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The impact of violent video gaming and adverse childhood experiences on fear conditioning, pain-related empathy, pain perception and pain tolerance

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ACC	anterior cingulate cortex
ACE	adverse childhood experiences
ALFF	amplitudes of low-frequency fluctuations
ANOVA	analysis of variance
BET	brain extraction tool
CI	confidence interval
CS	conditioned stimuli
CSV-S	scale for the assessment of pathological computer-gaming
CTQ	childhood trauma questionnaire
(f)MRI	(functional) magnetic resonance imaging
FPQ-SF	fear of pain questionnaire
FOV	field of view
HADS	hospital anxiety and depression scale
Hz	hertz
IGD	internet gaming disorder
IRI	interpersonal reactivity index
KERF	“belastende Kindheitserfahrungen“ scale (german adaption of MACE)
M	mean
MACE	maltreatment and abuse chronology of exposure scale
MD	mean difference
mm	millimeter
ms	milliseconds
NEO-FFI	NEO five-factor inventory

NG	non-gamer
NVVG	nonviolent video gamer
PANAS	positive and negative affect schedule questionnaire
SAM	self-assessment-manikin
SD	standard deviation
SE	standard error
SCID-5-CV	structured clinical interview for DSM-5-disorders
STAI-S/STAI-T	state-trait anxiety inventory
TE	echo time
TICS	Trier inventory for assessment of chronic stress
TR	repetition time
US	unconditioned stimuli
VAS	visual analogue scale
VVG	violent video gamer
°C	degree Celsius

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1 INTRODUCTION

“No one is suggesting that [violent video games are] the only reason they went out and committed those horrific acts, but was it a tipping point? Was it something that pushed them over the edge? Was it a factor in that? Perhaps. That's a really big deal.”
- Jim Steyer (2013) -

This quote from John Steyer, CEO of Common Sense Media, comes in the wake of several mass shootings and increased gun violence in America in 2013. Since this time violent video games have been accused multiple times to be responsible for various violent acts and rampages all over the world. An urge to know more about consequences of violent video gaming arose and that same year the president of the United States of America at this time, Barrack Obama, suggested:

"Congress should fund research on the effects that violent video games have on young minds. We don't benefit from ignorance. We don't benefit from not knowing the science."
- Barrack Obama (2013) –

With this statement and the large presence of violent video games in the news, research on this topic has become increasingly important. Over the years, research on violent video games has focused on covering most aspects related to human behavior, aggression tendency, or similar connected factors. However, violent games and their design are constantly changing, as they are digital products influenced by the constant technological progress of our society, which makes the reproducibility of some research results difficult at this time if they are connected to current design that is not further dominant in game design. Most mainstream video games have evolved from a single-player model to a multiplayer model that also favors parasocial interaction patterns. Further technological advances have led to violent video games becoming more realistic with increasing graphical and structural capabilities, to the point where even virtual reality violent video games are available. Not only are violent video games constantly changing, but so is the digital environment in which they are embedded. All these factors make it even more difficult for research on violent video games to be replicable and generalizable to the population. Ongoing technological advancements can be seen as one of the greatest achievements of our modern society, but also lead to more and more digital products being consumed as opportunities and embedding in daily life increase. Recent events such as the lockdown measures of the COVID-19 pandemic also favored a perspective of spending more time in front of screens at home, and therefore also led to more people discovering violent video games. The video game industry reported a 9% annual growth in 2020, resulting in $159 billion in revenue (Wijman, 2020). By the end of 2022, sales are projected to reach $196 billion (Beattie, 2020). This increase in the consumption of violent video games makes it even more important to look at current research on the topic and related factors, as Barrack Obama suggested, to gain more insight into how violent video games can affect human behavior and which groups of people are most affected. Human behavior that is often

affected according to past research is pain perception and groups of people that display psychological disorders are often mentioned to be especially affected by consumption of violent video games.
Pain perception is also a main variable in our research construct for this study. Pain in general has been widely studied for many years and many different frameworks have been established to comprehend every aspect of pain including pain perception (Moayedi & Davis, 2013). Foremost pain is defined as unpleasant sensory and emotional experience that can result from actual harm and damage to the body but does not have to result from actual damage taken. In addition to pain there is also the phenomenon of nociception. Nociception differs from pain as it only refers to the neural encoding process of noxious stimuli and not the subjective experience like pain (Mischkowski et al., 2018). However, nociception and pain are not limited to momentary sensations. Model learning, sensibilization, classic and operant conditioning or priming can lead to central nervous changes which favor a chronification of pain (Flor, 2011). Also, chronic stressors, depression and other mental disorders can favor a chronic pain condition as they change the way in which pain is perceived (Pfingsten et al., 2011). In general, alternated pain perception is correlated with many mental disorders like PTBS, bipolar disorders, schizophrenia, eating disorders, anxiety, substance use disorders and depression. These connections are said to exist in a bidirectional manner (Hooten, 2016; IsHak et al., 2018; Klossika et al., 2006). Similar to some of the mentioned mental disorders pain processing is also connected to changes in brain activity. Main areas that are correlated with changes in activation for pain processing are anterior cingulate cortex (activated by noxious and contextual stimuli), hippocampus, middle temporal gyrus, supramarginal gyrus, pre-/post-central gyri, medial frontal and (para-)cingulate cortex, inferior/middle frontal gyri, frontal operculum and insula, thalamus, and putamen (Biggs et al., 2020; Naor et al., 2020; Xiao et al., 2021; Xiao & Zhang, 2018). In order to research on the implications of pain perception many different experimental pain inducement methods have been developed. The most common methods are mechanical pain via strain-gauge focal pressure stimulator-dull or pressure algometer-plunger that apply pressure to small, bony portions of the body, cold pressor pain via a cold water bath, ischemic pain via blood flow interrupt, thermal pain via application of temperature controlled objects to the skin or via radiant heat source focused upon skin and electrical stimulation pain via application of electrical current to the skin, teeth or neurons (Edens & Gil, 1995). A variety of these methods was also picked for our study to allow a replicable and diverse assessment of pain. A more in-depth view on present research regarding violent video gaming and pain is presented in the following chapter.

1.1 Violent video gaming

Past research on violent video games has contributed to a better understanding of the influencing factors and consequences, and has led to statements such as the American Psychological Association's 2020 resolution recommending very cautious consumption of violent video games based on past research (American Psychological Association, 2020). The therapeutic diagnostic and statistical manual DSM-5 even considered video gaming behavior and its potential psychological consequences in the form of a new disorder called Gaming Disorder, which was added to the diagnostic manuals and later to the international classification of diseases ICD-11. (Guha, 2014). Violent video gaming is not solely responsible for Gaming Disorder but plays a big role

for adolescents as violent video gaming is a phenomenon that is almost ubiquitous among young people. In Germany 83% of male adolescents play violent video games or have friends who play them regularly (Statista, 2022). Consequently it is no surprise that video games play an important role in socialization and friendship development among adolescents (Lenhart et al., 2015). Further individuals with high trait aggression potential prefer violent video games as opposed to nonviolent video games even though violent content in games does not increase enjoyment or immersion significantly (Przybylski et al., 2009). Preference for violent video game content also leads to homo-typical selection effects, as adolescents prefer peers with similar aggression traits and become more similar in aggression over time. (Verheijen et al., 2021). This supports the assumption that violent video games can have significant effects on personality development, especially in younger people, as peer group influence is often an important factor during adolescence.

Many aspects of personality or perception changes have been stated by past research. For example in terms of exposure to violent video games, early research on this topic found that frequent use of violent video games significantly increased aggressive behavior, aggressive thoughts and emotions, and desensitization to violent content (Whitaker & Bushman, 2009). The results of one of the first meta-analyses also strongly suggest that exposure to violent video games is a causal risk factor for increased aggressive behavior, aggressive cognition, and aggressive affect, as well as decreased empathy and pro-social behavior. It found weak evidence for cultural differences in susceptibility and no evidence for gender differences in susceptibility (Anderson et al., 2010). More recent research contradicts the lack of gender differences suggested by Anderson et al. (2010). Mediating pathways in children representing the mediating role of aggressive cognition on the relationship between violent video games and aggressive behavior were significantly stronger in boys than in girls (Zhang et al., 2021). The effect of reducing feelings of pleasure and displeasure toward emotional stimuli after playing violent video games has also been confirmed (Arriaga et al., 2011). However, further research suggests that the effects of increased aggression and hostile anticipation after playing violent video games last only for a short period of a few days and do not represent a permanent change in aggressive behavior (Hasan et al., 2013). Recent evidence from a parallel moderated-mediation regression analysis also supports the claim that violent video gaming is associated with decreased empathy concerns, aggression-related thoughts and increased aggressive behavior. However, this study also supports the perspective that the association between violent video gaming and aggressive behavior is strongly supported by the presence of negative environmental factors (Addo et al., 2021).

In addition to studying aggressive behavior and empathy components, scientists have also begun to investigate the links between violent video games and pain perception. One of the first studies in this area found that after playing a violent video game, cold pressor latency was increased and pain perception was decreased in violent video gamers (VVG) compared to nonviolent video gamers (NVVG; Stephens & Allsop, 2012). Subsequent studies confirmed this effect. For example, Teismann et al. (2014) showed that participants not playing video games in their everyday life assigned to a violent video gaming group tolerated cold pressure pain stimuli significantly longer than participants assigned to a racing game group and showed significantly enhanced risk-taking behavior (Teismann et al., 2014). Recent research on this topic has confirmed this effect on pain tolerance and pain intensity ratings for habitual VVG (Förtsch et al., 2021).

However, research results on VVG are less congruent than it seems and there is criticism for some research on violent video gaming so far. In contrast to some of the

presented studies so far research also suggested that the influence of violent video games on increased aggression, reduced prosocial behavior, reduced academic performance, depressive symptoms and attention deficit symptoms is minimal and issues related to degrees of freedom in previous studies, citation bias and publication bias may be the reason for studies that suggested a significant influence of violent video games on these variables (Ferguson, 2007, 2015). A recent meta-analysis also suggests a publication bias connected to research on violent video gaming, states that studies are not able to demonstrate valid short-term effects of aggressive video gaming content on aggressive behavior and advocates for methodological weakness and researcher expectancy effects being present in current research on violent video gaming up to this point (Drummond et al., 2020).

In addition to behavioral data there is also evidence regarding neuroimaging studies on the effects of violent video gaming. However, some research results are also doubtful about how violent video gaming could affect activation in the brain. For example, the effect of neural desensitization associated with violent video gaming. Event-related potentials were recorded while viewing violent and neutral images selected from the International Affective Picture System while participants were advised to a VVG or non-gamer (NG) condition respective to their video gaming habits. Event-related potentials did differ between image conditions but not between the VVG and NG groups suggesting that there is no neural desensitization among VVG (Goodson et al., 2021). In contrast to research results by Goodson et al. (2021) a study by Miedzobrodzka et al. (2022) detected significant effects for event-related potentials regarding a pain judgement task among VVGs. Event-related potentials revealed habituation to pictures displaying painful content among VVGs for top-down and bottom-up empathy regarding pain related responses. Neuroimaging studies focusing on tasks containing a presentation of pictures with negative emotional content or pain exposure displayed significantly lower activation for VVG compared to NG in the limbic system such as anterior and posterior cingulate cortex, amygdala, thalamus, posterior and superior parietal lobe, hippocampus, cerebellum, left lateral medial frontal lobe and entorhinal cortex (Montag et al., 2012; Palaus et al., 2017; Wang et al., 2009). Further gamers diagnosed with internet gaming disorder show significantly different brain activation during a cue-reactivity functional magnetic resonance imaging task compared to NG with 92.37% accuracy according to multi-voxel pattern analysis. Therefore, altered patterns of neural activity can be assumed for gamers with internet gaming disorder. Multi-voxel pattern analysis showed the strongest difference for precuneus, posterior lobe of the right cerebellum and middle frontal gyrus (Wang et al., 2022). In addition, VVGs underlying internet gaming disorder seem to display higher levels of withdrawal, tolerance and neglect of everyday life compared to NVVGs with internet gaming disorder (Kim et al., 2022). However, these effects are not fully consistent and accepted as other studies also suggested no difference in empathy ability between VVG and NVVG for amplitudes of low-frequency fluctuations (ALFF) and fractional ALFF (Pan et al., 2018).

In addition some research revealed no difference in empathy ability between action video game players mostly including games with violent content and NGs for drift-diffusion modeling which showed no difference in decision making stages for discrimination of facial emotions and reverse inference techniques which did not reveal any group differences for mental representation of facial emotion expressions (Pichon et al., 2021).

Tasks requiring empathy for pain in others and violent video gaming show similar brain activation patterns as increased activation in the anterior cingulate gyrus. However, they also differ in activation patterns as tasks for empathy for pain in others also show increased activation in fusiform gyrus, anterior central gyrus as well as insula and

violent video gaming is associated with increased activity in hippocampus, cerebellum, thalamus, amygdala, entorhinal cortex as well as posterior and superior parietal lobe (Fallon et al., 2020; Li & Wang, 2021; Montag et al., 2012; Palaus et al., 2017; Pan et al., 2018; Xiong et al., 2019).
All of the research presented on violent video games leads to the consensus that there are multiple factors influenced by violent video gaming. VVGs display lower pain sensitivity, increased aggressive behavior, aggressive thoughts, and emotions as well as desensitization to violent content. However, research to this point is not fully consistent as several studies also suggest methodological weaknesses among the conducted violent video gaming studies so far and more studies on violent video gaming are needed to gain a better understanding of interaction effects and to draw appropriate conclusions about whether and how violent video games influence human behavior or the way we perceive our environment.

1.2 Adverse childhood experiences

In addition to the mentioned influences of violent video gaming a change on how we perceive our environment is also stated as a common consequence of adverse childhood experiences (ACE). ACEs have first been described in a groundbreaking study conducted in 1998 by the Centers for Disease Control and the Kaiser Permanente health organization in California. In that study, ACEs referred to three specific types of stressors children faced in the home environment - various forms of physical and emotional abuse, neglect, and household dysfunction (Felitti et al., 1998). The relevance of these stressors and the interactional framework of ACEs are often explained via the biopsychosocial model (Engel, 1977). The biopsychosocial model defines mental illness as an impairment of the body-soul unit with consequences on every aspect of the individual's life. Three dimensions are defined in the model, which can be impaired: A psychological dimension (mental and spiritual), a physical dimension (biological) and an interaction-based dimension (social). This point of view for illness also represents a fundamental keystone for the biopsychosocial medicine today (Egger, 2005).
ACEs are a very common factor in our nowadays society. A meta-analysis for 38 studies across 96 countries revealed that approximately 50% of children in Asia, Africa and North America have experienced violence in the past (Hillis et al., 2016). A study including mostly German participants displayed that 43.7% of respondents reported at least one stressful childhood experience in the past. Four or more ACEs were reported by 8.9% of participants. Parental divorce/separation (19.4%), alcohol use and substance abuse in the family (16.7%), emotional neglect (13.4%), and emotional maltreatment (12.5%) were most frequently reported. In the cumulative model, the high-risk group with four or more ACEs showed significantly increased risk for depressiveness, anxiety, physical aggression, and impaired life satisfaction (Witt et al., 2019).
As relevance and research on ACEs increased several diagnostic tools have been developed. The adverse childhood experiences questionnaire is the first scale measuring ACEs (Felitti et al., 1998). It categorizes ACEs in 5 aspects: physical neglect and abuse, emotional neglect, and abuse as well as sexual abuse. However, as more data on ACEs was available, diagnostic tools also improved and nowadays the state of art to measure ACEs is the childhood trauma questionnaire (CTQ; Bernstein et al., 1998). The CTQ consists of the same 5 categories but is assessed more detailed as more item questions are included. The classification of ACE types into physical abuse and

neglect, emotional abuse and neglect and sexual abuse has proven sufficient over the years.
A meta-analysis on the current state of ACE research that includes 37 studies with a total of over 250.000 participants revealed that individuals with at least 4 types of ACEs have strong increase in risk for sexual risk taking, mental illness and problematic alcohol use as well as very strong risk for problematic drug use and interpersonal/self-directed violence (Hughes et al., 2017). A more recent meta-analysis also displayed a strong connection between various types of ACE and the probability for body dysmorphic disorders (Longobardi et al., 2022). Body dysmorphic disorders are very common among certain personality disorders and ACEs can be a trigger for them. Researchers found that ACEs disrupt the development of certain emotion regulation processes and favor changes in the structure as well as function of key areas in the brain (Sheffler et al., 2020). Body dysmorphic disorders and emotion regulation problems are also said to be often connected to altered perception of several stimuli that affect the body.
How pain is perceived plays not only a role in violent video gaming as described before but also for ACEs. In terms of pain sensitivity, studies found that participants with ACE had an increased tendency to catastrophize pain, regardless of the influence of other risk factors such as sociodemographic characteristics or anxiety and depression symptoms (MacDonald et al., 2021). Especially higher values in the CTQ subscale emotional abuse seem to be connected to lower pain tolerance in the form of heat pain (Pieritz et al., 2015). In addition, there is evidence suggesting decreased pain thresholds in individuals suffering from ACE (Tesarz et al., 2015, 2016).
Studies on the relationship between ACEs and empathy have detected that higher ACE levels are associated with less empathy. Furthermore, empathy and ACE seem to be linked, as child-centered play therapy can increase empathy in ACE patients (Burgin & Ray, 2022; Narvey et al., 2021). Previous research has also shown that ACEs are related to impaired emotion processing (Young & Widom, 2014) and individuals suffering from ACE have increased sensitivity to negative emotions (Curtis & Cicchetti, 2011; Masten et al., 2008; Pollak et al., 2000). These changes among participants are also evident on a neurological level. fMRI studies have shown that participants with ACEs show activation pattern changes in the amygdala and hippocampus compared to participants without ACEs (Assed et al., 2020; Dannlowski et al., 2012; Etkin et al., 2011).

1.3 Aims and hypotheses

The aim of this dissertation was to gain insights into how violent video gaming and ACEs influence pain sensitivity regarding multiple pain tests, how they affect the vulnerability for fear conditioning towards painful stimuli, how they impact empathy for pain in others and how they influence pain perception regarding painful stimuli. We wanted to clarify these potential interactions as previous research is not consistent on results for these variables or did not investigate interactional effects so far. Therefore, we conducted two studies to research on these effects. Both studies were conducted together via one larger study appointment. For the studies we recruited the participants for each of three groups: VVGs, NVVGs and NGs with 20 participants in each group. Both studies were conducted with the same sample of participants. ACE levels of each participant were assessed via the CTQ.

Study 1 consisted of a laboratory part and an MRI part. For the laboratory part of the study participants took part in several pain sensitivity assessments. These included an electric stimulation via a cupric electrode connected to a high voltage constant current stimulator, a cold pressor measurement using an ice bucket and a pressure pain measurement via a pressure algometer. Participants were asked to rate their pain threshold and pain tolerance for each of these pain tests. For the MRI part of this study participants took part in a classic fear conditioning experiment for painful stimuli. The fear conditioning experiment consisted of 4 phases: Habituation, Acquisition 1, Acquisition 2 and Extinction. The pain stimulus used for the fear conditioning experiment was also an electric stimulus via a cupric electrode connected to a high voltage constant current stimulator.
The following hypotheses were tested for Study 1:

1.1. NVVGs and NGs are expected to show significantly increased pain sensitivity compared to VVGs for all conducted pain measurements.

1.2. ACEs serve as moderator variables significantly enhancing the increased pain sensitivity among NVVGs and NGs compared to VVGs.

1.3. NVVG and NG display a significantly enhanced limbic response compared to VVG regarding brain activation for the exposure to conditioned fear stimuli.

1.4. ACEs significantly enhance brain activation patterns for the exposure to conditioned fear stimuli.

Similar to Study 1, hypotheses for Study 2 were also examined via a laboratory section and an MRI section. The MRI section of the study consisted of an empathy for pain in others experiment, in which participants were shown facial expressions via short videos (Presented emotions: pain, neutral expression, happiness and fear), and a pain perception experiment, in which participants were presented with painful stimuli at different stimulus frequencies via a cupric electrode connected to a high-voltage constant-current stimulator. The laboratory section of the study consisted of an extended version of the empathy for pain experiment with more presented emotions (Presented emotions: pain, neutral expression, happiness, fear, disgust, anger, sadness and surprise).
The following hypotheses were tested for Study 2:

2.1. NVVGs and NGs show significantly more ability to recognize pain-related emotions correctly and significantly more brain activation patterns to painful stimuli compared to VVGs.

2.2. NVVGs and NGs show significantly increased activation in prefrontal brain areas when exposed to pain-related emotions and significantly increased limbic responses to painful stimuli compared to VVGs.

2.3. Higher values in ACEs lead to significantly impaired correct identifications of pain-related emotions and significantly increased brain activation patterns regarding painful stimuli.

2.4. Higher values in ACEs lead to significantly increased activation of prefrontal brain areas and the limbic system when exposed to pain-related emotions or painful stimuli.

2 MATERIAL AND METHODS

2.1 Study 1

2.1.1 Participants

We examined 60 participants (23 female; mean age 30, SD 7.90, range 20-58 years). Previous studies on this fear conditioning paradigm yielded a high effect size (d = .80; α = .05) for N=18 per group (Flor et al., 2002; Flor et al., 1996; Rothemund et al., 2012). To account for potential loss of data, we recruited 20 participants per group for our experiment: Non-gamers (NG; 11 females; mean age 33, SD 7.39, range 21-54 years), nonviolent video gamers (NVVG; 7 females; mean age 30, SD 9.02, range 21-58 years) and violent video gamers (VVG; 5 females; mean age 29, SD 7.02, range 20-43 years). Participants in the VVG group needed to play videogames containing self-executed violence to virtual humans or humanoid beings for 15 hours or more per week for at least the year preceding the testing to qualify for the VVG group. Most common videogames in this group were “Call of Duty“, “Counter Strike“ and “Left 4 Dead“. Participants in the NVVG group needed to play videogames containing no violence for 15 hours or longer per week for at least the year preceding the testing. Strategic games with negligeable third person violence also classified as nonviolent videogames (e.g. League of Legends). The most common videogames were “Magic the Gathering: Arena”, “Hearthstone” and strategic world building games. Participants in the NG group needed to play videogames less than 5 hours per week for at least the year preceding the testing.
Exclusion criteria for the magnetic resonance imaging (fMRI) task and pain assessments were video gaming hours, neurological illness, kidney and liver illness, acute suicidality, peripheral coagulopathy or impacted hematopoiesis, pregnancy or breast-feeding, pacemaker or other metal inside the body, aneurysmal-clip or related cardio- or prosthetic clips, claustrophobia or related illness that make laying in the scanner difficult for the participant, brain damage or risk of seizures, organic brain diseases (e.g. Parkinson’s disease) and epilepsy or seizures in the past.
The local Ethics Committee of the Medical Faculty Mannheim of the University Heidelberg approved the study which adhered to the Declaration of Helsinki. Informed consent was obtained from all participants.

2.1.2 Study procedure

In the beginning of the study each participant completed informed consent and self-report questionnaires (see below). The participants were assessed via the Maltreatment and Abuse Chronology of Exposure (MACE; Teicher & Parigger, 2015) in its german adaption (KERF; Isele et al., 2014) and a structured clinical interview based on DSM-5 criteria (SCID-5-CV; Beesdo-Baum et al., 2019). After that pain threshold and pain tolerance were determined. Pain threshold refers to the transition point from rising

sense of pressure to the pain onset point and pain tolerance refers to the maximum level of pain a participant can endure. Pain measurements included electric stimulation during the fMRI experiment and after the fMRI measurement as well as pressure pain assessment and temperature-based pain stimuli after the fMRI measurement. These pain markers were then used in the subsequent fMRI measurement assessing fear conditioning where painful stimuli were employed as unconditioned stimuli. The fMRI measurement also included an empathy for pain and a pain perception task, which will be reported in a separate paper. The participants were examined in terms of electromyogram and skin conductance during the whole fear conditioning experiment. Additionally, participants were asked to give ratings of valence, arousal and contingency after every conditioning phase to check if fear conditioning took place. The overall study procedure is visualized in figure 1 below.

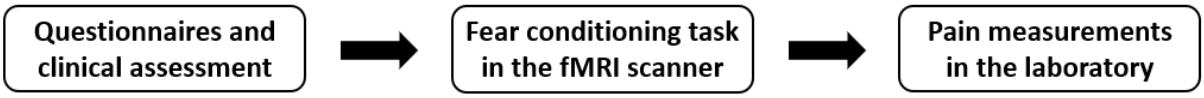

Figure 1. Study 1: The overall study procedure including questionnaires and clinical assessment, the fear conditioning task in the functional magnetic resonance imaging (fMRI) scanner and the pain measurements in the laboratory.

2.1.3 Questionnaires and clinical assessment

Participants completed the detailed clinical assessment via KERF and SCID-5-CV to assess possible mental disorders. In addition, they answered the german adaption (CTQ; Wingenfeld et al., 2010) of the “Childhood Trauma Questionnaire” (Bernstein et al., 1998), the “Positive and Negative Affect Schedule” (PANAS; Watson et al., 1988), the “NEO Five-Factor Inventory” (NEO-FFI; Costa & McCrae, 1989), the “State-Trait Anxiety Inventory” (STAI-S/STAI-T; Spielberger, 1983), the “Scale for the Assessment of Pathological Computer-Gaming” (CSV-S; Woelfling et al., 2010), the “Trier Inventory for Assessment of Chronic Stress” (TICS; Schulz & Schlotz, 1999), the personality questionnaire “Dirty Dozen” (Jonason & Webster, 2010), the “Fear of Pain Questionnaire” (FPQ-SF; Asmundson et al., 2008), the German version of the anxiety and depression inventory “Hospital Anxiety and Depression Scale” (HADS; Herrmann-Lingen et al., 2011) and the “Interpersonal Reactivity Index” (IRI; Davis, 1983).

2.1.4 Adverse childhood experiences assessment

The CTQ was the main source for assessing and calculating the level of ACE together with the in-person KERF and SCID-5-CV diagnostic. The CTQ consists of 5 subscales each rated on a scale from 5 (not or minimal occurred) to 25 (very common and extreme), which are emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect. Each subscale consisted of 5 items rated on a scale of 1 (not occurred) to 5 (very common) and the subscale value was computed via the sum-score of these items. Overall, the CTQ displays good internal consistency with a Cronbach α

ranging from .62 to .96 across all subscales and good construct validity ranging from .14 to .40 across all subscales.

2.1.5 Pain measurements

In order to assure adequate pain measurement for pain threshold and pain tolerance different sensory pain markers were assessed in the fMRI and laboratory part of the study. During the fMRI part prior to the fear conditioning experiment an electric stimulus for the experiment was determined via a cupric electrode connected to a high voltage constant current stimulator (Electric stimulation; Digitimer, DS7A, Welwyn GardenCity, UK). A steadily increasing pain stimulus (50-ms bursts, 12 Hz) was presented to the right thumb of the participants and they were asked to rate the point of pain threshold (first experience of pain) and pain tolerance (point where the pain became unbearable) 3 times. With these data points the targeted pain level was computed via the following formula: pain intensity = pain threshold mean + (pain tolerance mean – pain threshold mean) x 0.8.
After computing the desired pain level, the participants were exposed to the electric stimulus and were asked to rate it on a scale from 0 to 10 where 0 represented “no pain at all“ and 10 “the strongest imaginable pain”. The targeted response was 8 and the pain stimulus was adjusted via a manual high voltage constant current stimulator and respective participant rating if the first rating was not equal to 8. This computed stimulus was then used in the fear conditioning experiment. In the laboratory part of the study the electric stimuli were employed to compute pain threshold and pain tolerance in the same fashion.
Additionally, a cold pressor test on the right hand of the participants was conducted. The participant was asked to immerse their hand into an ice water bucket (0-4°C) for up to three minutes. This test was included in the experiment to allow for a more diverse pain threshold and pain tolerance assessment. The participants were asked to rate pain threshold and pain tolerance during the cold pressor measurement and further they indicated the pain intensity every 10 seconds on a scale from 0-100, where 0 represented “no pain at all” and 100 “the strongest imaginable pain“.
Before and after the cold pressor measurement a pressure pain test via a pressure algometer was conducted to measure sensitivity of the muscle. The participants were asked to report their pain threshold while being exposed to constantly increasing pressure via the pressure algometer applied on the left palm between the thumb and the forefinger. This measurement was conducted three times before the cold pressor measurement and three times after the cold pressor measurement. A visualization for the sequence of the pain measurements and rating scales can be viewed in figure 2 below.

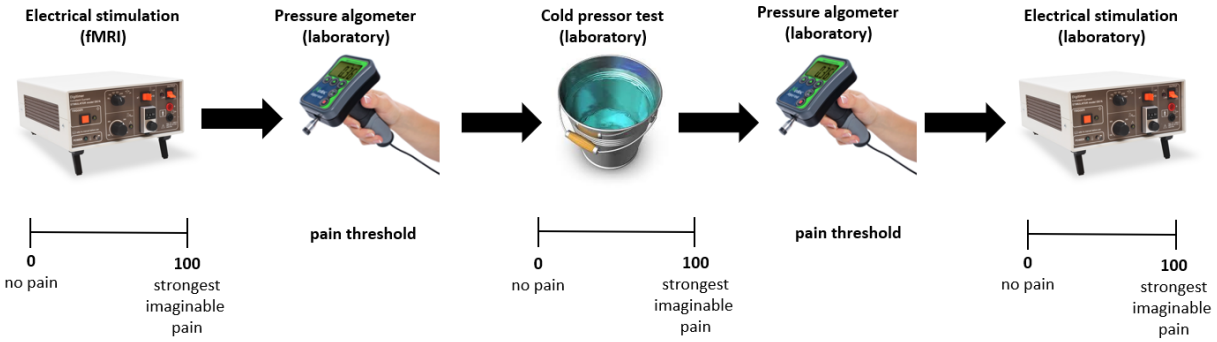

Figure 2. Study 1: Sequence of all conducted pain tests across the experiment with respective ratings for every pain test.

2.1.6 Experimental design for the fear conditioning task

The study used an established fear conditioning paradigm, shown to provide adequate conditioning to fearful stimuli (Baeuchl et al., 2019; Fullana et al., 2016; Rothemund et al., 2012; Suarez-Jimenez et al., 2020). In this differential conditioning paradigm geometrical shapes in the form of a circle and a triangle served as conditioned stimuli, with one stimulus (CS+) predicting the occurrence of a painful unconditioned stimulus (US) while being actually paired with the presentation of the US (CS+c), as well as a condition in which the same conditioned stimuli was not paired with the presentation of the US (CS+uc) to check for hemodynamic responses evoked by the CS+ without the confounding effects of the US. In addition, the other stimulus shape (CS-) signaled the absence of a painful US. In the habituation phase CS+, CS- and US were presented 6 times in a pseudo-randomized order. US presentation in this phase served the purpose to allow participants to habituate to the strong US and reduce motion artifacts in the scanner. Next two identical acquisition phases were presented to the participants. In the acquisition phases the CS+ was presented 19 times and CS- was presented 21 times each in a pseudo-randomized order. During the acquisition phases the US was presented 2.8 seconds together with the CS+ representing the fear conditioning. During extinction, the CS+ and the CS- were presented 8 times in a pseudo-randomized order without the presence of the US. The intertrial interval was always 7-10 s. (see Fig. 3). After each phase, participants were asked questions about the US, CS+ and CS- in terms of arousal, contingency and valence via a visual analogue scale (VAS). These ratings were used to observe if participants experienced the CS+ as a conditioned stimulus and if CS- was distinctively rated and not conditioned to the US. For the ratings the Self-Assessment-Manikin (SAM; Bradley & Lang, 1994) was used and adapted to a 9-point scale reaching from very unlikely (value equals 1 on the scale) to very likely (value equals 9 on the scale).

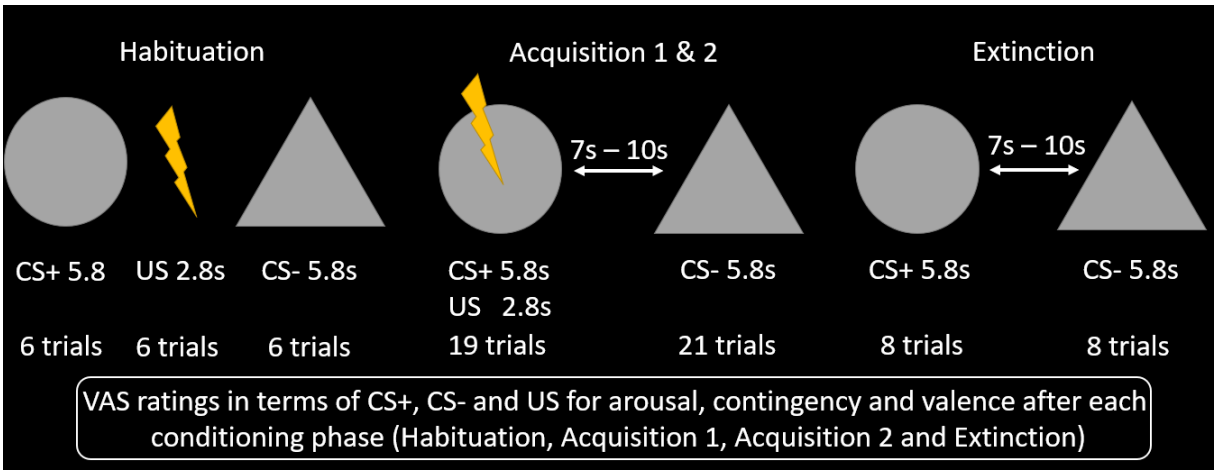

Figure 3. Study 1: The four conditioning phases of the fear conditioning experiment. The circle shape serves as conditioned stimuli paired with the unconditioned stimuli (CS+), the triangle shape as conditioned stimuli not paired with the unconditioned stimuli (CS-) and the lightning shape represents the electric stimulus labeled as unconditioned stimuli (US). After each phase a visual analogue scale (VAS) was presented to obtain ratings in terms of arousal, contingency and valence for the CS+, CS- and the US.

2.1.7 Magnetic resonance imaging acquisition

T1-weighted 3D images were compiled for every participant via a 3-Tesla MR-scanner (PRISMA, Siemens Medical Solutions, Erlangen, Germany) with a 64-channel head coil and rapid gradient echo sequence (TR/TE = 3.15/1.37 ms; 160 slices; 1.6 mm isotropic voxel size). MRI data were acquired during the fear conditioning task via a T2-weighted gradient-echo echo planar imaging sequence (TR/TE = 3100/30ms; 51 slices; FOV 192 mm; 2.0 x 2.0 x 2.5 mm voxel size).

2.1.8 Peripheral psychophysiological recordings

During the fear conditioning experiment in the MR scanner pulse and heart rate were assessed using the respective function of a 3-Tesla MR-scanner (PRISMA, Siemens Medical Solutions, Erlangen, Germany). The measurement was conducted during the whole fMRI experiment from the first trial presentation until the last rating to detect any abnormal physical activities that may have to be accounted for (TR = 3100 ms).

2.1.9 Statistical analysis

Behavioral data analysis:

Behavioral statistics regarding pain measurements and fear conditioning ratings for valence, arousal and contingency were conducted with IBM SPSS Statistics 21 (IBM Corp., 2012). To test for group differences between each observed video gaming group at once regarding the high voltage constant current stimulator electrode stimulation pain test, the cold pressor measurement pain test and the pressure algometer pain test, univariate and multivariate ANOVAs were computed for each pain measurement cause more than two groups were defined in the experimental design and we wanted to analyze the data for single as well as for multiple dependent variables simultaneously. Further linear regression models were used to check for the connection between each CTQ subscale and each pain measurement. Valence, arousal and contingency ratings for the fear conditioning experiment were analyzed using univariate ANOVAs. To test for a potential moderator effect of ACE on the association between video gaming and pain measurements we conducted several moderation analyses by HAYES via PROCESS v3.2 with ordinary least squares regression, yielding unstandardized coefficients for all effects (Hayes, 2017). For the moderation design we used video gaming groups as independent variable, pain threshold or tolerance of the respective pain tests as dependent variable and CTQ subscales as moderator variable. We calculated moderations for every possible combination of pain tests and CTQ subscales. Chi-square analyses were sampled for potential gender and ACE differences between groups to assess potential interfering confounding variables regarding the pain measurements and the fear conditioning experiment. The goal was to exclude confounding variables like gender or ACE-level differences between video gaming groups on pain threshold or pain tolerance for each of the conducted pain measurements and for the pain application of the fear conditioning task.

MRI data analysis:

FSL v6.0 (FMRIB, Oxford, UK) was used to analyze the MRI data for the first-level and higher-level analyses and RStudio version 4.1.2 was used to compile the regressor files of the fear conditioning experiment (Jenkinson et al., 2012; RStudio Team, 2020). The fMRI data was preprocessed in terms of motion correction, high-pass temporal filtering (cut-off = 100s) and was brain-extracted using the respective FSL-tool (http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/BET). A Gaussian kernel of full-width at half-maximum of 5 mm was used for image smoothing and MNI152_T1_2mm standard brain as well as the individual brain extracted MPRAGE were applied for volume registration via FMRIB´s Linear Registration Tool (http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/FLIRT). Correlation contrasts to check for interactions and main effects were designed in a full model setup to check the hypotheses regarding the first-level analysis.
The FSL higher-level analysis was used to test for video gaming group differences in brain activation for every fear conditioning phase in the form of independent t-tests with and without the covariate of CTQ-subscales representing ACE-dimensions as well as for a general correlation between brain activity and CTQ-subscale values. T-tests were conducted via contrast testing on the subject level with using the respective feat-folders of each participant. For video gaming group differences, two respective video gaming

groups were selected and contrasted against each another based on the defined first level analysis contrasts. This analysis was then extended by adding each CTQ subscale as a covariate to the analysis one by one. For the general influence of CTQ subscales on the overall sample we conducted a correlation analysis with the whole sample at once, not dividing the sample by video gaming groups. We also merged the acquisition phases 1 and 2 for a follow-up analysis to generate more power. Analysis methods were congruent to the analysis described above; however, we did not use the feat-folders, but the respective contrasts defined in the first level analysis one by one (Cope-level analysis). We used a mixed effects model: FLAME 1 with a cluster-z-threshold of 3.1 and a cluster-p-threshold of 0.05.

2.2 Study 2

2.2.1 Participants

Sixty participants were included in the study (23 females; mean age 30, SD 7.90, range 20-57 years). Several pain assessment studies with electric or olfactory stimuli yielded good effect size (d = .80; α = .05) for N=18 per group and therefore adequate study metrics can also be assumed for this study (Flor et al., 2002; Flor et al., 1996; Rothemund et al., 2012). Sixty participants were recruited for the study to account for potential loss of data. These participants were allocated to 3 groups with 20 participants each: non-gamers (NG; 11 females; mean age 33, SD 7.39, range 21-54 years), nonviolent video gamers (NVVG; 7 females; mean age 30, SD 9.02, range 21-58 years), and violent video gamers (VVG; 5 females; mean age 29, SD 7.02, range 20-43 years). Participants were included in the NG group if they consumed video games for less than 5 hours per week for at least the year preceding participation. Participants were allocated to the NVVG group if they spent at least 15 hours per week for at least the year preceding participation on video games without violent content. Strategic games were accepted and negligeable third person violence was also classified as nonviolent. Magic the Gathering: Arena, Hearthstone and strategic world building games were the most popular games in this group. Participants qualified for the VVG group if they played video games containing self-executed violence to virtual humans or humanoid beings for at least 15 hours per week for at least one year prior to the testing. Call of Duty, Counter Strike and Left 4 Dead were the most popular games played by this participant group. The NVVG group and VVG groups had to play the respective category of games for at least 90% of their gaming time to qualify for their gaming group.
The following exclusion criteria were used in the study: neurological illness, kidney and liver illness, acute suicidality, periphery coagulopathy or impacted hematopoiesis, pregnancy or breastfeeding, pacemaker or other metal inside the body, aneurysmal-clip or related cardio or prosthetic clips, claustrophobia or related illness that made lying in the scanner difficult for the participant, brain damage or affinity for seizures, organic brain diseases (e.g. Parkinson's) and epilepsy or seizures in the past.
The local Ethics Committee of the Medical Faculty Mannheim of the University Heidelberg approved the study, which adhered to the Declaration of Helsinki. Written informed consent was obtained from all participants.

2.2.2 Study Procedure

To assess violent video gaming criteria and ACE level estimations, participants were asked to complete self-assessment questionnaires and take part in a clinical assessment at the beginning of the study. The clinical assessment consisted of the Maltreatment and Abuse Chronology of Exposure (MACE; Teicher & Parigger, 2015) in its German adaption (KERF; Isele et al., 2014) and a structured clinical interview based on DSM-5 criteria (SCID-5-CV; Beesdo-Baum et al., 2019). Subsequently individual electrical pain thresholds and pain tolerance were determined in order to select the later used painful stimuli for the pain perception task.
After this assessment participants were invited to the fMRI part of the study. Two experiments were conducted in the fMRI session: An empathy for pain task, followed by a pain perception task. In addition, skin conductance and electromyogram from all participants were continuously recorded. After the fMRI measurement the empathy for pain task was conducted again in the laboratory with a larger stimulus quantity and more presented emotions (described below).

Figure 4. Study 2: The overall study procedure including questionnaires and clinical assessment, the empathy for pain task and the pain perception task in the magnetic resonance scanner using functional recordings (fMRI).

2.2.3 Questionnaires and clinical assessment

After the clinical assessment via KERF and SCID-5-CV, participants were asked to complete the "Childhood Trauma Questionnaire" (CTQ; Bernstein et al., 1998), the "Positive and Negative Affect Schedule" (PANAS; Watson et al., 1988), the "NEO Five-Factor Inventory" (NEO-FFI; Costa & McCrae, 1989), the "State-Trait Anxiety Inventory" (STAI-S/STAI-T; Spielberger, 1983), the "Scale for the Assessment of Pathological Computer-Gaming" (CSV-S; Woelfling et al., 2010), the "Trier Inventory for the Assessment of Chronic Stress" (TICS; Schulz & Schlotz, 1999), the personality questionnaire "Dirty Dozen" (Jonason & Webster, 2010), the "Fear of Pain Questionnaire" (FPQ-SF; Asmundson et al., 2008), the German version of the anxiety and depression inventory "Hospital Anxiety and Depression Scale" (HADS; Herrmann-Lingen et al., 2011) and the "Interpersonal Reactivity Index" (IRI; Davis, 1983).

2.2.4 Adverse childhood experiences assessment

The CTQ was the main factor in assessing ACE history together with the KERF screening. The CTQ includes 5 types of early childhood trauma: emotional abuse, physical

abuse, sexual abuse, emotional neglect and physical neglect. Each subscale consists of 5 items rated on a scale from 5 (not or minimal occurred) to 25 (very common and extreme) and the subscale score was computed via sum-score addition of the items. The CTQ displays good internal consistency with a Cronbach α ranging from .62 to .96 across all subscales and good construct validity ranging from .14 to .40 across all subscales.

2.2.5 Pain measurements

The pain perception task conducted in the scanner required an individual assessment of pain threshold and pain tolerance. Therefore, participants were exposed to a precisely calculated pain stimulus on the right thumb via a cupric electrode connected to a high voltage constant current stimulator prior to the fMRI measurement (electric stimulation; Digitimer, DS7A, Welwyn GardenCity, UK). The pain stimulus was constantly increasing in intensity (50-ms bursts, 12 Hz) and the participants were asked to rate the point of pain threshold and pain tolerance three times across three trials. These datapoints were used to compute a targeted electrical stimulus intensity. The following pain intensity formula was used to compute the individual pain intensity for the pain perception task: pain intensity = pain threshold mean + (pain tolerance mean – pain threshold mean) x 0.8. This computed pain intensity was then presented to the participants and they rated the stimulus on a scale from 0 (“no pain at all”) to 10 (“the strongest imaginable pain”). The targeted response by participants to the pain stimulus was 8. The pain stimulus was adjusted manually via a high voltage constant current stimulator until a response of 8 was given by the participants, if the first rating was not equal to the targeted response of 8. This stimulus was then used in the pain perception experiment. This pain measurement was also conducted in the laboratory in order to detect any setting-based pain sensitivity changes.

2.2.6 MRI and laboratory – Experimental design for the empathy for pain task

The rating for empathy for pain in others was conducted by an established empathy for faces task in the fMRI as well as a longer version in the laboratory (Simon et al., 2006; Vachon-Presseau et al., 2011). For the fMRI task, participants were presented short videos of people with a video duration of one second. After that they were asked to rate the shown emotion. Participants had to choose between neutral expression, surprise, happiness, anxiety, disgust, anger, pain or sadness. Pain was the main interest for hypotheses regarding this study and other emotions were assessed as control emotions. A Self-Assessment-Manikin (SAM; Bradley & Lang, 1994) and additional manikin visualizations partially adopted from prior research (Simon et al., 2006; Vachon-Presseau et al., 2011) were shown after each trial, asking participants about the perceived arousal, pleasantness/unpleasantness and suffering of the presented person via nonverbal pictures. The values were then converted to a 9-point scale.
These ratings were included in the experiment to gain insight about the impact different emotions had on the participants. The fMRI version of the task consisted of 16 trials showing facial expressions for the emotions fear, happiness, neutral and pain. Each of these emotions was shown four times in a pseudo-randomized order. For the

laboratory version of the task 32 trials were included with facial expressions of the emotions fear, happiness, neutral, pain, anger, sadness, disgust and surprise, each emotion presented 4 times. The experiment is depicted in figure 5.

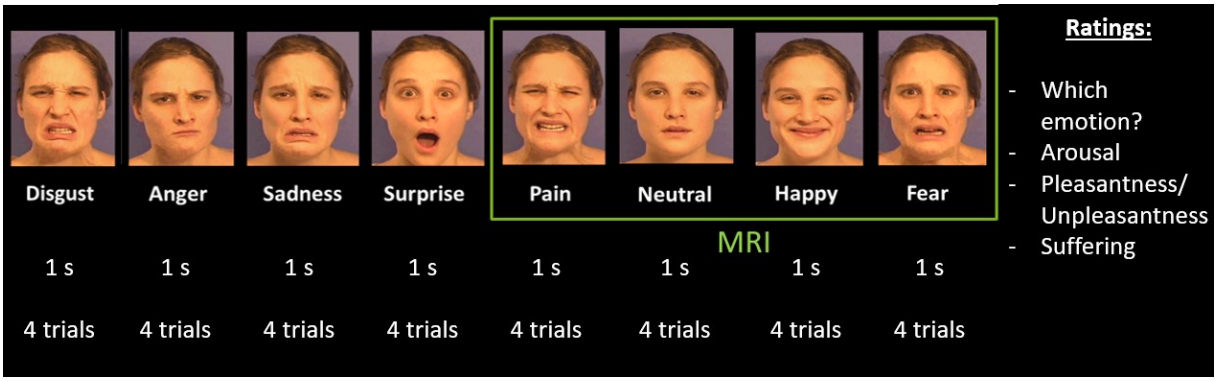

Figure 5. Study 2: The empathy for pain task structure for the functional magnetic resonance imaging part (MRI) and for the laboratory experiment (all shown emotions included) with ratings asking which emotion was shown as well as ratings of perceived arousal, pleasantness/unpleasantness and suffering.

2.2.7 MRI - Experimental design for the pain perception task

In order to assess self-experienced pain originating from different pain stimulus frequencies and to gain insights about how pain perception changes/increases with varying stimulus frequencies the study contained a pain perception experiment, which was also conducted in the MRI scanner. The experiment used a previously self-adjusted pain stimulus on the right thumb via a cupric electrode connected to an electric device (Digitimer, DS7A, Welwyn GardenCity, UK) for pain induction during the task. The pain perception experiment consisted of 15 intervals and each interval included 10 painful stimuli. Three different pain stimulus frequencies were applied via electric stimulation to the thumb of the participants at 0.2 Hz, 1 Hz and 2 Hz. Each pain stimulus frequency was presented five times in a pseudo-randomized order. We conducted the experiment with the chosen frequencies to detect a possible effect of temporal summation with increased pain stimulus frequency and hinder habituation. After each stimulation interval the participants were asked to rate the perceived intensity and unpleasantness of the experienced pain on a visual analogue scale (VAS) with 23.81 cm length reaching from “no pain”/“not unpleasant at all” to “strongest imaginable pain”/ “as unpleasant as possible”. The VAS was later transformed to a 0 to 100 scale. The task structure for the pain perception experiment is shown in figure 6 below.

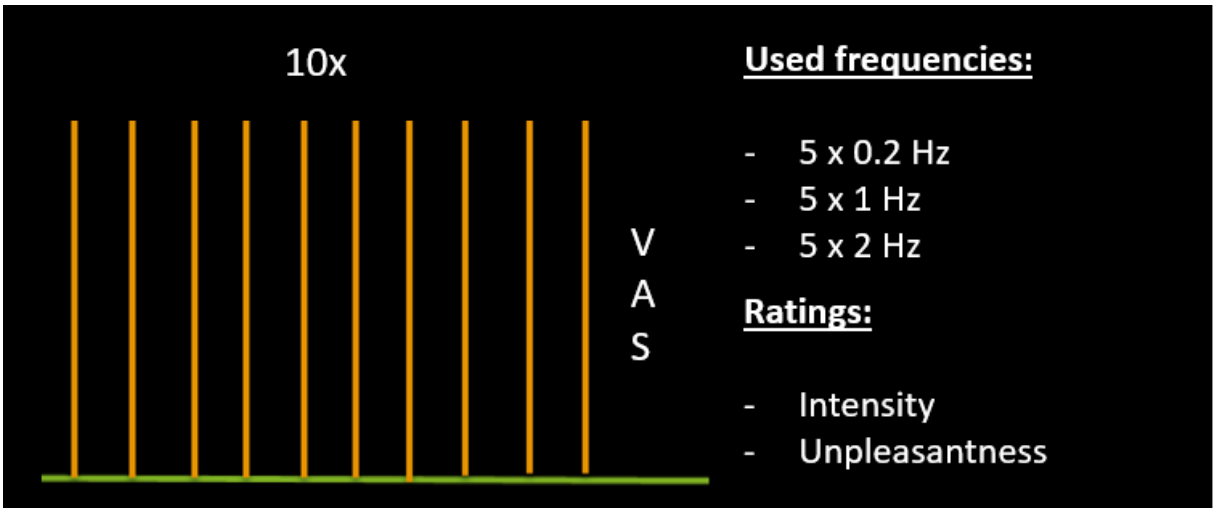

Figure 6. Study 2: Task structure for the pain perception fMRI experiment including visual analogue scale (VAS) ratings and each pain stimuli interval.

2.2.8 MRI acquisition

T1-weighted 3D images were compiled for every participant via a 3-Tesla MR-scanner (PRISMA, Siemens Medical Solutions, Erlangen, Germany) with a 64-channel head coil and rapid gradient echo sequence (TR/TE = 3.15/1.37 ms; 160 slices; 1.6 mm isotropic voxel size). MRI data were acquired during the empathy for pain and pain perception task via a T2-weighted gradient-echo echo planar imaging sequence (TR/TE = 3100/30ms; 51 slices; FOV 192 mm; 2.0 x 2.0 x 2.5 mm voxel size).

2.2.9 Peripheral psychophysiological recordings

During the empathy for pain and pain perception task in the MRI heartrate was recorded using the body physiology function of the MR scanner. Heartrate was assessed continuously during the entire MRI measurement for each task beginning from the first trial presentation until the last rating to detect any physiological confounding variables that may have to be considered during the fMRI analysis. No physiological recordings were assessed for the laboratory part of the empathy for pain task.

2.2.10 Statistical analysis

Behavioral data analysis:

Arousal, pleasantness/unpleasantness and perceived suffering ratings of the empathy for pain experiment as well as intensity and unpleasantness ratings of the pain perception experiment were analyzed using IBM SPSS Statistics 21 (IBM Corp., NY, USA,

2012). For group differences regarding each researched variable univariate and multivariate ANOVAs were used. Linear regressions were computed to check for the influence of CTQ subscales on correct emotion identification. Differences in ratings for arousal, pleasantness/unpleasantness and perceived suffering in the empathy for pain task were analyzed via ANOVAs using computed means of the respective rating group for each used emotion.
In the pain perception experiment we used univariate ANOVAs to determine video gaming group differences regarding intensity and unpleasantness ratings for pain stimuli regarding each stimulus frequency. The influence of CTQ subscales on intensity and unpleasantness ratings was analyzed via linear regression.
To ensure the assessment of potential confounding variables like sex-based differences regarding classification of emotions, ratings for arousal, pleasantness/unpleasantness, suffering and intensity as well as gaming group dependent ACE-levels, several analyses were conducted. ACE-level differences between groups and potential sex distribution across groups were checked via chi-square analyses and violated sphericity assumptions were considered via a Greenhouse-Geiser correction for all ANOVA analyses.

MRI data analysis:

All fMRI data were analyzed at first-level and higher-levels using FSL v6.0 (FMRIB, Oxford, UK). The regressor files for timing of the empathy for pain and pain perception experiment were compiled with RStudio version 4.1.2 (Jenkinson et al., 2012; RStudio Team, 2020). Preprocessing for the fMRI data included motion correction, high-pass temporal filtering (cut-off = 100s) and brain extraction of field maps and MPRAGE images using the FSL BET-tool (http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/BET). Image smoothing was done with the Gaussian kernel of full-with at half-maximum of 5mm and volume registration via FMRIB's linear registration tool was compiled with the MNI152_T1_2mm standard brain as well as the individual brain extracted MPRAGE image (http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/FLIRT). A full model setup for the first-level analysis was compiled for hypothesis testing setting up correlational contrasts to check for main effects of different emotions in the empathy for pain experiment and different frequencies of pain stimuli in the pain perception experiment as well as interactional effects of main effects for each of these experiments.
The FSL higher-level analysis section of fMRI data was done via computing contrasts for video gaming group differences regarding brain activation for different shown emotions as well as for brain activation in the pain perception experiment regarding different frequencies of pain stimuli. We used independent t-tests with and without the covariate of CTQ subscale values for the analysis of both experiments. Analyses without the covariates were compiled in order to determine the pure effect of video gaming behavior in the respective experiment and analyses with covariates were conducted to analyze if the video gaming behavior persisted after accounting for the effect of CTQ subscales. FSL higher level analyses were also conducted for the relationship of CTQ subscale values as ACE dimensions and brain activity for different emotions in the empathy for pain experiment as well as with brain activity of the pain perception experiment regarding each used electric pain stimulus frequency. Respective single subject result folders from the first-level analysis were used for contrast testing on group levels. We compared performance between different video gaming groups by selecting two respective video gaming groups per analysis and contrasted them against each other. To check for influence of ACE on these contrasts we also added CTQ subscales as

covariates into that analysis one by one after checking for the pure influence of video gaming group differences on the postulated hypotheses. In order to check for the correlational influence of ACE on the whole sample we computed correlational analyses with one CTQ subscale per analysis using the whole experimental sample at once not divided according to video gaming groups. Mixed effects model: FLAME 1 was used applying a cluster-z-threshold of 3.1 and a cluster-p-threshold of 0.05 and no further correction was applied.

3 RESULTS

3.1 Study 1

3.1.1 Sample characteristics

Chi-square tests showed no significant differences for the observed pool of participants between the video gaming groups regarding sex (male vs female distribution; $\chi^2(2) = 3.95$, $p = .136$) and ACE levels (ACE in the past vs no ACE in the past; $\chi^2(2) = 3.60$, $p = .165$). An illustration for the means and standard deviations of CTQ subscale values across the different video gaming groups can be seen in Table 1. One-factorial ANOVAs yielded no significant differences for the CTQ subscales across the video gaming groups for emotional abuse ($F(2,57) = .51$, p=.606), physical abuse ($F(2,57) = 2.19$, p=.121), sexual abuse ($F(2,57) = .60$, p=.555), emotional neglect ($F(2,57) = .20$, p=.821) and physical neglect ($F(2,57) = .66$, p=.519).

Table 1. Study 1: Means and standard deviations of each individual CTQ subscale assessing ACE (emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect) for every video gaming group (violent video gamer (VVG), non-violent video gamer (NVVG) and non-gamer (NG))

Video gaming group	VVG	NVVG	NG
Emotional abuse	13.00 (*SD*=6.72)	11.30 (*SD*=6.05)	11.30 (*SD*=5.71)
Physical abuse	9.45 (*SD*=6.40)	6.50 (*SD*=2.93)	7.45 (*SD*=3.56)
Sexual abuse	8.35 (*SD*=6.36)	6.70 (*SD*=3.50)	7.65 (*SD*=4.06)
Emotional neglect	13.55 (*SD*=7.53)	12.25 (*SD*=6.12)	13.05 (*SD*=6.00)
Physical neglect	9.40 (*SD*=5.79)	7.65 (*SD*=3.90)	8.40 (*SD*=4.58)

Note. N = 60; N=20 per video gaming group.
VVG = violent video gamer; NVVG = nonviolent video gamer; NG = non-gamer

3.1.2 Pain measurements and video gaming groups

In terms of the conducted pain measurements the cold pressor measurement test results displayed significant pain threshold differences for the video gaming groups ($F(2,26.60) = 5.28$, $p = .012$). VVG showed a significantly higher pain threshold in the cold pressor measurement test than NG (36.90, 95%-CI[4.24, 69.56], $p = .023$) but not

significantly higher than NVVGs (18.20, 95%-CI[-14.46, 50.86], p = .379). NVVG displayed no significant difference for the pain threshold in the cold pressor measurement test compared to NG (18.70, 95%-CI[-13.96, 51.36], p = .359).
The cold pressor measurement test also displayed significant pain tolerance differences across the video gaming groups (F(2,31.43) = 6.19, p = .005). VVG showed significantly higher cold pressor pain tolerances than NG (43.40, 95%-CI[1.20, 84.80], p = .038), but VVG did not show significantly higher cold pressor measurement test pain tolerance than NVVG (4.25, 95%-CI[-37.15, 45.65], p = .967) and NVVG did not show a significant difference in cold pressor measurement test pain tolerance compared to NG (39.15, 95%-CI[-2.25, 80.55], p = .068).
Electric stimulation in the laboratory displayed significant differences in pain threshold across the video gaming groups (F(2,56) = 5.70, p = .006). VVG showed higher pain thresholds than NG (1.59, 95%-CI[.38, 2.80], p = .007) and NVVG (1.28, 95%-CI[.08, 2.47], p = .034), but NVVG did not display a significant difference to NG (.32, 95%-CI[-.89, 1.53], p = .803). Electric stimulation in the laboratory also showed significant pain tolerance differences across video gaming groups (F(2,56) = 3.36, p = .042). VVG displayed significantly higher pain tolerance than NG (1.76, 95%-CI[.09, 3.43], p = .036) but not significantly higher pain tolerance than NVVG (1.16, 95%-CI[-.49, 2.81], p = .215). NVVG also showed no significantly higher pain tolerance than NG (.60, 95%-CI[-1.07, 2.27], p = .664). Electric stimulation in the MRI displayed significant video gaming group differences regarding reaching the targeted pain intensity rating of eight by participants (F(2,56) = 4.92, p = .011). VVG displayed higher pain intensity ratings than NG (5.20, 95%-CI[.98, 9.42], p = .012), but VVG did not show significantly higher pain intensity ratings than NVVG (1.03, 95%-CI[-3.25, 5.30], p = .833) and NVVG did not show a significant difference in pain intensity ratings compared to NG (4.17, 95%-CI[-.10, 8.45], p = .057).
The pressure algometer test before the cold pressor measurement displayed significant differences in pain thresholds across video gaming groups (F(2,34.80) = 4.06, p = .026). VVG displayed significantly higher pain thresholds than NG (20.86, 95%-CI[4.99, 36.72], p = .007) but not significantly higher pain threshold than NVVG (11.64, 95%-CI[-4.23, 27.51], p = .191). NVVG also showed no significantly higher pain threshold than NG (9.22, 95%-CI[-6.65, 25.10], p = .349). Pressure algometer values after the cold pressor measurement also displayed significantly different pain threshold ratings across the video gaming groups (F(2,35.02) = 4.83, p = .014). VVG displayed higher pain threshold than NG (25.12, 95%-CI[7.32, 42.91], p = .004), but VVG did not show significantly higher pain threshold than NVVG (14.09, 95%-CI[-3.70, 31.89], p = .146) and NVVG did not show a significant difference in pain threshold compared to NG (11.02, 95%-CI[-6.78, 28.82], p = .303).
Results for all conducted pain assessments and respective video gaming group differences can be viewed in Table 2 below.

Table 2. Study 1: Means and standard deviation derived from one-factorial ANOVA testing for each conducted pain measurement across the video gaming groups

Video gaming group	VVG	NVVG	NG
Cold pressor measurement pain threshold	M = 48.30 SD = 61.01	M = 30.10 SD = 41.74	M = 11.40 SD = 7.84
Cold pressor measurement pain tolerance	M = 92.35 SD = 65.30	M = 88.10 SD = 63.10	M = 48.95 SD = 25.19
Electric stimulation pain threshold (laboratory)	M = 4.06 SD = 1.87	M = 2.78 SD = 1.36	M = 2.47 SD = 1.40
Electric stimulation pain tolerance (laboratory)	M = 5.93 SD = 2.23	M = 4.77 SD = 2.20	M = 4.17 SD = 2.05
Electric stimulation (MRI)	M = 11.32 SD = 5.59	M = 10.29 SD = 5.85	M = 6.12 SD = 5.18
Pressure algometer pain threshold pre cold pressor measurement	M = 65.15 SD = 29.14	M = 53.51 SD = 12.29	M = 44.30 SD = 17.44
Pressure algometer pain threshold post cold pressor measurement	M = 69.40 SD = 33.25	M = 55.31 SD = 13.87	M = 44.29 SD = 18.51

Note. N = 60; N=20 per videogaming group.
VVG = violent video gamer; NVVG = nonviolent video gamer; NG = non-gamer
M = mean; SD = standard deviation

We observed significant differences in means between the pain threshold ratings of the pressure algometer test pre cold pressor measurement and pressure algometer test post cold pressor measurement accounting for the entire sample (p=.043). However, the only group that showed a significant difference in pain ratings regarding the pressure algometer test before the cold pressor measurement and after the cold pressor measurement was VVG (p=.043). VVG showed significantly higher pressure pain tolerance after the cold pressor measurement than before. NVVG (p=.331) as well as NG (p=.995) did not show significant differences for the pressure algometer test pre cold pressor measurement and pressure algometer test post cold pressor measurement. A table containing the results is visualized below.

Table 3. Study 1: Differences between the pressure algometer test pre cold pressor measurement and pressure algometer test post cold pressor measurement pain threshold ratings for the whole sample as well as divided by groups

Video gaming group	Whole sample	VVG	NVVG	NG
Pressure algometer pre cold pressor measurement	M=54.32 SD=22.23	M=65.15 SD=29.14	M=53.51 SD=12.29	M=44.29 SD=17.45
Pressure algometer post cold pressor measurement	M=56.33 SD=25.22	M=69.40 SD=33.25	M=55.31 SD=13.87	M =44.29 SD=18.51
t	-2.07*	-2.17*	-1.00	.01

Note. N = 60; N=20 per video gaming group.
VVG = violent video gamer; NVVG = nonviolent video gamer; NG = non-gamer
* = $p < .05$

3.1.3 Pain measurements and adverse childhood experiences

The pressure algometer pain threshold pre cold pressor measurement showed a significant predictability of variance by the level of ACE ($F(5,54) = 5.58$, $p < .001$). Twenty-eight percent of variance of sensitivity in the pressure algometer test pre cold pressor measurement was explained by the level of ACE. Emotional neglect was the strongest predictor for sensitivity in the pressure algometer test pre cold pressor measurement with respect to the condition of listwise inclusion. Higher levels of emotional neglect were connected to significantly lower scores of pain threshold in the pressure algometer test pre cold pressor measurement. CTQ subscales emotional abuse ($\beta = 1.457$; $t(54) = 1.964$; $p = .055$), physical abuse ($\beta = 1.567$; $t(54) = 1.897$; $p = .063$), sexual abuse ($\beta = .917$; $t(54) = 1.317$; $p = .193$) and physical neglect ($\beta = .704$; $t(54) = .788$; $p = .434$) were also added to the model via listwise inclusion.
The pressure algometer pain threshold post cold pressor measurement also revealed variance predictability of pain threshold ratings by levels of ACE ($F(5,54) = 4.67$, $p = .001$). Twenty-four percent of variance for the sensitivity of the pressure algometer test post cold pressor measurement was explained by the level of ACE. Emotional neglect represented the strongest factor as a single subscale in explaining the influence of ACE on this pain test. Higher levels of emotional neglect were connected to significantly lower scores in terms of pressure algometer threshold for the pressure algometer test post cold pressor measurement ($\beta = -2.253$; $t(54) = -2.522$; $p = .015$). CTQ subscales emotional abuse ($\beta = 1.662$; $t(54) = 1.873$; $p = .066$), physical abuse ($\beta = 1.678$; $t(54) = 1.740$; $p = .088$), sexual abuse ($\beta = 1.189$; $t(54) = 1.462$; $p = .149$) and physical neglect ($\beta = .301$; $t(54) = .288$; $p = .774$) were added to the model via listwise inclusion. CTQ subscales did not significantly predict variance of other pain measurements than the pressure algometer test.

3.1.4 Pain measurements, adverse childhood experiences and video gaming groups

Next, a moderation analysis was performed to determine whether CTQ subscales moderate the connection between gaming groups and the sensitivity in pain tests. Physical neglect moderated the pain sensitivity in multiple pain tests (see fig. 2). The moderation regarding the pressure algometer test pre cold pressor measurement explained 32.68% of variance (F (3,56) = 9.06, $p < .001$) with a significant interaction between physical neglect and video gaming groups ($\Delta R^2 = 11.06\%$, F (1, 56) = 9.20, $p = .004$, 95% CI [0.611, 2.991]). Conditional effects analyses with simple slope interaction showed non-significant predictor qualities for low levels of physical neglect (- 1 SD; $b = 2.97$, $SE = 3.74$, $t = .80$, $p = .430$). In contrast, a mean level of physical neglect (MEAN; $b = 9.24$, $SE = 2.98$, $t = 3.10$, $p = .003$) as well as high levels of physical neglect (+1 SD; $b = 17.87$, $SE = 3.98$, $t = 4.49$, $p < .001$) were significant moderators for the relation between gaming groups and pressure algometer test pre cold pressor measurement sensitivity. Table 4 below shows the data for this moderation model.

Table 4. Study 1: Moderation model results for the moderating effect of physical neglect on the relation between the independent variable video gaming groups and the dependent variable pressure algometer test pre cold pressor measurement

	b	*SE(b)*	*t*	*p*
Constant	53.72 [48.86; 58.58]	2.43	22.15	< .001
Video gaming group	9.24 [3.28; 15.20]	2.98	3.10	.003
Physical neglect	.88 [-.17; 1.92]	.52	1.68	.100
Video gaming group x Physical neglect	1.80 [.61; 3.00]	.59	3.03	.004

Note. N = 60; N=20 per video gaming group.

Physical neglect as a moderator also explained 29.13% of variance on the relation between video gaming groups and the pressure algometer test post cold pressor measurement pain threshold (F (3,56) = 7.67, $p < .001$) with a significant interaction between gamer groups and physical neglect ($\Delta R^2 = 9.28\%$, F (1, 56) = 7.33, $p = .009$, 95% CI [0.489, 3.256]). Similar to the pressure algometer test pre cold pressor measurement pain threshold low levels of physical neglect did not show significant conditional effects (- 1 SD; $b = 4.97$, $SE = 4.35$, $t = 1.14$, $p = .257$) but mean (MEAN; $b = 11.49$, $SE = 3.47$, $t = 3.32$, $p = .002$) and high scores (+1 SD; $b = 20.46$, $SE = 4.64$, $t = 4.41$, $p < .001$) served as significant moderators for the relation between gaming groups and pressure algometer test post cold pressor measurement sensitivity. Table 5 below shows the data for this moderation model.

Table 5. Study 1: Moderation model results for the moderating effect of physical neglect on the relation between the independent variable video gaming groups and the dependent variable pressure algometer test post cold pressor measurement

	b	*SE(b)*	*t*	*p*
Constant	55.71 [50.06; 61.36]	2.82	19.74	< .001
Video gaming group	11.49 [4.55; 18.43]	3.47	3.32	.002
Physical neglect	.58 [-.64; 1.80]	.61	.95	.346
Video gaming group x physical neglect	1.88 [.49; 3.26]	.69	2.71	.009

Note. N = 60; N=20 per video gaming group.

In addition, the sensitivity of electric stimulation pain thresholds in the laboratory was also moderated by physical neglect. Physical neglect explained 25.17% of variance on the relation between video gaming groups and sensitivity of the electric stimulation pain threshold ($F(3,55) = 6.17$, $p = .001$) with a significant interaction between physical neglect and gaming groups ($\Delta R^2 = 9.78\%$, $F(1, 55) = 7.19$, $p = .010$, 95% CI [0.040, 0.270]). Conditional effects of low (- 1 SD; $b = 0.32$, $SE = 0.31$, $t = 1.05$, $p = .297$), mean (MEAN; $b = 0.82$, $SE = 0.24$, $t = 3.39$, $p = .001$) and high (+1 SD; $b = 1.52$, $SE = 0.36$, $t = 4.25$, $p < .001$) physical neglect as focal predictor at values of the moderator showed similar patterns as the previous mentioned pain tests. Table 6 below shows the data for this moderation model.

Table 6. Study 1: Moderation model results for the moderating effect of physical neglect on the relation between the independent variable video gaming groups and the dependent variable pain threshold markers obtained via electric stimulation in the lab

	b	*SE(b)*	*t*	*p*
Constant	3.03 [2.63; 3.42]	.20	15.27	< .001
Video gaming group	.82 [.34; 1.31]	.24	3.39	.001
Physical neglect	-.08 [-1.18; .02]	.05	-1.58	.120
Video gaming group x physical neglect	.15 [.04; .27]	.06	2.68	.010

Note. N = 60; N=20 per video gaming group.

Pain tolerance for electric stimulation in the laboratory was also significantly moderated by physical neglect and physical neglect explained 17.20% of variance on the relation between gaming groups and this pain test (F (3,55) = 3.81, *p* = .015) with a significant interaction between physical neglect and gaming groups (ΔR^2 = 6.76%, F (1, 55) = 4.50, *p* = .039, 95% CI [0.009, 0.333]). Similar to the other reported pain tests low levels of physical neglect did not show significant conditional effects (- 1 SD; *b* = 0.35, *SE* = 0.43, *t* = 0.81, *p* = .423) but mean (MEAN; *b* = 0.90, *SE* = 0.34, *t* = 2.65, *p* = .011) and high scores (+1 SD; *b* = 1.67, *SE* = 0.50, *t* = 3.34, *p* = .002) did. Table 7 below shows the data for this moderation model.

Table 7. Study 1: Moderation model results for the moderating effect of physical neglect on the relation between the independent variable video gaming groups and the dependent variable pain tolerance markers obtained via electric stimulation in the lab

	b	*SE(b)*	*t*	*p*
Constant	4.87 [4.32; 5.43]	.28	17.56	< .001
Video gaming group	.90 [.22; 1.56]	.34	2.65	.011
Physical neglect	-.08 [-.22; .06]	.07	-1.13	.265
Video gaming group x physical neglect	.17 [.01; .33]	.08	2.12	.039

Note. N = 60; N=20 per video gaming group.

We obtained no additional moderating effects of CTQ subscales besides physical neglect on the conducted pain tests. All moderation-based interactions are visualized in figure 7 below.

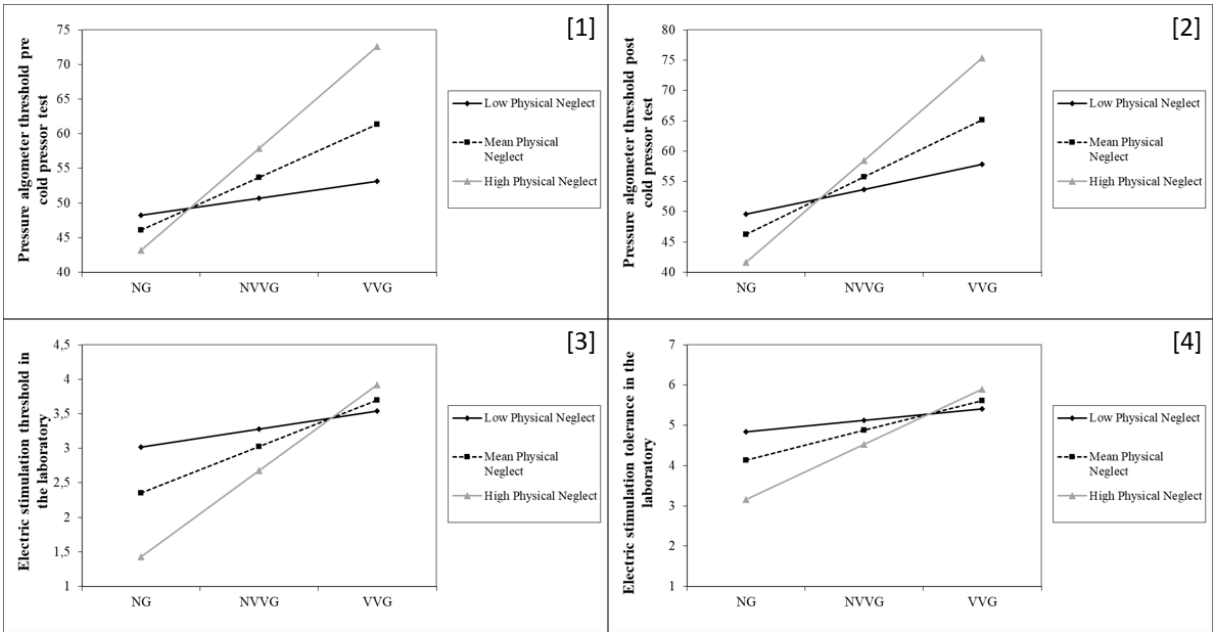

Figure 7. Study 1: Illustrated moderations of 1] physical neglect (M) – gamer groups (X) – pressure algometer threshold pre cold pressor test (Y); [2] physical neglect (M) – gamer groups (X) – pressure algometer threshold post cold pressor test (Y); [3] physical neglect (M) – gamer groups (X) – electric stimulation threshold in the laboratory (Y); [4] physical neglect (M) – gamer groups (X) – electric stimulation tolerance in the laboratory (Y).

NOTE. X = moderator variable; X = independent variable; Y = dependent variable
VVG = violent video gamer; NVVG = nonviolent video gamer; NG = non-gamer

3.1.5 Arousal, valence and contingency ratings for the fear conditioning task conducted in the MRI

Arousal, valence and contingency ratings were observed to assess if fear conditioning was induced. Arousal, valence and contingency ratings indicated successful fear conditioning, because all ratings showed a significant distinction in mean ratings between phases that differed in stimulus presentation due to the conditioning paradigm and no significant distinctions were observed between the two acquisition phases which did not differ in the conditioning stimulus paradigm (see Table 8, Table 9 and Table 10). No significant differences for the arousal, valence and contingency ratings between the video gaming groups were observed and CS+ and CS- were not correlating in the acquisition phases in terms of ratings of arousal, valence or contingency as designed. Visualization for arousal ratings (see figure 8), valence ratings (see figure 9) and contingency ratings (see figure 10) are listed below.

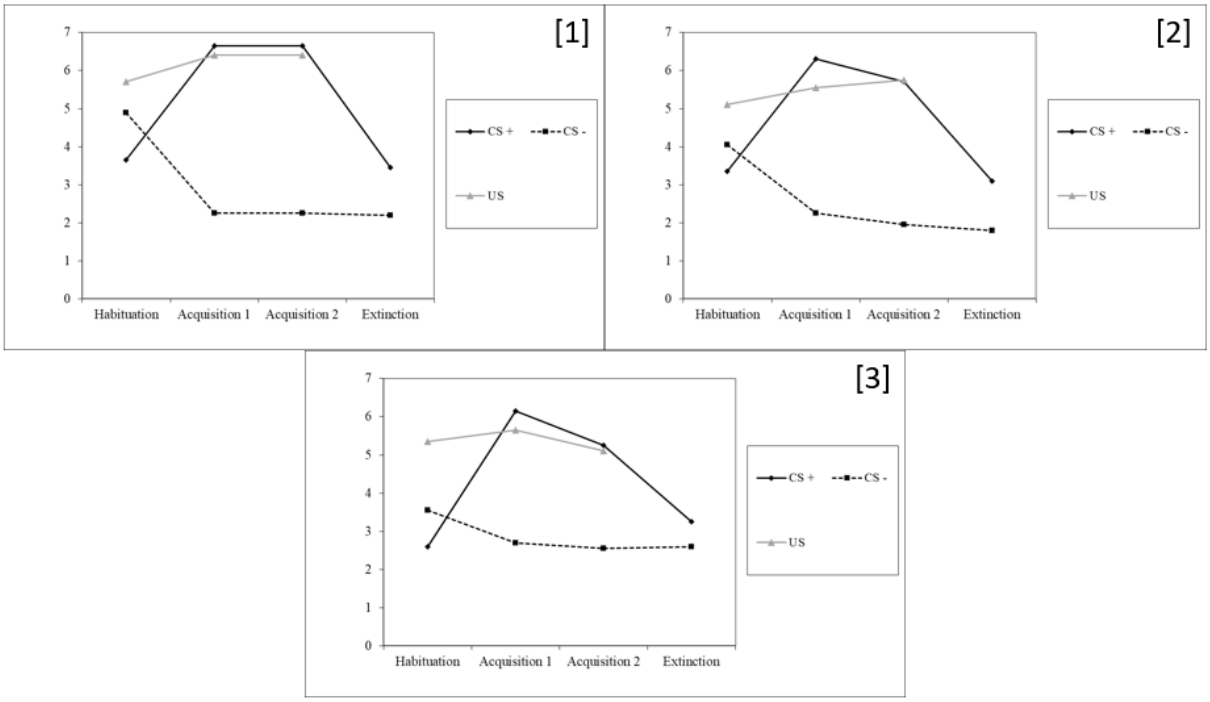

Figure 8. Study 1: Fear conditioning arousal ratings regarding each fear conditioning phase for [1] violent video gamers, [2] nonviolent video gamers and [3] non-gamers.

Table 8. Study 1: Pairwise comparisons for arousal ratings regarding mean difference between all used conditioning phases for the conditioning stimuli conditioned stimulus paired (CS+), conditioned stimulus unpaired (CS-) and unconditioned stimulus (US)

	CS+	CS-	US
Habituation - Acquisition 1	MD= -3.17***	MD= 1.77***	MD= -.48
Habituation - Acquisition 2	MD= -2.67***	MD= 1.92***	MD= -3.67
Habituation - Extinction	MD= -.07	MD= 1.97***	
Acquisition 1 - Acquisition 2	MD= .50	MD= .15	MD= .12
Acquisition 1 - Extinction	MD= 3.10***	MD= .20	
Acquisition 2 - Extinction	MD= 2.60***	MD= .05	

Note. N = 60, MD = mean difference
CS+ = conditioned stimulus paired; CS- = conditioned stimulus unpaired; US = unconditioned stimulus
*** = $p < .001$

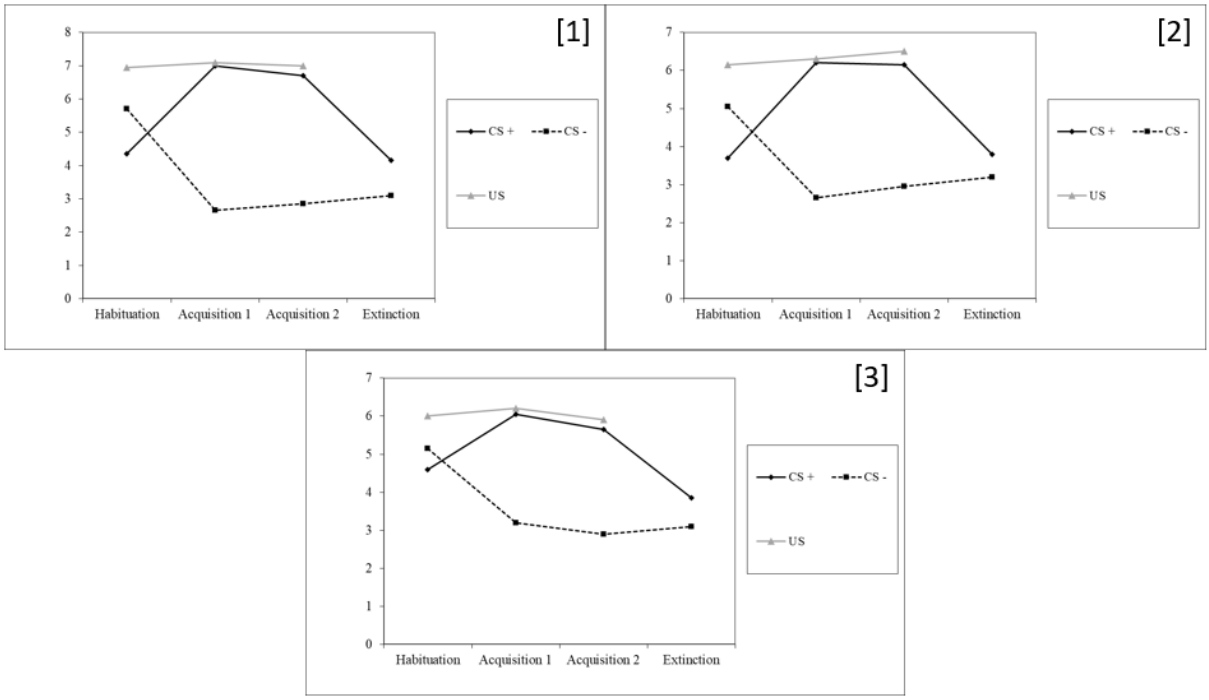

Figure 9. Study 1: Fear conditioning valence ratings regarding each fear conditioning phase for [1] violent video gamers, [2] nonviolent video gamers and [3] non-gamers.

Table 9. Study 1: Pairwise comparisons for valence ratings regarding mean difference between all used conditioning phases for the conditioning stimuli conditioned stimulus paired (CS+), conditioned stimulus unpaired (CS-) and unconditioned stimulus (US)

	CS+	CS-	US
Habituation - Acquisition 1	MD= -2.20***	MD= 2.47***	MD= -.19
Habituation - Acquisition 2	MD= -1.95***	MD= 2.40***	MD= -.13
Habituation - Extinction	MD= .28	MD= 2.17***	
Acquisition 1 - Acquisition 2	MD= .25	MD= -.07	MD= .06
Acquisition 1 - Extinction	MD= 2.48***	MD= -.30	
Acquisition 2 - Extinction	MD= 2.23***	MD= -.23	

Note. N = 60, MD = mean difference
CS+ = conditioned stimulus paired; CS- = conditioned stimulus unpaired; US = unconditioned stimulus
*** = $p < .001$

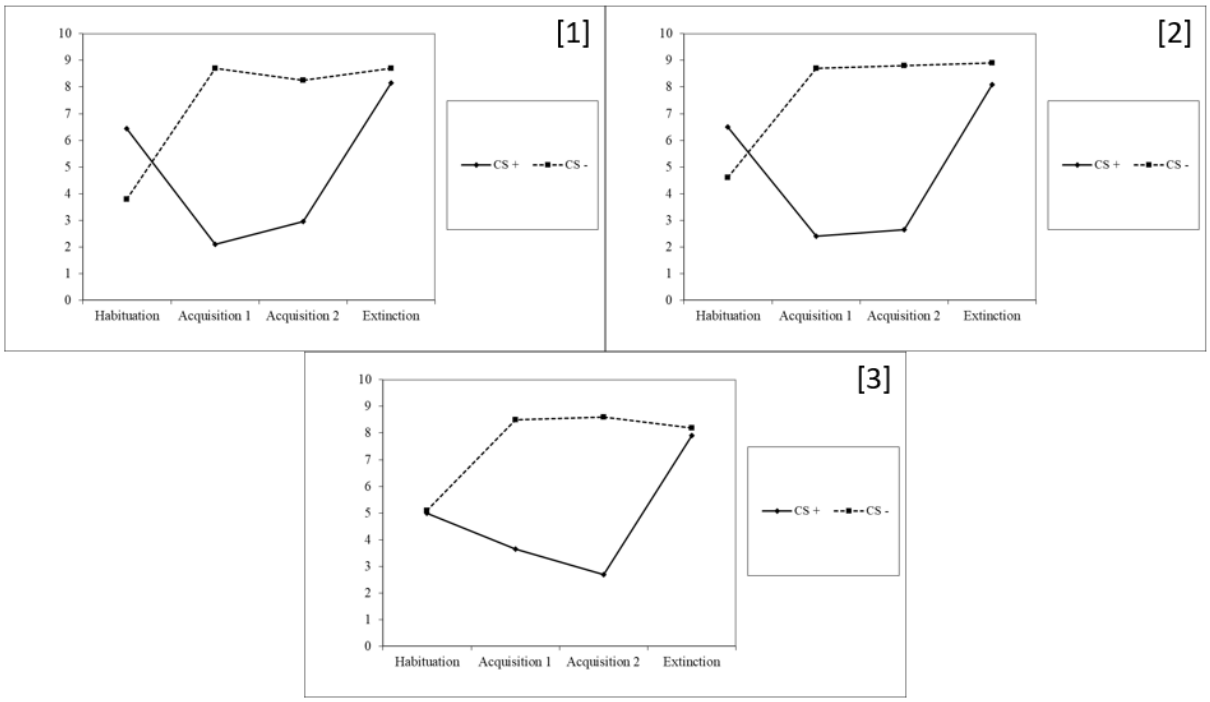

Figure 10. Study 1: Fear conditioning contingency ratings regarding each fear conditioning phase for [1] violent video gamers, [2] nonviolent video gamers and [3] non-gamers.

Table 10. Study 1: Pairwise comparisons for contingency ratings regarding mean difference between all used conditioning phases for the conditioning stimuli conditioned stimulus paired (CS+), conditioned stimulus unpaired (CS-) and unconditioned stimulus (US)

	CS+	CS-
Habituation - Acquisition 1	MD= 3.27***	MD= -4.13***
Habituation - Acquisition 2	MD= 3.22***	MD= -4.05***
Habituation - Extinction	MD= -2.07***	MD= -4.10***
Acquisition 1 - Acquisition 2	MD= -.05	MD= .08
Acquisition 1 - Extinction	MD= -5.33***	MD= .03
Acquisition 2 - Extinction	MD= -5.28***	MD= -.05

Note. N = 60, MD = mean difference
CS+ = conditioned stimulus paired; CS- = conditioned stimulus unpaired; US = unconditioned stimulus
*** = p < .001

3.1.6 Brain activation patterns for the fear conditioning paradigm

Because the conducted fear conditioning experiment used the same stimuli design for Acquisition phase 1 and 2, we combined these phases regarding fMRI data analysis. The most important contrast for fear conditioning is the difference in activation for the presentation of a conditioning cue that is paired with a painful stimulus (CS+c) minus the same conditioning cue but without it being paired with a painful stimulus (CS+uc). Therefore, we focused on this contrast for evaluating the fear conditioning task, because this contrast allowed us to gain insight on brain activation patterns for the conditioning design. For the combined phase of Acquisition 1 and Acquisition 2 (using the contrast CS+c minus Cs+uc) NG showed a significantly higher activation in the ACC, juxtapositional lobule cortex and paracingulate gyrus compared to VVG. Figure 11 shows brain activation patterns for each significantly active cluster regarding the respective design. Table 11 shows peak voxels (MNI coordinates), *t*-values and cluster size of brain areas that show significantly higher activations for NG compared to VVG on this contrast. We did not observe significant group differences in terms of respective brain activations regarding an inclusion of NVVG as one of the groups.

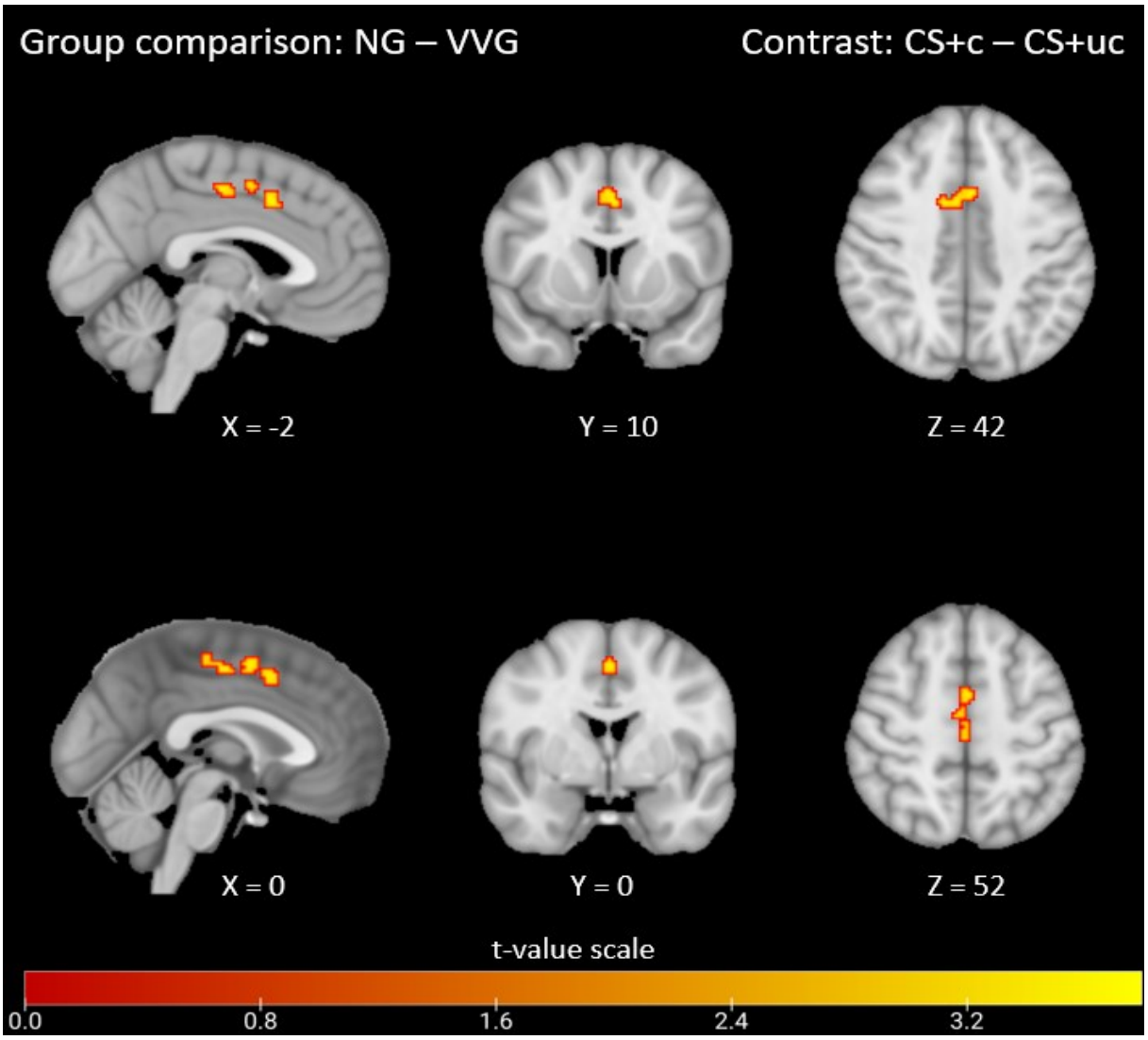

Figure 11. Study 1: Brain response contrast for the combined Acquisition phase between non-gamer (NG) minus violent video gamer (VVG) on the contrast CS+c minus CS+uc.

Table 11. Study 1: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significant higher activations for non-gamers (NG) compared to violent video gamers (VVG) on the contrast CS+c minus CS+uc

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Cingulate gyrus, anterior division	4	6	42	3.52	120
Paracingulate gyrus	-2	11	45	3.68	
Juxtapositional lobule cortex	4	4	56	3.18	95

Note. N = 60; N=20 per video gaming group.

Values in the CTQ subscale physical neglect correlated significantly negatively with activation in precuneus and intracalcarine cortex in the same contrast (CS+c minus CS+uc). Figure 12 shows brain activation patterns for each significantly active cluster regarding the respective design. This means that higher values on the CTQ subscale physical neglect were connected to lower brain activation on the conditioning contrast in the shown brain regions and vice versa. Table 12 displays peak voxels (MNI coordinates), *t*-values and cluster size of brain areas that show significant lower activation with increasing level of physical neglect.

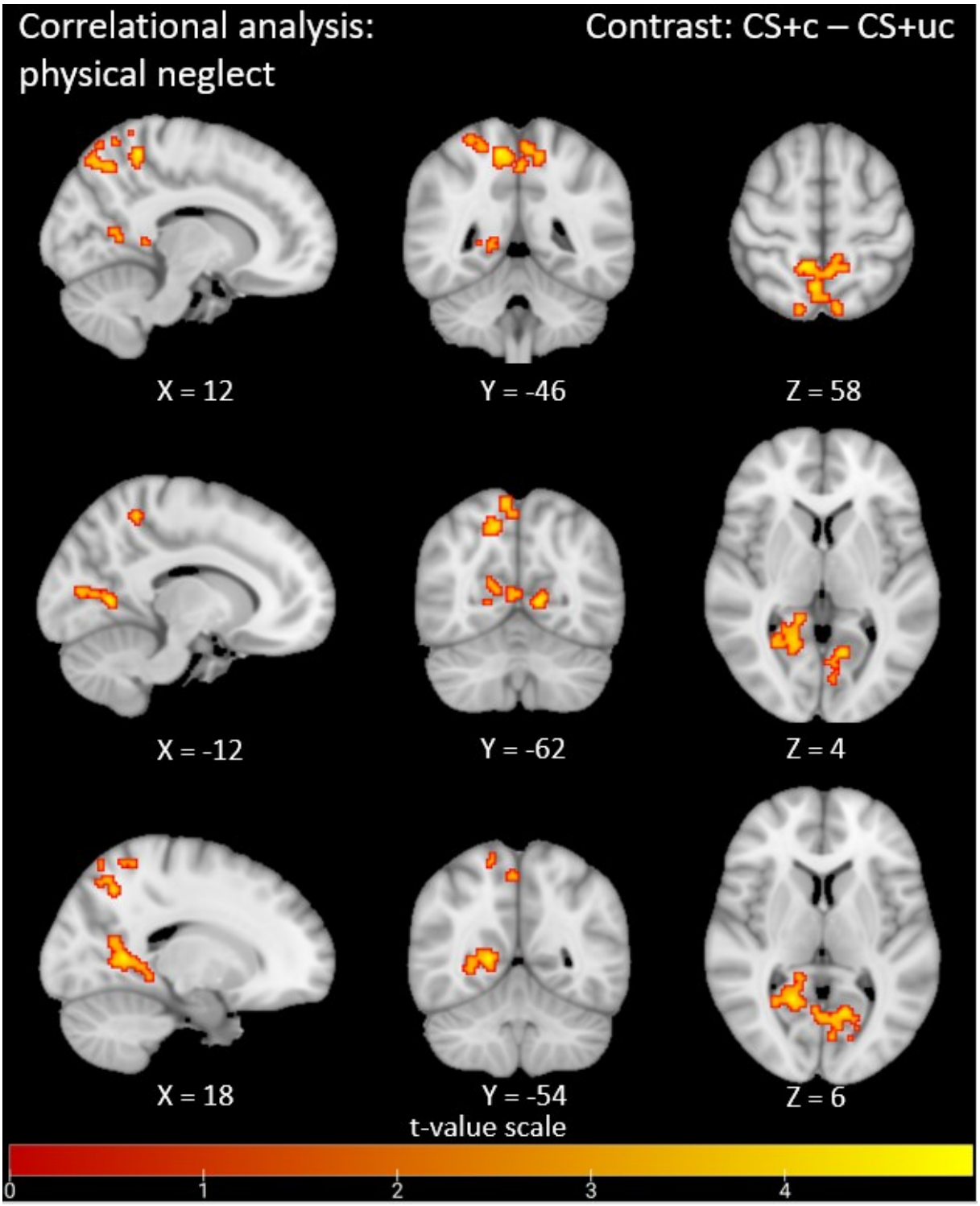

Figure 12. Study 1: Brain response contrast for the neural correlate in respect to the between-subject variance of the CTQ subscale physical neglect in the contrast CS+c minus CS+uc.

Table 12. Study 1: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significant lower activation with increasing level of physical neglect

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Precuneus	8	-46	54	4.42	696
Intracalcarine cortex	-8	-72	10	3.91	364
Precuneous cortex	20	54	8	4.31	286

Note. N = 60; N=20 per video gaming group.

In addition to these two analyses, we designed a combined analysis addressing the respective group comparison as well as ACE subscale influence of physical neglect on brain activation in respect to the contrast CS+c minus CS+uc. Therefore, we added physical neglect as covariate to the group comparison of NG minus VVG. Results still displayed significant brain activation patterns for ACC and supramarginal gyrus (posterior division). Figure 13 shows brain activation patterns for each significantly active cluster regarding the respective design. Table 13 visualizes peak voxels (MNI coordinates), *t*-values and cluster size of brain areas that show significant higher activation in the NG group compared to the VVG group even with physical neglect serving as a covariate.

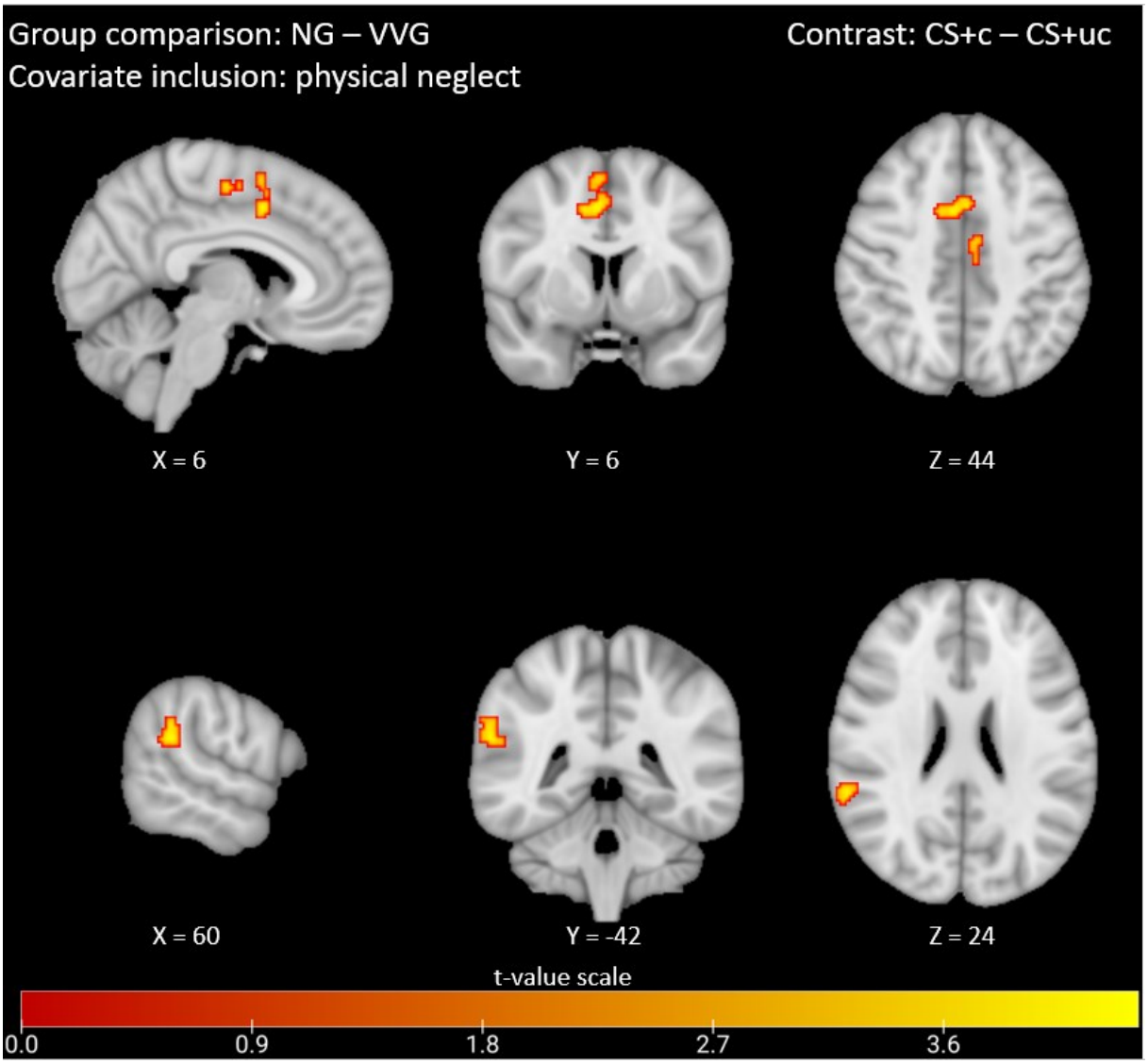

Figure 13. Study 1: Brain response of ACC and supramarginal gyrus for the contrast of non-gamers (NG) minus violent video gamers (VVG) with physical neglect as a covariate on the contrast CS+c minus CS+uc.

Table 13. Study 1: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significant higher activation in the non-gamer group (NG) compared to the violent video gamer group (VVG) even with physical neglect serving as a covariate

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Cingulate gyrus, anterior division	3	7	43	4.13	275
Supramarginal gyrus, posterior division	62	-42	22	3.74	94

Note. N = 40; N=20 per video gaming group.

Data for the fear conditioning phases habituation and extinction are not reported as the acquisition phases are the main interest for the conditioning hypotheses and no significant differences for the video gaming groups or CTQ subscales were observed. This is also true for the reporting of additional fear conditioning contrasts. As physical neglect showed the most interesting brain activation patterns and was the main interest in reporting pain measurement results, no further CTQ subscales are reported.

3.2 Study 2

3.2.1 Sample Characteristics

Neither gender (male vs female distribution; $\chi^2(2) = 3.95$, $p = .136$) nor ACE levels (ACE in the past vs no ACE in the past; $\chi^2(2) = 3.60$, $p = .165$) showed any significant difference across the observed gaming groups. Table 14 displays the distribution of CTQ subscales across the video gaming groups. We observed no significant differences for the CTQ subscales across the video gaming groups for emotional abuse ($F(2,57) = .51$, $p=.606$), physical abuse ($F(2,57) = 2.19$, $p=.121$), sexual abuse ($F(2,57) = .60$, $p=.555$), emotional neglect ($F(2,57) = .20$, $p=.821$) and physical neglect ($F(2,57) = .66$, $p=.519$).

Table 14. Study 2: Means and standard deviation of each individual CTQ subscale assessing ACE (emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect) for every video gaming group (violent video gamer (VVG), nonviolent video gamer

Video gaming group	VVG	NVVG	NG
Emotional abuse	13.00 (*SD*=6.72)	11.30 (*SD*=6.05)	11.30 (*SD*=5.71)
Physical abuse	9.45 (*SD*=6.40)	6.50 (*SD*=2.93)	7.45 (*SD*=3.56)
Sexual abuse	8.35 (*SD*=6.36)	6.70 (*SD*=3.50)	7.65 (*SD*=4.06)
Emotional neglect	13.55 (*SD*=7.53)	12.25 (*SD*=6.12)	13.05 (*SD*=6.00)
Physical neglect	9.40 (*SD*=5.79)	7.65 (*SD*=3.90)	8.40 (*SD*=4.58)

Note. N = 60; N=20 per video gaming group.
VVG = violent video gamer; NVVG = nonviolent video gamer; NG = non-gamer
SD = standard deviation

In addition to the distribution of CTQ subscales we also observed the mean ratings of every video gaming group for targeted electric stimulation intensity in the MRI as well

as pain threshold and pain tolerance of electric stimulation for the additional laboratory assessment. The results can be viewed in table 15.
Regarding the electric stimulation in the MRI data revealed significant video gaming group differences for reaching the targeted pain intensity rating of eight by participants (F(2,56) = 4.92, p = .011). VVG showed increased pain intensity ratings compared to NG (MD=5.20, 95%-CI[.98, 9.42], p = .012), but VVG did not reveal significantly increased pain intensity ratings compared to NVVG (MD=1.03, 95%-CI[-3.25, 5.30], p = .833) and NVVG did not display a significant difference in pain intensity ratings compared to NG (MD=4.17, 95%-CI[-.10, 8.45], p = .057). The data for electric stimulation in the laboratory also revealed significant differences in pain threshold across the video gaming groups (F(2,56) = 5.70, p = .006). VVG displayed increased pain thresholds compared to NG (MD=1.59, 95%-CI[.38, 2.80], p = .007) and NVVG (MD=1.28, 95%-CI[.08, 2.47], p = .034), but NVVG did not show a significant difference to NG (MD=.32, 95%-CI[-.89, 1.53], p = .803). Further, electric stimulation in the laboratory also revealed a significant pain tolerance differences across the video gaming groups (F(2,56) = 3.36, p = .042). VVG showed significantly increased pain tolerance compared to NG (MD=1.76, 95%-CI[.09, 3.43], p = .036) but not significantly increased pain tolerance compared to NVVG (MD=1.16, 95%-CI[-.49, 2.81], p = .215). NVVG also displayed no significantly increased pain tolerance compared to NG (MD=.60, 95%-CI[-1.07, 2.27], p = .664).

Table 15. Study 2: Means (M) and standard deviation (SD) for electric stimulation measurement (mA x10) in the MRI and the laboratory across the video gaming groups

Video gaming group	VVG	NVVG	NG
Electric stimulation pain threshold (laboratory)	M = 4.06 SD = 1.87	M = 2.78 SD = 1.36	M = 2.47 SD = 1.40
Electric stimulation pain tolerance (laboratory)	M = 5.93 SD = 2.23	M = 4.77 SD = 2.20	M = 4.17 SD = 2.05
Electric stimulation (MRI)	M = 11.32 SD = 5.59	M = 10.29 SD = 5.85	M = 6.12 SD = 5.18

Note. N = 60; N=20 per videogaming group.
VVG = violent video gamer; NVVG = nonviolent video gamer; NG = non-gamer

3.2.2 Behavioral results: Empathy for pain experiment (MRI + laboratory)

In contrast to the expected relation between video gaming behavior/ACEs and empathy for pain in others we did not detect a significant connection between these variables. In addition, we did not observe any significant differences in the identification of the correct emotion among the three video gaming groups for the empathy for pain experiment conducted in the MRI scanner. The MRI version of the experiment did not show any significant influence by the level of ACE as assessed by the CTQ subscales on correct emotion identification. Arousal, pleasantness/unpleasantness and suffering

ratings did significantly differ between the emotions except for the difference for arousal between the emotions fear and happy, indicating appropriate emotional distinction for the shown emotion-based facial expression stimuli. ANOVA results for this analysis can be seen in Appendix A.
The empathy for pain experiment conducted in the laboratory also did not show significant differences in emotion identification across the different used video gaming groups (VVG, NVVG, NG). However, post hoc tests in the form of a linear regression analysis regarding the influence of ACE levels (CTQ subscales) on emotion identification stimulus “surprise” revealed significant results. Increasing levels of several CTQ subscales were related to lower correct estimation of the emotion “surprise”. The CTQ subscale “emotional abuse” explained 8.5% of variance in the estimation of the emotion “surprise” ($F(1,56) = 5.22$, $p = .026$). The CTQ subscale “physical abuse” explained 16.2% ($F(1,56) = 10.85$, $p = .002$) and the CTQ subscale “physical neglect” explained 9.8% of variance ($F(1,56) = 6.10$, $p = .017$). Further results for the relation between these CTQ subscales and the emotion rating “surprise” can be seen in table 16 below.

Table 16. Study 2: Linear regression results for the influence of ACE levels in the form of CTQ subscales on emotion ratings of “surprise” used in the laboratory part of the empathy for pain experiment

	b	*SE(b)*	*t*	*p*
Emotional Abuse	-.34 [-.06; .00]	.02	-2.29	.026
Physical Abuse	-.06 [-.10; -.02]	.02	-3.30	.002
Physical Neglect	-.05 [-.08; -.01]	.02	-2.47	.017

Note. N = 60

The most frequent wrong classification of “surprise” was “fear”. 78.95% of wrong ratings on the emotion-stimuli “surprise” were rated as the emotion “fear”. Other wrong identifications were “happiness” (15.79%) and “anger” (5.26%).
Arousal, pleasantness/unpleasantness and suffering ratings differed significantly between most shown emotions, suggesting appropriate emotional distinction for the emotion-based facial expression stimuli. ANOVA results for this analysis can be seen in Appendix B.

3.2.3 MRI results: Empathy for pain experiment

Even though there was no significant difference regarding emotion-based stimuli ratings (arousal, pleasantness/unpleasantness, suffering) between video gaming groups or between different ACE levels in form of CTQ subscales for the empathy for pain experiment in the MRI scanner, we observed a significant brain activation increase for the entire sample in respect to higher CTQ subscale levels of physical abuse for fear stimuli via post hoc testing. Higher levels of physical abuse experience in the past correlated with more activation in superior frontal gyrus (see figure 14). Table 17 displays peak voxels (MNI coordinates), *t*-values and cluster size of the superior frontal gyrus that shows significantly higher activation with increasing levels of physical abuse. We did not observe any additional significant results for other emotions or video gaming group differences.

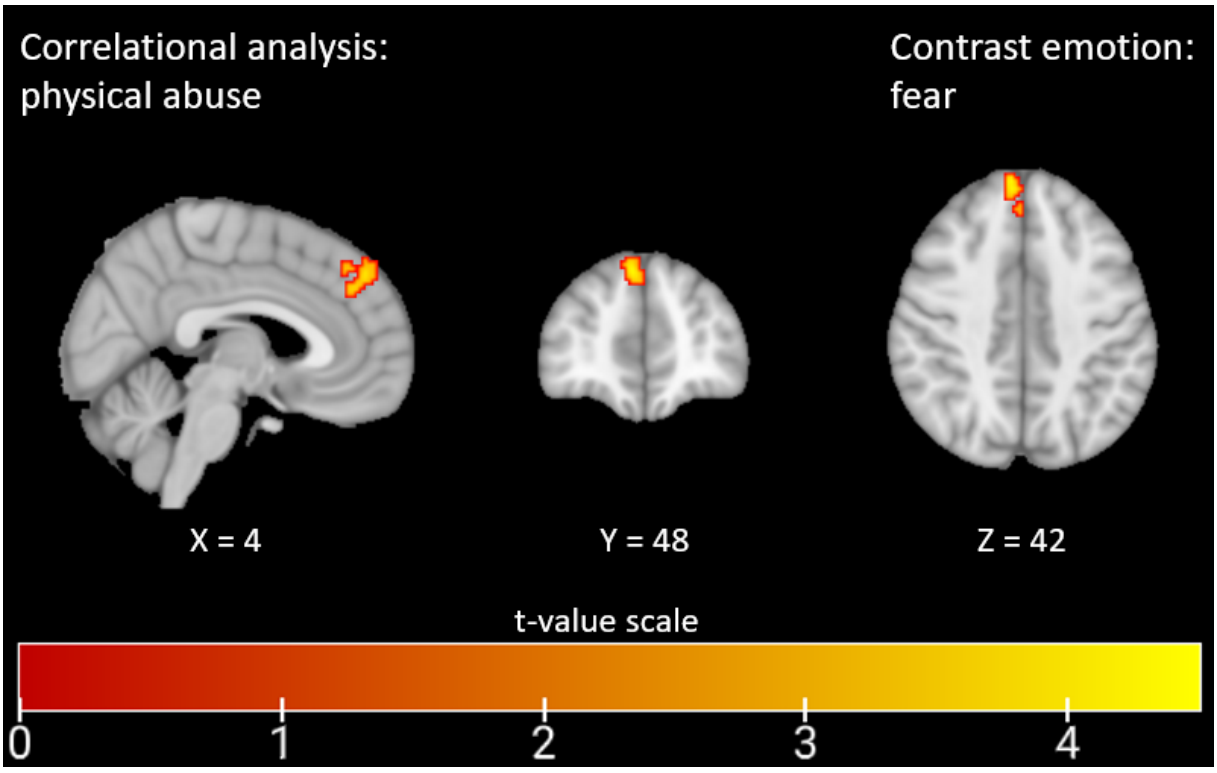

Figure 14. Study 2: Brain response increase of superior frontal gyrus with increasing levels of the CTQ subscale physical abuse for the emotion-based stimuli “fear” in the empathy for pain experiment regarding the whole sample.

Table 17. Study 2: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significantly higher activation with increasing level of physical abuse

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Superior frontal gyrus	4	48	42	4.12	142

Note. N = 60

3.2.4 Behavioral results: Pain perception experiment

Next, we computed ANOVAs to gain insights on rating differences for intensity and unpleasantness across the different pain stimulus frequencies (frequencies: 0.2 Hz, 1 Hz, 2 Hz). Regarding the stimulus type participants showed significant differences for intensity ratings across the used frequencies (F (1.57,92.63) = 56.39, $p < .001$). Isolated video gaming group evaluations showed significant intensity rating differences for NG (F (1.45,27.57) = 25.23, $p < .001$) for each pain stimulus frequency except for 1 Hz vs 2 Hz ($p = .078$), a significant intensity rating difference for NVVG (F (1.39,26.49) = 25.15, $p < .001$) and a significant intensity rating difference for VVG (F (2,38) = 9.62, $p < .001$). However, pairwise comparisons for VVG revealed that intensity rating differences of 0.2 Hz vs 1 Hz ($p = .059$) and 1 Hz vs 2 Hz ($p = .126$) were not significantly different. Post-hoc ANOVA results for intensity rating differences regarding electric pain stimuli frequencies can be seen in table 18 below.

Table 18. Study 2: Intensity ratings for all electric pain stimulus frequencies (0.2 Hz, 1 Hz, 2 Hz) for each video gaming group

Video gaming group	VVG	NVVG	NG
0.2 Hz	M = 54.67 SD = 35.85 CI[44.98, 64.36]	M = 46.72 SD = 32.80 CI[37.86, 55.58]	M = 52.66 SD = 32.13 CI[43.98, 61.35]
1 Hz	M = 60.19 SD = 38.76 CI[49.72, 70.66]	M = 54.50 SD = 27.89 CI[46.97, 62.03]	M = 61.99 SD = 30.98 CI[53.62, 70.36]
2 Hz	M = 63.85 SD = 39.55 CI[53.16, 74.54]	M = 60.33 SD = 26.29 CI[53.23, 67.43]	M = 66.18 SD = 31.51 CI[57.67, 74.70]
0.2 Hz vs 1 Hz	MD = -5.52 SD = 16.79 *P* = .059 CI[-11.21, .17]	MD = -7.78 SD = 13.86 *P* = .001 CI[-12.48, -3.08]	MD = -9.33 SD = 11.72 *P* < .001 CI[-13.30, -5.36]
0.2 Hz vs 2 Hz	MD = -9.18 SD = 18.67 *P* = .004 CI[-15.51, -2.85]	MD = -13.61 SD = 19.03 *P* < .001 CI[-20.06, -7.16]	MD = -13.52 SD = 19.13 *P* < .001 CI[-20.00, -7.04]
1 Hz vs 2 Hz	MD = -3.66 SD = 13.00 *P* = .126 CI[-8.07, .75]	MD = -5.83 SD = 10.65 *P* = .001 CI[-9.44, -2.22]	MD = -4.19 SD = 13.43 *P* = .078 CI[8.74,-.36]

In addition to intensity ratings, we also computed ANOVAs to compare stimulus type unpleasantness rating differences regarding all used electric pain stimuli frequencies. We detected significant differences for unpleasantness ratings for the entire sample of participants across the used frequencies (F (1.42,83.46) = 40.51, $p < .001$). Isolated

video gaming group evaluations displayed a significant unpleasantness rating difference for NG (F (2,38) = 15.72, $p < .001$) regarding each rating, a significant Greenhouse-Geisser-corrected unpleasantness rating difference for NVVG (F (1.21,22.90) = 21.96, $p < .001$) and a significant Greenhouse-Geisser-corrected unpleasantness rating difference for VVG (F (1.22,23.09) = 6.53, $p = .013$) except for 0.2 Hz vs 1 Hz ($p = .377$). Table 19 visualizes post-hoc ANOVA results for the electric pain stimuli frequency comparisons regarding the whole sample and each video gaming group.

Table 19. Study 2: Unpleasantness ratings across all used electric pain stimulus frequencies (0.2 Hz, 1 Hz, 2 Hz) for each video gaming group

Video gaming group	VVG	NVVG	NG
0.2 Hz	MD = 59.07 SD = 42.19 CI[47.67, 70.47]	MD = 47.42 SD = 32.78 CI[38.56, 59.28]	MD = 54.91 SD = 33.34 CI[45.90, 63.91]
1 Hz	MD = 63.26 SD = 39.11 CI[52.69, 73.83]	MD = 57.08 SD = 29.40 CI[49.14, 65.02]	MD = 61.11 SD = 29.43 CI[53.16, 69.07]
2 Hz	MD = 68.75 SD = 42.40 CI[57.29, 80.21]	MD = 64.00 SD = 30.73 CI[55.70, 72.30]	MD = 67.44 SD = 30.94 CI[59.08, 75.80]
0.2 Hz vs 1 Hz	MD = -4.19 SD = 20.27 *P* = .377 CI[-11.06, 2.68]	MD = -9.66 SD = 16.25 *P* = .001 CI[-15.17, -4.15]	MD = -6.21 SD = 15.10 *P* = .015 CI[-11.33, -1.09]
0.2 Hz vs 2 Hz	MD = -9.68 SD = 27.29 *P* = .038 CI[-18.93, -.43]	MD = -16.58 SD = 26.15 *P* < .001 CI[-25.44, -7.72]	MD = -12.54 SD = 19.23 *P* < .001 CI[-19.05, -6.02]
1 Hz vs 2 Hz	MD = -5.49 SD = 11.97 *P* = .006 CI[-9.55, -1.43]	MD = -6.92 SD = 13.75 *P* = .003 CI[-11.58, -2.26]	MD = -6.33 SD = 17.36 *P* = .033 CI[-12.21,-.44]

Stimulus type intensity and unpleasantness ratings for all three used frequencies revealed no significant difference in ratings between the video gaming groups. Additionally, ACE (CTQ subscales) did not show any significant influence on intensity or unpleasantness ratings for any of the three used electric pain stimulus frequencies across the entire sample. However, considering the group NG the CTQ subscale physical neglect predicted 52.2% of variance regarding unpleasantness ratings for the electric pain stimuli presented in a frequency of 2 Hz ($F(1,18) = 6.75$, $p = .018$). Higher values of physical neglect for NG were connected to higher unpleasantness ratings for the condition of electric pain stimuli presented in a frequency of 2 Hz ($\beta = 2.036$; t (18) = 2.60; $p = .018$). NVVG and VVG did not show a significant influence of physical neglect on unpleasantness ratings in this condition.

3.2.5 MRI results: Pain perception experiment

Video gaming group results:
First, we evaluated the MRI data in terms of video gaming group differences for brain activations. There was no significant difference in brain activation patterns between NG and NVVG and between NVVG and VVG for any main effect of pain stimulus frequencies or contrasts of pain stimulus frequencies. However, we observed significant differences between VVG and NG on the pain stimulus frequency of 2 Hz. VVG showed significantly stronger activation in superior frontal gyrus, lateral occipital cortex (superior division), juxtapositional lobule cortex, postcentral gyrus and precentral gyrus compared to NG. Figure 15 displays brain activation patterns for each significantly active cluster regarding the group comparison of VVG minus NG on the main effect of a pain stimulus frequency of 2 Hz. Table 20 shows peak voxels (MNI coordinates), *t*-values and cluster size of brain areas that show significantly higher activations for VVG compared to NG on the contrast of a pain stimulus frequency of 2 Hz.

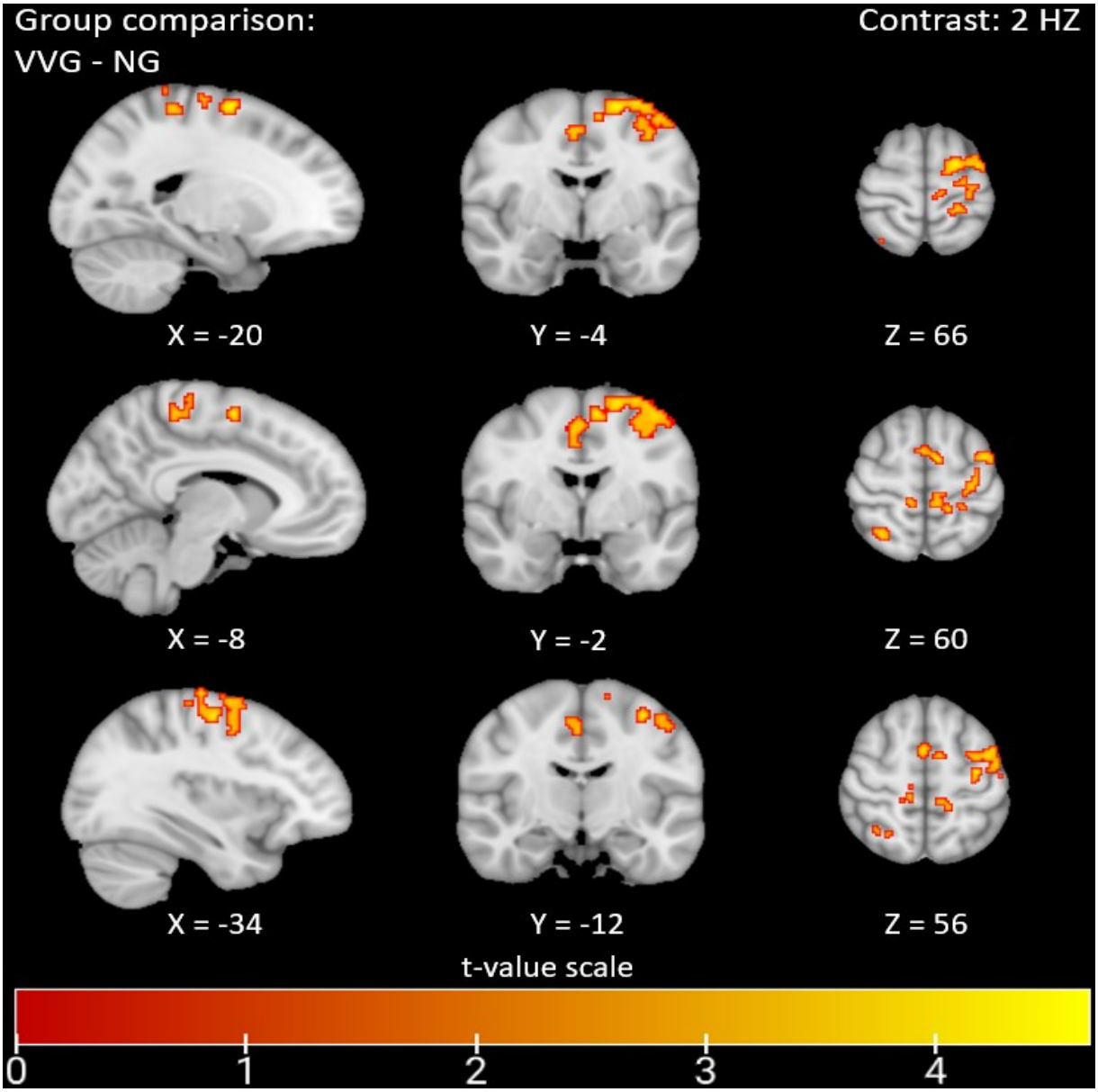

Figure 15. Study 2: Brain response contrast between violent video gamer (VVG) minus non-gamer (NG) on the pain stimulus frequency 2 HZ.

Table 20. Study 2: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significantly higher activation for violent video gamer (VVG) compared to non-gamer (NG)

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Superior frontal gyrus	-20	-4	68	4.02	417
Lateral occipital cortex, superior division	28	-62	64	3.31	310
Juxtapositional lobule cortex	4	0	54	3.82	240
Postcentral gyrus	-24	-36	66	3.62	180
Precentral gyrus	-46	-8	54	3.62	172

Note. N = 40; N=20 per video gaming group.

ACE level results:

In addition to videogaming group differences in brain activation we also checked for the connection of ACE in the form of CTQ subscales on pain perception regarding different pain stimulation frequencies. Physical abuse and physical neglect were significantly related to brain activations for a pain stimulus frequency of 1 Hz. Increasing levels of physical abuse were connected to significantly higher activation in middle frontal gyrus, superior frontal gyrus and frontal pole. Figure 16 visualizes the correlational analysis of physical abuse on the pain stimulus frequency of 1 Hz and table 21 displays peak voxels (MNI coordinates), *t*-values and cluster size of brain areas that show significant higher activation with increasing level of physical abuse.

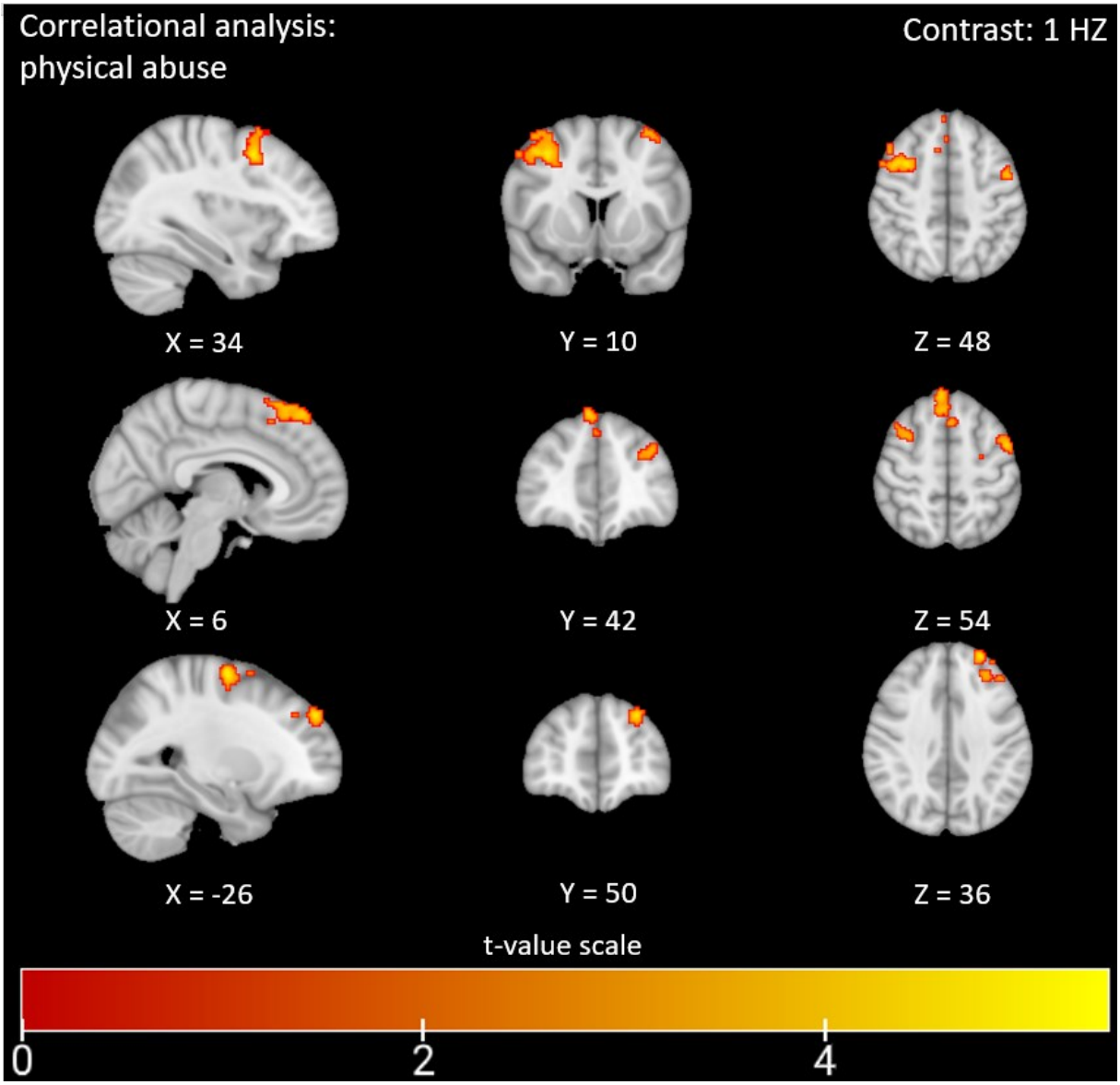

Figure 16. Study 2: Brain response contrast for the neural correlate in respect to the between-subject variance of the CTQ subscale physical abuse on the contrast of the pain stimulus frequency 1 HZ.

Table 21. Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significantly higher activation with increasing levels of physical abuse for the pain stimulus frequency 1 Hz

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Middle frontal gyrus	36	12	60	3.48	442
Superior frontal gyrus	6	30	58	4.19	305
Frontal pole	-24	52	34	4.83	262

Note. N = 59.

For ACE, we observed that the CTQ subscale physical neglect revealed significantly higher brain activation in the frontal pole with increasing levels of physical neglect in the pain stimulus frequency of 1 Hz (see figure 17). Table 22 visualizes peak voxels (MNI coordinates), *t*-values and cluster size of brain areas on this contrast for the entire sample of participants. We did not observe any other significant connection of other CTQ subscales with brain activations in the pain perception experiment.

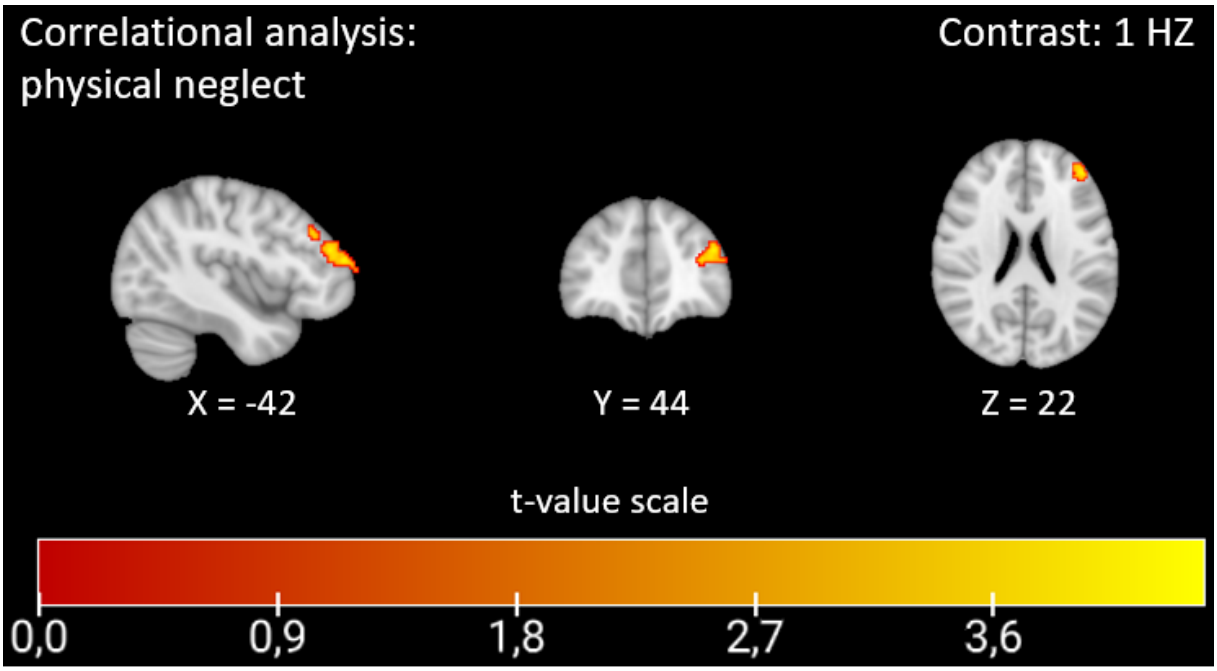

Figure 17. Study 2: Brain response contrast for the neural correlate in respect to the between-subject variance of the CTQ subscale physical neglect on the contrast of the pain stimulus frequency 1 HZ.

Table 22. Study 2: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significantly higher activation with increasing levels of physical neglect for the pain stimulus frequency 1 Hz

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Frontal pole	-42	44	22	4.39	331

Note. N = 59.

Video gaming group + ACE level results:
Additionally, an analysis for the interaction of group differences and ACE levels was conducted. Therefore, we added the CTQ subscales physical neglect and physical abuse as covariates to the group comparison of VVG minus NG. As we primarily wanted to gain more insight about the detected main effect of the VVG-NG contrast we designed no follow-up analysis for NVVG. We conducted one covariate analysis for physical abuse and one for physical neglect to detect any specific individual influence of the CTQ-subscales. Results displayed that physical abuse as covariate did not

change the brain activation difference between VVG minus NG significantly for most brain regions. The analysis, however, revealed significantly higher activation for the superior frontal gyrus, precentral gyrus and lateral occipital cortex for a pain stimulus frequency of 2 Hz. Figure 18 visualizes the significantly higher brain activation areas for VVG compared to NG for the pain stimulus frequency of 2 Hz. Table 23 displays peak voxels (MNI coordinates), *t*-values and cluster size of brain areas that show significantly higher activation in the VVG group compared to the NG group with physical abuse serving as a covariate.

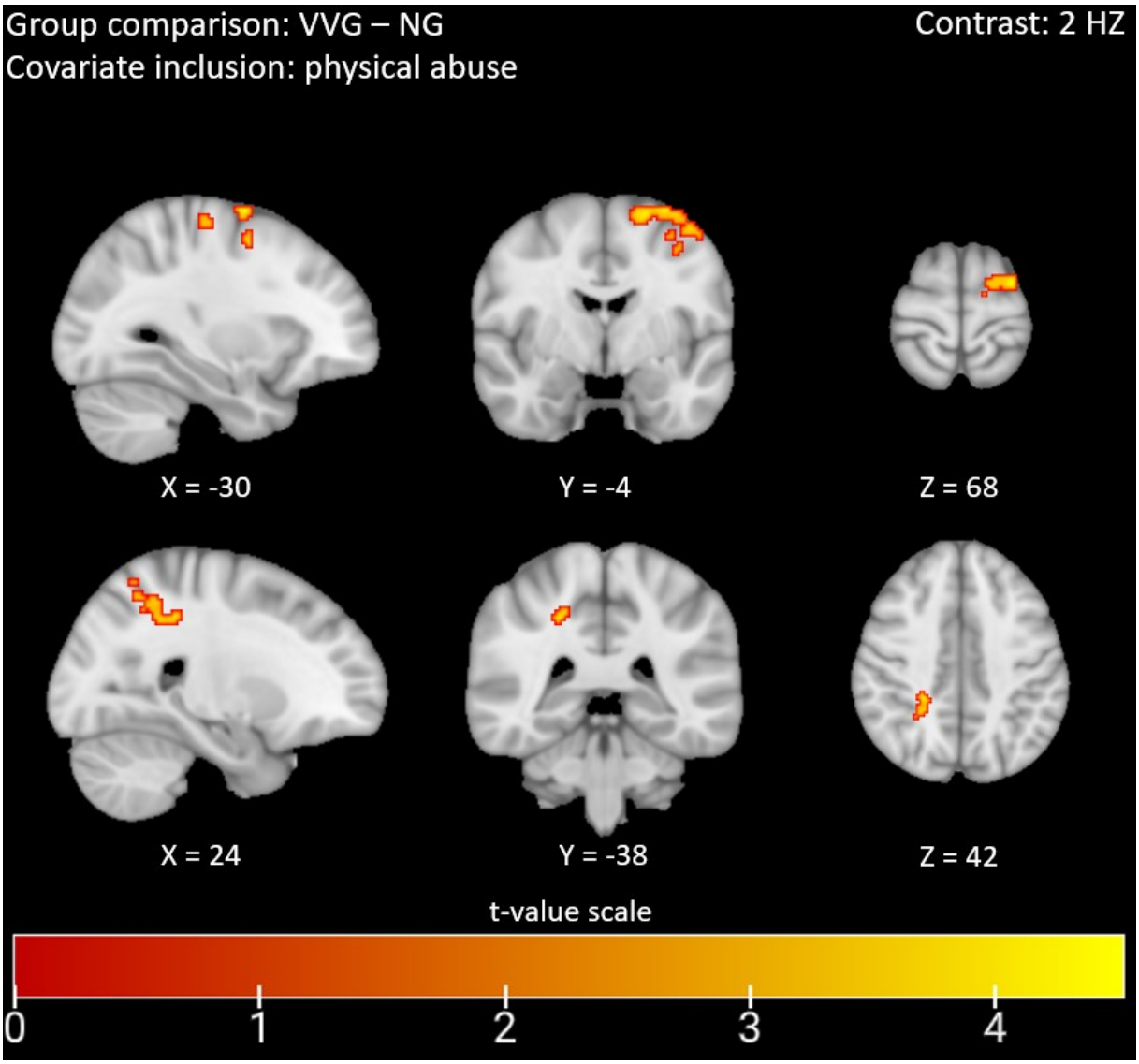

Figure 18. Study 2: Brain response for the contrast of violent video gamers (VVG) minus non-gamers (NG) with physical abuse as a covariate on the pain stimulus frequency of 2 Hz.

Table 23. Study 2: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significant higher activation in the violent video gamer group (VVG) compared to the non-gamer group (NG) even with physical abuse serving as a covariate

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Superior frontal gyrus	-20	-4	68	3.74	321
Precentral gyrus	-44	-8	56	3.35	
Lateral occipital cortex	31	-60	64	3.28	179

Note. N = 40; N=20 per video gaming group.

Next a covariate analysis with physical neglect serving as the covariate on the group comparison of VVG minus NG for the pain stimulus frequency of 2 Hz was conducted. Even with the inclusion of physical neglect as a covariate VVG still showed significantly higher activation in superior frontal gyrus, precentral gyrus, lateral occipital cortex and juxtapositional lobule cortex compared to NG. Figure 19 displays the significant higher brain activation areas for VVG compared to NG for the pain stimulus frequency of 2 Hz. Table 24 shows peak voxels (MNI coordinates), *t*-values and cluster size of brain areas that show significant higher activation in the VVG group compared to the NG group even with physical neglect serving as a covariate.

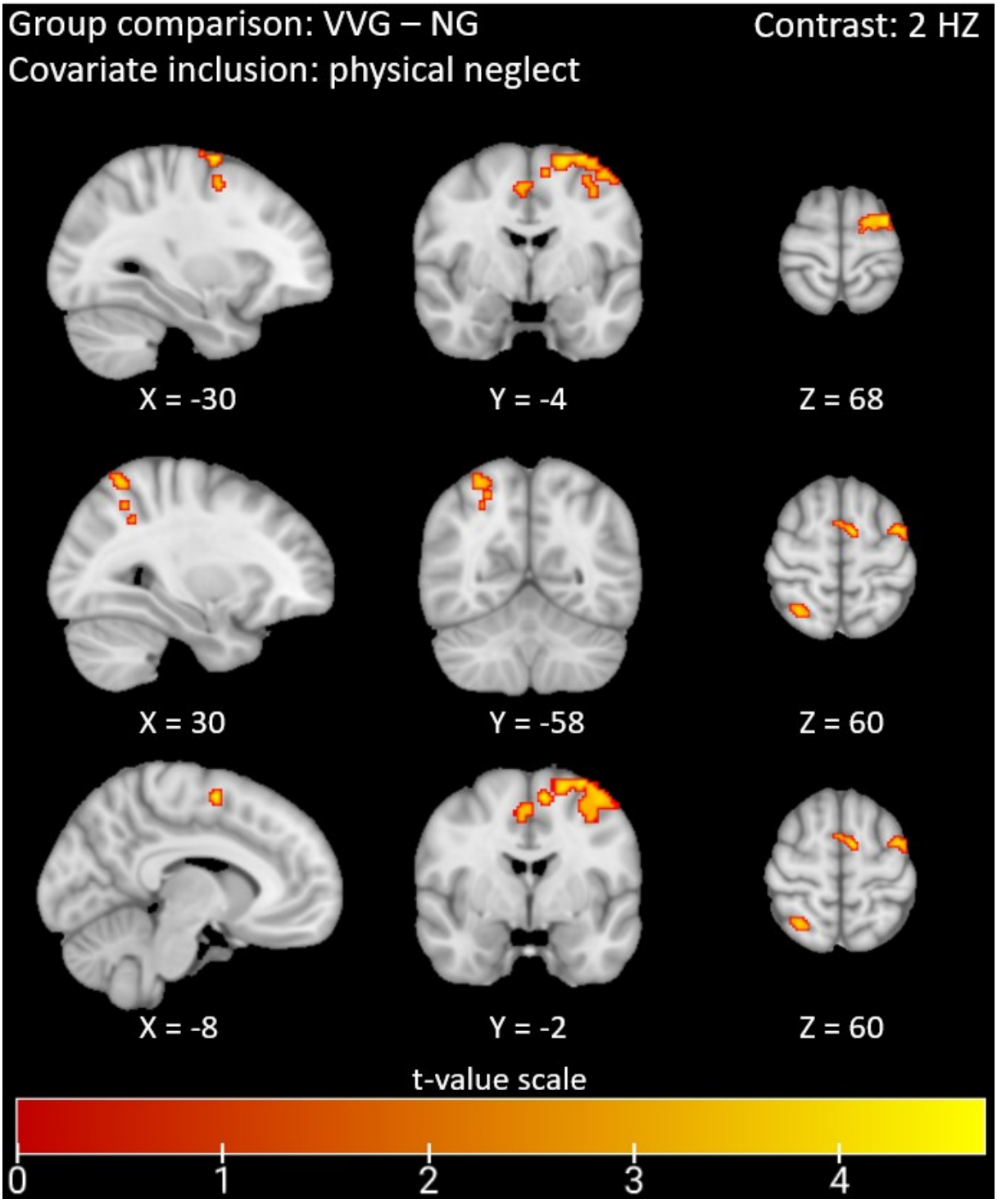

Figure 19. Study 2: Brain response for the contrast of violent video gamers (VVG) minus non-gamers (NG) with physical neglect as a covariate on the pain stimulus frequency of 2 Hz.

Table 24. Study 2: Peak voxels (MNI coordinates), t-values and cluster size of brain areas that show significant higher activation in the violent video gamer group (VVG) compared to the non-gamer group (NG) even with physical neglect serving as a co-variate

Brain areas	X (mm)	Y (mm)	Z (mm)	*t*-values	Cluster size voxels
Superior frontal gyrus	-16	-2	68	3.41	341
Precentral gyrus	-46	8	54	3.53	
Lateral occipital cortex	30	-62	64	3.24	183
Juxtapositional lobule cortex	4	0	54	3.67	166

Note. N = 40; N=20 per video gaming group.

3.2.6 Appendix: Supplementary analyses

Appendix A. Study 2: Mean (M), mean difference (MD), standard deviation (SD) and confidence intervals (CI) for arousal, pleasantness/unpleasantness and suffering regarding means of all used emotions in the empathy for pain experiment in the MRI scanner.

Emotion based stimuli mean comparisons	Arousal	Pleasantness/ Unpleasantness	Suffering
Pain	M = 4.88 SD = 1.51 CI[4.48, 5.27]	M = -2.08 SD = .82 CI[-2.29, -1.87]	M = -4.04 SD = 1.57 CI[-4.44, -3.63]
Fear	M = 3.78 SD = 1.15 CI[3.48, 4.07]	M = -1.28 SD = .49 CI[-1.41, -1.15]	M = -2.64 SD = 1.47 CI[-3.02, -2.26]
Happy	M = 3.43 SD = 1.24 CI[4.48, 5.27]	M = 1.71 SD = 1.04 CI[1.44, 1.98]	M = -.19 SD = .56 CI[-.33, -.04]
Neutral	M = 1.36 SD = 1.40 CI[.99, 1.72]	M = -.38 SD = .47 CI[-.50, -.25]	M = -1.05 SD = 1.23 CI[-1.73, -.73]

Pain - Fear	MD = 1.10 SD = 1.04 *P* < .001 CI[.73, 1.47]	MD = -.80 SD = .76 *P* < .001 CI[-1.07, -.54]	MD = -1.40 SD = 1.31 *P* < .001 CI[-1.86, -.93]
Pain - Happy	MD = 1.45 SD = 1.30 *P* < .001 CI[.99, 1.91]	MD = -3.79 SD = 1.47 *P* < .001 CI[-4.31, -3.27]	MD = -3.85 SD = 1.26 *P* < .001 CI[-4.43, -3.28]
Pain - Neutral	MD = 3.52 SD = 1.58 *P* < .001 CI[2.96, 4.08]	MD = -1.71 SD = .75 *P* < .001 CI[-1.97, -1.44]	MD = -2.99 SD = 1.43 *P* < .001 CI[-3.49, -2.48]
Fear - Happy	MD = .35 SD = 1.04 *P* = .070 CI[-.02, -.72]	MD = -2.99 SD = 1.19 *P* < .001 CI[-3.41, -2.57]	MD = -2.46 SD = 1.46 *P* < .001 CI[-2.97, -1.94]
Fear - Neutral	MD = 2.42 SD = 1.32 *P* < .001 CI[1.95, 2.88]	MD = -.91 SD = .53 *P* < .001 CI[-1.09, -.72]	MD = -1.59 SD = 1.42 *P* < .001 CI[-2.09, -1.09]
Happy - Neutral	MD = 2.07 SD = 1.53 *P* < .001 CI[1.53, 2.61]	MD = 2.08 SD = 1.21 *P* < .001 CI[1.66, 2.51]	MD = .87 SD = 1.33 *P* < .001 CI[.40, 1.33]

Note. N = 60

Appendix B. Study 2. Mean (M), mean difference (MD), standard deviation (SD) and confidence intervals (CI) for arousal, pleasantness/unpleasantness and suffering regarding means of all used emotions in the empathy for pain experiment in the laboratory

Emotion-based stimulus-mean comparisons	Arousal	Pleasantness/ Unpleasantness	Suffering
Anger	M = 4.01 SD = 1.27 CI[3.68, 4.34]	M = -1.51 SD = .68 CI[-1.69, -1.34]	M = -2.88 SD = 1.50 CI[-3.27, -2.50]
Sadness	M = 3.63 SD = 1.31 CI[3.28, 3.97]	M = -1.71 SD = 0.71 CI[-1.89, -1.52]	M = -3.64 SD = 1.56 CI[-4.04, -3.24]
Fear	M = 5.27 SD = 1.32 CI[4.92, 5.61]	M = -2.71 SD = .69 CI[-2.35, -1.99]	M = -4.32 SD = 1.70 CI[-4.76, -3.88]
Happiness	M = 4.37 SD = 1.36 CI[4.02, 4.72]	M = 2.51 SD = .78 CI[2.31, 2.72]	M = -.29 SD = .75 CI[-.49, -.10]
Neutral	M = 1.14 SD = 1.43 CI[.78, 1.51]	M = -0.13 SD = .39 CI[-.23, -.03]	M = -0.66 SD = 1.07 CI[-.94, -.38]
Pain	M = 5.23 SD = 1.19 CI[4.92, 5.54]	M = -2.39 SD = .69 CI[-2.57, -2.21]	M = -4.69 SD = 1.57 CI[-5.10, -4.29]
Disgust	M = 4.95 SD = 1.28 CI[4.62, 5.28]	M = -1.92 SD = .76 CI[-2.12, -1.72]	M = -3.53 SD = 1.73 CI[-3.97, -3.08]
Surprise	M = 4.18 SD = 1.20 CI[3.87, 4.49]	M = -0.16 SD = .82 CI[-.37, .05]	M = -1.64 SD = 1.43 CI[-2.01, -1.27]
Anger - Sadness	MD = .38 SD = .95 *P* = .078 CI[-.02, .79]	MD = .19 SD = .81 *P* = 1.00 CI[-.15, .54]	MD = .75 SD = 1.21 *P* < .001 CI[.24, 1.27]
Anger - Fear	MD = -1.26 SD = 1.21 *P* < .001 CI[-.02, .79]	MD = .66 SD = .72 *P* < .001 CI[.35, .96]	MD = 1.43 SD = 1.12 *P* < .001 CI[.96, 1.91]
Anger - Happiness	MD = -.36 SD = 1.26 *P* = .843 CI[-.89, 0.17]	MD = -4.03 SD = 1.23 *P* < .001 CI[-4.55, -3.51]	MD = -2.60 SD = 1.64 *P* < .001 CI[-3.28, -1.90

Anger - Neutral	MD = 2.87 SD = 1.51 *P* < .001 CI[2.23, 3.50]	MD = -1.38 SD = .75 *P* < .001 CI[-1.70, -1.06]	MD = -2.23 SD = 1.37 *P* < .001 CI[-2.80, -1.65]
Anger – Pain	MD = -1.22 SD = 1.23 *P* < .001 CI[-1.74, -.70]	MD = .87 SD = .68 *P* < .001 CI[.59, 1.16]	MD = 1.81 SD = 1.36 *P* < .001 CI[1.23, 2.39]
Anger – Disgust	MD = -.94 SD = 1.11 *P* < .001 CI[-1.41, -.47]	MD = .41 SD = .76 *P* = .004 CI[.08, .73]	MD = .64 SD = 1.65 *P* = .008 CI[.10, 1.19]
Anger – Surprise	MD = -.17 SD = 1.08 *P* = 1.00 CI[-.63, .28]	MD = -1.35 SD = 1.05 *P* < .001 CI[-1.80, -.91]	MD = -1.25 SD = 1.38 *P* < .001 CI[-1.83, -.66]
Sadness – Fear	MD = -1.64 SD = .89 *P* < .001 CI[-2.02, -1.26]	MD = .47 SD = .74 *P* < .001 CI[.15, .78]	MD = .68 SD = 1.08 *P* < .001 CI[.22, 1.14]
Sadness – Happiness	MD = -.75 SD = 1.28 *P* = .001 CI[-1.29, -.20]	MD = -4.22 SD = 1.25 *P* < .001 CI[-4.75, -3.69]	MD = -3.35 SD = 1.68 *P* < .001 CI[-4.06, -2.63]
Sadness – Neutral	MD = 2.48 SD = 1.51 *P* < .001 CI[1.84, 3.12]	MD = -1.58 SD = .71 *P* < .001 CI[-1.88, -1.27]	MD = -2.98 SD = 1.35 *P* < .001 CI[-3.55, -2.41]
Sadness – Pain	MD = -1.61 SD = 1.06 *P* < .001 CI[-2.06, -1.16]	MD = .68 SD = .78 *P* < .001 CI[.35, 1.01]	MD = 1.06 SD = 1.21 *P* < .001 CI[.55, 1.57]
Sadness – Disgust	MD = -1.33 SD = 1.08 *P* < .001 CI[-1.79, -0.87]	MD = .21 SD = .74 *P* = .825 CI[-.10, .53]	MD = -.11 SD = 1.21 *P* = 1.00 CI[-.62, .40]
Sadness – Surprise	MD = -.56 SD = 1.17 *P* = .014 CI[-1.05, -0.06]	MD = -1.55 SD = 1.04 *P* < .001 CI[-1.99, -1.10]	MD = -2.00 SD = 1.43 *P* < .001 CI[-2.60, -1.40]
Fear – Happiness	MD =.90 SD = 1.35 *P* < .001 CI[.32, 1.47]	MD = -4.69 SD = 1.19 *P* < .001 CI[-5.19, -4.18]	MD = -4.02 SD = 1.82 *P* < .001 CI[-4.80, -3.25]

Fear – Neutral	MD = 4.12 SD = 1.66 *P* < .001 CI[3.42, 4.82]	MD = -2.04 SD = .76 *P* < .001 CI[-2.36, -1.72]	MD = -3.66 SD = 1.59 *P* < .001 CI[-4.33, -2.99]
Fear – Pain	MD =.03 SD = .87 *P* = 1.00 CI[-.33, .40]	MD = .22 SD = .72 *P* = .678 CI[-.09, .52]	MD = .38 SD = 1.20 *P* = .506 CI[-.13, .89]
Fear – Disgust	MD =.32 SD = .95 *P* = .358 CI[-.09, .72]	MD = -.25 SD = .72 *P* = .253 CI[-.56, .05]	MD = -.79 SD = 1.19 *P* < .001 CI[-1.30, -.29]
Fear – Surprise	MD = 1.08 SD = 1.06 *P* < .001 CI[.64, 1.53]	MD = -2.01 SD = 1.05 *P* < .001 CI[-2.46, -1.56]	MD = -2.68 SD = 1.31 *P* < .001 CI[-3.23, -2.12]
Happiness – Neutral	MD = 3.23 SD = 1.66 *P* < .001 CI[2.52, 3.93]	MD = 2.64 SD = .85 *P* < .001 CI[2.28, 3.00]	MD = .37 SD = 1.31 *P* = .42 CI[-.11, .84]
Happiness – Pain	MD = -.86 SD = 1.33 *P* < .001 CI[-1.43, -.30]	MD = 4.90 SD = 1.23 *P* < .001 CI[4.38, 5.42]	MD = 4.40 SD = 1.83 *P* < .001 CI[3.63, 5.18]
Happiness – Disgust	MD = -.58 SD = 1.07 *P* = .003 CI[-1.04, -.12]	MD = 4.44 SD = 1.33 *P* < .001 CI[3.87, 5.00]	MD = 3.23 SD = 1.88 *P* < .001 CI[2.44, 4.03]
Happiness – Surprise	MD = .19 SD = 1.23 *P* = 1.00 CI[-.33, .71]	MD = 2.68 SD = 1.18 *P* < .001 CI[2.18, 3.18]	MD = 1.35 SD = 1.52 *P* < .001 CI[.70, 1.99]
Neutral – Pain	MD = -4.09 SD = 1.56 *P* < .001 CI[-4.75, -3.43]	MD = 2.26 SD = .78 *P* < .001 CI[1.93, 2.59]	MD = 4.04 SD = 1.62 *P* < .001 CI[3.35, 4.72]
Neutral – Disgust	MD = -3.81 SD = 1.41 *P* < .001 CI[-4.40, -3.21]	MD = 1.79 SD = .86 *P* < .001 CI[1.42, 2.16]	MD = 2.87 SD = 1.59 *P* < .001 CI[2.19, 3.54]
Neutral – Surprise	MD = -3.04 SD = 1.51 *P* < .001 CI[-3.68, -2.40]	MD = .03 SD = .83 *P* = 1.00 CI[-.32, 0.38]	MD = .98 SD = 1.21 *P* < .001 CI[0.47, 1.49]

Pain – Disgust	MD =.28 SD = 1.00 *P* = .945 CI[-.14, .71]	MD = -.47 SD = .68 *P* < .001 CI[-.76, -.18]	MD = -1.17 SD = 1.32 *P* < .001 CI[-1.73, -.61]
Pain – Surprise	MD = 1.05 SD = 1.03 *P* < .001 CI[-.14, .71]	MD = -2.23 SD = .92 *P* < .001 CI[-2.68, -1.83]	MD = -3.06 SD = 1.47 *P* < .001 CI[-3.68, -2.43]
Disgust - Surprise	MD = .77 SD = .80 *P* < .001 CI[0.43, 1.11]	MD = -1.76 SD = 1.16 *P* < .001 CI[-2.25, -1.27]	MD = -1.89 SD = 1.32 *P* < .001 CI[-2.45, -1.33]

Note. N = 60

4 DISCUSSION

4.1 Summary of findings

The purpose of this dissertation was to gain insights on how violent video gaming and ACEs interact with fear conditioning, pain-related empathy as well as pain perception. We conceptualized and conducted an empirical study to research on these interactional patterns and divided the executed experiments into two studies. In Study 1 we focused on the interaction of violent video gaming and ACEs on fear conditioning as well as pain sensitivity. Study 2 investigated the interactive pattern of violent video gaming and ACEs on pain-related empathy as well as pain perception.
Study 1 revealed an increased pain threshold and pain tolerance for VVGs across all conducted pain tests (electric stimulation, temperature-based stimulation and pressure pain stimuli) compared to NGs. This finding supports parts of hypothesis 1.1. stating that VVGs display a significantly decreased pain sensitivity compared to NGs but does not support the aspect that VVGs also show this difference compared to NVVGs. Additionally, we observed support for hypothesis 1.2. as well, meaning ACEs serve as a moderator variable on the connection between video gaming behavior and pain sensitivity for the different conducted pain tests. In terms of the fear conditioning experiment conducted in the MRI scanner we observed higher brain activity in limbic areas for NGs compared to VVGs supporting parts of Hypothesis 1.3. However, we did not observe higher brain activation patterns for NVVGs compared to VVGs for the fear conditioning experiment. Higher values in the ACE subscale physical neglect were connected to lower brain activation patterns in precuneus, intracalcarine cortex and precuneous cortex regarding the fear conditioning experiment providing support for hypothesis 1.4.
Although we expected a significant difference for emotion recognition regarding different video gaming groups in hypothesis 2.1. Study 2 displayed no significant difference between violent gaming groups regarding the ability to recognize pain-related emotions in the empathy for pain experiment correctly. In addition, video gaming groups did not show significant differences in reaction to different frequencies of pain stimuli in the pain perception experiment. Even though we did not obtain significant results for hypothesis 2.1. we observed significant results for hypothesis 2.2 but in the opposite way of the hypothesized effect direction we expected. VVGs did show significantly higher brain activation in superior frontal gyrus, lateral occipital cortex (superior division), juxtapositional lobule cortex, postcentral gyrus and precentral gyrus compared to NGs in the pain perception experiment regarding the highest used pain stimuli frequency. In contrast, the empathy for pain experiment did not display any video gaming group differences contrary to expectations from hypothesis 2.2. Hypothesis 2.3. stated that ACE levels in the form of CTQ subscales can influence the correct identification of pain-related emotions in the empathy for pain experiment and significantly increase the reaction to painful stimuli in the pain perception experiment. Our data support the hypothesis regarding the empathy for pain experiment for several ACE subscales and higher levels of ACE subscales were also connected to higher unpleasantness ratings in the pain perception experiment. Our data also supports hypothesis 2.4. regarding increasing brain activation patterns for rising values of ACEs in the form of CTQ subscales for pain-related emotions in the empathy for pain experiment and for painful stimuli on different stimuli frequencies regarding the pain-processing experiment.

Different ACE subscales did display higher brain activation patterns across the empathy for pain experiment and the pain perception experiment as suggested.
Overall different video gaming groups as well as different values of ACEs are connected to different values in almost all our conducted experiments. Only values in the empathy for pain experiment did not seem to vary with different video gaming groups. Next a detailed interpretation of findings across both conducted studies is listed.

4.2 Interpretation of findings

4.2.1 Violent video gaming

We conducted several pain tests to gain more insight on how video gaming habits correlate with pain sensitivity in terms of pain threshold and pain tolerance among participants. Our data suggests that VVG show significantly higher pain threshold and pain tolerance for all these pain tests compared to NGs. These findings are in line with previous research that suggested lower pain sensitivity and desensitization among VVG (Anderson et al., 2010b; Bushman & Anderson, 2009; Carnagey et al., 2007; Miedzobrodzka et al., 2022). However, we did not observe significant pain threshold or pain tolerance differences between VVG and NVVG for most conducted pain tests unlike suggested by previous research (Stephens & Allsop, 2012; Teismann et al., 2014). Teismann et al. (2014) researched on short-term effects of violent and nonviolent videogaming behavior while our study focused on long-term effects as they assigned participants either to a racing game group or to a violent video gaming group and obtained results for a cold pressor measurement after the gaming scenario. It can be assumed that the difference in pain sensitivity for VVG compared to NVVG is stronger for a short-term experimental condition than long-term experimental design. Although there seems to be a trend effect as values for pain threshold and pain tolerance in our study were always located between values for NG and VVG. A effect of VVG on pain sensitivity compared to other video gaming habits is also confirmed by recent research on this topic (Förtsch et al., 2021). Self-executed media violence seems to play a big role in pain sensitivity and distorted pain perception for a variety of pain stimuli. From past research we know that permanent central nervous changes in pain sensitivity can occur through model learning, sensibilization, classic and operant conditioning or priming and lead to a chronification of pain (Flor, 2011). A possible interpretation could be that violent video gaming also works through some of these learning mechanisms but in an opposite way and favors a desensitization to pain stimuli. If so, it could be interesting to see if violent video gaming could help chronic pain patients to desensitize regarding their pain sensitivity and regulate it down to a more comfortable level. However, our research also focused on different pain stimuli frequencies in order to detect possible pain perception variations for this condition.
In order to acquire data for video gaming group differences for different pain stimuli frequencies we conducted the pain perception experiment in the MRI scanner. Video gaming groups did not show significant differences for ratings of unpleasantness or intensity for each used frequency, but VVG were the only video gaming group that did not reveal significant rating differences for unpleasantness and intensity of the electric pain stimuli between each of the used pain stimuli frequencies. In contrast NG and NVVG did show significant rating differences between every used pain stimuli

frequency for unpleasantness as well as intensity ratings. These findings that VVGs show less discriminability between different used pain stimuli frequencies can be interpreted in line with previous research suggesting impaired pain sensitivity among VVGs (Anderson et al., 2010; Bushman & Anderson, 2009; Teismann et al., 2014). Behavioral data of the pain perception experiment display this impaired pain sensitivity among VVG especially for lower frequencies of pain stimuli.
The MRI data obtained for the pain perception experiment in the scanner also revealed significant differences for VVG compared to other video gaming groups. VVG displayed significantly higher activation in superior frontal gyrus, lateral occipital cortex, juxtapositional lobule cortex, postcentral gyrus and precentral gyrus for a pain stimulus frequency of 2 Hz compared to NG which suggests higher somatosensory and somatomotor activity among VVG for high frequency pain stimuli. The higher brain activation patterns likely arise because of the different temporal summation induced via the experimental design. Temporal summation refers to a concept that repeated and equal-intensity noxious stimuli when presented at a high frequency lead to an increased pain experience. Previous research data regarding similar MRI designs revealed controversial results. There is evidence that neural desensitization or brain activation change is unusual for VVG recorded via event-related potentials (Goodson et al., 2021). On the other hand results of studies using multi-voxel pattern analysis suggest a clear distinction between VVG and healthy controls with 92.37% accuracy (Wang et al., 2022). We believe that our results for the pain perception experiment, as well as for the other MRI experiments described below, can shed light on some of these disparate findings from previous research on this topic.
Another MRI paradigm conducted in the scanner was the fear conditioning experiment. NG displayed higher activation in anterior cingulate cortex, paracingulate gyrus and juxtapositional lobule cortex than VVG regarding the conditioned stimuli in the acquisition phase of the experiment. The significant difference in activation relates to the anterior cingulate cortex, which is a part of the limbic system and is often identified as an important brain area in research on video gaming behavior when comparing VVG, NVVG, and NG. Attention, cognitive control and visuospatial skills represent factors that are often influenced by internet gaming disorder or VVG (Montag et al., 2012; Palaus et al., 2017; Y. Wang et al., 2009). However, this difference in brain activation patterns occurred only for the acquisition phase and not in any other used fear conditioning phase which underpins the assumption that especially the conditioning displays video gaming group differences and not mainly the stimuli reaction itself. This difference for fear conditioning towards a painful stimulus only resides between NG and VVG and is not occurrent for the comparison between NG and NVVG or NVVG and VVG. It seems that violent content in videogames does truly favor alter response to fearful stimuli affecting the individual body via painful stimulation but this difference is only significant when compared to NG and not NVVG.
In addition to the hypothesis how violent video gaming affects response to individual pain, we also wanted to research on how violent video gaming can affect how we see others suffering from pain and how we perceive pain-related emotions in others. Research so far about how emotional content influences different video gaming groups is not fully coherent to this point. Different MRI studies detected significantly reduced activation for VVG compared to NG for the limbic system including anterior and posterior cingulate cortex, amygdala, thalamus, posterior and superior parietal lobe, hippocampus, cerebellum, left lateral medial frontal lobe and entorhinal cortex regarding presentation of emotional content (Montag et al., 2012; Palaus et al., 2017; Wang et al., 2009). On the other hand, drift diffusion modelling studies have not revealed any significant difference for video gaming groups (Pichon et al., 2021). Studies focusing

on amplitudes of low-frequency fluctuations and fractional amplitudes of low-frequency fluctuations have also not detected differences in empathy skills among video gaming groups (Pan et al., 2018). We did not observe any difference between VVG, NVVG and NG for the empathy for pain experiment in our study. These findings are also contrary to findings from prior studies that suggested significantly more desensitization and habituation among VVG compared to NVVG (Miedzobrodzka et al., 2022). It appears that violent video gaming in the empathy for pain task does not affect the skill to distinguish between emotional expressions originating from various emotional cues. In addition, intensity, suffering and unpleasantness/pleasantness ratings as well as brain activation patterns did not differ between VVG, NVVG or NG. It is uncommon to be unable to observe an influence of violent video gaming on empathy for pain variables as different research from the past suggests similar brain regions that are active for violent video gaming and empathy for pain tasks like the anterior cingulate gyrus. In addition research also suggests regions that show more activation for empathy for pain like fusiform gyrus, anterior central gyrus and insula and regions that show more activation for violent video gaming like hippocampus, cerebellum, thalamus, amygdala, entorhinal cortex and posterior and superior parietal lobe, but our data doesn't support such conclusions (Fallon et al., 2020; Li & Wang, 2021; Montag et al., 2012; Palaus et al., 2017; Pan et al., 2018; Xiong et al., 2019).
Even though the empathy for pain experiment did not yield any differences for the video gaming groups, emotion ratings and brain activity patterns were affected by different levels of ACE which will be described in the next section. Overall violent video gaming did display as a moderate factor in the performance of the different conducted pain tests, the pain perception experiment and the fear conditioning experiment underpinning its important role in research for pain related research questions. Many therapy approaches consider pain sensitivity hence why it could also be important to examine violent video gaming for potential therapy interventions. In addition, violent video gaming also showed interesting interactional effects with different levels of ACE in the form of CTQ subscales described further in section 4.2.3.

4.2.2 Adverse childhood experiences

Based on the data we have acquired via the various pain tests included in Study 1, we detected a clear influence of ACEs on pain threshold and pain tolerance. Models including all ACE subtypes defined in the CTQ revealed a significant correlation of pain threshold for the cold pressor test measurements and the levels of ACE subscales. Research in the past also support these findings somewhat as they revealed that participants with ACE background tend to experience more pain, regardless of other risk factors like sociodemographic characteristics or anxiety and depression symptoms (MacDonald et al., 2021). Emotional neglect served as the highest predictor among CTQ subscales for pain sensitivity in the cold pressor test for our study. Previous studies revealed especially an influence of emotional abuse for temperature-based pain stimulation like heat pain (Pieritz et al., 2015). Overall, many different CTQ subscales revealed influence regarding our study parameters which will be discussed for the respective experiments below. Past research revealed that many mental disorders can affect pain sensitivity. PTBS, bipolar disorders, schizophrenia, eating disorders, anxiety, substance use disorders and depression revealed a connection to pain sensitivity. However, research also reveals that these connections are present in a bidirectional way (Hooten, 2016; IsHak et al., 2018; Klossika et al., 2006). Therefore, it could also

be speculated for our study that ACE values and pain sensitivity are connected in a bidirectional manner. However, our study only allows for correlational assumptions and no causality deductions.
Similar to the hypotheses about violent video gaming we also wanted to gain insight on how pain sensitivity among participants with different levels of ACE is influenced by different pain stimuli frequencies in the pain perception experiment. Past research displayed lower pain threshold for individuals with trauma background (Tesarz et al., 2015, 2016). Data regarding the pain perception experiment conducted in Study 2 also revealed positive correlation between the CTQ subscales physical abuse and physical neglect and brain activation for pain stimuli frequencies of 1 Hz. Data displayed significantly higher activation in the middle frontal gyrus, superior frontal gyrus as well as frontal pole for higher levels of physical abuse experience in the past among participants. Higher levels of physical neglect experience in the past were connected to higher activation of the frontal pole. These data support the perspective that ACEs based on physical neglect or abuse are related to higher attention and higher sensory awareness regarding painful stimuli and favor a perspective of higher monitoring of expected negative outcomes that arise from the exposure to painful stimuli. It is interesting that these kinds of ACEs mainly were presented with a significant interaction for medium to high levels of pain stimuli frequencies and not for lower levels. One possible interpretation could be that ACE related thoughts or automatic reactions are mainly triggered when exposure to painful stimuli becomes more noticeable due to a higher frequency and lower frequencies of pain do not trigger this reactional model that easy.
As pain sensitivity and pain perception is affected by different levels of ACE it was also a goal of the study to detect if conditioning to these painful/fearful stimuli is affected by different levels and kinds of ACE as well. Previous studies suggested decreased thresholds for rising values of ACEs which leads to the conclusion that the interaction between fear conditioning to painful stimuli and ACE values could be present as well (Tesarz et al., 2015, 2016). To gain insight about the interaction of fear conditioning to painful stimuli and ACEs we focused on the reaction to the conditioned stimuli in the acquisition phase of the conditioning paradigm. This contrast displayed significantly lower brain activation in precuneus and intracalcarine cortex areas with increasing levels of the ACE subscale physical neglect. Precuneus and intracalcarine cortex are areas that are widely associated with a variety of functions like affective response to pain, stimulus reactivity and visual processing. Differences in precuneus activations have also been observed in studies in the past by the comparison of participants with internet gaming disorder and healthy controls (Z. L. Wang et al., 2022). It seems that ACE values in the form of physical neglect can influence attention towards harmful situations or fear-inducing stimuli and reaction time and intensity to these stimuli may be impaired due to lower activation in several respective brain regions which is something that should be considered when working with individuals suffering from physical neglect experiences in their childhood or adolescence.
A lot of research in the past has also focused on empathy abilities among individuals with ACE background. Studies have revealed that ACEs can be related to hindered emotional processing and a tendency for increased sensitivity to negative emotions (Curtis & Cicchetti, 2011; Masten et al., 2008; Pollak et al., 2000; Young & Widom, 2014). Alongside these findings therapeutic studies also detected a link between empathy and ACE in patients using child-centered play therapy to increase empathy abilities in individuals suffering from ACE (Burgin & Ray, 2022; Narvey et al., 2021). Our behavioral data support the findings of prior studies as various ACE types in the form of the CTQ subscales physical abuse, physical neglect and emotional abuse did play a significant role in impaired recognition of the emotion “surprise” presented via facial

expressions. Individuals mostly recognized this emotion wrongly as “fear”. Increased sensitivity to negative emotions among individuals suffering from ACE suggested by various research in the past could be a possible explanation for the common wrong attribution of the emotion fear in our sample for the targeted emotion surprise revealed by post-hoc tests (Curtis & Cicchetti, 2011; Masten et al., 2008; Pollak et al., 2000; Young & Widom, 2014). Although this wrong attribution towards the emotion “fear” did not occur across all used emotions in the empathy for pain experiment and no additional influence of ACE displayed via CTQ subscales on the recognition of other emotions was observed. No therapeutic conclusions can be made due to our findings in Study 2 as it was not a therapeutic controlled setting, but it is recommended to keep the impaired ability to recognize several emotions correctly in mind and to account for the fact that increased perception of negative attributed emotions could arise regarding the field of working with individuals suffering from ACE.
In addition to behavioral data influence ACEs in the form of CTQ subscales also showed influence on brain activation patterns during the empathy for pain experiment in the scanner which is supported by previous neuroimaging studies researching on brain activity alterations of ACE participants (Assed et al., 2020; Dannlowski et al., 2012; Etkin et al., 2011). Especially the CTQ subscale physical abuse was connected to higher activation in the superior frontal gyrus whenever participants were exposed to the emotion “fear” visualized via facial expression videos. One of the main functions of the superior frontal gyrus is its association with impulse control and the working memory. Studies in the past have also revealed altered functional connectivity for the superior frontal gyrus among individuals suffering from ACEs (Sokołowski et al., 2022). In addition to altered functional connectivity there is also evidence for greater connectivity in the superior frontal gyrus among ACE individuals revealed by medial prefrontal cortex seed analysis (Dong et al., 2022). However, our data for the empathy for pain experiment did not show altered brain activation patterns for other presented emotions and it seems like especially the emotion “fear” leads to an altered reaction among individuals suffering from physically abuse in their childhood or adolescence. It could be that this higher activation pattern is somewhat explained by the findings from Dong et al. (2022) and relates to the higher connectivity among ACE participants or that “fear” based facial expressions tend to trigger physical abuse experiences from the past among ACE participants. However, it should be kept in mind that this interaction was discovered via post-hoc testing. Overall, our data on the various experiments conducted in Study 1 and Study 2 yielded highly significant results for both violent video games and ACEs and therefore it is very reasonable to look at possible interactional patterns to gain a better understanding of how these factors possibly interconnect for our experiments.

4.2.3 Interactional effects of violent video gaming and adverse childhood experiences

Moderation analyses for the effect of ACEs in the form of the CTQ subscale physical neglect on the connection between video gaming behavior and several pain tests displayed physical neglect as a significant moderator on the connection between video gaming behavior and electrical stimulation pain tests as well as pressure algometer tests. Increasing values of the CTQ subscale physical neglect did display a decreasing correlation regarding the sensitivity for electric stimulation, pressure algometer pain threshold and pain tolerance for VVG but also an increasing correlation for NG. However, this effect was more present with higher levels of physical neglect and is not

apparent with lower levels of physical neglect meaning that according to our data video gaming behavior could relate to a change in how strong experiences with physical neglect affect the individual's pain sensitivity. Implications could be a consideration for video gaming behavior when working with individuals that show a strong physical neglect background in their childhood or adolescence and take these results into account when working on pain sensitive topics like for example self-harming behavior. As our results revealed that violent video gaming habits are connected to even more decreased pain sensitivity compared to NG among people with strong physical neglect background it could be the case that these patients tend to show stronger self-harming behavior compared to NG to reach a similar effect. Our study is no therapeutic study and can therefore not confirm this effect but therapists should consider a stronger psychoeducation to address the situation and potential consequences.

The pain perception experiment also showed interesting interactional effects between video gaming behavior and ACEs. We did not observe any differences in rating for the different used pain stimuli frequencies between the video gaming groups, but we did detect ACE subscale effects for the group of NG that were not present in any other video gaming group. Regarding the highest used pain stimuli frequency of 2 HZ higher levels of ACE in the form of the CTQ subscale physical neglect were related to higher rating of unpleasantness among NGs which is in line with previous research on pain sensitivity among ACE participants (Tesarz et al., 2015, 2016). The fact that we only detected this interaction in the highest frequency used for the pain perception experiment rises up the question if people suffering from physical neglect experiences in the past that play video games regularly meaning NVVG and VVG develop some kind of skill to be less responsive to even high frequencies of pain stimuli. Future research should take a closer look on this interaction by researching on a wider range of different frequencies and different pain stimuli to shed light on the interaction between ACE in the form of the CTQ subscale physical neglect, video gaming behavior and different pain stimuli or different pain stimuli frequencies.

We reported differences in brain activation patterns for the pain perception experiment on the 2 Hz pain stimulus frequency between VVG and NG earlier. Covariate analysis revealed that even with the inclusion of the CTQ subscales physical abuse and physical neglect brain activation differences between VVG and NG did not change for the most part. Only region that did not show any significant difference after including the covariates was the postcentral gyrus. This supports the statement that there is a difference in brain activation patterns between VVG and NG regarding pain stimulus exposure for high used pain stimuli frequencies even though they may not report any difference in unpleasantness or intensity in the first place.

The fear conditioning experiment showed similar interaction patterns as the pain perception experiment between the video gaming groups and the ACE levels for the change in brain activation patterns. Similar to the analyses of individual effects we used the reaction to the conditioned stimuli in the acquisition phase of the fear conditioning to detect any interactional effects. We added ACE values in the form of the CTQ subscale as covariate to the comparison between VVG and NG, but physical neglect did not influence the main areas of different brain activation between VVG and NG namely the anterior cingulate gyrus. Only motor cortex areas were impaired when adding physical neglect as a covariate to the group comparison which suggests that there is a true difference between VVG and NG for fear conditioning in terms of awareness regarding fearful stimuli and emotional awareness toward them that cannot be explained by ACEs. We did not research on interactional effects of video gaming groups and ACE values in the form of CTQ subscales for the empathy for pain experiment as there were no significant video gaming group differences for this experiment. Overall, we detected

many main and interactional effects regarding video gaming behavior and ACEs that may help a bit to clarify the inconsistent research on these important topics so far, but our study also comes with some limitations which will be mentioned below.

4.3 Limitations

The experiments conducted in Study 1 and Study 2 have helped to gain a better understanding of the interaction between violent video gaming, ACEs, pain sensitivity, fear conditioning, empathy for pain and pain perception. However, our studies also have some limitations which are mentioned in this section.
Although the number of participants included in the studies are accepted for the field of neuroimaging research and pain research more participants could help to improve the power of statements for interactional patterns. Because past research has yielded such different results, a study with increased sample size could make results even more trustworthy and replicable. Especially a possible future meta-analysis including all kinds of research to this point like different classifications of ACEs and violent video gaming could help to broader the understanding we have on the interactional patterns of pain sensitivity, pain perception, fear conditioning and empathy abilities so far.
Regarding Study 1 we used a variety of different pain stimuli. However, past research does not suggest which kind of stimuli are optimal to research on pain sensitivity among violent video gamers and participants suffering from ACEs. It could be very well the case that other pain stimuli could be more suited for this participant group, because there is no research to this point revealing which pain stimuli are optimal for the design. In addition, it would be wise to include more physiological markers for all conducted experiments like skin conductance responses to observe more biological results reflecting arousal of pain stimuli on pain sensitivity, fear conditioning, empathy for pain or pain perception.
Another point worth mentioning is the number and frequency of pain stimuli, cues and conditioning parameters. We did construct our study design based on past research for pain sensitivity or fear conditioning (Baeuchl et al., 2019; Fullana et al., 2016; Rothemund et al., 2012; Suarez-Jimenez et al., 2020). However there is no clear guideline for timing, frequency and number of presented cues for the design as variations on these parameters could lead to different results overall and different conclusions. Future research should consider to alternate the number of presented cues per phase to see if different effects like video gaming group differences in habituation or extinction phase can be observed.
Regarding the pain perception experiment past research did not detect the optimal variation in pain stimuli frequency to observe an effect of temporal summation for a video gaming habit and ACE sample of participants. Therefore, our segmentation into 0.2 Hz, 1 Hz and 2 Hz could be not the optimal design to observe video gaming group or ACE level differences. In case upcoming studies want to research on these interactional effects it would be beneficial to use a broader variety of pain stimuli frequencies in the pain perception experiment from Study 2. Our data suggested a stronger effect for higher frequencies of pain stimuli that is why we recommend to add more high frequented pain stimuli for future studies supporting temporal summation.
Similar to the other experiments conducted in Study 1 and Study 2 we used a well-established paradigm for the empathy for pain experiment. Unfortunately, this paradigm was designed many years ago and has some restrictions. The empathy for pain

experiment we used does not account for any culturally diverse facial expressions. This does not affect our study as our pool of participants was also not very culturally diverse but should be accounted for future studies assessing a more diverse range of participants. In addition, some participants did report that the video length of 1 second was only barely enough to detect which emotion was shown via the facial expression presentation in the fMRI part of the experiment. The fMRI scanner noises and unfamiliar surroundings may have made it harder for participants to stay focused during this short time of facial expression presentation. The video length was selected by intention but feedback from participants should be taken serious in case of future designs. Future research should consider an experimental design for the fMRI with slightly increased video duration to prevent confusion on behalf of the participants. Additionally, it is recommended to implement a new set of facial expression videos that accounts for culturally diverse emotion recognition in case the participant sample requires it as interpretation of facial expressions can vary with change of cultural background.
Because the experimental procedure was already challenging in terms of the length for the participants, we had to reduce the fMRI part of the empathy for pain experiment by 50% regarding the amount of shown control emotions. Therefore, we recommend, that future fMRI studies on this paradigm should include a broader variety of shown emotions to gain more insight on this interaction.

4.4 Conclusions and outlook

Violent video gaming and ACEs have become very relevant topics in our modern society and will become even more relevant in the future as more interactive patterns are discovered. In this dissertation we tried to shed light on the possible influence violent video gaming and ACEs could have on different pain and empathy related factors via our experimental design.
We discovered that average pain perception and brain activation for anterior cingulate cortex and other relevant regions related to fear conditioning are impaired for individuals that play violent video games on a regular basis and experienced physical neglect in their childhood or adolescence. Impaired pain sensitivity among individuals suffering from ACEs was detected in prior research as well and this may help to broaden the understanding especially in the case of violent video gaming (Garrido et al., 2018). In addition, multiple pain tests showed that high ACE values in the form of the CTQ subscale physical neglect were mostly related to more pain sensitivity among NG but extensive use of violent video games inverted this connection. Therefore, VVG with high values of physical neglect were connected to impaired pain sensitivity. As outlook it would be interesting to explore more if violent video gaming affects not just pain sensitivity but also affects nociception reactions in the form of the neural encoding process of noxious stimuli.
To diversify the results on pain sensitivity among VVG and individuals with ACE we also tested pain sensitivity with different pain stimulus frequencies via the pain perception experiment. VVG did show significantly higher sensory-motor activation compared to other video gaming groups for the highest used pain stimulus frequency of 2 Hz. We suspect that this difference could be an effect of temporal summation among VVGs that does not appear in this intensity for the NVVGs and NGs. ACEs also played an important role in the perception of different pain stimuli frequencies for the pain perception experiment as higher levels of physical neglect or physical abuse were

associated with increased attention and sensory awareness towards the painful stimuli as well as higher monitoring of potential outcomes regarding the painful stimuli exposure for higher painful stimuli frequencies. Physical neglect was also connected to higher ratings of unpleasantness in the pain perception experiment.
On the other hand, video gaming behavior did not show an influence on discrimination between different emotions. VVGs were able to discriminate between emotions displayed via facial expressions just as well as NVVGs or NGs. ACEs in terms of the CTQ subscales emotional abuse, physical abuse and physical neglect however displayed a strong misattribution for the emotion “surprise”. The most common misattribution was to rate “surprise” as “fear”. The emotion “fear” seems to play a big role in individuals with ACEs as higher values in the CTQ subscale physical abuse were also related to increased brain activation patterns when exposed to facial expressions attributed to the emotion “fear”.
Alternated emotional and pain related perception among violent video gamers and individuals suffering from ACEs has been proven by our study and previous research and should be considered when working with these individuals. We did not research on therapeutic interventions or coaching aspects regarding these interactions but our study results suggest that awareness of possibly altered perception in any context that includes work with violent video gamers or individuals that suffer from ACE is recommended and possibly leads to more beneficial outcomes.
However, our studies also yielded some limitations that should be accounted and adjusted for in further research on the topics of this dissertation. Future studies on the paradigm used should consider examining a larger number of participants to increase power, changing the types of pain stimuli and pain frequencies to better understand how video game habits or types of ACE interact with pain stimuli such and different pain stimulus frequencies, should create an updated version of the empathy for pain experiment with facial expressions reflecting culturally sensitive parameters, and should use all available emotions in the form of facial expressions for an fMRI task.
Overall violent video gaming as well as ACE values did display significant influence in almost every of our conducted experiments. Physical neglect or abuse were strong factors for individual and interactional patterns on our research design. Violent video gaming and ACEs are very common characteristics in our modern society and this dissertation revealed that they also have very strong influence on many aspects of our daily live and should therefore be researched in many other aspects to gain a clear understanding on what consequences they can have and how we should approach them. This dissertation managed to describe a variety of different impacts that have not been researched before or are very controversially discussed in the research community. But all parameters presented in this study are very important factors in everyday life especially video gaming. We live in a society that becomes more and more digital day by day and is constantly changing its digital shape with less and less text on websites and apps and more and more visual information in the form of videos as well as shorter duration of these videos which can have impact on for example our attention span. We know that these new types of stimulation heavily influence how we perceive the rest of the world. Yet it is a very challenging task for research to keep up with these constantly shaping designs and produce results that are still reliable and valid years later. This dissertation manages to give a good understanding of very relevant factors to this point in time and should be used to give individuals more insights about the potential consequences of their behavior but should also give an impulse for therapeutic research to acknowledge the modern way of perceiving our environment. There are pitfalls but also opportunities revealed by this study. We should seek to

capitalize on these opportunities and use the interactions between violent video games and ACEs to our advantage to benefit from the change in our environment.

5 SUMMARY

Video gaming and adverse childhood experiences are very common in our nowadays society. Recent events like lockdown measures of the COVID-19 pandemic led to even more people discovering video gaming for themselves. One of the most common types of video gaming is violent video gaming. Previous research stated that violent video gaming and adverse childhood experiences affect various pain perception types like pain sensitivity or pain processing. In this dissertation we tried to evaluate how violent video gaming and adverse childhood experiences affect fear conditioning, pain perception and empathy for pain in others. In addition, we also wanted to see if violent video gaming and adverse childhood experiences show interactional effect patterns regarding behavioral data or MRI data on these topics. We examined three groups of participants (violent video gamers, nonviolent video gamers and non-gamers) and assessed adverse childhood experiences on 5 different subscales (emotional abuse, emotional neglect, physical abuse, physical neglect and sexual abuse) via the Child Trauma Questionnaire to observe a diverse spectrum of possible effects and interactions. Pain sensitivity and empathy for pain in others experiments were conducted via fMRI measurements in the scanner as well as in the laboratory. Fear conditioning and pain perception experiments were completely assessed via fMRI measurements in the scanner. We conducted one big assessment for these topics and divided the experiments into two studies. Study 1 included a pain sensitivity assessment as well as a fear conditioning task and Study 2 consisted of an empathy for pain in others task with facial expressions shown to the participants and a pain perception task with different frequencies of electrical pain stimulation that were presented to the participant. Results for Study 1 displayed significantly higher pain threshold and pain tolerance for violent video gamers compared to nonviolent video gamers and non-gamers, but no significant difference between nonviolent video gamers and non-gamers. Adverse childhood experiences in the form of physical neglect moderated this connection significantly. Violent video gamer also showed significantly lower activation in anterior cingulate cortex, juxtapositional lobule cortex and the paracingulate gyrus compared to non-gamers for painful stimuli in the acquisition phase of the fear conditioning task conducted in the fMRI scanner. Increasing levels of physical neglect were connected to lower activation of precuneus and intracalcarine cortex for the same contrast of the fear conditioning. Study 2 revealed no difference between video gaming groups for emotion recognition via facial expressions in the empathy for pain experiment, but higher values of adverse childhood experiences displayed higher superior frontal gyrus activations for fear-based facial expressions. We did not observe any significant differences for the pain emotion in the empathy for pain experiment. Regarding the pain perception experiment violent video gamers displayed significantly higher activation in sensory-motor brain activation than non-gamers for the highest used pain stimuli frequency and higher values of physical abuse and physical neglect were connected to increased activity of middle frontal gyrus, superior frontal gyrus and frontal lobe in the pain perception experiment for a pain stimuli frequency of 1 Hz.
Overall violent video gaming and adverse childhood experiences were connected to various kinds of pain perception like pain threshold, pain tolerance and temporal summation affecting the individual directly but violent video gaming did not seem to affect the perception of painful emotions in others. This dissertation reveals that violent video gaming and adverse childhood experiences can have a wide range of consequences for the individual and researching more on these factors may help to understand many other consequences as well and improve treatment.

6 REFERENCES

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7 CURRICULUM VITAE

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2016	Bachelor thesis: „Burnout and the Psy-Bel-Comic: An even more work-place moderated disease than expected? A cross-cultural evaluation”
30.06.2016	Bachelor of Science, Grade: 1,2
2016 - 2018	Studies in Psychology (Master of Science) At Ludwig-Maximilians-Universität, München
2018	Master thesis: „Kulturelle Intelligenz und Persönlichkeitsprofile als Einflussfaktoren auf Prozesse des Design Thinking -Eine quantitative und qualitative Analyse“
01.08.2018	Master of Science, Grade: 1,3
January 2020 – present	Doctoral candidate in Psychology in the project GRK 2350, Medical Faculty Mannheim, Heidelberg University

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