# The Role of Fear and Anxiety in the Subjective Pain Experience

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# 1. Abstract

Dette er et kandidatspeciale som forsøger at undersøge hvordan angst og frygt kan ændre den subjective oplevelse af smerte, og problemformuleringen lyder ”*Kan angst eller frygt for et elektrisk stød sænke den oplevede intensitet af ”the thermal grill illusion*”?” Udforskningen af dette gøres gennem en ”teori og baggrunds” sektion, hvor de vigtigste teorier omkring smerteperception, Gate control theory of pain og neuromatrix theory of pain bliver presenteret. Disse argumenterer i stigende grad for at smerte ikke skal forstås som en 1:1 relation mellem nerveinput og oplevelse af smerte. Smerte er en subjektiv oplevelse som bliver skabt af, og reguleret igennem, det centrale nervesystem.

Disse to teorier har fostret meget research, bland andet fordi forskere har set dem for argument for at følelser måske kan være i stand til at ændre hvordan smerte perciperes, og efter disse er præsenteret, præsenteres noget af den research som er gjort som har vist at både tristhed og angst kan ændre hvor intenst smerte opleves. Derefter præsenteres to modeller som forskere har brugt til at forklare resultaterne fra sådanne studier. Disse er ”the attentional model of pain perception” og ”the motivational model of pain perception”. På basis af disse modeller laves en hypotese som lyder “*Induceret frygt eller angst for et elektrisk stød vil højne smertetærsklen på en thermal grill opgave*”.

Hypotesen bliver eksperimentelt testet i et eksperiment hvor deltagere deles op i tre grupper. En kontrol gruppe, en angst gruppe og en frygt gruppe. For at inducere angst eller frygt får deltagere en elektrode på pegefingeren. I angstgruppen får de at vide at de måske kan få et smertefuldt stød af elektroden, men bliver ikke tildelt et stød på noget tidspunkt under forsøget. Frygt gruppen får samme instruktion, men får et smertefuldt stød inden de placerer hånden på the thermal grill. Deltagere bliver spurgt om hvor intenst thermal grill stimulansen opleves seks gange under forsøget, og får at vide at en intensitet på ti svarer til deres smertetærskel, som er målt inden forsøgets start.

Efter en præsentation af eksperimentet og resultaterne, diskuteres resultaterne af forsøget. Dette fører videre til en general diskussion af de to modeller som forsøger at forklare emotioners indflydelse på som er præsenteret. Til sidst i diskussionen foreslås at en ny model bør indeholde elementer fra begge modeller, da de ikke alene kan forklare resultater fra alle forsøg som er lavet indenfor feltet.

Baseret på dette er der lavet en konklusion som inddrager begge modeller. Konklusionen er at angst eller frygt for et elektrisk stød godt kan sænke den oplevede smerteperception, men at det kommer an på flere faktorer. Disse faktorer emotionel valens og rettethed af individets opmærksomhed.

## 2. Introduction

The quest for understanding pain has undergone for centuries. One of the earliest known theories of pain dates back to the French philosopher Rene Descartes whose theory of pain is still accepted outside of the scientific community. Descartes idea was that the intensity of a stimulus would transmit directly to the brain, and cause an equivalent disturbance. The experienced intensity of the pain would in this theory be based solely on the intensity of the stimulus.

Early research on pain was based on the idea of Descartes', and the earliest scientific theory of pain is *specificity theory of pain*. The central idea of specificity theory is the existence of specialized neurological pathways, which sole function is to react to painful stimuli (Moayedi & Davis, 2013, p. 8). The specificity theory could, however, was unsuccessful in explaining some phenomena of pain, such as how *temporal summation* (continuous stimulation of an otherwise non-painful stimulus) could cause pain. This led to the suggestion of other theories, such as *intensity theory* were proposed. According to intensity theory there are no specific "pain pathways", the experience of pain is rather created by the same nerves that register any other somatosensory stimulus, and is experienced as painful when the stimulus intensity exceeds a certain threshold (ibid.).

These early theories have been important in shaping the field of pain research. An especial important contribution of the "battle" between the theories, was that much effort was put into finding the specialised pain pathways, suggested by specificity theory, since this would seem to settle the debate once and for all. This led to the important discovery of these pain pathways, large afferent myelinated fibres in 1965 and small afferent unmyelinated fibres in 1967 (ibid.,).

The discovery of the specialized pain pathways did, however, not settle the debate, as the "winner", specificity theory, still was not able to account for some specifics of pain experience, such as the earlier mentioned temporal summation, but also it did not account of psychological factors could have on pain perception. If there is a 1:1 relationship between the activation of pain receptors and pain perception, then the only factor influencing pain should be stimulus intensity. That, however, did not seem to be the case. For this reason, several new theories were suggested. These theories are known as *pattern theories.* This is an umbrella term for several theories which tried to unify specificity and intensity theory. This led to one

of the most important theories of pain, *gate control theory of pain* (GT). This theory will be covered much more extensively in section 2.1.1, however, it was important to mention here, because it is the theory that started putting emphasis on the central nervous system in the experience of pain.

The emphasis on the role of the central nervous system opened up the idea that maybe individual psychological characteristics or affective states could influence pain. Since the emergence of GT much research has been conducted trying to understand the emotional aspects of pain. Since then a lot of research has been made, which has investigated the link between traits, emotions and pain. We will turn to a more thorough discussion of some of these studies in section 2.2.

Since the invention of neuro imagining techniques such as position emission tomography (PET) scans and Functional magnetic resonance imaging (fMRI), researchers have sought to finding the neural correlates of pain, and several areas have been identified such as the primary somatosensory cortex (S1), and the secondary somatosensory cortex (S2) (Wager, & Atlas, 2015, p. 407). But most important for this thesis is that, areas usually associated with emotion have also been found to be activated during the sensation of pain. Areas such as the anterior cingulate cortex, which is part of the limbic system and the dorsal posterior insula (ibid., p. 407).

The realization that emotion is an integral part of the sensation of pain is of great interest to clinicians. The most important treatment of chronic pain is still pharmaceutical intervention, but considering the detrimental side effects of years of substance abuse, alternative forms of treatment with less side effects would be preferable. In recent years' research has been investigating how to manipulate pain sensitivity. Rhudy. and Meagher (2000), found that anxiety (threat of shock) lowered pain thresholds, whereas fear (experience of shock) elevated thresholds, Boettger et al. (2011) found that inducing sad mood elevated experienced pain perception. Some studies on how psychological intervention can alter pain perception are accounted for in section 2.2.2.

Even though it is now well established that emotions can affect how we perceive pain, it is still not well understood exactly how. The difficulty arises because different methodologies seem to yield different results. For example, some studies suggest that arousal and valence are

the most important predictors of pain perception (Rhudy & Meagher, 2001), while others suggest that the most important factor is attention (Arntz et al., 1991).

The aim of this master's thesis is to try to gain a better understanding of why different methodologies might yield different results. Specifically, it tries to investigate if level of arousal correlates with the perceived intensity of a painful stimulus.

To investigate this, an experiment was designed, which purpose was to investigate the difference between induced fear and anxiety. These emotions are similar in valence, but differ in arousal. The idea is that it can help shed light on the problem statement:

*"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?"*

I use the definitions of fear and anxiety put forward by Rhudy. and Meagher, (2000).

*"Fear is an immediate alarm reaction to present threat, characterized by impulses to escape, and typically results in surge of sympathetic arousal (Barlow et al., 1996). Anxiety, on the other hand, is a future-oriented emotion characterized by negative affect and apprehensive anticipation of potential threats"* (Rhudy & Meagher, 2000, p. 65).

According to this definition the distinction between fear and anxiety is that fear is felt towards a threat that is known (in this experiment a painful experienced shock) and anxiety is felt when the stimulus is unknown (The threat of a painful shock).

The definition I use for intensity is as a subjective experience of the stimulus, and is measured by a *visual analogue scale(*VAS) going from 0-20. 0 is defined as no sensation at all, 10 as the subjective pain threshold and 20 as unbearable pain. This also means that in the assignment lowering experienced intensity and raising pain thresholds are used interchangeably.

The experiment is similar to one of Rhudy & Meagher, in which they also tested the difference between fear and anxiety on pain perception. In the study, they concluded that there were "divergent effects" (ibid., p. 65). Specifically, that anxiety heightened pain perception, whereas fear reduced it (ibid.). They conclude that this is due to fear causing a higher level of autonomic arousal, and hence it blocks perception of pain (ibid.).

The study of Rhudy & Meagher serves as an argument for the *aversive-appetitive* or *motivational* model of pain perception, which is discussed in section 2.3.1. The reason I believe it is necessary to replicate is that they got results that are different than results of other studies (such as Arntz et al, 1992), which suggest that attention has a higher predictive power than level of autonomic arousal. The study of Rhudy and Meagher used a slightly unconventional measure of pain threshold (a finger withdrawal task). Because of this, I reason that to properly discuss my problem statement *"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?"*, a reproduction of their study, using a more solid measure of pain sensitivity, is needed A more extensive discussion of why design by Rhudy and Meagher might be problematic are presented in section 7.1.

In this assignment will try to answer the problem statement by first accounting for the background of the main theories of pain, the gate theory and the neuromatrix theory. These theories led to the understanding that pain is not as simple as understanding the peripheral nervous system, but that the brain plays an important role in interpreting and altering the incoming signals, which is the theoretical basis for the interest in studying the role emotions play in pain perception.

Second part of the "theory and background" will account for the emergence of an understanding of a pain network in the brain. In this section, the emergence of evidence for the role of emotional centres in the brain will be presented. This evidence comes from neuroimaging and studies on how personality traits and experimental manipulation of mood seems able to effect pain perception.

The "theory and background" section will conclude with a summary and the hypothesis that I have about the experiment.

After the "theory and background", there is a "methodology section. In this, I account for, and discuss, the experimental setup that is used in the experiment. Arguments are presented for the choice of psychological tests, apparatus and procedure.

The next section is the "method and results" section this explains in detail how the experiment was set up and the procedure. Here the results are also presented.

Then comes the "discussion", which is divided into a results discussion and a general discussion. The results discussion will interpret the statistical analysis and end with a

conclusion on the data from my experiment. This conclusion will be used in the overall discussion to suggest an implication for the current theories of how emotion influences pain perception. In the end of the assignment there will be a conclusion.

# 3. Theory and background

To answer the problem statement *"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?"* the main theories of pain are accounted for, and how they led to the focus on the brain in pain research. Afterwards research already done on the subjects will be presented. This will lead to hypothesis on the experiment, which are based on the previous research.

## 3.1. Theories of pain

As touched briefly upon on the introduction, several theories of pain have been formulated. They have all contributed to the field of pain research, and it can be said that current theories contain elements from the older theories. This section will only account for the newer theories, as these are the ones relevant for the discussion. I will, however, touch on some of the criticisms of specificity and pattern theory. This is because the realizations of the limitations of these theories is what led to the formulation of the theories that we still use today, the gate theory of pain and the neuromatrix' theory of pain. These theories are not mutually exclusive; rather neither theory is complete on its own. Both theories will be accounted for in this section in order of when they were formulated.

### 3.1.1. Gate control theory of pain

Gate control theory of pain first came as a criticism of specificity theory and pattern theory. Specificity theory holds two central assumptions, which both have their problems. The first assumption is what is known as the *physiological assumption*, namely that specific receptors respond to specific forms of stimulation and then transmit that directly to the brain (Melzack & Wall, 1965, p. 971). This assumption, however, fail to explain several phenomena of pain, for example how surgical lesions fail to abolish pain and how gentle touch or vibration can cause pain (ibid.). Even more problematic is what is called the *psychological assumption*, that there is a 1:1 relationship between stimulus and pain, as it is well established that pain can be abolished by psychological factors. Melzack and Wall mention two cases 1) soldiers of the at the battle of Anzio who felt no pain following extensive battle wounds, presumably

because of the bliss of having survived and 2) Pavlov's dogs who would be applied with a painful shock and afterwards be pretended with food. After a while, the dogs did no longer experience the shock as being painful as they had learned to associate it with food (ibid.) According to Melzack and Wall a reaction against the psychological assumption is the main reason for what is known as pattern theory. Pattern theory suggests the experience of pain is not created by the firing of specialized neurons, but rather the pattern of firing that occurs. The biggest issue with pattern theory is that is does not recognize the existence of specialized pain receptors. Receptors for which evidence was starting to emerge (ibid., p. 973).

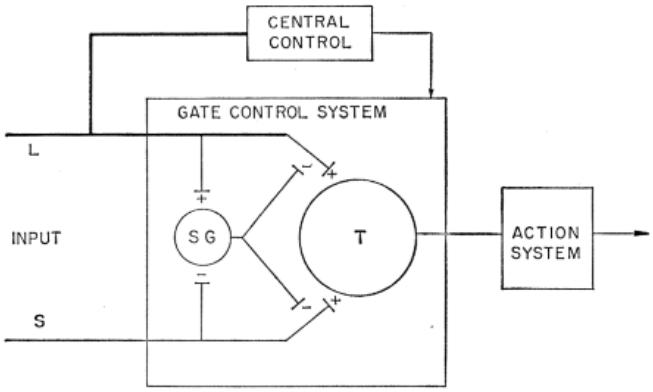
The gate control theory tries to unify pattern theory and specificity theory, by stating that there are specialized pain pathways, known as Aδ- and C-fibres, but that the pattern of firing of the different neurons can alter the perception of pain.

The gate control theory does this by suggesting three mechanisms which work together to cause the sensation of pain, the gate control system, the central trigger, and the action system.

#### *3.1.1.1. The gate control system*

According to the gate control theory in order to explain pain you must look at the pattern of specialized touch and pain receptors. Fig 1 shows illustrates the gate control system. The skin contains two kinds of afferent nerve fibres; Aδ fibres, which are large myelinated fibres and C fibres which are small unmyelinated fibres. Both of these fibres transmit to the substantia gelatinosa (SG) and the transmission cells (T-cells) of the spinal cord (ibid., p. 975). The T-cells transmit to the brain, which starts what is called an *action system*, an action system is simply defined a response to a stimulus. As fig. 1 shows, cells of the SG inhibit the transmission cells. This means that a more active SG inhibits the action system. The large diameter Aδ or "touch" fibres excite the SG whereas the small C or "pain" fibres inhibits the SG (ibid.). This is the gate control. The "pattern" of the "specialized" fibres determines the pain response

*Fig 1 - Taken from Melzack & Wall (1965)*




### 3.1.1.2. The central control trigger

The second component of the gate control theory is the "central control trigger", which is a mechanism, which allows input from the brain to effect the feeling of pain. The mechanism of how this would function was according to Melzack not well understood, but the theory was that there was a central control trigger, which consists of a nerve impulse sent to the brain before it reaches the gate control system (ibid., p. 976). Since it was already well established that stimulation of the brain triggers efferent fibres, which can influence earlier synaptic levels, this allows for a feedback system, which "asks" the brain if the gate should be open to the pain or closed.

There is some evidence for a mechanism such as this. The authors bring up the examples of the soldiers injured in war who feels no pain from their wound, but complain bitterly about an inept vain puncture, or Pavlov's dogs who would not feel pain from a shock after having learned they would get food right after, but still feel pain from shock applied to another part of the body (ibid.).

### 3.1.1.3. The action system

The "action system" is the last level of the gate control system and is activated when the integrated firing of the T-cells exceeds a critical level (ibid.). The reason for calling it the action system, rather than for instance the "pain response" is that pain is not considered a "single ring of the appropriate central bell", but rather it is an ongoing process (ibid.). Sudden unexpected damage leads to a series of actions such as, a startle reflex, withdrawal of the limb, reorientation of the head, vocalization and so on (ibid.)

#### 3.1.1.4. Merits of the gate control theory

This gate control theory helped explain many previously unexplained phenomena of pain that were previously not explainable by the existing theories. Here are some examples of things that could now be explained.

1) Temporal summation – The painful sensation of repeated or long-term application of a non-painful stimulus can be explained by fatiguing the L-neurons, thereby leaving more S-neurons to fire relative to the L-neurons.
2) Spatial summation – The experienced intensity of a painful stimulus seems to increase with stimulus area, even if the intensity of the stimuli is kept the same. This can easily be explained by the gate control theory, as any synaptic input that converges on the same cell would contribute to the output (ibid., p. 977)

The new understandings granted by gate theory also changed the way chronic pain is treated, now instead of severing nerves methods of peripheral stimulation were used. Most famously transcutaneous electrical nerve stimulation (TENS) which is non-painful electrical stimulation at the area where pain is felt. This stimulation can help relieve pain, through the mechanism of "closing the gate" (Melzack, 1999, p. 122).

The most important contribution for our purpose is, however, the introduction of a "gate", that can be opened or closed by the central control trigger. A mechanism within the brain. With this theory the focus shifted from the peripheral nervous system to the central nervous system, and it laid the foundation for the neuromatrix theory, which emphasis on the brain is even stronger. This theory will be discussed in the next section

#### 3.1.1.5. Criticism of the gate control theory

There are some criticisms to the gate control theory, but most of them are physiological in nature and concern the structure of the spinal cord and the localization of the model (Moayedi & Davis, 2013, p. 9). These criticisms can be ignored for the purpose of this assignment, as the important part is the conceptual idea.

The most important criticism of the gate control theory that is relevant for psychological research, however, comes from Ronald Melzack himself and are about the limited understanding the gate control theory gives to the phenomenon of phantom limb pain. Melzack gives a thorough analysis of the experience of phantom limb pain in his article

"Phantom Limbs, The Self And The Brain" (1989), in which he stresses the incredible feeling of realness phantom limbs have. They can feel touch, pressure, warmth, they can feel so real that individuals missing a leg can even try to jump out of bed and land on the missing leg (Melzack, 1989, pp. 2 & 3).

This lead Melzack to conclude that the brain is even more important than previously suggested, even after the introduction of the gate control theory and the central control trigger.

### 3.1.2. The neuromatrix theory of pain

From his work with phantom limbs, Melzack came with four important conclusions, which he in 1989 used to formulate, the *neuromatrix theory of pain*.

1) Phantom limbs seem to feel every bit as real as actual limbs. Patients report prosthetic hands to feel every bit as real as a real hand, and even treatment with medicine that causes tremors, would lead patients to feel tremors in amputated limbs. This led to the conclusion that "The experience of a phantom limb has the quality of reality because it is produced by the same brain processes that underlie the experience of the body when it is intact." (Melzack, 1989, p. 4).
2) The quality of the sensation of phantom limbs is as diverse as that of real limbs. They are reported to feel warm, cold, wet and even sweaty. This lead to the conclusion that "Neural networks in the brain generate all the qualities of experience that are felt to originate in the body; inputs from the body may trigger or modulate the output of the networks but are not essential for any of the qualities of experience." (Melzack, 1989, p. 4).
3) Phantom limbs feel every bit as part of the body as real limbs. They move with the body. This led to the conclusion "The experience of the body has a unitary, integrated quality which includes the quality of the "self"—that the body is uniquely one's own and not that of any other individual." (Melzack, 1989, p. 5).
4) Children born without certain limbs, seem to feel the phantom limbs, just like patients who got a limb amputated later on. This led to the following conclusion "The neural network that underlies the experience of the body-self is genetically determined but can be modified by sensory experience." (Melzack, 1989, p. 6).

From these four conclusions come Melzack's definition of the neuromatrix; "the entire network, whose spatial distribution and synaptic links are initially determined genetically, and are later sculpted by sensory inputs, as a neuromatrix" (Melzack, 1989, p. 8).

The neumatrix theory of pain is a model consisting of four levels, the body-self neuromatrix, the neurosignature, the sentient neural hub and the action neuromatrix. I will explain these levels in turn

#### *3.1.2.1. The body self neuromatrix*

The body self neuromatrix is a network of neurons in the brain which grants possibility to "feel" your body. The important notions of the neuromatrix in Melzack's theory is its ability to change with experience, which means that how the body is influenced by pain can change, and the second property is that it is widespread throughout the brain (Melzack, 1989, p. 11)

#### *3.1.2.2. The neurosignature*

The neurosignature is the the distinct patten of signals within the body self neuromatrix which leads to the perception of pain. It functions through cyclical processing and synthesis, meaning that signals go back and forth in the neuromatrix and in the end it synthesises in the sentient neural hub (ibid.).

#### *3.1.2.3. The sentient neural hub*

The sentient neural hub can be thought of as the conscience of pain. According to the theory, this is where an awareness of the pain comes. This leads to an action neuromatrix (ibid.).

#### *3.1.2.4. The action neuromatrix*

The theory proposes that the information from the neuromatrix synthesises in the brainstem and there produces the "output", which is determined by two systems. The neuromatrix in the brainstem, which produces the awareness of the output, and a neuromatrix that is involved in overt action (ibid., p. 12). This distinguishing is important, as it says not all pain is related to action, but there are two different systems, which means that it is possible to feel pain even in the absence of useful responses.

#### *3.1.2.5. Merits of the neuromatrix theory of pain*

The merits of the neuromatrix theory of pain are mainly twofold. Firstly, it serves to explain how pain, which is usually thought of as an adaptive component of human existence, can sometimes be pathological. Namely because the action neuromatrix is divided in two systems

as mentioned in the previous chapter. The one giving awareness of the pain and the one leading to overt action. It is easy to speculate the system would work that way, as the awareness is adaptive as a function of changing future behaviour, whereas the overt action is to protect the individual from immediate danger. Melzacks own example is the phantom limb pain, where he believes that the pain from phantom limbs is created in the sentient neural hub, due to a lack of neural input from the missing limb (ibid.). The second merit of the theory is that it explains why surgical removal of parts of the brain, for example the thalamus, fails to relieve patients with chronic pain. The neuromatrix theory states that it is a huge network in the brain, and not localized to a "pain center" as previously believed (ibid.).

#### *3.1.2.6. Criticism of the neuromatrix theory of pain*

To my knowledge, the theory has few criticisms. The main problem is finding out whether there really is a "pain matrix" as it has later been called. Specific neurons, which respond only to pain. Based on evidence from neuroimaging techniques it seems that there might not be, since the neurons responding to pain also seem to respond to other stimuli (Iannetti, & Mouraoux, 2010, p. 8).

### 3.1.3. Summary and concluding remarks on theories of pain

There are currently two leading theories of pain, the gate control theory and the neuromatrix theory. These theories are probably best seen as complementing each other and not as competing theories.

The gate control theory mainly tries to grasp the mechanisms in the periphery nervous system and in the spinal cord, and proposes that there is a "gating" mechanism, which can block inputs from the peripheral nervous system. The gate can be closed both by mechanisms in the peripheral nervous system and by processes in the brain. The main contribution of the gate control theory from our perspective is that it leads the focus of pain research towards the central nervous system.

The neuromatrix theory goes a step further, and tries to understand how pain is processed in the brain. Even though this is not stated explicitly by Melzack, it can be seen as the system in the brain which can also "open and close" the gate. The main contribution of the neuromatrix theory is to move the focus of research into the brain and try to find what one might call "the neural correlate of pain". The theory however does state that many parts of the brain are involved.

There is some criticism of the theories, but those are mainly technical and not important for the purpose of this assignment. These criticisms are about the exact mechanisms in the spinal cord which produces the gating mechanism, and if the neuromatrix actually only processes pain, or if it also processes other stimuli. The major takeaway from these theories is that pain is in the brain, and might be influenced by several different brain regions, and that this gives reason to believe that it might be modulated by other factors than stimuli intensity.

## 3.2. The pain in the brain

Nowadays the brain is considered an integral part of the pain experience, and there has been a boom in the field of research that researches how brain processes can affect pain perception. As early as the 1960s lesion studies of humans suggested that damage to the anterior cingulate cortex could ease the distress associated with pain (Apkarian, 2013, p. 111), and several animal models of pain were made during the 1980s, which suggested that several parts of the cerebral cortex had a function in mediating pain. This is interesting to psychologist, as it means you can theorise that it might be possible to developing novel behavioural/psychological therapies that can aid in pain reduction.

The mapping of pain centres in the brain really gained momentum in the 1990s when brain-scanning methods such as positron emission tomography (PET) become more widely used. The first studies on pain using these modern scanning methods were published in 1991 and 1992, and even though they had different results, they supported Melzack's theory of a widespread neural network of pain (ibid.)

### 3.2.1. The pain network

Hundreds of studies have now researched the neural correlate of pain and scanning techniques are continuously improving, with the introduction of functional magnetic resonance imaging (fMRI), electroencephalographic (EEG) analysis and magnetoencephalographic (MEG) analysis. These techniques have helped us gain a good map of areas, which effect pain perception.

The cortical regions that are most commonly seen activated are the primary somatosensory cortex (S1), the secondary somatosensory cortex, and the prefrontal cortex (PFC) (ibid., p. 112). Several subcortical areas have also shown to be active during pain processing, such as the anterior cingulate cortex (ACC), the insular cortex (IC). The amygdala, the thalamus, the cerebellum and the periaqueductal grey area (PAQ) (ibid.).

Many of the areas that are activated have been associated with emotional processing. The insula is activated during feelings of depression (Breedlove et al., 2010, p. 462) and disgust (Kalat, 2009, p. 348), and the anterior cingulate cortex is activated in both love and in sadness (Breedlove et al., 2010, p. 462). The amygdala is part of the limbic system, which is generally considered the emotional centre of the brain. The PFC is generally part of decision making and planning.

Interestingly, we now know that the ventromedial prefrontal cortex, the hypothalamus and the amygdala all synapse on to the periaqueductal grey area (PAG) in the brain, which is theorized as being part of the descending pathway, which closes the gate, due the strong correlation with activation of this area placebo induced hypoalgesia. (Wager & Atlas, 2015, p. 409). This suggests a pathway through which emotion can alter the experience of pain.

In fact, it is generally accepted that emotion does play a role in the pain experience, and several studies has been done showing that it is possible to alter pain perception. One field of interest is the field of placebo research. Here it is possible to investigate which specific areas are less or more activated when a placebo effect is present compared to when it is not. Areas such as the dorsal ACC, the thalamus and the anterior insula are all consistently less activated in the research when subjects experience a placebo effect (ibid., p. 407).

### 3.2.2. Psychological intervention and their effect on pain perception

Since it is accepted that a network of neurons in the brain is responsible for altering the perception of pain, and since several parts of this network can be activated or inhibited by emotional states, it is reasonable to believe that emotions such as depression, fear or anxiety can alter the perception of pain. The question is; does it also work in practice? In order to be able to confirm or disconfirm this with any sort of confidence we need placebo controlled experimental trials. Otherwise we cannot be very confident that other factors do not play a role. Such experiments have already been conducted. In order to answer my problem statement *"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?"*, I will fist account for research done on how manipulating subjects to feel sad or depressed might alter pain perception, as sad mood is similar in valence, but lower in arousal.

#### 3.2.2.1. Pain and depressive states

Some of the earlier observational studies of the influence of depression in pain perception are from 1992 when Dworkin and colleagues observed that elevated depression and anxiety scores led to a higher chance of patients developing a condition known as postherpetic neuralgia. The same year Affleck and colleagues found that depression assessed prior to a diary of pain reports led to higher daily pain reports during a 75-day period (Keefe, 2001, p. 589).

This shows a correlation between mood and pain perception, but even more interesting would be if a manipulation of mood could alter the perception of pain. Already in 1991, such a correlation was found on a cold pressor task where subjects induced with a sad mood had lower pain tolerance on a cold pressor task (Zelman et al., 1991). However, a study using a similar method was conducted in 2011, which is more relevant to my purpose because they used the thermal grill illusion instead of a cold pressor task.

In the 2011 study by Boettger, et al subjects were put in one of two groups, sad or neutral mood. Both groups would have their hands placed on a thermal grill device with interlacing cold and hot bars, where the cold bars would be 5 degrees Celsius warmer than their cold pain threshold and the hot bars would be 5 degrees colder than their heat pain threshold rating the sensation on an unpleasantness scale. After this the subjects would go through a Velten mood-induction procedure, where they would be asked to say 21 statements that would either be neutral (neutral mood group), such as “citrus is a fruit” or sad (sad mood group) such as “life is a heavy burden. Subjects were asked to really feel the moods suggested by the statements. After the induction phase, subjects would again place their hand on the thermal grill device and rate the sensation (Boettger et al, 2011).

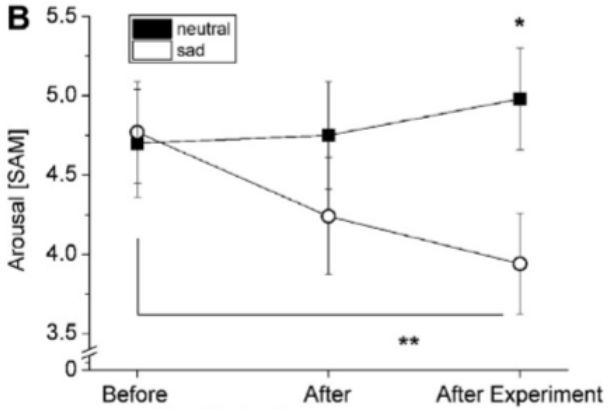



*Fig 2 – taken from Boettger et al, 2011*

The authors used different scales to validate their mood induction procedures, and the induction methods did show. Fig 1 shows the subjects moods before and after the procedure as assessed by a self-assessment manikin (ibid., p. 127).

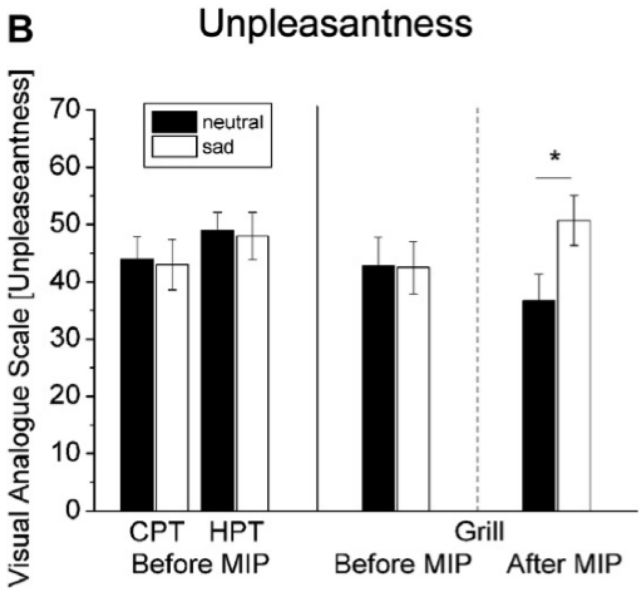



*Fig 3 - taken from Boettger et al, 2011*

The results from the experiment is shown in figure 3, and clearly suggests that subjects with induced sad moods experienced the thermal grill pain as more unpleasant than subjects with a neutral mood state (ibid.).

In fact, the influence of sad mood on pain perception has been established in several studies, such as Pinerua-Shuhaibar et al. (1999 and 2010) and Terhaar, et al. (2010).

#### 3.2.2.2. Pain, fear, and anxiety

In recent years, the field of the influence of anxiety on pain perception has received increasing attention. The results from correlational experiments (measuring traits, and see how they correlate with pain) show a significant relationship between anxiety and pain perception. The scales used to measure anxiety, however, differ between experiments. Traits that has been found to have a positive correlation with pain sensitivity is anxiety sensitivity (the same as fear of pain) (Keefer et al, 2001, p. 592) and catastrophizing (ibid., p. 591). Both of these measures have shown to increase sensitivity to pain.

Especially *Catastrophizing*, which can be defined as *"… as a tendency to magnify or exaggerate the threat value or seriousness of pain sensations* "(Quartana, et al., 2009, p.111) correlates highly with pain perception. It has been shown to be associated with several pain related outcomes, such as higher self-reported pain, increased use of pain medication and greater overall health care utilization (ibid.). Even more specifically the part of the pain catastrophizing scale that investigates the trait "rumination" seems to correlate most strongly with pain sensitivity (Sullivan, et al., 1998, p. 253, Burns et al., 2015, p. 28). The rumination subscale consists of the items "*I keep thinking about how badly I want the pain to stop*.", "*I anxiously want the pain to go away*.", *"I can't seem to keep it out of my mind*." and "*I keep thinking about how much it hurts*." (Sullivan, et al., 1995, p. 526)

The relationship between fear and anxiety has also been studied in the laboratory. For example, in a study by Jamie Rhudy & Mary Meagher (2000) electric shock was to induce either fear or anxiety in subjects. The authors argue that fear and anxiety are qualitatively different experiences. Fear is response to an immediate threat, whereas anxiety is a future oriented emotion (ibid., p. 65). This distinction is also supported by animal research, showing significantly different reactions to fear (actual threats) and anxiety (supposed threats) (ibid.) Therefor the authors found it interesting to study if fear and anxiety also produces different responses in humans.

Fear was induced by instructing subjects that they "might receive a shock" from an electrode attached to their finger, afterwards they would get three painful shocks. The instructions for the anxiety group was the same, except this group would not receive any shock. Their pain sensitivity would then be measured by having them place a hand on a hot platform and see

how long it would take before they withdrew their hand (Rhudy & Meagher, 2000, pp. 66-67).

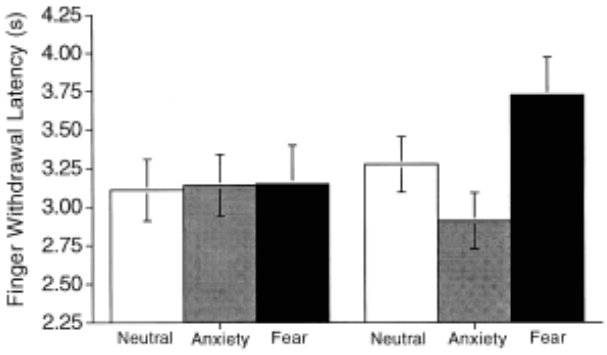



*Fig 4 - taken from Rhudy & Maegar (2000)*

The results can be seen in fig 4. The 3 columns on the left are the pre-test withdrawal measures. The results suggest that anxiety causes increased pain perception as they withdrew their fingers significantly faster after the induced anxiety, whereas fear would make subjects less sensitive to pain (ibid.).

One issue with interventional research on the effect of anxiety on pain perception is that different studies yield different results, even with similar designs. Al Absi, & Rokke (1991) for example used the same induction method for anxiety as Rhudy & Meagher, but found that irrelevant anxiety (anxiety related to the electrode), led to reporting less pain than subjects with anxiety related to the cold pressor task they used (Al Absi & Rokke, 1991, p. 43). Other research has found a significant correlation between attention and pain, showing that distracting anxiety-inducing stimuli might reduce pain. (Arntz et al., 1991, p. 41; Arntz et al., 1994, p. 307).

## 3.3. Models of the influence of emotion on pain

The theory that emotion influences pain seems well established, as I have argued throughout this section. The question that remains is; how does emotion influence pain? In this section, I will account for two models, which try to explain the results from the studies of emotion and pain.

### *3.3.1. A motivational model of pain*

Since both depressed mood and anxiety, (section 2.3.1.) seem to have a positive correlation with pain perception a theory could be that negative emotion or stress would increase pain

perception. This, however, seems too simple, as it for example would not explain the results of the Rhudy & Meagher study mentioned in section 2.3.1, as both fear and anxiety should then increase pain sensitivity.

The motivational model of pain suggests that human beings have innate appetitive and aversive systems, which modulate defensive systems (Rhudy & Meagher, 2001, p. 243). That is; If a stimulus is positive in valence, such as a nude sexual partner or an appetizing meal, the appetitive system is activated, whereas if it is negative in valence such as rotting food, or a threatening animal, the aversive system is activated (ibid., p. 241).

The degree of activation in either the appetitive or aversive system is known as "arousal". Together this gives a two-factor model of emotion, where low negative valence-low arousal would be described as a "sad" mood, negative valence high arousal would be anxiety, positive valence, and low arousal would be a "calm" mood and positive valence high arousal would be "happy".

This two-factor model was originally proposed to describe emotions (ibid.), but since it seems clear that pain is partly understood as an emotion, there is a theoretical argument that valence and arousal might explain some of the variance in pain perception.

The valence-arousal model has been tested in some experiments, such as one by Rhudy & Meagher, using the international affective picture system (IAPS). These pictures are validated to induce a specific valence and level of arousal. In the study, they used slides that evoked fear, disgust, and neutral pictures. They found that the slides inducing a negative valence (fear and disgust), reduced thresholds for pain unpleasantness and intensity, and the fear slides were also found to decrease pain tolerance (ibid., p. 243). A similar study was made using picture with positive valence (erotic pictures and nurturing pictures), these would vary in arousal, with the erotic pictures being more arousing than the nurturing. The study found that the erotic pictures increased pain intensity and unpleasantness thresholds (ibid.)

Along with the data from the experiment accounted for in section 2.3.1, showing that anxiety (medium arousal) can cause increased pain sensitivity and fear (high arousal) can decrease pain sensitivity, it was suggested that negative valence and low arousal would lead to increased pain sensitivity, and negative valence and high arousal would lean to decreased pain

sensitivity. Positive valence along with sufficient arousal would also lead to decreased pain sensitivity (ibid., p. 244).

These findings are also consistent with an evolutionary theory, in which pain is an adaptive system which function is to protect the individual from harm. When an individual has positive emotions, there is little reason to be feeling pain, as no immediate threat is present. On the other hand, when an individual is highly aroused as when he is in fear, it is probably that there is a need to deal with a situation before it is relevant to get to rest and mend wounds.

### *3.3.2. An attentional model of pain*

The motivational model of pain serves well to explain several aspects of anxiety, as mentioned in the previous section, however, several studies have shown the importance of attention in pain perception. This is largely neglected in the motivational model, but these results are important to also take into consideration.

There has never been an empirical consensus on whether, or how, anxiety influences pain. Studies show inconsistent results, in some studies it seems to influence pain perception, in others it seems not to. Already back in 1991 Arnoud Arntz and colleagues noticed that in most studies done on anxiety and pain perception, attention was never controlled for (Arntz et al., 1991, p. 42). The authors theorize that this might be the reason that earlier studies had found conflicting results.

This led to an experiment, which sought to test anxiety relevant to a painful stimulus, vs anxiety that was irrelevant to a painful stimulus.

The researchers took 52 women and 3 men and divided into 4 groups, all of which were spider phobic. They then induced anxiety either by exposure to spiders (irrelevant anxiety) or by enhancing the fear towards an experimental stimulus, by telling them that random trials could be much more painful than the normal stimulus condition. The subjects were furthermore divided into high and low anxiety, in the irrelevant anxiety group by controlling the exposure to spiders, and in the relevant anxiety group by laying more emphasis on the variability of the shocks (ibid., 1991, p. 44)

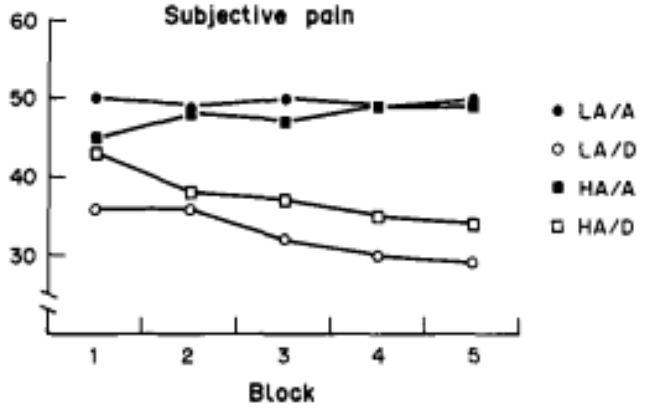



*Fig 5 - Taken from Arntz et al. (1991)*

As can be seen in fig 5, the subjective pain sensation of the electric shocks administered as evaluated on a VAS-scale only differ significantly between attention, and not level of arousal (ibid., p. 46).

Similar results have been attained in other studies, with similar methodology such as Al Absi & Rokke (1991), which used test anxiety as the irrelevant anxiety, and Arntz & Janssen (1996).

According to this theory anxiety serves as a mediator, rather than having a direct influence on pain. Higher anxiety makes people more likely to have attention towards a stimulus, and people higher in anxiety are therefore more likely to experience more pain because they are more focussed upon it.

### 3.3.3. Summary of emotional models of pain and hypothesis

The conclusion to this chapter can be summed up well by Seymour & Dolan's conclusion in "Emotion, Motivation and Pain" (2013), which states that *"Pain is unlikely to be underpinned by a single unified motivational value system. Rather, evidence points to a number of distinct systems of action, including innate, habit-like, and goal-directed systems" (Seymour & Dolan, 2013, p. 453)* Stated in a simpler manner, the link between emotion and pain is not a straight forward one. Using different methodologies seems to yield different results about which is more important, attention, appetitive-aversiveness, or expectation. This is important because it suggests that neither of the models is accurate, and an improved model should be able to explain all the findings, instead of just focus on some of the findings.

My summary of the findings is the following: It seems well established that mood can affect the perception of pain, and we are fairly confident that both sadness, anxiety and fear

influence pain thresholds. Most data, in my opinion, supports the theory that fear or anxiety can either decrease or increase pain perception, depending on what it is directed against.

The study of Rhudy & Meagher (2000) that I described earlier, where participants are given an electric shock to elevate their anxiety are, however, inconsistent with this. The anxiety or fear they induced is not relevant to the painful stimulus, and therefore it should rather decrease pain perception. The reason I choose to slightly disregard this study is that I think the measure of pain sensitivity is less than optimal. They use a finger withdrawal task rather than a task in which participants have to rate the intensity of a given stimulus. This means that I think the experiment should be reproduced, using another measure of pain sensitivity. My hypothesis for this experiment, which is described in further detail in section 4, are as follows:

*Induced fear or anxiety for an electric shock will increase pain thresholds on a thermal grill task*

# 4. Methodology

In this section, I will argue about the reasons for choosing the psychological scales I have used in the experiment and for using electrical shock as my method of anxiety induction, and why I used the thermal grill as the pain-inducing stimulus.

## 4.1. Psychological measurement

The psychological assessment tool used was state/trait anxiety inventory (STAI-S and STAI-T), pain catastrophizing scale and the McGill pain questionnaire, short form. All of these tests were chosen based on the extensive amount of validation there has been done on all of the tests. Additionally, the convenience of all the tests being short, meaning they all could be filled out in a few minutes.

## 4.2. The thermal grill illusion

The thermal grill illusion was discovered by the Swedish physician Torsten Thunberg in 1896. In his article “förnimmelserna vid till samma ställe lokaliserad, samtidig pågående köld- och värmeretning” he describes the paradoxical phenomenon that when subjects touch interlaced cold and warm bars of 20 and 40 degrees Celsius, subjects would experience a sensation of strong heat. This sensation could not be explained by the sensation of the

individual bars, as 40 degrees is way below heat threshold for normal individuals and 20 degrees is way above the usual cold threshold for individuals (Thunberg, 1896.

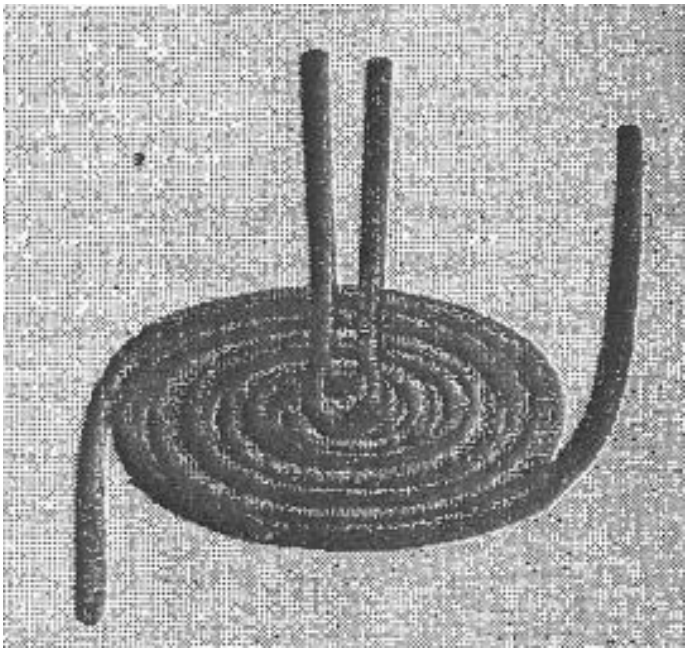


*Fig 6 - Taken from Thunberg (1896). This is the first thermal grill device used in the original study by thunberg*

The first attempt to explain the painful sensation caused by the thermal grill illusion was a sort of pattern theory explanation. The temporal summation of the cold and warm stimulus would "push" the sensation above the threshold and make it feel painful (Craig & Bushnell, 1994, p. 252). After the discovery of several receptors for bodily sensation such as warm receptors and heat receptors, showing that the pain perceiving heat receptors only respond to stimuli above the heat pain threshold at around 45 degrees Celsius, this "fusion theory", however, started to seem unlikely (ibid.).

A more likely explanation for the thermal grill illusion came in 1994 when Craig & Bushnell published their article "The Thermal Grill Illusion: Unmasking the Burn of Cold Pain". In their experiment, they recorded activity of neurons in the spinal cord of a cat while exposing it to the thermal grill. Craig and Bushnell found significantly reduced activity in "COLD-neurons" (neurons responding to innocuous cold) during the thermal grill illusion. This led them to suggest that stimulating heat receptive neurons would inhibit the COLD-neurons, which usually inhibit the neurons that responds to noxious cold. This was called an "unmasking phenomenon" in that the heat would "unmask" the effect of the usually inhibited

cold pain neurons of the spinal cord (Craig & Bushnell, 1994, p. 254). More simply put, it is regarded as a purely physiological response, operating on the level of the spinal cord.

The study by Craig & Bushnell was initially critiqued because it seems, optimistic to assume that the somatosensory system of a cat is closely related to that of a human, but later studies seem to support their conclusion. With the help of neuroimaging techniques, the thermal grill illusion has shown to activate similar areas of the brain as pain caused by mechanical stimuli or heat stimuli such as the ACC, PFC, S1, S2 (Leung et al., 2014, p. 1), and the thalamus (Lindstedt et al, 2011, p. 12). The isolation of COLD and WARM fibres has also been tried through application of chemicals such as capsaicin (Schaldemose et al, 2015), leading to allodynia (pain sensitisation), formaldehyde and menthol, enhancing the grill-evoked sensation (Averbeck, et al., 2012). This evidence suggests that the thermal grill evoked painful sensation follows the same pathway to the brain as any other painful sensation, and hence triggers similar responses in the brain.

That the paradoxical pain caused by the thermal grill is elicited by similar mechanisms as heat and cold pain is also supported by findings by Frederic Adam and colleagues in a study of magnitude of differences between the cold and the warm bars used for the illusion. The researchers simply measured the number of "responders" (people rating the grill as being painful) to the thermal grill during different temperatures of the warm and cold bars. What they found was that the larger the difference between the bars, the higher a percentage of responders there would be (Adam et al., 2014, p. 2612). Also suggesting similar mechanisms as in normal pain perception.

Some data, however, suggests that the thermal grill illusion is more of a psychological phenomenon than regular pain sensation. Fredrick Lindstedt and colleagues found in an fMRI study found significant activation in the right mid/anterior insula, which has been proposed to have a role in subjective feelings (Lindstedt et al., 2011, p. 12), and Leung and colleagues have similar findings from an fMRI study (Leung et al., 2014, p. 9).

### 4.2.1. Stimulus parameters

As the parameters used for the thermal grill in my design differ from that of the original study by Thunberg, I have included a section that accounts for the reason that this should not affect the results.

First key difference is that I use a design with straight bars, instead of the spiral design from the original study. The straight bar design is much more convenient, both in construction of the device and in that it allows the possibility of re-arranging the bars. In terms of effect on the results, it should have none. All modern thermal grills use the straight bar design, and the illusion still exists. See the previous section for references, as all studies, except Thunberg, that are referenced use a straight bar design.

The spacing between the bars and the number of bars used are standard for today's thermal grill design, but it is still important to note that a study has been done comparing different parameters, such as spacing of 2mm-10mm and number of bars of 2-6. Neither had any significant effect on the experience of the thermal grill (Li,et al., 2009, p. 1)

The most controversial part of my design is using higher spans between the cold and warm bars than in some other experiments. 17 degrees and 42 degrees respectively, as opposed to the 20 and 40 degree bars in the original study, which is still used in much (but not all) of the research. The reason for this is the study by Frederick Adam and colleagues, which shows that more people "respond" to the thermal grill when the temperatures are further apart. Hence, it should help lower the variance of the experiment a little, while still being safely above and below the thresholds of normal adults.

### 4.2.2. Strengths and weaknesses of using a TGI design

#### *4.2.2.1. Strengths*

In my opinion, the TGI is one of the best ways of studying the role of emotion on pain. As argued above, the pain pathway to the brain is likely to be very similar to that of other pain responses, as you would get for example from a cold pressor task (having subjects dip their hands in cold water for as long as they can) or a pressure-pain sensitivity test. A distinct reason for having an interest in the thermal grill is that since it is an illusion of pain, all pain will be innocuous, whereas with a cold pressor task you need to limit the timespan allowed for the participants to have their hands submerged, and for the pressure-pain test, you have to have a maximum amount of pressure you are allowed to apply. This of course becomes especially relevant if you want to test pain perception in fragile individuals such as the elderly, but is always a relevant consideration.

A second reason is that the TGI seems to have a stronger emotional component than other forms of pain, as accounted for in the previous chapter. Studies have been conducted that

seems to support this as well. Raymonde Scheuren et al. for example showed that personality traits such as rumination and interoceptive accuracy served as "major predictors" of whether subjects would respond to the TGI or not (Scheuren, et al., 2014, p. 1). This emotional component might also be enhanced by the fact that none of the participants used are familiar with the paradigm, whereas cold water for instance is known by all people. This seems also to be agreed on by psychological researchers as quite a lot of research on the influence of emotion on pain has been done on the thermal grill (see for example: Boettger et al., 2011; Boettger et al., 2013; Scheuren et al., 2014).

#### *4.2.2.2. Weaknesses*

By far the biggest problem with a TGI design is the variance. Most studies report that only about 1/3 of participants are responders to the illusion. This is also shown in the previously mentioned study by Frederick Adams and colleagues, where in the most extreme condition (temperature of the bars being more than 26 degrees apart) still only had a bit more than 1/3 of the participants be responders (42%) (Adams et al., 2014, p. 2615). This variance does not have to be a problem if you have unlimited capability of acquiring subjects, but since recruiting for a pain experiment is already difficult, it can be problematic because it means that you will require more participants to be able to get statistical significance. This also means that a design using the thermal grill will generally be more time consuming.

An additional problem is keeping the temperature consistent. Most thermal grill devices (including the one I used) are water driven which means you have to pour in warm water if it is a bit too cold and cold water if it is a bit too warm. This means that the precision of the temperature you can produce mostly will not be too good and usually the precision is only as good as ±1 degree Celsius. This can lead to similar problems as the previous weakness; you need more participants because it raises the variance of the condition, which is only likely to even out with more participants. In addition, there is the potential danger that this will lead to unconscious confirmation bias, as the temperature is controlled by the experimenter, and he might unknowingly raise or lower the temperature to fit his subconscious bias.

The last weakness I will mention is that the measure is not continuous in the same degree as other paradigms such as the cold pressure. The usual way to do analysis when using a TGI design is by dividing into a group of "responders" and "non-responders". This means that the paradigm is less sensitive to small changes in pain perception. In a cold pressor test, if a

subject last for 5 seconds longer, it will always be included in the statistical analysis, whereas if someone assesses the thermal grill as 0.5 points more painful on the visual analogue scale (VAS), he might still not be a responder, and hence it will not change the analysis, even though there is a difference. This leads to the same problem as the previous weaknesses, you need more subjects.

#### 4.2.2.3. Summary of TGI

The thermal grill illusion is a good paradigm for investigating the effect of emotion on pain, as it seems to follow similar pathways to the brain as other pain induction methods. Benefits of this paradigm specifically is that it is very safe due to it being an illusion and stimuli way below the noxious threshold can be used, and that it seems to be slightly more effected by emotion. It does have a big weakness in that it needs more participants than other paradigms, and carries a significant risk of making a type 2 error (a false negative), in case you have too few participants.

# 5. Design

My experiment is very close to the one referenced by of Rhudy & Meagher's (2000) in section 2.2.2., except I used a TGI design rather than a finger withdrawal task, as I argued in the previous section has some advantages in this kind of experiment.

## 5.1. Method

### 5.1.1. Subjects

21 subjects were recruited to the experiment, 12 male and 9 female. Subjects were mainly university students and staff. One subject had to be excluded due to being an extreme outlier (3 standard deviations from the mean), leaving me with one less male in the control group compared to the other groups. Mean age was 28 years. None had prior knowledge of TGI. Subjects were excluded if they had any chronic pain or contagious illnesses.

### 5.1.2. Apparatus

#### *5.1.2.1. Electric stimulation*

The electric shocks were administered by 2 mm round electrode attached to their finger, which was connected to a noxi-stimulator. They would receive three shocks of 12 mA lasting 2 Ms and spaced 1 Ms apart.

#### 5.1.2.2. Medoc

Pain thresholds were measured using a MEDOC-device with a 9cm^2 thermode with a peltier element.

#### 5.1.2.3. Thermal grill

The thermal grill device consisted of 6 hollow brass bars. The bars were perfused with either cold or hot water, heating 3 bars to 42 degrees (± 1 °C) and 3 to 17 degrees (± 1 °C). The bars were 10 mm X 120 mm and placed 2 mm apart, hot and cold bars interlaced. The bars were re-arrangable, making it possible to place either only hot or cold bars adjacent to each other.

### 5.1.3. Experimental Procedure

Subjects were divided randomly into three groups, control, fear and anxiety. Groups were controlled so the all groups would have the same amount of male and female subjects. Before the experiment participants were asked to fill out a STAI-S, a STAI-T and a pain catastrophizing scale, their cold and heat thresholds would then be measured using the MEDOC device. The participants would then be seated next to the thermal grill and have an electrode attached to the index finger of their dominant hand. Subjects in the experimental groups would be given the instruction “you might receive a painful electric shock”, but only the “fear” group would receive a shock. Subjects in the control condition would receive the instruction “you are in the control group, and will therefore not receive any shock”, and their electrode would be detached from the noxi-stimulator. Subjects were then asked to place their hand on the thermal grill device for 1 minute, rating the intensity of the sensation every 15 seconds on the visual analogue scale (VAS)-scale, ranging from “no sensation”, to “light pain” to “unbearable pain”. Afterwards they were again asked to fill out the STAI-S, but asked to answer how they felt while holding the hand on the thermal grill, and they were asked to fill out a Mcgill pain questionnaire, short form.

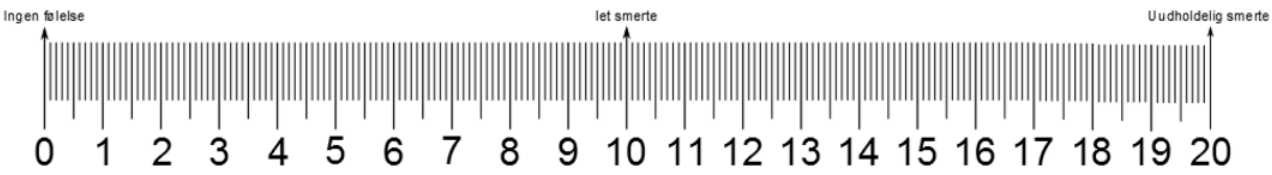



*Fig 7 - Visual analogue scale. The participants were told that a "10" on the scale would be the equivalent intensity to that of their measured pain threshold*

## 6. Results

Data was analysed using R-statistics. I will first account for the descriptive statistics, then the initial data analysis, and hence for the post hoc analysis I did as well.

### 6.1. Descriptive statistics

Data for all groups passed the D'agostino skewness test, which tests against the alternative hypothesis that "data has a skewness". P-values are; Catastrophizing (0.64), Pre-test STAI-S (0.84), STAI-T (0.52), VAS-score (0.62), and post-test STAI-S (0.30). As tests for normality are always slightly controversial, mainly because the null hypothesis is that the data does follow a normal distribution. Based on this, the data can be treated as normally distributed.

| | Pre STAI-S | STAI-T | Catastrophizing | VAS-score | Post STAI-S |
|---|---|---|---|---|---|
| Mean | 30.67 | 36.2 | 15.15 | 8.97 | 32.9 |
| Min | 21 | 21 | 0 | 0 | 20 |
| Max | 45 | 56 | 29 | 16 | 51 |
| Median | 31.00 | 36.00 | 14.50 | 9.40 | 30.00 |
| St. deviation | 7.69 | 9.32 | 7.96 | 4.34 | 8.73 |

Table 1.

Table 1 shows the mean of all the data, the minimum and maximum value, the median and the standard deviation. The median and the standard deviation being close together supports the theory that the data is normally distributed. The min and max values are there to give perspective to the size of the standard deviation, which is reasonably high in all of the measures. The significance of the mean will be used in the results discussion to give a perspective to how my sample compare to the average population. The data shows little difference between the pre-test STAI-S and the post-test STAI-S.

| Means | Pre STAI-S | STAI-T | Catastrophizing | VAS-score | Post STAI-S | Responders |
|---|---|---|---|---|---|---|
| Control | 32 | 33.5 | 15.33 | 9.08 | 32 | 3/6=50% |
| Fear | 30.20 | 33.71 | 10.85 | 9.14 | 33.28 | 3/7=45% |
| Anxiety | 31.43 | 41 | 19.29 | 8.69 | 33.29 | 3/7=45% |

Table 2.

Table 2 shows the means for the different tests when subjects are divided into groups. The VAS-scores are similar across groups, the state anxiety measure is similar from pre to post test, and the number of responders I have to the grill is 45%, which is quite high (compared to around 1/3 in other thermal grill experiments). The number of responders is equal in each group. There is not significant between group differences on the pre-test STAI-S, however, the anxiety group scored much higher on trait anxiety and catastrophizing. The difference between the groups on STAI-T and catastrophizing is not significant (STAI-T ($F(2, 17)=1.502$, $P=0.251$), catastrophizing ($F(2, 17)=2.21$, $P=0.14$).

## 6.2. Initial analysis

### *6.2.1. Validation of the induction protocol*

The initial analysis simply sought to investigate the hypothesis *"Induced fear or anxiety for an electric shock will increase pain thresholds on a thermal grill task"* I had to make sure the anxiety induction worked first. ANOVAS were conducted on the post-test STAI-S test ($F(2, 18)=0.23$, $P=0.795$). Not finding any significant effect of the mood induction

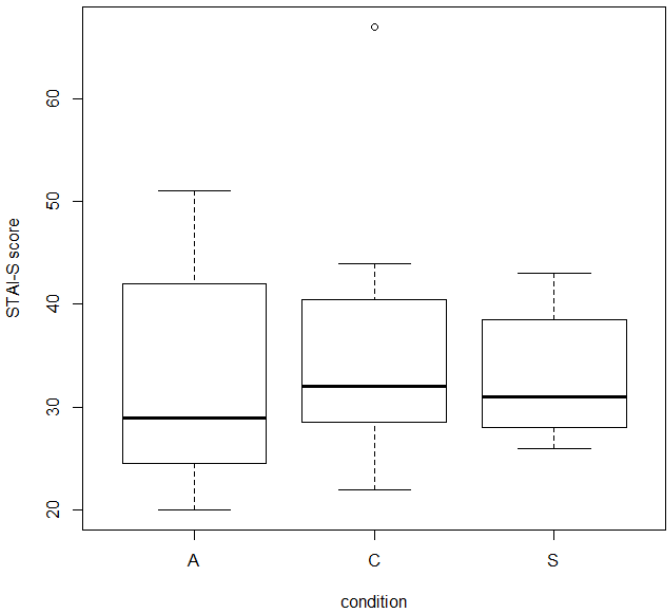



*Fig 8 - Boxplot over post STAI-S scores*

As shown in figure 9, the three groups are almost identical after the induction. A is the anxiety group and S is the fear group.

### 6.2.2. Difference between groups

Although the induction method seems to have been unsuccessful, I still made an analysis between the different groups, to see if there was any significant difference in the perceived intensity of the illusion, but no significant relationship was found ($F(2, 18)=0.158$, $P=0.855$).

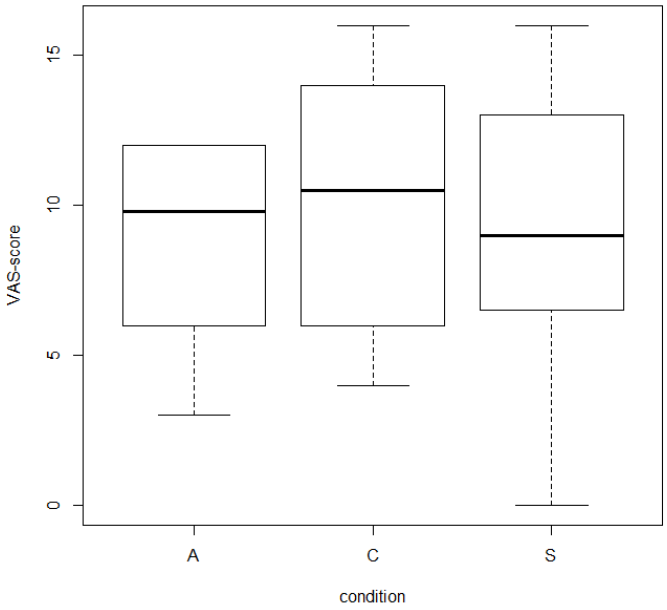



*Fig 9 - Difference in VAS scores between groups*

As evident from fig 10, the average unpleasantness score is close to identical between groups.

### 6.2.3. Post hoc analysis

Since the initial analysis did not show any significant relationships, I decided to make a thorough post hoc analysis of the data in order to investigate if I could find any interesting relationships. Post hoc analysis suffers the problem that in order to establish any relationship with any confidence you need to make a correction to your P-value, such as a Bonferroni correction. This is because the accepted significance level we use in psychology is 95%, this means that on average, for every 20 tests made (because 5% is 1/20), a type 1 error (false positive) will occur. In order to avoid this, usually you adjust the P-value to make up for this. However, due to very high variance in an experiment like mine, this adjustment would make it impossible to find anything of significance, and instead I will still operate with the 95% confidence interval, while noting that any significant correlation can only be used for speculation and future research purposes.

#### *6.2.3.1. High trait anxiety, high state anxiety, high catastrophizing and pain perception*

Still to investigate the hypothesis that *"Induced fear or anxiety for an electric shock will increase pain thresholds on a thermal grill task"* a division of subjects into groups of high, medium and low for trait- and state anxiety as well as for catastrophizing. It was done in 33%

quartiles so the low group would be the 33% lowest scores, medium would be between 33% in the middle and the high groups would be the 33% highest scores. No difference was found on grill scores for catastrophizing (F(2, 18)=0.034, P=0.967), nor for pre-test STAI-S (F(2, 18)=1.538, P=0.243) or pre-test STAI-T (F(2, 18)=3.227, P=0.06). However, the relationship between STAI-T and pain perception comes close to significance.

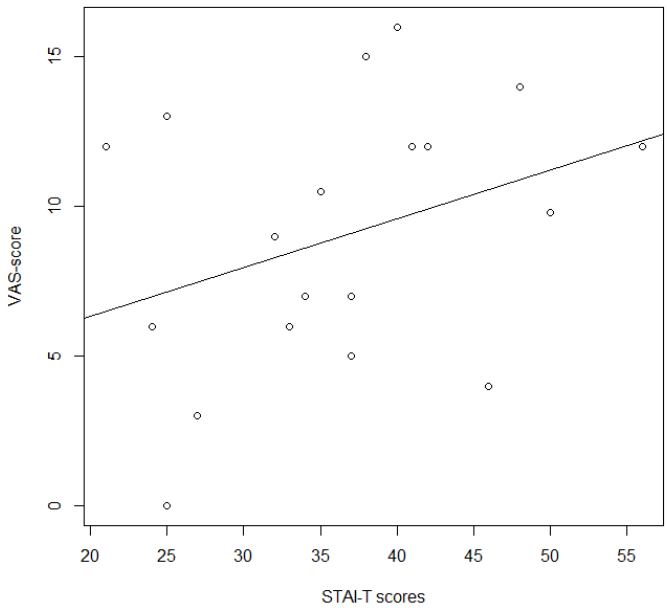



*Fig 10 - Correlation between STAI-T and the VAS-score for the thermal grill. The P for the calculated pearson's r is 0.13*

Fig 11 shows the linear regression of the correlation between STAI-T score and perceived pain intensity. The correlation is insignificant, but it is clear that the standard deviation is quite high on the grill scores (4.33) with a span of only from 0-17 and with a mean of 9.3.

#### *6.2.3.2. Gender differences*

Some earlier studies have shown gender differences, such as a study by Edmund Keogh and Julie Birkby who found that anxiety sensitivity correlated significantly with pain perception, but only in women (Keogh, E. & Birkby, J., 1999). Therefore, the dataset was split up in male and female, in search for any gender differences. There was no significant effect on condition for either gender (F(2, 6)=0.02, P=0.98) for females and (F(2, 6)=0.29, P=0.756) for males.

### *High trait anxiety, high state anxiety, high catastrophizing and pain perception – Gender divided groups*

Correlation tests between VAS-score and all the different psychological tests were also conducted for the gender divided groups. No significant correlations were found for women between catastrophizing and VAS-score (t=-2.07, df=7, P=0.08), nor pre-test STAI-S (t=0.596, df=7, P=0.57), pre-test STAI-T (t=0.68, df=7, P=0.52), or post-test STAI-S (t=1.24, df=7, P=0.25). Similar results were found for the male group Catastrophizing and VAS-score (t=0.34, df=9, P=0.34), nor pre-test STAI-S (t=2.13, df=9, P=0.06), pre-test STAI-T (t=1.71, df=9, P=0.12), or post-test STAI-S (t=0.84, df=9, P=0.42).

The only correlations, which were close to significance, were catastrophizing and VAS-score for females and pre-test STAI-S for males. The linear regression models for these are depicted below.

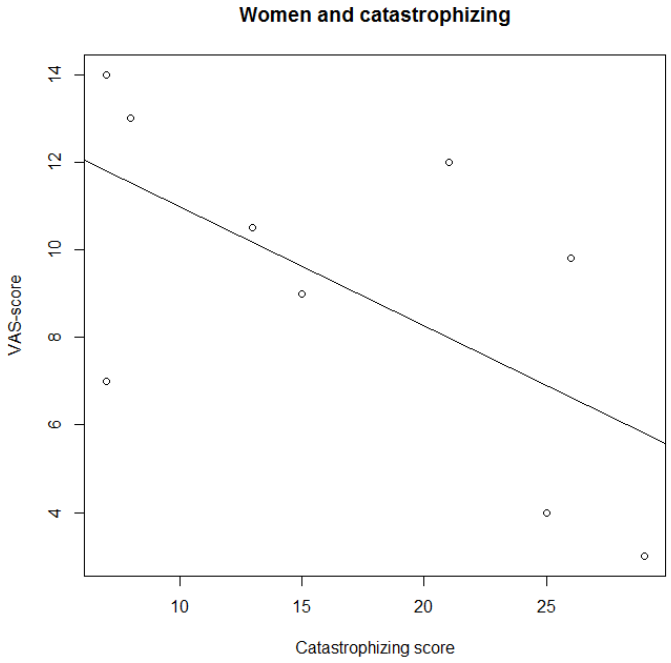



*Fig 11- Correlation between VAS-score and catastrophizing. The correlation is negative (-0.27)*

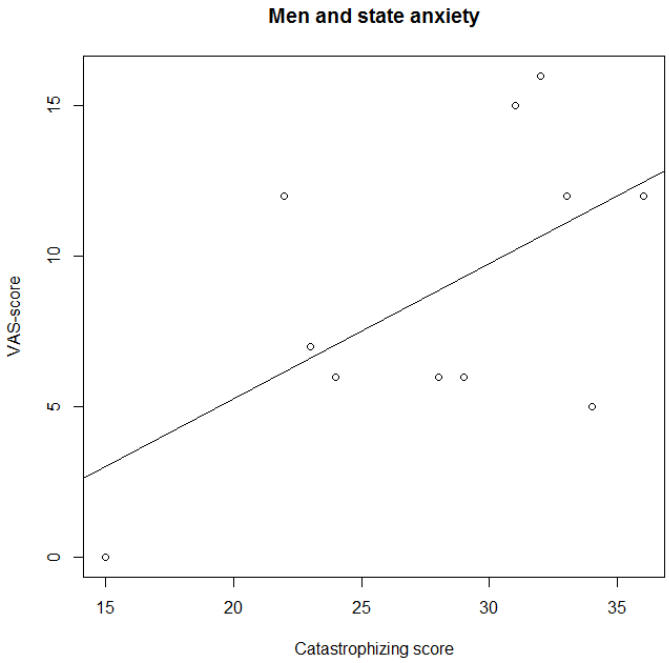



*Fig 12 - Correlation between VAS-score and pre-test STAI-S. The correlation is positive (0.57)*

# 7. Discussion

Rhudy and Meagher's research on anxiety, fear and pain perception have shown anxiety to have a positive correlation with perceived pain intensity, and fear to have a negative correlation with perceived pain intensity (see section 2.3.1). In this section, I will discuss how my results differ from the results from other research. First, I will discuss the statistics from the results section, then I will discuss the relation my results in respect to other research.

## 7.1. Results discussion

As the fear and anxiety induction method used failed to be validated the between groups ANOVA cannot be used to investigate the hypothesis *"Induced fear or anxiety for an electric shock will increase pain thresholds on a thermal grill task",* other methods of analysis had to be utilized to investigate the hypothesis

The ANOVA's of the divided groups of subjects into low, medium and high scores on the pre-test STAI-S, the STAI-T, the catastrophizing scale, and the post-test STAI-T, were all insignificant. The same trend is shown by Pearson's R correlation tests, which also gives insignificant P-values. These result cannot be explained by gender differences or interactions, as a division into a male and a female group give the same results.

In this section, I will discuss how to interpret the data from the experiment, and which conclusions might be made. I will discuss what my results suggest in terms of how trait/state anxiety and catastrophizing influence pain perception. I will also discuss whether electric shock might be an ineffective mood induction procedure.

### 7.1.1. Does state and trait anxiety effect pain perception?

#### *7.1.1.1. Trait anxiety*

To my knowledge, no previous studies have been made investigating state anxiety and the influence it has on pain perception. However, as previously argued, some data suggest that there could be a positive correlation, as other measures such as fear of pain have shown to increase pain ratings on a cold pressor task, and it has been shown to have some prediction property of which patients develop chronic pain after having some disease, such as herpes (see section 2.2.).

Though there is a theoretical basis for believing that trait anxiety might have an effect on pain perception, that relationship is not present in this experiment, as no significant results were

found. It is, however, interesting that there is a "trend" towards significance for the male group, with a rather high correlation of 0.57 between VAS-score and STAI-T. This suggests that a larger experiment might show a significant correlation.

This is of course speculative, but the state-anxiety scores in the experiment were generally in the lower end with 75% of subjects scoring lower than 41. Usually a score of 39-40 is considered a cut-off point, and an individual scoring higher might have problems with anxiety. A larger scale study could include more people in the high end of the spectrum. I therefore consider it likely that a positive correlation would be present.

#### *7.1.1.2. State anxiety*

As accounted for in section 2.3.1., state anxiety has been shown to correlate with pain sensitivity in some studies. I would therefore be reasonable to expect similar results in this experiment. This, however, was not the case for either group (all subjects, just men, and just women). The data for state anxiety did not even show a trend towards significance.

There are a few possible explanations for this that are worth considering.

The first is that there is a correlation, but that it is generally weak. As was the case with state anxiety, the subjects that agreed to participate in my experiment are generally low in state anxiety as well. 75% of subjects scored lower than 34, even on the post-test 75% scored under 39. This means that only very few of the participants in my experiment could actually be called anxious. If the effect of state anxiety is generally weak it could easily be hidden, when generally you only get subjects, which are low in anxiety.

Another reasonable theory is that a measure such as state anxiety simply has more unpredictable effects on pain perception. Some people might be mostly afraid of the electric shock, some of the thermal grill some of the experimental situation and performing up to the expectations of the experimenter.

#### *7.1.1.3. Catastrophizing*

Catastrophizing is usually regarded as one of the best predictors of pain perception. As mentioned in the "pain, fear, and anxiety" section, much research has shown this. My experience, however, did not find any such correlation. In fact, the only close to significant measure is a negative correlation between women and catastrophizing. However, when you look a little deeper into the results, they might still be interesting.

In a study by Frederik Kristiansen and colleagues, they investigated whether even small differences in catastrophizing could influence pain perception if the pain seems threatening. Stimulation of the gastrointestinal tract was used as their threatening stimulus. The study showed that low catastrophizers felt significantly more pain than non-catastrophizers. (Kristiansen, et al., 2013, p. 136). What is most interesting for this discussion is that the "Low-catastrophizer" group ranged from a score from 8-25 on the catastrophizing scale (ibid., p. 140). This is relevant to my study because the highest score in my study was 29, and the mean 15.15 (see table 1). Only 0.1% of participants would be classified higher than "low catastrophizers", and even these subjects did not score especially high.

This means that the results from my experiment a very homogenous group of low catastrophizers. What the present study suggests is then that pain perception does not correlate with moderate levels of catastrophizing for cutaneous stimuli. One could speculate, based on the mentioned Kristensen et al. study that perceived level of threat from the pain-inducing stimulus could have a significant effect on how much catastrophizing matters.

#### *7.1.1.4. Induction method*

Since the induction method I used did not seem to cause much distress to subjects, it is worth considering if it is an ineffective method of induction.

Since this method has been validated in other studies, it seems unreasonable to suggest that it is ineffective based on one study. On the other hand, the results do suggest that this method of induction might not always be effective. A factor that might play a role here is that the thermal grill was unknown to all participants, which might take focus away from the electrical stimulation. The low state/trait anxiety and catastrophizing of the participants might also mean that they are less sensitive to a fear/anxiety inducing stimulus.

Another hypothesis is that the method of recruitment was asking people around the university campus if they were willing to participate in an experiment in which there was a chance they might receive a painful electric shock. This might have meant that only participants with low aversion towards electricity were recruited.

### 7.2. Criticism and Conclusion on the results discussion

There are some criticisms that can be made to the internal validity, mainly the recruitment method. The participants were informed about the nature of the study before agreeing to it,

which is probably the reason that only participants low in anxiety participated in the experiment. This made the problem statement hard to answer from this study *"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?"*, as the subjects did not seem to fear or feel anxious about the shock.

An analysis of trait/state anxiety or catastrophizing also did not show any significant correlation with VAS-score. The data also showed no significant different between men and women, and multivariate analysis did not show any significant effect either.

Taken together, the results from this study suggest that anxiety and catastrophizing has little effect on perceived pain intensity for cutaneous pain, at least when the scores fall within the low/normal range. It also seems that induction of anxiety for individuals within the low to normal range of anxiety and catastrophizing can be difficult.

In terms of answering my hypothesis that "Induced fear or anxiety for an electric shock will increase pain thresholds on a thermal grill task", the results from the present study would suggest that the null hypothesis cannot be rejected. However, my results are inconclusive due to the anxiety induction not seeming to work well.

## 7.3. Overall discussion

The two models of the role of emotion in pain perception, the motivational and the attentional model of pain might help shed some light on the hypothesis. However, since results from experiments seem to differ a bit in the results, I will here discuss the two models, and try to figure out which shortcomings each of them has. In this discussion I will use the results from my experiment which I find relevant, and data from previous experiments.

### 7.3.1. Discussion of a motivational model

As accounted for in section 2.3.1. a motivational model of pain suggests that there are two distinct systems that govern human experience; an appetitive and an aversive system. These can be described by valence. If an individual is in a bad mood he will have negative valance, and if he is in a good mood, his valence will be positive. The theory suggests that our experience of the world is different depending on whether the negative or the positive system is activated. In terms of pain Positive valence would, according to this theory, be protective. The more aroused you are while in positive valence the less likely you are to feel pain. Your body knows there is no immediate danger, and hence there is no reason to warn you. Only if

the pain is strong enough that it exceeds your arousal, will you feel pain. We know this for example from when we are running or lifting heavy weights, we will not feel the blister until after. Sometimes we can even finish an important sports match after having sprained an ankle, because the pain is dulled by the arousal. The negative valence system is different. When we have negative valence we need to tend to our injuries, flee or fight. This is when we are vulnerable. This theory suggests that low arousal and negative valence will make us feel pain even more. This is when we are injured, but in no immediate danger. The motivation of this system will be to mend our injuries. It is most beneficial not to use the injured limb. However, when we are negative of valence and high in arousal, there is immediate danger. This corresponds in nature for example to when we see a lion in the bush. We might have a sprained ankle that needs to heal, but if we do not get away from the lion, our ankle is pretty irrelevant. In this state, we will feel less pain, because there is no time to mend our wounds.

Since fear is directed towards a present danger and anxiety is not, according to this theory, fear will increase pain thresholds, whereas anxiety will lower it. This is used in Rhudy and Meagher's (2000) study to explain why they get the results that the expectation of maybe getting an electric shock lowers pain thresholds, whereas having actually experienced that shock raises pain thresholds. The motivational theory would also suggest a similar hypothesis for my experiment.

I, however, had a different hypothesis. Namely that "Induced fear or anxiety for an electric shock will increase pain thresholds on a thermal grill task". There are a couple of reasons for this. Firstly, other experiments similar to Rhudy and Meagher's (2000), which use different irrelevant anxiety such as cognitive testing or spider phobia (section 2.3.2), has shown anxiety to raise pain thresholds. There could here be an argument for arousal level being high enough so it could be defined as fear, but since they used different levels of spider phobia and different exposure to spiders, that does not seem too likely. Rather the effect seems to be cumulative. Higher arousal towards an irrelevant stimulus protects more against pain than lower arousal. Hence I do not think as Rhudy and Meagher (2000), when concluded that there are "divergent" effects of fear and anxiety, rather a linear protective effect. It is likely that their results rather come from the method of measuring pain, as it is not a measure in which participants rate their perceived pain, but rather a withdrawal task. I do think it might be a stretch to equate vigilance with pain perception.

This is also tentatively supported by the results from my study, which showed no difference between groups regarding pain thresholds. All three groups had the same amount of responders to the thermal grill task. The reason I say tentatively supported, is that there was no significant difference between the pre-test STAI-S and the post-test STAI-S as mentioned in section 5.2. This means that there is a lot of uncertainty regarding whether they were anxious. The insignificant difference between pre and post STAI-S scores, could however have happened for other reasons, such as participants feeling anxious about the experiment during the pre STAI-S, and then when calming down from that, redirecting their anxious feelings towards the stimulus. This is of course merely speculation, but it does strike me as odd that participants do not feel anxious or fearful about a painful electric shock. In short, if my experiment suggests anything on this matter, it is that fear does not lower pain thresholds.

### 7.3.2. Discussion of an attentional model

For reasons discussed in the previous section, I think it is clear that a motivational model at least does not account completely for the effect of anxiety and fear on pain, but what about the attentional model discussed in section 2.3.2? Does that hold stronger explanatory power, or are there problems with both models?

The attentional model suggests that the most important mediating factor of pain. If an individual is exposed to a painful stimulus, the experienced intensity is modulated by how attentive the individual is towards the stimulus. As mentioned both in the previous section and in section 2.3.2., this theory is supported by data from experiments from arntz et al (1990) and Al Absi & Rokke (1991). These studies show that induced anxiety that is not relevant to the stimulus, makes subjects feel less pain, whereas inducing stimulus specific anxiety, such as by saying that the stimulus is incredibly painful, makes them rate it as more painful than the control group.

This would suggest another hypothesis for my experiment. Fear and anxiety should both raise pain thresholds, because the electrode should take at least some of the attention from the thermal grill. Level of arousal would then determine how much the shock would protect against the sensation of pain, meaning that the group with the least responders would be the control group, in the middle would be the anxious group and the fear group would feel the least pain from the thermal grill task.

I cannot really claim that my experiment supports this hypothesis, even though the control group does have a lower percentage of responders due to one participant being excluded due to being an extreme outlier in terms of state anxiety. This means that in the control group 50% rated the thermal grill illusion as being painful, whereas only 45% in the two other groups. This is, however, insignificant and cannot be used to conclude much. What on the other hand can be said is that the results from my experiment does not directly go against this hypothesis. All participants were very low in anxiety and it seems probable that it would have an effect if the level of anxiety was higher.

There are, however, a few things arguing against this theory as well. For example, the study of Pavlov's dogs mentioned in section 2.1.1., in which Pavlov taught the dogs to associate an electric shock with getting food, after which they stopped acting as if it was painful. There is no reason to believe that the attention shifted, however, the appetitiveness changed. The otherwise painful electric shock was now less painful, maybe because the appetitive system was activated. We also all know something similar, sometimes pain feels pleasant even if we are completely attentive towards it. Some individuals go as far as having something close to an addiction to painful mutilations of the body, such as getting branded with a symbol, or getting tattooed or pierced in a very painful area. Pain does not have to be painful, even if focussed on.

### 7.3.3. Conclusion on the discussion of the two models

As argued, it seems that none of the models can in itself give a good explanation of my results or the results from other studies, and hence will not on its own be able to answer my problem statement "Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?". On the other side, both of them seem to explain some part of how anxiety can influence pain perception. Appetitiveness and attention both seem to be able to alter the experience of pain, the question that remains is how they interact.

One clue comes from the correlational research. How traits effect pain perception. As accounted for in section 2.2.2, there seems to be a correlation between measures of anxiety and both chronic pain and pain measured in the laboratory. What is interesting is that catastrophizing as a trait has been found to have an especially high correlation with, and to an even higher extend rumination, rumination is measured in the catastrophizing scale with the items items "*I keep thinking about how badly I want the pain to stop*.", "*I anxiously want the*

*pain to go away*.", *"I can't seem to keep it out of my mind*." and "*I keep thinking about how much it hurts*.". These are, interestingly all items which are about being anxious specifically about the pain, which is bound to strengthen negative attention towards the pain.

Another clue comes from the Kristensen et al. (2013) study mentioned in section 6.1.1.3. in which even low catastrophizing seemed to have a positive correlation with experienced pain, if the pain feels like it poses a threat. A stimulus that is negative in valence by being frightening, and that demands attention is much more affected by catastrophizing scores. This once again emphasizes the importance of negative valence and attention. If you are very unlikely to direct negative attention towards a stimulus, even a frightening stimulus will not feel as painful as it will to controls.

A third clue to this comes from the practice of mindfulness meditation. The point of mindfulness meditation is not to direct your attention elsewhere, but rather to passively observe feelings that stream into your consciousness. Yet, several studies have shown mindfulness to significantly alleviate pain (Zeidan et al., 2015, p. 15308). This shows that attention only causes pain when it is negative in valence.

I think that the evidence suggests that motivation and attention both play an important role in regulating pain perception. If the valence of your emotions are negative, they serve as a protection if they are directed towards something else, such as a tiger or an electric shock, whereas if they are directed towards the stimulus they serve to make the pain feel more intense. How the mechanics work with positive emotion, is less studied and therefor harder to asses, but a good guess is that it serves to protect no matter the directedness. I think this because your valence will shift if a painful stimulus exceeds a certain threshold, it is difficult to be in severe pain and yet be happy, so when you are feeling positive, you will not feel as much pain, no matter what you feel pain about.

What can be called "directed aversiveness" suggests that anxiety mediates pain it two ways. Firstly, it directs your attention. If every time you feel anything unusual in your body fear it might be cancer or some other dangerous condition, you will focus more on it. Secondly, that attention towards something you are afraid of, will change your valence and therefor make the pain feel even more painful. This can explain why people high in anxiety are more likely to develop chronic pain than others (section 2.2.2.), and might help to explain why sufferers

from conditions such as fibromyalgia have been shown to often report childhood maltreatment (Häuser et al., 2016, p. 2).

## 8. Conclusion

This master's thesis tried to answer the problem statement *"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?".* It did that by first accounting for the main theories of pain, the gate control theory and the neuromatrix theory of pain, and accounting for how these theories shifted much of the focus of pain research from the peripheral nervous system and into the central nervous system. The entire idea of Patrick Melzack's neuromatrix theory is that there is a wide network in the brain, which creates and modulates the experience of pain. The importance of this argument was to show how well established it is that the brain regulates pain, and that on a theoretical basis it is possible for anxiety to regulate the experience of pain. To further support this, I then accounted for some of the research that has been done showing the relationship between pain and emotional states. Research has shown that emotion can influence pain perception, and there has also been done some research showing that anxiety can have an effect on pain perception.

Following these basic theories about how pain works in the brain, I went on to account for some of the models that seek to explain how anxiety can influence pain perception. These are the motivational and the attentional model of pain. Both of these state that anxiety can regulate how pain is perceived, but disagree on how it does so.

After accounting for these theories I made the hypothesis that "*Induced fear or anxiety for an electric shock will increase pain thresholds on a thermal grill task*" Because I had the theory that the study by Rhudy and Meagher, which the motivational model is partly built on, suffered some methodological problems, name that it was a withdrawal task, rather than a measure of subjective pain experience, and hence made a hypothesis built on the attentional model.

To test the hypothesis, I conducted an experiment which was similar to the study of Rhudy and Meagher but using a thermal grill illusion design, in which participants had to rate the intensity of the experienced sensation, with a score of 10 being defined as their pain threshold. This experiment did not show any difference between the groups, and the control, anxiety and fear group, all rated the thermal grill illusion similarly in intensity. This might have been due

to factors such as the participants being very low in state/trait anxiety and catastrophizing, which was measured during the experiment. It might also have been due to the thermal grill illusion being novel, and hence the electrode causing less distress, because the focus was on the grill.

All in all, the experiment does not support neither the hypothesis of the attentional or the motivational model, because fear and anxiety seemed neither to protect against or enhance the experienced intensity. Yet, many studies have established a correlation between experienced pain and anxiety, for which reason I do not think I can answer my problem statement *"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?",* but rather hypothesize that it did not in my experiment due to the factors mentioned above.

Therefore, to try to give a good answer, I discussed the two models, using data from other experiments, as well as some of the data from my own, to conclude that it seems that there is a relationship both between motivation and pain and attention and pain. Namely that both of them are an important factor. Attention needs to be present to feel pain, but if it is positive of valence, it can protect against pain.

The suggestion of a new model, which encompass both valence and attention would suggest that fear and anxiety for an electric shock indeed can raise pain thresholds on a thermal grill task, however with some caveats. If the thermal grill illusion seems more frightening than the electric shock, it will not protect against it. If the participant is more anxious about the experience of being in an experiment, that might be more important. If the participant feels very safe in the situation, or has little anxiety about electricity it will not raise the pain thresholds.

In short. The answer to my problem statement *"Can anxiety or fear for an electrical shock lower the experienced intensity of the thermal grill illusion?",* is “yes” and “no”. It depends who you are and what the situation is. But importantly, motivation and attention, can definitely alter the subjective experience of pain, there are just a few factors to consider in order to be able to use this information in advantageous ways.

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