journal of psychophysiology : official journal of the International Organization of Psychophysiology*, *42*(2), 147–159.
https://doi.org/10.1016/s0167-8760(01)00163-5

Day, M. A., Matthews, N., Newman, A., Mattingley, J. B., & Jensen, M. P. (2019). An evaluation of the behavioral inhibition and behavioral activation system (BIS-BAS) model of pain. *Rehabilitation psychology*, *64*(3), 279–287.
https://doi.org/10.1037/rep0000274

De Pascalis, V., Sommer, K., & Scacchia, P. (2018). Resting Frontal Asymmetry and Reward Sensitivity Theory Motivational Traits. *Scientific reports*, *8*(1), 13154.
https://doi.org/10.1038/s41598-018-31404-7

Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: dopamine, facilitation of incentive motivation, and extraversion. *The Behavioral and brain sciences*, *22*(3), 491–569.
https://doi.org/10.1017/s0140525x99002046

Derryberry, D., & Rothbart, M. K. (1997). Reactive and effortful processes in the organization of temperament. *Development and psychopathology*, *9*(4), 633–652.
https://doi.org/10.1017/s0954579497001375

Dowman, R., & Schell, S. (1999). Evidence that the anterior cingulate and supplementary somatosensory cortices generate the pain-related negative difference potential. *Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology*, *110*(12), 2117–2126.
https://doi.org/10.1016/s1388-2457(99)00196-0

Dueñas, M., Ojeda, B., Salazar, A., Mico, J. A., & Failde, I. (2016). A review of chronic pain impact on patients, their social environment and the health care system. *Journal of pain research*, *9*, 457–467.
https://doi.org/10.2147/JPR.S105892

Elvemo, N. A., Landrø, N. I., Borchgrevink, P. C., & Håberg, A. K. (2015). Reward responsiveness in patients with chronic pain. *European journal of pain (London, England)*, *19*(10), 1537–1543.
https://doi.org/10.1002/ejp.687

Evans, D. E., & Rothbart, M. K. (2007). Developing a model for adult temperament. Journal of Research in Personality, 41(4), 868-888.
https://doi.org/10.1016/j.jrp.2006.11.002

Feijó, L.M., Tarman, G. Z., Fontaine, C., Harrison, R., Johnstone, T., & Salomons, T. (2018). Sex-Specific Effects of Gender Identification on Pain Study Recruitment. *The journal of pain : official journal of the American Pain Society*, *19*(2), 178–185.
https://doi.org/10.1016/j.jpain.2017.09.009

Fields H. L. (2018). How expectations influence pain. *Pain*, *159 Suppl 1*, S3–S10.
https://doi.org/10.1097/j.pain.0000000000001272

Fillingim R. B. (2005). Individual differences in pain responses. *Current rheumatology reports*, *7*(5), 342–347.
https://doi.org/10.1007/s11926-005-0018-7

Fillingim, R. B., King, C. D., Ribeiro-Dasilva, M. C., Rahim-Williams, B., & Riley, J. L., 3rd (2009). Sex, gender, and pain: a review of recent clinical and experimental findings. *The journal of pain : official journal of the American Pain Society*, *10*(5), 447–485.
https://doi.org/10.1016/j.jpain.2008.12.001

Fillingim, R. B., & Maixner, W. (1995, December). Gender differences in the responses to noxious stimuli. In Pain forum(Vol. 4, No. 4, pp. 209-221). Elsevier.
https://doi.org/10.1016/S1082-3174(11)80022-X

Furman, A. J., Meeker, T. J., Rietschel, J. C., Yoo, S., Muthulingam, J., Prokhorenko, M., Keaser, M. L., Goodman, R. N., Mazaheri, A., & Seminowicz, D. A. (2018). Cerebral peak alpha frequency predicts individual differences in pain sensitivity. *NeuroImage*, *167*, 203–210.
https://doi.org/10.1016/j.neuroimage.2017.11.042

Furman, A. J., Prokhorenko, M., Keaser, M. L., Zhang, J., Chen, S., Mazaheri, A., & Seminowicz, D. A. (2020). Sensorimotor Peak Alpha Frequency Is a Reliable Biomarker of Prolonged Pain Sensitivity. *Cerebral cortex (New York, N.Y. : 1991)*, *30*(12), 6069–6082.
https://doi.org/10.1093/cercor/bhaa124

Furman, A. J., Thapa, T., Summers, S. J., Cavaleri, R., Fogarty, J. S., Steiner, G. Z., Schabrun, S. M., & Seminowicz, D. A. (2019). Cerebral peak alpha frequency reflects average pain severity in a human model of sustained, musculoskeletal pain. *Journal of neurophysiology*, *122*(4), 1784–1793.
https://doi.org/10.1152/jn.00279.2019

Garcia-Larrea, L., & Jackson, P. L. (2016). *Pain and the conscious brain*. Lippincott Williams & Wilkins. Retrieved from
https://jyu.finna.fi/Record/jykdok.1884443

Hadjistavropoulos, T. & Craig, K. D. (2004). Pain: Psychological perspectives. Mahwah, N.J.: Lawrence Erlbaum. Retrieved from
https://jyu.finna.fi/Record/jykdok.1729922

Haegens, S., Cousijn, H., Wallis, G., Harrison, P. J., & Nobre, A. C. (2014). Inter- and intra-individual variability in alpha peak frequency. *NeuroImage*, *92*(100), 46–55. https://doi.org/10.1016/j.neuroimage.2014.01.049

Hall, E. L., Robson, S. E., Morris, P. G., & Brookes, M. J. (2014). The relationship between MEG and fMRI. *NeuroImage*, *102 Pt 1*, 80–91. https://doi.org/10.1016/j.neuroimage.2013.11.005

Hansen, P., Kringelbach, M., & Salmelin, R. (Eds.). (2010). *MEG: an introduction to methods*. Oxford university press. Retrieved from https://jyu.finna.fi/Record/jykdok.2047894

Hashemi, A., Pino, L. J., Moffat, G., Mathewson, K. J., Aimone, C., Bennett, P. J., Schmidt, L. A., & Sekuler, A. B. (2016). Characterizing Population EEG Dynamics throughout Adulthood. *eNeuro*, *3*(6), ENEURO.0275-16.2016. https://doi.org/10.1523/ENEURO.0275-16.2016

Haslam, N. (2007). Introduction to personality and intelligence (Annotated edition.). Thousand Oaks, CA: Sage. Retrieved from https://jyu.finna.fi/Record/jykdok.1826587

Hauck, M., Lorenz, J., & Engel, A. K. (2008). Role of synchronized oscillatory brain activity for human pain perception. *Reviews in the neurosciences*, *19*(6), 441–450. https://doi.org/10.1515/revneuro.2008.19.6.441

Hundt, N. E., Brown, L. H., Kimbrel, N. A., Walsh, M. A., Nelson-Gray, R., & Kwapil, T. R. (2013). Reinforcement sensitivity theory predicts positive and negative affect in daily life. *Personality and Individual Differences*, *54*(3), 350–354. https://doi.org/10.1016/j.paid.2012.09.021

The International Association for the Study of Pain (IASP). (21.11.19) https://www.iasp-pain.org

Jensen, M. P., Ehde, D. M., & Day, M. A. (2016). The Behavioral Activation and Inhibition Systems: Implications for Understanding and Treating Chronic Pain. *The journal of pain : official journal of the American Pain Society*, *17*(5), 529.e1–529.e18. https://doi.org/10.1016/j.jpain.2016.02.001

Jensen, O., Gelfand, J., Kounios, J., & Lisman, J. E. (2002). Oscillations in the alpha band (9-12 Hz) increase with memory load during retention in a short-term memory task. *Cerebral cortex (New York, N.Y. : 1991)*, *12*(8), 877–882. https://doi.org/10.1093/cercor/12.8.877

Jensen, O., Spaak, E., & Zumer, J. M. (2019). Human brain oscillations: from physiological mechanisms to analysis and cognition. *Magnetoencephalography: From signals to dynamic cortical networks*, 471-517. https://link.springer.com/chapter/10.1007/978-3-642-33045-2_17

Jensen, M. P., Tan, G., & Chua, S. M. (2015). Pain Intensity, Headache Frequency, and the Behavioral Activation and Inhibition Systems. *The Clinical journal of pain*, *31*(12), 1068–1074. https://doi.org/10.1097/AJP.0000000000000215

Jepma, M., Koban, L., van Doorn, J., Jones, M., & Wager, T. D. (2018). Behavioural and neural evidence for self-reinforcing expectancy effects on pain. *Nature human behaviour*, *2*(11), 838–855. https://doi.org/10.1038/s41562-018-0455-8

Johnsen, N. & Agerskov, R. (2009). Somatosensory cortex: Roles, interventions and traumas. New York: Nova Science Publishers. Retrieved from https://jyu.finna.fi/Record/jykdok.1773344

Jung, Y. H., Lee, U. S., Jang, J. H., & Kang, D. H. (2016). Effects of Mind-Body Training on Personality and Behavioral Activation and Inhibition System According to BDNF Val66Met Polymorphism. *Psychiatry investigation*, *13*(3), 333–340. https://doi.org/10.4306/pi.2016.13.3.333

Kalso, E., Kalso, E., Haanpää, M., Hamunen, K., Kontinen, V., Vainio, A., . . . Rusanen, S. (2018). Kipu (4., uudistettu painos ed.). Helsinki: Kustannus Oy Duodecim. Retrieved from https://jyu.finna.fi/Record/jykdok.1871601

Keltikangas-Järvinen, L. (2009). Temperamentti - persoonallisuuden biologinen selkäranka. teoksessa R-L. Metsäpelto, & T. Feldt (Toimittajat), *Meitä on moneksi: persoonallisuuden psykologiset perusteet* (Sivut 49-69). PS-kustannus. Retrieved from https://jyu.finna.fi/Record/jykdok.1494534

Kennis, M., Rademaker, A. R., & Geuze, E. (2013). Neural correlates of personality: an integrative review. *Neuroscience and biobehavioral reviews*, *37*(1), 73–95. https://doi.org/10.1016/j.neubiorev.2012.10.012

Kitamura, Y., Kakigi, R., Hoshiyama, M., Koyama, S., Shimojo, M., & Watanabe, S. (1995). Pain-related somatosensory evoked magnetic fields. *Electroencephalography & Clinical Neurophysiology*, *95*(6), 463–474. https://doi.org/10.1016/0013-4694(95)00139-5

Klein, V. C., Gaspardo, C. M., Martinez, F. E., Grunau, R. E., & Linhares, M. B. (2009). Pain and distress reactivity and recovery as early predictors of temperament in toddlers born preterm. *Early human development*, *85*(9), 569–576. https://doi.org/10.1016/j.earlhumdev.2009.06.001

Klimesch W. (2012). α-band oscillations, attention, and controlled access to stored information. *Trends in cognitive sciences*, *16*(12), 606–617. https://doi.org/10.1016/j.tics.2012.10.007

Krabbe, S., Gründemann, J., & Lüthi, A. (2018). Amygdala Inhibitory Circuits Regulate Associative Fear Conditioning. *Biological psychiatry*, *83*(10), 800–809. https://doi.org/10.1016/j.biopsych.2017.10.006

Li, D., Sun, F., Jiao. J. (1996) Frontal EEG characters in aging and the correlativity with some cognitive abilities. Acta Psychol Sinica, 28 (1) pp. 76-81
http://journal.psych.ac.cn/xlxb/EN/Y1996/V28/I01/76

Nakamura, Y., Paur, R., Zimmermann, R., & Bromm, B. (2002). Attentional modulation of human pain processing in the secondary somatosensory cortex: a magnetoencephalographic study. *Neuroscience letters*, *328*(1), 29–32.
https://doi.org/10.1016/s0304-3940(02)00447-0

Navratilova, E., Morimura, K., Xie, J. Y., Atcherley, C. W., Ossipov, M. H., & Porreca, F. (2016). Positive emotions and brain reward circuits in chronic pain. *The Journal of comparative neurology*, *524*(8), 1646–1652.
https://doi.org/10.1002/cne.23968

Navratilova, E., & Porreca, F. (2014). Reward and motivation in pain and pain relief. *Nature neuroscience*, *17*(10), 1304–1312.
https://doi.org/10.1038/nn.3811

Nelson, D. (2013). The mystery of pain. London; Philadelphia: Singing Dragon. Retrieved from https://jyu.finna.fi/Record/jykdok.1464409

Neugebauer V. (2015). Amygdala pain mechanisms. *Handbook of experimental pharmacology*, *227*, 261–284.
https://doi.org/10.1007/978-3-662-46450-2_13

Nir, R. R., Sinai, A., Raz, E., Sprecher, E., & Yarnitsky, D. (2010). Pain assessment by continuous EEG: association between subjective perception of tonic pain and peak frequency of alpha oscillations during stimulation and at rest. *Brain research*, *1344*, 77–86. https://doi.org/10.1016/j.brainres.2010.05.004

Martucci K. T. (2017). Disentangling mood and pain: a commentary on 2 manuscripts. Pain, 158(1), 4–5.
https://doi.org/10.1097/j.pain.0000000000000746

Mathewson, K. E., Lleras, A., Beck, D. M., Fabiani, M., Ro, T., & Gratton, G. (2011). Pulsed out of awareness: EEG alpha oscillations represent a pulsed-inhibition of ongoing cortical processing. *Frontiers in psychology*, *2*, 99.
https://doi.org/10.3389/fpsyg.2011.00099

Mogil J. S. (2012). Sex differences in pain and pain inhibition: multiple explanations of a controversial phenomenon. *Nature reviews. Neuroscience*, *13*(12), 859–866. https://doi.org/10.1038/nrn3360

Muris, P., Meesters, C., van den Hout, A., Wessels, S., Franken, I., & Rassin, E. (2007). Personality and temperament correlates of pain catastrophizing in young adolescents. *Child psychiatry and human development*, *38*(3), 171–181.
https://doi.org/10.1007/s10578-007-0054-9

Paquet, A., Plansont, B., Labrunie, A., Malauzat, D., & Girard, M. (2017). Past Pain Experience and Experimentally induced Pain Perception. *Issues in mental health nursing*, *38*(12), 1013–1021. https://doi.org/10.1080/01612840.2017.1354103

Pfurtscheller, G., & Lopes da Silva, F. H. (1999). Event-related EEG/MEG synchronization and desynchronization: basic principles. *Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology*, *110*(11), 1842–1857. https://doi.org/10.1016/s1388-2457(99)00141-8

Peng, W., Huang, X., Liu, Y., & Cui, F. (2019). Predictability modulates the anticipation and perception of pain in both self and others. *Social cognitive and affective neuroscience*, *14*(7), 747–757. https://doi.org/10.1093/scan/nsz047

Ploner, M., Gross, J., Timmermann, L., Pollok, B., & Schnitzler, A. (2006). Pain suppresses spontaneous brain rhythms. *Cerebral cortex (New York, N.Y. : 1991)*, *16*(4), 537–540. https://doi.org/10.1093/cercor/bhj001

Ploner, M., Gross, J., Timmermann, L., & Schnitzler, A. (2002). Cortical representation of first and second pain sensation in humans. *Proceedings of the National Academy of Sciences of the United States of America*, *99*(19), 12444–12448. https://doi.org/10.1073/pnas.182272899

Ploner, M., Sorg, C., & Gross, J. (2017). Brain Rhythms of Pain. *Trends in cognitive sciences*, *21*(2), 100–110. https://doi.org/10.1016/j.tics.2016.12.001

Price D. D. (2000). Psychological and neural mechanisms of the affective dimension of pain. *Science (New York, N.Y.)*, *288*(5472), 1769–1772. https://doi.org/10.1126/science.288.5472.1769

Purves, D. (2012). Neuroscience (5th ed.). Sunderland, Mass: Sinauer Associates. Retrieved from https://jyu.finna.fi/Record/jykdok.1434703

Rainville, P., Duncan, G. H., Price, D. D., Carrier, B., & Bushnell, M. C. (1997). Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science (New York, N.Y.)*, *277*(5328), 968–971. https://doi.org/10.1126/science.277.5328.968

Ranger, M., & Campbell-Yeo, M. (2008). Temperament and pain response: a review of the literature. *Pain management nursing : official journal of the American Society of Pain Management Nurses*, *9*(1), 2–9. https://doi.org/10.1016/j.pmn.2007.09.005

Rathee, S., Bhatia, D., Punia, V., & Singh, R. (2020). Peak Alpha Frequency in Relation to Cognitive Performance. *Journal of neurosciences in rural practice*, *11*(3), 416–419. https://doi.org/10.1055/s-0040-1712585

Rolls E. T. (2004). The functions of the orbitofrontal cortex. *Brain and cognition*, *55*(1), 11–29. https://doi.org/10.1016/S0278-2626(03)00277-X

Rothbart, M. K. (1989). *Temperament and development.* In G. A. Kohnstamm, J. E. Bates, & M. K. Rothbart (Eds.), *Temperament in childhood* (p. 187–247). John Wiley & Sons. https://www.researchgate.net/publication/290821062_Temperament_and_Development

Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. L. Brown (Eds.), Advances in developmental psychology (Vol. 1, pp. 37–86). Hillsdale, NJ: Erl- baum. https://www.researchgate.net/publication/285885145_Development_of_individual_differences_in_temperament

Rothbart, M. K., & Sheese, B. E. (2007). Temperament and emotion regulation. Handbook of emotion regulation, 331-350 https://www.researchgate.net/publication/232477868_Temperament_and_emotion-regulation

Rothbart, M. K., & Bates, J. E. (2006). Temperament in children's development. In W. Damon, R. Lerner, & N. Eisenberg (Eds.), Handbook of child psychology (6th ed.): Vol 3. Social, emotional, and personality development (pp. 99–166). New York: Wiley. https://www.researchgate.net/publication/229633680_Handbook_of_Child_Psychology

Rothbart, M.K.& Gartstein, M.A. (2008), Temperament. Editor(s): Marshall M. Haith, Janette B. Benson,Encyclopedia of Infant and Early Childhood Development,Academic Pres,Pages 318-332, https://doi.org/10.1016/B978-012370877-9.00161-4

Roxo, M. R., Franceschini, P. R., Zubaran, C., Kleber, F. D., & Sander, J. W. (2011). The limbic system conception and its historical evolution. *TheScientificWorldJournal*, *11*, 2428–2441. https://doi.org/10.1100/2011/157150

Ruau, D., Liu, L. Y., Clark, J. D., Angst, M. S., & Butte, A. J. (2012). Sex differences in reported pain across 11,000 patients captured in electronic medical records. *The journal of pain : official journal of the American Pain Society*, *13*(3), 228–234. https://doi.org/10.1016/j.jpain.2011.11.002

Rudy, J. W. (2014). The neurobiology of learning and memory (Second edition.). Sunderland, Massachusetts: Sinauer Associates, Inc. Publishers. Retrieved from https://jyu.finna.fi/Record/jykdok.1504726

Sanson, N., Hach, S., Moran, R., & Mason, J. (2020). Behavioural activation and inhibition systems in relation to pain intensity and duration in a sample of people experiencing chronic musculoskeletal pain. *Musculoskeletal science & practice*, *47*, 102129. https://doi.org/10.1016/j.msksp.2020.102129

Schneider, M., Chau, L., Mohamadpour, M., Stephens, N., Arya, K., & Grant, A. (2016). EEG asymmetry and BIS/BAS among healthy adolescents. *Biological psychology*, *120*, 142–148. https://doi.org/10.1016/j.biopsycho.2016.09.004

Seifert, F., Schuberth, N., De Col, R., Peltz, E., Nickel, F. T., & Maihöfner, C. (2013). Brain activity during sympathetic response in anticipation and experience of pain. *Human brain mapping*, *34*(8), 1768–1782. https://doi.org/10.1002/hbm.22035

Sep, M., Steenmeijer, A., & Kennis, M. (2019). The relation between anxious personality traits and fear generalization in healthy subjects: A systematic review and meta-analysis. *Neuroscience and biobehavioral reviews*, *107*, 320–328. https://doi.org/10.1016/j.neubiorev.2019.09.029

Serrano-Ibáñez, E. R., López-Martínez, A. E., Ramírez-Maestre, C., Esteve, R., & Jensen, M. P. (2019). The behavioral inhibition and activation systems and function in patients with chronic pain. *Personality and Individual Differences*, *138*, 56-62. https://doi.org/10.1016/j.paid.2018.09.021

Serrano-Ibáñez, E. R., Ramírez-Maestre, C., López-Martínez, A. E., Esteve, R., Ruiz-Párraga, G. T., & Jensen, M. P. (2018). Behavioral inhibition and activation systems, and emotional regulation in individuals with chronic musculoskeletal pain. Frontiers in psychiatry, 9, 394. https://doi.org/10.3389/fpsyt.2018.00394

Simons, L. E., Moulton, E. A., Linnman, C., Carpino, E., Becerra, L., & Borsook, D. (2014). The human amygdala and pain: evidence from neuroimaging. *Human brain mapping*, *35*(2), 527–538. https://doi.org/10.1002/hbm.22199

Singh S. P. (2014). Magnetoencephalography: Basic principles. *Annals of Indian Academy of Neurology*, *17*(Suppl 1), S107–S112. https://doi.org/10.4103/0972-2327.128676

Sullivan, M. J., Thorn, B., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., & Lefebvre, J. C. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *The Clinical journal of pain*, *17*(1), 52–64. https://doi.org/10.1097/00002508-200103000-00008

Tarkka, I. M., & Treede, R. D. (1993). Equivalent electrical source analysis of pain-related somatosensory evoked potentials elicited by a CO2 laser. *Journal of clinical neurophysiology : official publication of the American Electroencephalographic Society*, *10*(4), 513–519. https://doi.org/10.1097/00004691-199310000-00009

Timmermann, L., Ploner, M., Haucke, K., Schmitz, F., Baltissen, R., & Schnitzler, A. (2001). Differential coding of pain intensity in the human primary and secondary somatosensory cortex. *Journal of neurophysiology*, *86*(3), 1499–1503. https://doi.org/10.1152/jn.2001.86.3.1499

Uhl, K., Litvinova, A., Sriswasdi, P., Zurakowski, D., Logan, D., & Cravero, J. P. (2019). The effect of pediatric patient temperament on postoperative outcomes. *Paediatric anaesthesia*, *29*(7), 721–729. https://doi.org/10.1111/pan.13646

Villemure, C., & Bushnell, M. C. (2009). Mood influences supraspinal pain processing separately from attention. *The Journal of neuroscience : the official journal of the Society for Neuroscience*, *29*(3), 705–715. https://doi.org/10.1523/JNEUROSCI.3822-08.2009

Vlaeyen, J. W., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain*, *85*(3), 317–332. https://doi.org/10.1016/s0304-3959(99)00242-0

Walton, K. D., Dubois, M., & Llinás, R. R. (2010). Abnormal thalamocortical activity in patients with Complex Regional Pain Syndrome (CRPS) type I. *Pain*, *150*(1), 41–51. https://doi.org/10.1016/j.pain.2010.02.023

Wiesenfeld-Hallin, Z. (2005). Sex differences in pain perception. Gender medicine, 2(3), 137-145. https://doi.org/10.1016/S1550-8579(05)80042-7

Wiltink, J., Vogelsang, U., & Beutel, M. E. (2006). Temperament and personality: the German version of the Adult Temperament Questionnaire (ATQ). *Psycho-social medicine*, *3*, Doc10. https://www.researchgate.net/publication/26800395_Temperament_and_personality_The_German_version_of_the_Adult_Temperament_Questionnaire_ATQ

Yoshino, A., Okamoto, Y., Onoda, K., Shishida, K., Yoshimura, S., Kunisato, Y., Demoto, Y., Okada, G., Toki, S., Yamashita, H., & Yamawaki, S. (2012). Sadness enhances the experience of pain and affects pain-evoked cortical activities: an MEG study. *The journal of pain : official journal of the American Pain Society*, *13*(7), 628–635. https://doi.org/10.1016/j.jpain.2011.12.005

Zappasodi, F., Pasqualetti, P., Tombini, M., Ercolani, M., Pizzella, V., Rossini, P. M., & Tecchio, F. (2006). Hand cortical representation at rest and during activation: gender and age effects in the two hemispheres. *Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology*, *117*(7), 1518–1528. https://doi.org/10.1016/j.clinph.2006.03.016

## APPENDIX

Figure 5. Scatter plots of significant correlations that did not pass the threshold criterion