

**EFFECTS OF EXCESSIVE VIDEO GAME PLAYING ON WORKING
MEMORY LOAD: AN EVENT-RELATED BRAIN OSCILLATION STUDY**



ÖZÜM KARYA SAKMAN

MAY 2022

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MEMORY LOAD: AN EVENT-RELATED BRAIN OSCILLATION STUDY**

**A THESIS SUBMITTED TO THE
GRADUATE SCHOOL
OF
BAHÇEŞEHİR UNIVERSITY**

ÖZÜM KARYA SAKMAN

**IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR
THE DEGREE OF MASTER OF ARTS
IN
THE DEPARTMENT OF COGNITIVE NEUROPSYCHOLOGY**

MAY 2022



T.C.
BAHCESEHIR UNIVERSITY
GRADUATE SCHOOL

09/05/2022

MASTER THESIS APPROVAL FORM

Program Name:	Cognitive Neuropsychology
Student's Name and Surname:	Özüm Karya SAKMAN
Name Of The Thesis:	Effects of Excessive Video Game Playing on Working Memory Load: An Event-Related Brain Oscillation Study
Thesis Defense Date:	09/05/2022

This thesis has been approved by the Graduate School which has fulfilled the necessary conditions as Master thesis.

Prof. Dr. Ahmet ÖNCÜ
Institute Director

This thesis was read by us, quality and content as a Master's thesis has been seen and accepted as sufficient.

	Title/Name	Signature
Thesis Advisor's	Prof. Metehan IRAK	
Member's	Prof. Canan BAŞAR EROĞLU	
Member's	Assist Prof. Hale ÖGEL BALABAN	



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Name, Last Name: Özüm Karya SAKMAN

Signature:

ABSTRACT

EFFECTS OF EXCESSIVE VIDEO GAME PLAYING ON WORKING MEMORY LOAD: AN EVENT-RELATED BRAIN OSCILLATION STUDY

SAKMAN, Özüm Karya

M.S., Cognitive Neuropsychology

Supervisor: Prof. Dr. Metehan Irak

May 2022, p. 53

Like other behavioral addictions, excessive video gaming may affect several cognitive mechanisms like working memory, but previous behavioral and neuroimaging studies show controversial results. Thus, the current study aims to understand the effects of excessive violent video gaming on manipulating, maintaining, and monitoring working memory and its neural underpinnings. We measured theta and alpha responses of excessive video game players (EVGP) ($n = 16$) and non-players (NP) ($n = 19$) at 70 to 130 ms, 70 to 140 ms, 130 to 240 ms, 180 to 250 ms, and 270 to 400 ms after the stimulus onset during the N-back task. 2- and 3-back responses were analyzed separately for behavioral and event-related oscillations (EROs). Contrary to our hypothesis, the behavioral results indicated that the two groups had similar WM performance. On the other hand, EVGPs showed later 70 to 140 ms post-stimulus theta latency and 270 to 400 ms post-stimulus alpha latency in contrast to NPs, especially in the occipital region in the 3-back task. The findings were interpreted as excessive gaming might negatively affect the processes of encoding and storing visual information when the working memory load increases.

Keywords: Excessive violent video game playing, working memory, N-back task, event-related oscillation

ÖZ

AŞIRI DÜZEYDE VİDEO OYUNU OYNAMANIN ÇALIŞMA BELLEĞİ YÜKÜ ÜZERİNDEKİ ETKİSİ: OLAY-İLİŞKİLİ BEYİN OSİLASYONU ÇALIŞMASI

SAKMAN, Özüm Karya

Yüksek Lisans, Bilişsel Nöropsikoloji

Tez Danışmanı: Prof. Dr. Metehan Irak

Mayıs 2022, s. 53

Diğer davranışsal bağımlılıklara benzer olarak, aşırı düzeyde video oyunu oynamak çalışma belleği gibi bilişsel mekanizmaları etkileyebilir. Buna karşın, önceki davranışsal ve beyin görüntüleme çalışmaları, aşırı düzeyde oyun oynamanın özellikle bilişsel süreçler üzerindeki etkileri konusunda tartışmalı sonuçlar sunmaktadır. Mevcut çalışma, aşırı oyun oynamanın, bilginin çalışma belleğinde değişimlenmesi, saklanması ve izlemesi süreçlerinin nöral temelleri üzerindeki etkilerini araştırmayı amaçlamıştır. Bu amaçla, aşırı düzeyde video oyunu oynayan oyuncular (ADO) ($n = 16$) ve oyun oynamayan kişilerin ($n = 19$) olay ilişkili teta ve alfa bantı aktiviteleri 70-130 ms, 70-140 ms, 130-240 ms, 180-250 ms, ve 270-400 ms zaman aralıklarında, N-geri görevi kullanılarak ölçülmüştür. 2- ve 3- geri aşamalarından elde edilen davranışsal (doğru ve yanlış tepkiler ve bunların tepki süreleri), ve olay ilişkili salınımlar birbirinden bağımsız olarak analiz edildi. Beklentimizin aksine, davranışsal analizler sonucunda gruplar arasında çalışma belleği performansı açısından anlamlı fark bulunmamıştır. Buna karşın, özellikle oksipital elektrotlarda, yalnızca 3-geri görevinde ADO grubunun 70-140 ms zaman aralığında teta ve 270-400 ms zaman aralığında alfa latanslarının oyun oynamayan gruba göre daha gecikmiş olduğu sonucu elde edilmiştir. Bulgular, çalışma belleği yükü arttığında, görsel bilginin kodlanması ve saklanması süreçleri üzerinde aşırı oyun oynamanın olumsuz etkileri olabileceği şeklinde yorumlanmıştır.

Anahtar Kelimeler: Aşırı düzeyde şiddet içerikli video oyunu oynama, Çalışma Belleği, N-back görevi, olay ilişkili salınımlar



ACKNOWLEDGEMENTS

In the process of writing this thesis, I approximately drank 3282 cups of coffee, listen 1850 hours of music, eat lots of fries and the world has been conquered by a deadly virus. Despite all the panic, confusion, and hardships, I never felt alone in this process. When everything is complicated and separated like ripples on a blank shore, it felt nice to have people who support and love me unconditionally. I am grateful to have all of you by my side.

First and most importantly, I would like to express my gratitude and thankfulness to my thesis advisor Prof. Metehan IRAK, for always having time for me, guiding me, and laughing at my jokes. He was always there whenever I asked for help and was supportive and knowledgeable. I am grateful to have an opportunity to work with a scientist like him.

I would like to express my gratitude to my committee members, Prof. Canan BAŞAR-EROĞLU and Asst. Prof. Hale Ögel BALABAN. They were so kind, supportive and reassuring and they improved this thesis to its best form with their precious contributions. It has been a privilege to me to have your comments and guiding in this process, I cannot thank you enough.

My dear fellow people in the lab, I am so lucky that I can count on exceptional people like you. Can Soylu, you have been a great supporter of me through all the difficulties and sufferings I had. Your contributions to this thesis and your sensible words meant a lot to me, I owe you big-time. Moreover, my dearest friend, Elif Güldemir, thanks for your compassion and companionship. I am delighted that I shared this journey with you, your support was always with me. My lovely friend Büşra Selduz, even though we did not work together on this thesis you were still one of the best colleagues I got. Thank you for your friendship.

My dear mother and father, Zehra and Özgür Mazhar Sakman, I am so lucky to be the daughter of such loving and supportive parents. You made me the independent and strong woman I am, I love you so much. I also send my love and gratitude to my aunt

Ümral Topaktaş who bought me the first book that I have read and supported me from the very first day of my education. I am forever grateful for your moral and material support. My other family members who always supported and loved me, thank you for always being there and being the best family that one could have.

My beloved friends! You all have a strong faith and believe in me; I don't know how I got so lucky to have you on my side. I must have used all my good fortune in this area. You sat behind me in my study room while I was writing this thesis and falling into pieces at the same time. You understood me, never let go of me, and made me smile when most needed. My dear Tuncer Ayakta, you were always splendid while encouraging me since you came into my life. Your patience and confidence in me gave me strength, thank you. My friends Yusuf Furkan Çeşnial and Ahmet Said Metin, even though there was a great distance between us, you were always one call away from listening to all my struggles and always cared for me. Thank you for being my dearest friends. My roommates Aslıhan Sarı and Sevgi Arslan, thank you for being a beautiful extension to my family in the last four years, I am so lucky to have both of you and your friendship. Last but not least, I would like to thank Volkan Gözütok for acting as a final push for me for completing this thesis. Your contribution is greater than you can imagine. Consequently, thanks for all the love, hugs, laughs, conversation, and support my friends. I love you with all my heart.

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LIST OF ABBREVIATIONS

ACC	Anterior Cingulate Cortex
ADHD	Attention Deficit and Hyperactivity Disorder
ADO	Aşırı Düzeyde Video Oyunu Oynayan Oyuncular
AUD	Alcohol Use Disorder
CE	Central Executive
dIPFC	Dorsolateral Prefrontal Cortex
EEG	Electroencephalography
ERO	Event-Related Oscillation
ERP	Event-Related Potentials
EVGP	Excessive Video Game Players
fMRI	Functional Neuroimaging
GD	Gaming Disorder
IA	Internet Addiction
IPC	Inferior Parietal Cortex
LTM	Long-Term Memory
MDD	Major Depressive Disorder
NP	Non-players
NSW	Negative Slow Wave
PFC	Prefrontal Cortex
PPC	Posterior Parietal Cortex
PSW	Positive Slow Wave
RT	Reaction Time
VIP	Ventral Intraparietal Area
vlPFC	Ventrolateral Prefrontal Cortex
vPFC	Ventromedial Prefrontal Cortex
WM	Working Memory

Chapter 1

Introduction

Video games serve humans as a tool for having fun, recreation, and entertainment and have a social interaction value (Ma et al., 2013). New software developments offer intense and realistic graphics with more complex rules for video game players. It has been estimated that there were approximately 2.5 billion video game players worldwide and the number is increasing (Newzoo, 2016). According to the Turkey Game Market Report (2020), 79% of adults play video games regularly and action-adventure video games are the most played genre among the Turkish population. As a result of the limitless world that the video game industry offered, some players may give excessive priority to gaming instead of daily activities, may lose control over gaming, be socially isolated, be hyper-focused in-game, and have mood swings (King, Haagsma, Delfabbro, Gradisar & Griffiths, 2013). Gaming overuse or excessive video game playing was defined with two-hour-per-day limit by The Council on Science and Public Health for the American Medical Association (AMA) (Kuss & Griffiths, 2012) and as suggested by the “Internet Gaming Disorder” section in DSM-V, it can be measured by difficulty in interpersonal relationships, withdrawal from work, school, and social commitments due to gaming. Since the latest video games offer a more realistic and complex nature, it requires more advanced attention, eye-hand coordination, and problem-solving skills. Excessive video gaming may have effects on impulse control, monitoring ongoing behavior, and disinhibition (Weng et al., 2013) but may increase the visual attention system and working memory (WM) capacity since video games require the gamer’s active participation, holding, and manipulation of the rules in the mind, usage of the game’s map, and decision making and consequently, WM (Green and Bavelier, 2006; Boot et al., 2008).

In the current study, we will focus on the relationship between working memory, working memory load, and excessive video game players' (EVGPs) WM performance and its neural assemblance.

1.1 Working Memory

Working memory (WM) is a multicomponent memory model with a limited capacity which underpins complex cognitive activities and is responsible for the storage, manipulation, and usage of the information for a short period (Chai, Hamid, Abdullah, 2018; Baddeley, Eysenck & Anderson, 2015, p. 124; Baddeley, 2012). Originally, WM consisted of four different components which are the central executive, the phonological loop, and the visuospatial sketch pad (Baddeley & Hitch, 1974), and the episodic buffer (Baddeley, 2000). While the phonological loop is responsible for the temporary maintenance of the verbal information, the visuospatial sketchpad is responsible for the visual and spatial information. These two and WM is ruled by the central executive (CE) and any attentionally demanding task, other than the simple maintenance of the information, requires the activation of the CE. CE focuses and divides the attention between targets or stimuli and then helps us to switch between tasks, with specific control mechanisms. Finally, episodic buffer helps to hold perceived stimulus or chunks in interrelation with CE.

WM assessments can be made with WM span tasks and these tasks can be in a range of simple to complex. One of the most widely used WM paradigms is called as *N*-back task (Mackworth, 1959; Kirchner, 1958). *N*-back task assesses storage, continual updating and manipulating of information held in WM and during the task, participants must update and monitor WM and maintain the information (Colzato et al., 2012; Owen et al., 2005). In *N*-back, the stimulus list is being shown to participants in a serial manner, and then, they decide whether the current item matches with the item *n* trials ago. For to make tasks more difficult and increase the working memory load, the number *n* can be increased (Au et al., 2015). Research indicated that, while the *N*-back task is a reliable and valid measurement of manipulation, maintenance and monitoring of WM (Schmiedek, Lövdén & Lindenberger, 2014; Kane, Conway, Miura, & Colflesh, 2007; Ragland et al., 2002), it might be sensitive to individual differences and different samples (Jarrold & Towse, 2006) but this disadvantage can be overcome by making the task harder through increasing the load (Jaeggi, Buschkuhl, Perrig & Meier, 2010).

1.1.1 Neurobiology of working memory. While it is controversial whether there are a specific structure or localization for the WM in the brain, it is accepted that the prefrontal and posterior parietal cortices play a significant role in WM processes (Qi et al., 2010; Baldo & Dronkers, 2006; Rowe et al., 2000; Goldman-Rakic, 1996). While neurons from the prefrontal cortex (PFC) and ventral intraparietal area (VIP) work together in encoding and monitoring of the attended stimulus (Qi et al., 2010), neurons in the posterior parietal cortex (PPC) help to encode distractors and unattended targets (Suzuki & Gottlieb, 2013). Hence, different areas work in a balancing manner to control WM processes (Jacob & Nieder, 2014).

Functional Magnetic Resonance Imaging (fMRI) research with healthy subjects (Fiehler et al., 2011; Honey et al., 2002) and human brain lesion patients (Barbey, Koenigs & Grafman, 2013) reported that, inferior parietal cortex (IPC) and dorsolateral prefrontal cortex (dlPFC) are important areas for the WM processes, especially for the manipulation of the verbal and spatial knowledge and further reasoning of the stimulus. Verbal WM processes were found to be related to IPC, dlPFC, and right ventrolateral PFC (vlPFC) activations and additional anterior cingulate cortex (ACC) activation with the increase in WM load (Gould et al., 2003; Ragland et al., 2002; Barch et al., 1997), which can be assessed by the means of N-back tasks. Increase in WM load also results with the heightened activity in the neurocognitive network for WM which includes frontoparietal and prefrontal connectivity (Honey et al., 2002). Furthermore, patients with dlPFC lesions made more errors in 3-back condition than non-dlPFC patients but not in other conditions (Barbey et al., 2012). The performance deficit of dlPFC patients may be related to the increased memory load but not monitoring of the information because they showed similar performance with the healthy subjects when the task was simple.

1.1.2 Neurobiology of excessive video gaming. EVGP's suppression of distracting stimuli is found to be better than non-players (NPs) because EVGP's P300 component to the target stimulus had a larger amplitude than that of NPs (Mishra et al., 2011). Moreover, increased interconnectivity in the central executive and salient networks of EVGPs and individuals with internet gaming disorder (IGD) in contrast to controls were reported (Gong et al., 2016; Ding et al., 2013). This may indicate higher WM, planning, and reasoning skills of EVGPs (Menon, 2015; Donohue, 2011;

Green & Bavelier, 2007). However, these results had not been replicated by other studies (e.g. Boot et al., 2008; van Ravenzwaaij et al 2014.) and this fail in replication of enhanced attention and memory capacity on a neural basis may be related to the game genre, playing hours, and game playing onset (in years) (Green et al., 2017). Negative effects on brain structure mostly seen in EVGPs who played action video games (Nuyens, Kuss, Lopez-Fernandez & Griffiths, 2018), but this effect have not been observed in logic/puzzle game players (Kühn & Gallinat, 2014). It is also known that professional and pathological game players show different brain structures due to gaming such as EVGPs' left thalamus showed a decreased volume in contrast to professional players (Han, Lyoo & Renshaw, 2012).

The circuit between the left IPC and the right inferior temporal gyrus has weakened (Ding et al., 2013) and cerebral blood flow was reported to be diminished in EVGPs' brain (Feng et al., 2013). Decline in gray matter volume in the right ventromedial prefrontal cortex (VPFC), left dlPFC and, thinner left superior parietal lobe (He, Turel, Wei & Bechara, 2020), higher left ventral striatum volume (Kühn et al., 2011), regardless of any increase in a specific region (Mohammadi et al., 2019) were observed in EVGPs; similar with the pathological gamblers (Mohammadi et al., 2016; Joutsa et al., 2011). Together, these regions are related to the reward system of the brain (Bechara, 2005) and gray matter alterations in those areas are related to the impulsive and addictive behaviors in biological addictions (Dalwani et al., 2011; Schmidt et al., 2018). A meta-analysis by Nuyens, Kuss, Fernandez and Griffiths (2017) supported this by claiming that excessive video game playing mostly affects perception, inhibition, and decision-making processes and, excessive and pathological game playing mostly result in a negative way for the brain and cognitive skills.

In short, alterations in the brain structure may be related to the behavioral addiction itself, not the gaming activity and they may be an indicator of the unbalanced reward system of EVGPs (Han et al., 2012). Given that excessive video game playing is highly enriching activity and may result in prefrontal changes, excessive video game playing may have similar results with substance abuse (He et al., 2012) or internet addiction (Mohammadi et al., 2019; Liu et al., 2010), eating addiction (Schmidt et al., 2018) and problematic gambling (Mohammadi et al., 2016; Joutsa et al., 2011); in such it may result in alterations in dopamine circuitry and reward processing (Weinstein & Lejoyeux, 2013) and structural grey matter changes in a negative way.

1.1.3 ERPs in working memory and excessive video gaming. Event-related potentials (ERPs) have been used to examine the temporal dynamics of WM. ERP studies are effective in assessing WM capacity (Lefebvre, Marchand, Eskes & Connolly, 2005) and individual differences (Dong et al., 2015), effects of WM load (Wei & Zhou, 2020; Gomarús, Althaus, Wijers & Mindera, 2006) and cognitive control (Scharinger, Soutschek, Schubert & Gerjets, 2017). Neural effects of an increase in WM load can be observed in larger N200 amplitude with the longer RTs (Wei & Zhou, 2019), the smaller amplitude for early positive components like P200, prolonged positive slow wave (PSW) for the incorrect responses (Gomarús et al., 2006) and later frontal negativity and suppressed P300 amplitude in response to errors (Lefebvre et al., 2005). Among several WM tasks, decreased P300 amplitude was most noticeable in the *N*-back task, meaning that the *N*-back task put specific demands on WM as the load increases, and these neural changes might be correlated to the behavioral differences (Scharinger et al., 2017). People with lower WM capacity, which was classified according to the performance in modified digit span task, demonstrated reduced P300 amplitude in comparison to people with higher WM capacity as the WM load increased from 1- to 4-back (Dong et al., 2015).

In a visual WM task, EVGPs P100 and P200 were larger and noticeable in posterior areas but while EVGPs' showed smaller P300 and smaller negative slow wave (NSW) amplitudes than NPs in the parietal area for misleading trials, the opposite was true for resolvable trials which may reflect the problems with WM maintenance (Irak & Soylu, 2021). EVGP's superior visual WM performance in terms of RTs and accuracy can be explained by the increase in P100, N100 and N200 amplitudes which might be related to the early sensory processing and attentional orienting to stimulus, respectively (Birch-Hurst et al., 2021). In another research by Irak, Soylu, Sakman and Turan (2020), no differences were found between EVGPs and NPs in terms of ERP components during the *N*-back task. However, the P100 and P200 amplitudes were higher for EVGPs than NPs, and slower RTs were significantly related to the smaller amplitudes in frontal and parietal areas in the 300-400 ms time window for both groups. Thus, effects of excessive game playing or internet use on ERPs show inconsistencies, but evidence signaled alterations in neural activity associated with the excessive behavior, especially in P300 latency and amplitude.

1.1.4 Working memory in excessive video gaming. On a behavioral level, EVGPs seem to outperform their NP counterparts in visual (Donohue, 2010) and auditory aspects of the perception (Bavelier & Green, 2007), visuospatial attention (Wilms, Petersen & Vangkilde, 2012; Bavelier & Green, 2006); speed-processing (Pardina-Torner, Carbonel & Castejón, 2019; Gorbet & Sergio, 2018; Dye, Green & Bavelier, 2009) and, WM and mental rotation skills (Blackler, Curby, Klobusicky & Chein, 2014) but not in long-term memory (LTM) access (Mcdermott, Green & Bavelier, 2014). Despite the findings of better behavioral performance of EVGPs, they do not align with the neurobiological findings of excessive video gaming. This contradiction may be due to differences in sample characteristics or design differences between behavioral and neurological studies (Powers et al., 2013). Thus, each finding shall be interpreted considering its sample characteristics and definition of excessive video gaming should be reach to a consensus within literature.

EVGPs who played mobile, action, and adventure video games showed better performances in verbal and visuospatial WM and N-back tasks, when controlled for age, education, and depression levels (Waris et al., 2019). EVGPs processing speed (McDermott et al., 2014), locating the target information and monitoring their attention (Green & Bavelier, 2003; 2006) were better than NPs. People who played action video games more than 20 hours in a week only had slightly better mental rotation skills than NPs (Boot et al., 2008). It is possible that action video games do not directly affect the visuospatial WM but instead, as the total playing time increased some improvements in the encoding of the information can be seen (Wilms et al., 2012). EVGPs' superiority in visuospatial WM might be due to the changes in the visual processing system (Irak, Soylu & Çapan, 2016) but some studies showed that excessive video gaming is more related to the parietal, but not occipital activity changes (Wu et al., 2012). Thus, excessive game playing might affect different components of WM and if their superior performance is related to the parietal activity changes, then we can relate EVGPs better performance to their attentional processes, not their visuospatial capacities, or vice versa.

In contrast to the evidence in favor of EVGPs, Blackler and Curby (2014) did not find any differences regarding visuospatial WM performance between EVGPs and NPs. Another research that used Wechsler Adult Intelligence Scale-IV's Full Scale Intelligence Quotient, Verbal Comprehension Index, Working Memory Index, and

Processing Speed Index subscales to measure general cognitive function with over-gaming activity, indicated that EVGPs who played action video games had lower scores in verbal comprehension, WM, and processing speed than NPs (Jang et al., 2021). Moreover, Irak and Soylu (2021) reported that, EVGP's general WM performance was not different than NP's but EVGPs had higher RTs for incorrect responses with the decreased P300 and NSW, which may indicate diminished WM capacity. Others linked excessive game playing to more interference effect in the Stroop task (Mathews et al., 2005) and decreased medial frontal negativity and a frontal slow wave of EVGPs (Bailey et al., 2010) serves as evidence. This finding may indicate an altered proactive control, which is a future-oriented form of regulation before the display of stimulus which allows more goal-directed behavior in EVGPs in contrast to NPs.

In conclusion, EVGPs mostly showed better behavioral performances during WM tasks, and this may be a result of better attentional resources or visuospatial capacities. However, there are no neurological evidence for EVGPs being superior to NPs in terms of WM processes. Other factors like individual differences, game specifications or differences in research design again might play a role in contradictory results.

1.1.5 N-back task in excessive video gaming. *N*-back is a reliable and valid task for assessing the WM as it requires participants to actively store, update, monitor, and manipulate the information in WM. Since video game players need to monitor a lot of different stimuli while playing, they may show different results than NPs during the *N*-back task (Green & Bavelier, 2006). *N*-back study designs show variety with the used stimulus type which can be a simple stimulus like letter or vowel or, complex stimuli such as objects from everyday encountering (Palomaki et al., 2012), faces (Schreppel et al., 2008) and pictures with emotional valance (Grissman et al., 2017). Moreover, *n* can be increased up to seven to increase WM load and to eliminate ceiling or floor effects (Verhaeghen et al., 2004). Comparable to the contradictory results of the EVGPs' WM processes, findings of EVGPs' *N*-back performances show some inconsistencies.

In 1- and 2- back paradigms, EVGPs had lower RTs for correct responses in both conditions, and they were more accurate than NPs, when age and IQ levels were

controlled (Colzato et al., 2013). Like adult EVGPs, adolescent EVGPs showed similar performance with NPs in the 1-back condition, but they were better than NPs in the 2-back condition both in speed and accuracy (Moisala et al., 2016). In a spatial 2-back task, professional players who played more than 20 hours a week also had lower RTs for correct responses, but their accuracy was similar with the NPs (Boot et al., 2008). In a similar vein, McDermott et al. (2014) found that with the increase in the memory load (up to 7-back), EVGP became faster than NP, though this RT difference has not been observed in simpler 1- and 2-back conditions. Finding no performance differences between EVGP and NP in the 1-back task may point out to the similar monitoring skills (However, Llamas-Alonso et al., 2019; Boot et al., 2008). In addition, EVGPs with heavy media multitasking habits (consumption of multiple streams of media at once simultaneously) did not show better performance both in 2- and 3-back conditions, but EVGPs with intermediate multitasking levels were still better than NPs in terms of speed and accuracy (Cardoso-Leite et al., 2015).

Thus, these findings may indicate that EVGPs may show better performance in case of speed and accuracy than NP in the N-back task, only if the task is complex. It is vital to note that none of this research measured pathological game playing and excess of game playing measured by weekly gaming hours only, which may not be enough by itself. Correspondingly, meta-analysis study by Powers et al., (2013) did not support the notion that excessive video game playing had a positive effect on WM components. Sample characteristics (e.g. age, gender, playing time) and N-back study design, which previous research had no consensus, had a major effect on the findings and their interpretations.

1.2 Event-Related Brain Oscillations

The concept of neuro-oscillatory dynamics assumes that complex brain signals are composed of oscillations of different frequencies (Karakas & Barry, 2016). When the oscillatory activity of the brain during a sensory or a cognitive process is analyzed, it is referred to as event-related oscillations (EROs) (Karakas Erzengin, & Başar, 2000). There are various frequency decomposition techniques and all of them have revealed that spontaneous activity in EEG consists of oscillatory components of different frequency ranges, namely delta (0.5-3.5 Hz), theta (4.0-7.5 Hz), alpha (8.0-

13.5 Hz), beta (14.0-29.0 Hz) and gamma (30.0-70.0 Hz) bands (Karakas & Barry, 2016). Certain frequency bands within the oscillatory responses might be associated with different cognitive functions (Başar, 2012, 2013; Guntekin & Başar, 2014). Moreover, oscillatory processes may have a key and significant role in brain networks concerning memory (Başar, Başar-Eroğlu, Karakaş & Schürmann, 2001). It has been thought that there were only three basic categories of electrophysiological brain responses which are evoked, event-related, and induced (Karakas & Barry, 2016) but now we know that oscillatory responses fall on a continuum ranging from strong to weak (Makeig et al., 2016). Brain oscillations' functional role has a conventional principle in distinct frequency bands. For example, slow oscillatory responses like delta, theta, and alpha may connect distant areas of the brain while fast oscillations like gamma seem to take place in more local circuits (Moran & Hong, 2011). Low-frequency oscillations also may have a vital role in the regulation of gamma activities and their role in attention (Schroeder & Lakatos, 2009).

Oscillations are also used in identifying biomarkers of neuropsychiatric disorders (Başar & Guntekin, 2013). Balconi et al., (2017) suggested that ERO slow-wave and resting-state fast-wave brain activity may be related to impulsive behavior, impaired behavioral monitoring, and self-error detection in internet addiction (IA). Moreover, previous research indicated that EVGPs maintenance and manipulation of the information during N-back task show differences than NPs. Thus, investigating ERO N-back task related ERO components (e.g. theta and alpha) in EVGPs help us to comprehend the relationship between neural mechanisms and excessive video game playing.

1.2.1 Brain oscillations in working memory and video gaming. WM depends on the activity of several cortical regions, primarily the prefrontal and parietal regions (Basar-Eroglu et al., 2007). Alpha band seems to be in a significant relationship with WM processes and long-term memory (LTM) traces (Başar et al., 1997; Klimesch, Schimke & Schwaiger, 1994). During a visual WM task, the encoding stage results in a decrease in alpha response in occipital, parietal and temporal areas, and the maintenance stage result in increased alpha response in frontal and parietal areas (Bashivan, Bidelman & Yeasin, 2014). Increased theta activity in the frontal midline and left temporal areas were found to be related to the increase in WM load (Onton,

Delorme & Makeig, 2005) and mental effort to the decreased alpha (Aftanas & Golosheikine, 2001). Moreover, during WM retention (Sarnthein et al., 1998), encoding and retrieval (Sauseng et al., 2004) processes, synchronous theta activity between prefrontal and temporo-parietal areas were observed, especially in the right hemisphere. Sauseng et al., (2004) also suggested that increased theta activity in posterior regions originally initiated by the frontal cortex. As a reflection of the organization of multiple items into sequential WM representations, higher frequency oscillations like gamma may be found in theta cycles and interregional theta band synchronization may represent different sub-functions of WM processes (Sauseng, Griesmayr, Freunberger & Klimesch, 2010). Thus, encoding, retention, and retrieval processes of WM and effect of increase in WM load are closely related to the alpha and theta activities in the brain, especially in the frontal, temporal, and parietal areas.

Unfortunately, there is a limited number of studies regarding the oscillatory responses of EVGPs' WM processes. Expecting to see differences in oscillatory activity between EVGP and NP is reasonable because when inexperienced people play video games for a period, neural changes such as increase in grey matter volume in right hippocampal formation, right dlPFC and bilateral cerebellum can occur due to plasticity (Kühn et al., 2014). In a dual-task paradigm, which used Corsi block-tapping and N-back tasks, EVGPs showed higher electroencephalographic correlation (rEEG) in the alpha2 (10.5–13.5 Hz), beta1 (13.5–19.5 Hz), beta2 (19.5–30 Hz), and gamma (31–50 Hz) bands than NPs (Llamas-Alonso et al., 2019). Increase in alpha power from 300 to 700 ms and delta power from 250 to 600 ms following the successful detections of the specific target in the game, were related to the better learning rates and this connection was at its peak in the left frontal area which was found to be slightly related to P300 amplitude (Mathewson et al., 2012; Maclin et al., 2011).

A recent meta-analysis by Burleigh et al., (2020) indicated that decreased delta and theta with the lower beta activity might be a neurophysiological marker of gaming disorder (GD). In a resting state EEG study, participants with GD demonstrated higher delta and decreased theta band powers in contrast to NPs (Kim et al., 2017) and lower absolute beta power than people with alcohol use disorder (AUD) and NPs (Son et al., 2015). In addition, decreased theta, and alpha activity in left frontal areas was observed in people with internet GD, in comparison to NPs, which may be an indicator of impairments in cognitive control (Kim et al., 2019). Researchers also claimed that

lower frontal theta activity may be an indicator of the severity of the behavioral addiction. On the other hand, another research found that participants with GD showed differences in higher frequency band gamma and beta in contrast to AUD and NPs and as the gamma band increased, the total Internet Addiction Test score also increased (Park et al., 2017a). Moreover, Choi et al., (2013) claimed that high impulsivity and impaired inhibitory control in internet addiction (IA) are associated with lower beta and higher gamma responses in a resting state.

In consequence, a neural network with several brain regions was linked to the WM processes and effects of WM load can be traced back to the alpha and theta oscillations in those areas. Alterations in alpha and theta responses may be distinguishing for EVGPs, and similar behavioral addictions like IGD. It should be noted that, those neural responses may be affected by other psychiatric and neurological disorders like attention deficit and hyperactivity disorder (ADHD), anxiety, and major depressive disorder (MDD) (Park et al., 2017b; Youh et al., 2017) as these psychiatric conditions show high comorbidity rates with excessive gaming activity (Toker & Baturay, 2015; Bioulac, Arfi, & Bouvard, 2008).

1.2.2 Brain oscillations and N-back task. According to Basar-Eroglu et al., (2007), healthy subjects used various cognitive strategies depending on task difficulty. Changes in neural mechanisms and brain oscillations due to WM load can be traced by parametric changes in n . It is not clear in which frequency bands neural oscillatory changes occur in WM by varying WM load during the N-back task. Evidence showed that (e.g. Jensen & Tesche, 2002; Sauseng et al., 2004; Pesonen et al., 2007; Gevins & Smith, 2000; Scharinger, Soutschek, Schubert & Gerjets, 2017; Klimesch, Schack & Lipka, 2004; Klimesch, 1996), increase in WM load can be characterized with the increased theta along with the decreased alpha and beta activities at parietal and frontal sites. In healthy participants, there was a decrease in posterior alpha power and increase in temporal region-distributed beta power in the N-back task as the WM load increased from 1- to 2-back, but no increase in the theta power distributed over the frontal midline which also emanates from the anterior cingulate cortex (Chen & Huang, 2016). In an n-back task with complex stimulus, from 0- to 3- back conditions, theta wave power show increases approximately 50-2200 ms after stimulus onset and this effect were observed in all electrodes, but the increase was prominent for target

responses than non-targets (Palomäki et al., 2012). Moreover, they also found that alpha and beta powers decreased with the WM load in 0-, 1- and 2- back conditions, but not in 3-back conditions, between the duration of 400 to 2100 ms. An increase in theta activity regardless of the age and a decrease in alpha band activity with the increase in WM load was found to be related to the decrease in P300 amplitude in the parietal region in young adults (Pergher et al., 2019). Thus, it is plausible to assume that theta and alpha oscillations are susceptible to WM load, and it show a pattern across different participants. To our knowledge, no research investigated the oscillatory activity during the N-back task in EVGP sample. Llamas-Alonso et al., (2019) found that, during 1-back task EVGPs' alpha2, beta1 and -2, and gamma bands showed a greater interhemispheric correlation in the frontal area. However, in this research 1-back task was used as a dual-task, and as researchers claimed, it might have worked as a distractor, more than the main task, and did not show the effect of WM load.

Since there is a limited amount of research on EVGPs, we can look at the other behavioral addictions' effect on oscillatory activity during the N-back task. People with IA had higher alpha frequency bands and lower RTs than control group, as the WM load increased from 1- to 2- back and IA severity and WM performance were negatively correlated (Wang, Sun, Lan & Liu, 2020). In addition, internet GD, nicotine dependence, and other substance use disorders show similarities in some of their neural mechanisms (Yan, Chen, Liu & Zheng, 2021; Ge et al., 2017; Ko et al., 2013; Smith, Hummer & Hulvershorn, 2015). During the N-back task, nicotine users' behavioral performance was like the non-users, but their alpha band decreased while theta wave increased in frontal and parietal areas as the WM load increased (Fisher et al., 2011). After excessive marijuana use, users showed slower and less accurate responses, which was shown with decreased theta and alpha power as the task became more difficult (Ilan et al., 2004). Therefore, while theta and alpha bands are closely related to the WM load, differences in alpha and theta activity can be seen between different addiction groups like IA, internet GD or nicotine and non-addicted groups with the increase in WM load. These changes may not be observed on a behavioral level but addiction, whether it is behavioral or biological, can affect alpha and theta responses when given the WM load.

1.3 The Goal of the Study

Although some studies investigated neural mechanism of behavioral addictions (e.g. IA), and indicated their functional mechanisms in terms of WM by using *N*-back task, the changes in neural mechanism due to excessive gaming is still unclear. IA studies and findings may be ambiguous and include some confounding variables because IA includes many different activities such as gambling, shopping, and excessive gaming which can be performed both online and offline and may be in different genres (Pontes & Griffiths, 2014) and internet may be a gateway for the actual addicted stimuli. In this research, we focused on the effects of excessive video gaming since there is a gap in literature and biomarkers of the pathological gaming should be classified.

Playing video games requires active WM processes for a successful performance. In non-pathological players, video gaming was associated with better performance in WM tasks in terms of RTs and accuracy (e.g. Donohue, 2010; Bavelier & Green, 2007, 2006; Pardina-Torner, Carbonel & Castejón, 2019; Gorbet & Sergio, 2018; Dye, Green & Bavelier, 2009; Wilms, Petersen & Vangkilde, 2012; Blacker, Curby, Klobusicky & Chein, 2014) and this positive effect of video gaming have been associated to the its boosting effects of brain's plasticity (Gong et al., 2019) because it can be increased with training or practice (Green & Bavelier, 2015). In contrast, pathological gamers did not exhibit positive effects of video gaming experience on their WM capacity. Instead, they demonstrate WM impairments which can be seen from neuropsychological findings (e.g. Ding et al., 2013; Lin et al., 2015; Moulton et al., 2013; Feng et al., 2013; He et al., 2020; Park et al., 2017a; Park et al., 2017b). Given the gaming experience's close relation to WM processes, investigating the EVGPs' performance on *N*-back task both at a behavioral and neurophysiological level may give us insights for where the pros of video gaming end and where the cons begin.

In this research, we used EROs since they are important for understanding the neural dynamics and when a brain cognitively change in a pathological way, it can be observed all over the cortex (Başar, 2013). Since the biomarkers of GD still not completely understood, findings from ERO research may be useful in this area. *N*-back task was also widely used in addiction and ERO research, and proven to be useful in

creating and investigating effect of WM load on the brain through EEG (Deng et al., 2021; Khoonsinthaweekul, Rintra & Sittiprapaporn, 2017; Brouwer et al., 2012).

In the light of the literature, it may be deduced that increase in theta and alpha band activities are related to the WM processes (Sauseng et al., 2015). In detail, increase in WM load emerge itself with a decrease in alpha, and increase in theta band power, in parietal and frontal areas, respectively (Klimesch, Schack & Lipka, 2004; Klimesch, 1996). As the resting state EEG studies with GD and ID revealed, alterations in overall theta and alpha activity might be important indicators of the addiction severity (Kim et al., 2019). Since both theta and alpha activities are notable in WM processes, *N*-back task, and GD, in this research we specifically investigated theta and alpha bands.

The goal of the current study is to investigate the effects of excessively violent video gaming on theta and alpha band activity during the *N*-back task and to see how selected bands are affected by the increase in the WM load, in contrast to NP. Furthermore, this study will also consider the effect of the WM load on the performance of the EVGPs.

1.4 Hypotheses of the Study

The research questions and hypotheses of the current study will be like the following:

1. Does excessive violent video gaming have a different effect on 2- and 3-back conditions? We hypothesized that EVGPs have faster RTs but lower accuracy than NPs during 3-back condition when memory load increased, but these two groups will show similar performance in 2-back condition.
2. Do the amplitude and latency values of the theta responses of the two groups differ in 2-back and 3-back conditions? We hypothesized that EVGPs have higher amplitude and prolonged latency of P100-theta than non-players in 3-back condition when memory load increased, but these two groups will not be different in 2-back condition.
3. Do the amplitude and latency values of the alpha responses of the two groups differ in 2-back and 3-back conditions? We hypothesized that EVGPs have lower amplitude and prolonged latency of P300-alpha than non-players during 3-back

condition when memory load increased, but these two groups will not be different in 2-back condition.



Chapter 2

Method

2.1 Participants

A survey containing the weekly game time and the names of the five most played games were distributed to the Bahçeşehir University students ($n = 500$). Then, initially, 37 university students participated in the study. However, 2 participants were excluded from the analysis because of the higher number of missing data. Thus, the current study was conducted with 35 (20 female) undergraduate and graduate university students from different departments, with a mean age of 22.31 ($SD = 2.82$). Participants were divided into two groups as excessive video game players (EVGPs) and non-players (NPs). All participants were right-handed and reported normal or corrected-to-normal vision and no neurological or psychiatric disorder history. The study conformed to the Declaration of Helsinki and was approved by the University Research Ethics Committee. Participants provided informed consent and were given financial compensation and/or course credits where applicable.

Inclusion criteria for the EVGP group were as follows: Following the previous studies, time spent playing violent video games of more than 5 h/week were accepted as inclusion criteria for the EVGP group ($n = 11$, 5 female). The mean of their weekly game play was 30.50 hours ($SD = 23.74$), as stated by the participants. EVGP group reported at least more than two symptoms ($M = 5.25$, $SD = 2.67$) on the Pathological

Game Addiction Symptoms List (Gonnerman and Lutz, 2011), and their mean score from the Game Addiction Scale (Lemmens et al., 2009) was 53.69 ($SD = 13.90$). The mean age of the EVGP group was 21.13 ($SD = 2.90$).

Inclusion criteria for the NP group ($n = 19$, 15 female) were having no or little experience with any type of violent video games. The mean of their weekly game play was 2.68 hours ($SD = 1.86$), as stated by the participants. NP group reported less than 2 symptoms on the Pathological Game Addiction Symptoms List ($M = 2.16$, $SD = 1.86$) and obtained a mean score of 31.68 on the Game Addiction Scale ($SD = 11.50$). The mean age of the NP group was 23.32 with a standard deviation of 2.38. The mean of their weekly game play was 1.90 hours ($SD = .55$), as stated by the participants.

In addition, demographic characteristics of the participants, age, gender, and level of education can be seen in detail in Table 1.

Table 1

Demographic characteristics of the participants

Variable		EVGP (n = 16)	NP (n = 19)	Total (n = 35)
Age (SD)		21.13 (2.90)	23.32 (2.38)	22.31 (2.82)
Gender				
	Female	5 (31.3%)	15 (78.9%)	20 (57.1%)
	Male	11 (68.8%)	4 (21.1%)	15 (42.9%)
Level of Education				
	Undergraduate	15 (93.8%)	15 (78.9%)	30 (85.7%)
	Graduate	1 (6.3%)	4 (21.1%)	5 (14.3%)

2.2 Materials

In this section, materials that were used in this research were explained.

2.2.1 Pathological gambling section on DSM-IV. The original list was created from pathological gambling symptoms according to DSM-IV criteria by Gonnerman and Lutz (2011). In the Pathological Game Addiction Symptom List, the word “gambling” was changed to the word “gaming” for this study. The scale has 16 items, and the Turkish translation was made by Arslan-Durna (2015) and Başer (2015). The scores that can be obtained from the listed range between 0-16. Higher scores indicate higher levels of game addiction symptoms.

2.2.2 Game Addiction Scale. This scale was originally developed by Lemmens et al., (2009) to measure the degree of game addiction. The scale has 21 items with seven factors: salience, tolerance, mood modification, relapse, withdrawal, conflict, and problems. Participants’ responses were given on a 5-point Likert scale from 1 (never) to 5 (very often). A Turkish adaptation was completed by Arslan-Durna (2015)

and Başer (2015). Participants can obtain a minimum score of 21 and a maximum of 105 points. High scores indicate higher levels of game addiction symptoms.

2.2.3 State-Trait Anxiety Inventory (STAI). STAI was originally developed by Spielberger et al., (1983), and it was standardized by Oner and Le Compte (1985) by using both healthy and clinical samples. It assesses both state and trait anxiety and includes 20 items with a 4-point Likert scale where each item is scored from 1 (rarely) to 4 (almost always). While the trait anxiety subscale assesses general anxiety and related feelings, the state subscale measures a person's anxiety related to the moment and context they are in. Trait anxiety subscale's Cronbach alpha was between .83 and .87 and test-retest reliability was between .71 and .86. Moreover, the state anxiety subscales' Cronbach's alpha was between .94 and .96 and its test-retest reliability was between .26 and .68. Item reliability correlation was between .42 and .85 for STAI.

2.2.4 Beck Depression Inventory-I (BDI-I). BDI-I is a self-report measure that included 21 items with a 4-point Likert scale (0 as I do not feel sad and 3 as I am so sad, I cannot stand it). This inventory was developed by Beck, Steer, and Brown (1996), and its Turkish adaptation was conducted by Hisli (1988). The total score obtained from the inventory can be ranged between 0 and 63 and, higher points indicate higher depression scores.

2.2.5 N-back task. The N-back task is used to investigate the neural bases of WM by increasing the mental workload. Since the task requires participants to online monitor, update, and manipulate the remembered stimuli, it places great demands on several key processes in WM. For each stimulus, participants need to decide if the current stimulus is the same as the one presented *N* trials ago (2 trials, 3 trials, etc.). In this study, the N-back procedure involved the presentation of letters as stimuli, one at a time on a screen, for a period of 2000 milliseconds (ms), with an interstimulus interval of 500 ms. Letters were selected randomly from the alphabet. Only 2-back and 3-back versions were used in this study. Both 2-back and 3-back tasks were presented in epochs of 20-second duration. Each 2-back and 3-back epoch was preceded by a 5-second instruction epoch and followed by a 20-second rest epoch. During instruction epochs, the instruction 'Press the X if the current letter is the same as the letter 2 items

ago' for 2-back condition and 'Press the X if the current letter is the same as the letter 3 items ago' for the 3-back condition was presented on the screen. During rest epochs, the word 'REST' was presented, and the subject was not required to make any motor response. There were 144 letter stimuli per participant: 72 letter stimuli for each condition.

2.3 Electrophysiological Recording and Pre-processing

The N-back task was conducted in an electrically shielded and soundproof room and ERPs and EROs were recorded during the N-back task. Data were analyzed during the 2-back and 3-back conditions separately for 200 ms before and 1000 ms after the stimulus onset. EEG/EOG signals were recorded using 32 Ag/AgCl electrodes mounted in elastic Quick-caps (Neuromedical Supplies, Compumedics, Inc., Charlotte). The EOG signal was measured from two bipolar channels: one was formed by two electrodes placed at the outer canthus of each eye; another by two electrodes below and above the left eye. EEG signal was recorded from 30 (FP1, FP2, F7, F8, F3, F4, Fz, FT7, FT8, FC3, FC4, FCz, T7, T8, C3, C4, Cz, TP7, TP8, CP3, CP4, CPz, P7, P8, P3, P4, Pz, O1, O2, Oz) electrodes arranged according to the standard 10–20 system, with additional electrodes placed at BP1/BP2 and also on the left and right mastoids (M1/M2). All EEG electrodes were referenced on-line to an electrode at the vertex and re-referenced off-line to linked mastoids. EEG and EOG signals were amplified and recorded at a 1000 Hz sampling rate using Synamp2 amplifier at AC mode (Neuroscan, Compumedics, Inc., Charlotte) with a high- and low-pass filter set at 0.15 and 100 Hz, respectively. EEG electrode impedance was kept below 5 k Ω .

EEG data pre-processing was conducted using Edit 4.51 (Neuroscan, Compumedics, Inc. Charlotte) and applied to each participant's dataset. Data were down-sampled to 250 Hz to reduce computational demands and then low-pass filtered at 30 Hz and high-pass filtered at 0.15 Hz. EEG segments were extracted with an interval of 200 ms preceding and 1000 ms following the stimulus onset. Artifact rejection is applied in two steps. First, trials containing activity exceeding a threshold of ± 100 μ V at vertical and horizontal EOG and EEG channels were automatically detected and rejected. Second, we manually removed trials with saccades identified over the horizontal EOG channel. For the computation of ERPs, artifact-free segments

were baseline corrected using a 100 ms pre-stimulus period and then averaged for the experimental conditions. EROs were obtained by stimulus-locked averaging of EEG recorded during two phases, namely 2-back and 3-back conditions.

2.3.1 Data analysis. In this section, first the behavioral analysis then ERO analysis were presented.

2.3.1.1 Behavioral analysis. To analyze the behavioral performance of subjects and compare two groups in each condition, we examined accuracy and response time for 2- and 3-back conditions by using an independent sample t-test. The responses to the stimuli were divided into four categories: hit (target and button press), false alarm (non-target and button press), correct rejection (non-target and no button press), miss (target and no button press).

2.3.1.2 ERO Analysis. In the present study, the average EROs was determined in a temporal direction. As discussed earlier, while the ERO correlates of the N-back task are known, the WM process of excessive video game players during the N-back task was not identified. Thus, we analyzed the grand average ERO between the time windows of -200 to 1000 ms by filtering 4.1 and 20 Hz. ERO's were calculated separately for the frontal (F3, Fz, F4), central (C3, Cz, C4), parietal (P3, Pz, P4), and occipital (O1, O2, and Oz) electrodes which represented four brain regions.

As stated earlier two EROs were analyzed for this study, namely theta and alpha oscillations at 70 to 130 ms, 70 to 140 ms, 130 to 240 ms, 180 to 250 ms, and 270 to 400 ms time windows. Theta frequencies were obtained by filtering the data between 4.1 and 8 Hz and alpha frequencies were obtained by filtering the data between 8.1 and 12 Hz.

According to grand averages and following by the previous literature, the P100 amplitudes, calculated as the average over 70 to 140 ms; N100 amplitudes, calculated as the average over 70 to 130 ms; P200 amplitudes, calculated as the average over 130 to 140 ms; N200 amplitudes, calculated as 180 to 250 ms; and P300 amplitudes, calculated as the average over 270 to 400 ms as their largest peaks in those intervals. Theta band was investigated at 70 to 130 ms, 180 to 250 ms, 130 to 240 ms, and 270 to 400 ms time intervals, for N100, N200, P200 and P300 peaks at the frontal region

(F3, F4, and Fz, electrodes); at 70 to 140 ms, 70 to 130 ms, 180 to 250 ms, and 270 to 400 ms time intervals, for N100, N200, P100, P300 peaks at the parietal region (P7, P8, and Pz electrodes); and at 70 to 140 ms, 130 to 240 ms and 270 to 400 ms time intervals for P100, P200, P300 peaks at occipital region (O1, O2, and Oz Electrodes).

The alpha band was investigated at 180 to 250 ms time interval for N200 peak at frontal region (F3, F4, and Fz); at 270 to 400 ms time interval for P300 peak at parietal region (P7, P8, and Pz); and, at 70 to 130 ms, 180 to 250 ms, 70 to 140 ms, 130 to 240 ms, and 270 to 400 ms time intervals for N100, N200, P100, P200 and P300 peaks at occipital region (O1, O2, and Oz).

ERO topographies and head plots for the 12 electrodes are presented for each experimental condition. Statistical analyses were performed with theta and alpha band frequencies in given channels for both latency and amplitude values, using mixed design 2 (group: EVGP and NP) x 3 (Hemisphere: Left, Right, and Midline) x 3 (Electrode: Frontal, Parietal and Occipital) ANOVA that included Greenhouse–Geisser corrections in cases where factors had more than two levels. All reported differences are significant at least at a level of $p < .05$ and were followed by Bonferroni post-hoc comparison tests, if necessary. For post-hoc comparison, α was assigned the value of 0.05 for each p among a set of p values, such that for a set of p values did not exceed a critical value. The number of missing values was less than 3 for each channel both in amplitude and latency values. After artifact rejection, the total number of usable trials was 5328 for all the participants (2664 for each condition). For the non-player group, the number of usable trials was 1291 for each condition, and for the EVGP group, the number was 1368 for each condition.

2.4 Procedure

After the approval of the Bahcesehir University Research Ethic Committee, the data collection of the current study was started.

Selected volunteer participants were invited to Bahçeşehir University Brain and Cognition Research Laboratory to the assessments ($n = 42$). To prevent participants' history effect on the study, they were requested to sleep well, not to be hungry, and not use alcohol and caffeine one night/day before the experiment.

Before the experiment, participants were informed about the aim, potential risks, and benefits of the study. Participants were asked to complete a battery of self-report which included a demographic information form, pathological gambling scale, game addiction scale, BDI, and STAI. An EEG cap (Quick-Caps) fitted with sintered Ag/AgCl electrodes were placed on participants' heads. Then, the participants were instructed about the procedure of the N-back task. During the task, participant EEG was continuously recorded.

Visual Studio 2010 Ultimate was used for the presentation of the task that is based on C# (sharp) programming language. During the experiment, a Windows XP computer with a 21-inch monitor was used. At the end of the study, participants were given a debriefing form and were thanked for their time and cooperation. Data collection took approximately 2 hours for each participant.

Chapter 3

Results

3.1 Behavioral Results

In this section, group comparisons on psychological scales and N-back performance were presented.

3.1.1. Group comparisons on psychological scales and gaming hours. An ANOVA was conducted to test the difference on the psychological scale scores and gaming hours between the groups (Table 2). Table 2 also shows the mean, standard deviations, minimum and maximum scores of the variables, and ANOVA results.

Table 2

Descriptive Statistics of Violent, Non-Violent, And Total Gaming Hours, Game Addiction Scale, STAI-State, STAI-Trait and BDI Of Each Group, And ANOVA Results

Variable	EVGP		NP		F(1, 34)	P
	Mean (SD)	Min- Max	Mean (SD)	Min- Max		
Violent Gaming Hours	22.13 (18.35)	5-59	.53 (1.26)	0-4	26.340	.000**
Weekly Non-Violent Gaming Hours	8.19 (11.46)	0-35	1.95 (2.07)	0-7	5.457	.026*
Weekly Total Gaming Hours	30.50 (23.74)	7-94	2.68 (2.36)	0-7	25.932	.000**
Pathological Game Addiction Symptoms List	5.25 (2.67)	2-11	2.16 (1.86)	0-6	16.166	.000**
Game Addiction Scale	53.69 (13.90)	21-77	31.68 (11.50)	21-62	26.284	.000**
STAI-State	114.67 (14.85)	67-127	75.68 (21.84)	47-121	34.924	.000**
STAI-Trait	59.27 (9.97)	26-70	39.52 (14.42)	18-65	21.366	.000**
BDI	6.69 (4.61)	1-16	6.63 (6.73)	0-27	.001	.978

* $p < .05$; ** $p < .01$

The results showed that EVGP group had significantly higher violent gaming ($M = 22.13$, $SD = 18.35$) and non-violent gaming ($M = 8.19$, $SD = 11.46$) hours than NPs' violent ($M = .53$, $SD = 1.26$) and non-violent ($M = 1.95$, $SD = 2.07$), $F(1, 34) = 26.34$, $p < .01$; $F(1, 34) = 5.46$, $p < .05$, respectively. Similarly, EVGP group ($M = 30.50$, $SD = 23.74$) had significantly higher total gaming hours than NPs' ($M = 2.68$, $SD = 2.36$), $F(1, 34) = 25.93$, $p < .01$.

EVGP group ($M = 5.25$, $SD = 2.67$) had significantly higher scores than NP group ($M = 2.16$, $SD = 1.86$) on pathological game addiction symptom list, $F(1, 34) = 16.17$, $p < .01$. EVGP group ($M = 53.69$, $SD = 13.90$) also had significantly higher scores than NP group ($M = 31.68$, $SD = 11.50$) on game addiction scale, $F(1, 34) = 26.28$, $p < .01$. Moreover, there was a significant difference between the groups on STAI-State, $F(1, 34) = 34.92$, $p < .01$, and, STAI-Trait scores, $F(1, 34) = 21.37$, $p < .01$. EVGP group ($M = 114.67$, $SD = 14.85$) had higher STAI-State scores than NP group ($M = 75.68$, $SD = 21.84$). Likewise, STAI-Trait scores were higher in EVGP group ($M = 59.27$, $SD = 9.97$) than non-players ($M = 39.52$, $SD = 14.42$). On the other hand, there was no significant difference between the groups on BDI, $F(1, 34) = .00$, $p > .05$.

3.1.2 Group comparisons on n-back task. Before analyses, data were screened for missing values and outliers (Tabachnick & Fidell, 2007). True (Hits and correct rejection) and false responses (False alarms and misses) were accepted as dependent variables both in the 2-back and 3-back phase separately. Reaction times (ms, RTs) were only recorded for hits and false alarms. An ANOVA was conducted to see the difference between true and false answers and their RTs in both 2- and 3- back conditions (Table 3).

Table 3

Mean and Standard Deviations of 2- and 3-back Responses: ANOVA Results

Variable	EVGP	NP	<i>F</i> (1, 34)	<i>p</i>
	Mean (SD)	Mean (SD)		
2-back True Responses	21.38 (1.71)	20.68 (2.75)	.760	.389
2-back False Responses	1.42 (.52)	1.92 (.96)	2.659	.117
2-back Hits RTs (ms)	724.06 (160.68)	729.18 (122.23)	.011	.915
2-back False Alarms RTs (ms)	812.04 (202.63)	723.35 (204.84)	1.182	.288
3-back True Responses	18.19 (3.33)	16.11 (3.23)	3.508	.070
3-back False Responses	2.38 (1.19)	2.38 (1.81)	.000	1.000
3-back Hits RTs (ms)	779.61 (152.42)	795.71 (164.83)	.089	.768
3-back False Alarms RTs (ms)	868.32 (183.92)	852.87 (196.25)	.043	.838

* $p < .05$; ** $p < .01$

True and false answers were investigated for each group (EVGP and NP) in 2-back and 3-back conditions, separately. Results indicated that there were no significant group differences between the true answers both in 2-back, $F(1, 34) = .76$, $p > .05$, and 3-back conditions, $F(1, 34) = 2.66$, $p > .05$. Similarly, no significant differences were found between two groups in their false responses in 2-back, $F(1, 34) = 2.67$, $p > .05$, and 3-back conditions, $F(1, 34) = 3.51$, $p > .05$ (Figure 1.).

In addition, RTs for hits and false alarms were investigated for 2- and 3-back conditions, separately. Results indicated that there were no significant group differences between the RTs for hits both in 2-back, $F(1, 34) = .01$, $p > .05$, and 3-back conditions, $F(1, 34) = .09$, $p > .05$. Similarly, no significant group differences were found between two groups in their false responses in 2-back, $F(1, 34) = 1.18$, $p > .05$, and 3-back conditions, $F(1, 34) = .04$, $p > .05$ (Figure 2.).

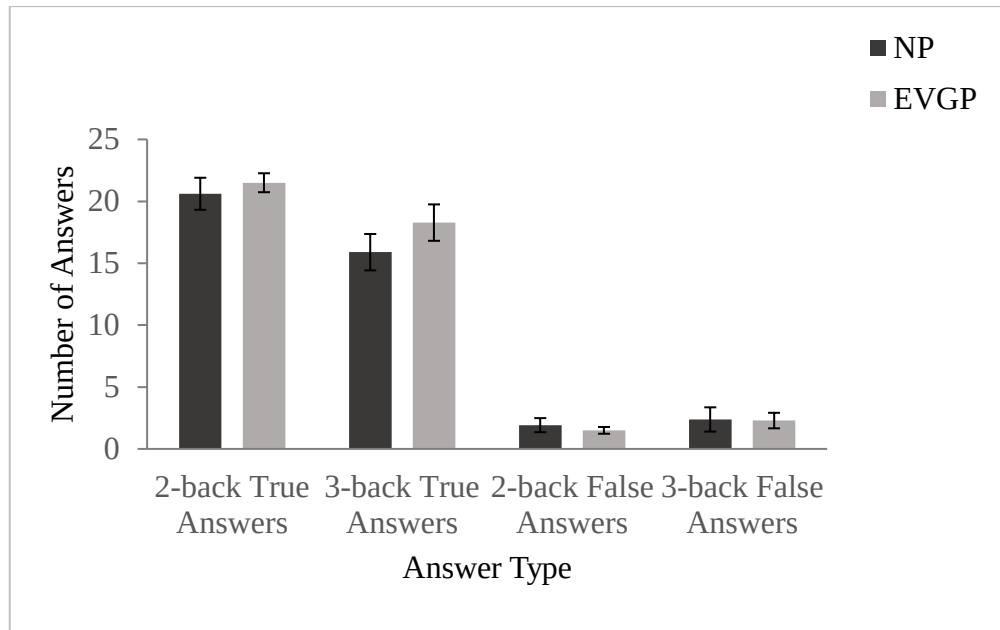


Figure 1 The number of true and false answers for two groups in 2-Back and 3-Back conditions.

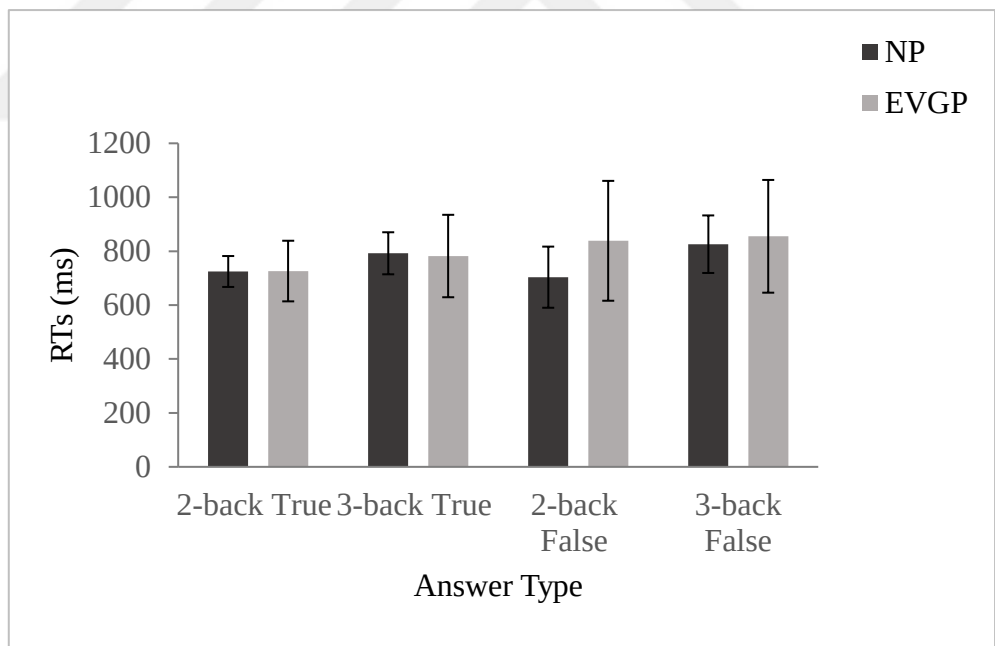


Figure 2 Reaction times (ms) of true and false answers in 2-Back and 3-Back conditions.

3.2 ERO Results

3.2.1 Visual analysis. ERO grand averages were calculated for 2-back and 3-back conditions for the two groups separately (see Figure 3-6). Specifically, at the 2-back and 3-back conditions significant theta responses were recorded at 70 to 130 ms and 180 to 250 ms time intervals at frontal and parietal electrodes; at 70 to 140 ms time interval at occipital and parietal electrodes; at 130 to 240 ms time interval in frontal and occipital electrodes, and; at 270 to 400 ms time interval at frontal, occipital and parietal electrodes. Alpha responses were observed at both the 2-back and 3-back conditions at 70 to 130 ms and 70 to 140 ms time intervals in occipital electrodes; at 180 to 250 ms time intervals at frontal and occipital electrodes, and at 270 to 400 ms time intervals at occipital and parietal electrodes.

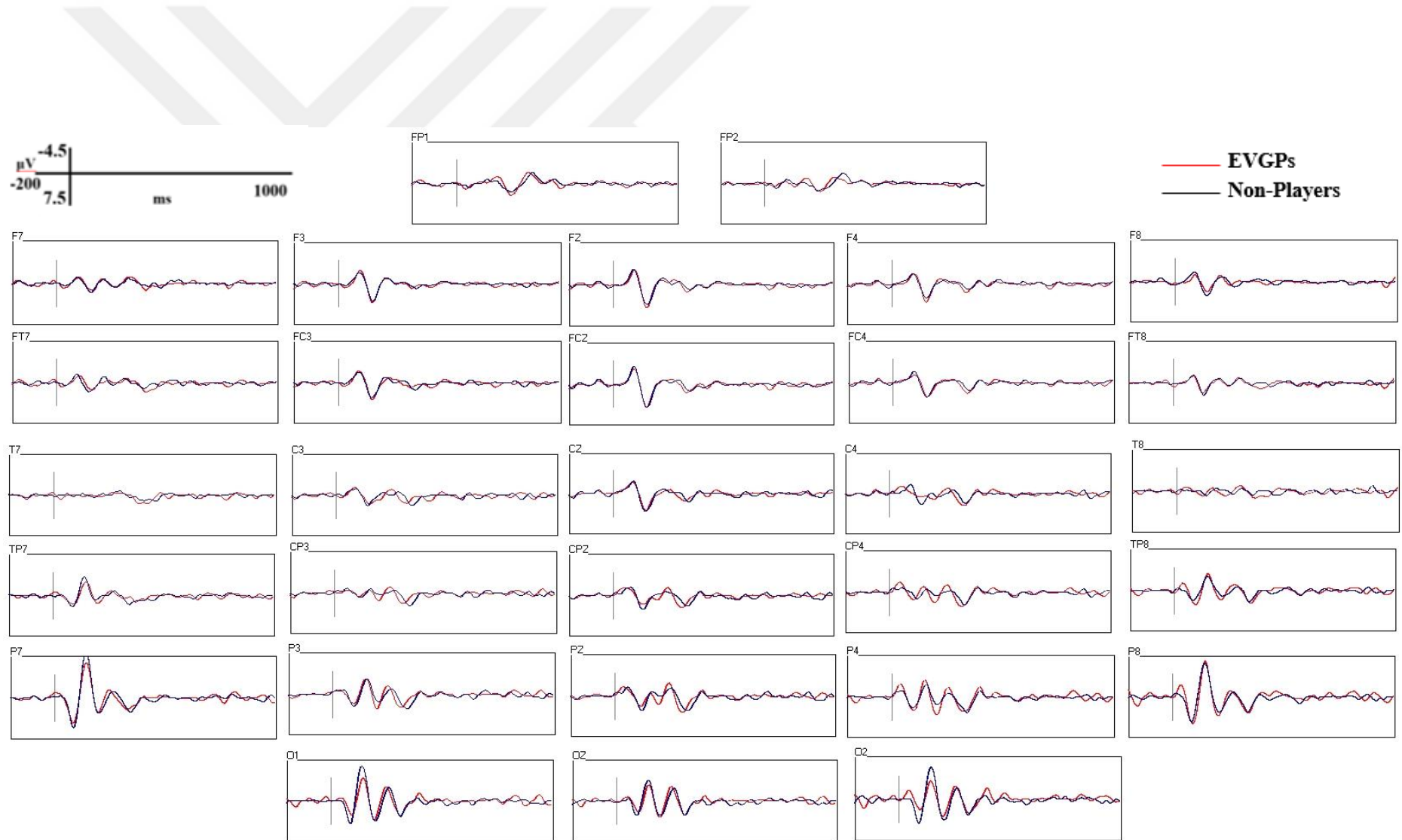


Figure 3 Grand average alpha waveforms topography during 2-back conditions for the two groups.

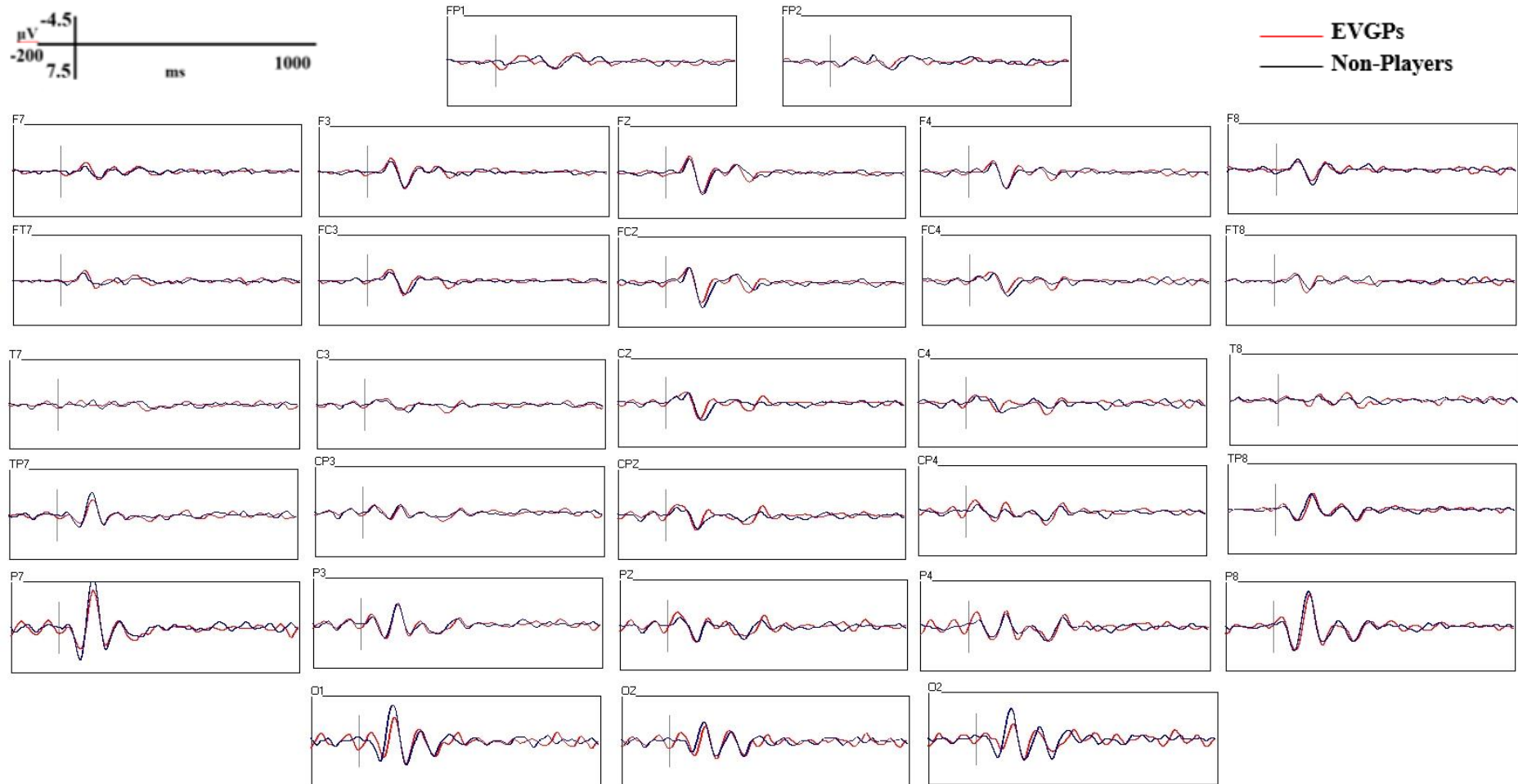


Figure 4 Grand average alpha waveforms topography during 3-back conditions for the two groups.



Figure 5 Grand average theta waveforms topography during 2-back conditions for the two groups.

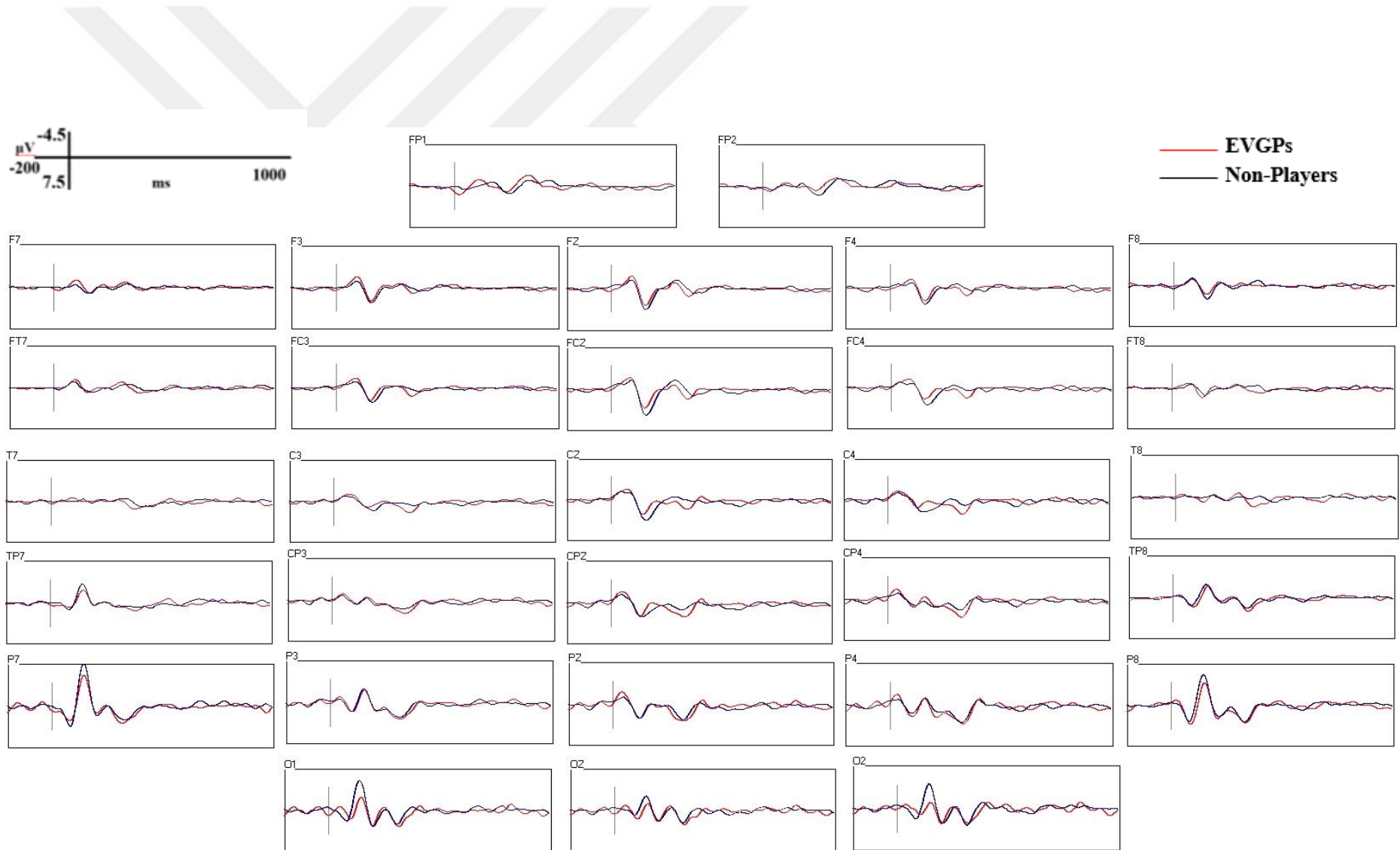


Figure 6 Grand average theta waveforms topography during 3-back conditions for the two groups.

Mean amplitude and latency values obtained at the frontal (F3, F4, and Fz), occipital (O1, O2, and Oz), and parietal (P7, P8, and Pz) regions were analyzed in the range between -200 to 1000 ms. A 2 group (EVGPs and non-players) x 3 hemispheres (lefts, medial, and right) x 2 electrode region (frontal and parietal) repeated measures ANOVA was conducted for N100 and N200 time intervals for theta response; a 2 group (EVGPs and non-players) x 3 hemispheres (lefts, medial, and right) x 2 electrode region (occipital and parietal) repeated measures ANOVA was conducted for 70 to 140 ms time interval for theta response and 270 to 400 ms time interval for alpha response; a 2 group (EVGPs and non-players) x 3 hemispheres (lefts medial, and right) x 2 electrode region (frontal and occipital) repeated measures ANOVA was conducted for 130 to 240 ms time interval for theta response and 180 to 250 ms time interval for alpha response; a 2 group (EVGPs and non-players) x 3 hemispheres (left, medial, and right) x 3 electrode region (frontal, occipital and parietal) repeated measures ANOVA was conducted for 270 to 400 ms time interval in theta response, and, a 2 group (EVGPs and non-players) x 3 hemispheres (lefts, medial, and right) repeated measures ANOVA was conducted for 70 to 130 ms and 70 to 140 ms time intervals in alpha response. Separate Greenhouse-Geisser corrected analyses employed a Bonferroni adjustment for inflated Type 1 error and α was assigned the value of 0.05 for each p among a set of p 's, such that a set of p values did not exceed a critical value. Analyses were carried out and reported for two experimental phases: 2-back and 3-back separately. However, only statistically significant results namely the 70 to 140 ms time interval for theta band and 270 to 400 ms time interval for the alpha band were reported here. On the other hand, other results were given in the Appendices section (see Appendix 1 to 6).

3.2.2 Results for theta oscillation. In this section, results for theta oscillation were presented for 2- and 3- back conditions, separately.

3.2.2.1 2-back condition. For theta latency values at 70 to 140 ms after stimulus onset, the main effect of hemisphere was significant, $F(2, 66) = 28.05$, $p < .001$, $\eta^2 = .50$. Follow-up analyses showed that, theta latency at 70 to 140 ms time interval were later at midline ($M = 108.74$, $SE = 3.04$) when compared to right ($M = 90.03$, $SE = 2.48$) and left hemisphere ($M = 88.55$, $SE = 2.17$). The interaction effect between

hemisphere and electrode were significant, $F(2, 66) = 13.74, p < .001, n^2 = .29$. Theta latency at Oz ($M = 101.90, SE = 4.05$) and Pz ($M = 115.58, SE = 3.64$) electrodes were later than the theta latency at O1 ($M = 93.85, SE = 3.04$) and P1 ($M = 83.25, SE = 1.93$) electrodes at 70 to 140 ms time interval, respectively (Table 4.).

Table 4

A 2x3x2 mixed design ANOVA results for Theta Latency at 70 to 140 ms After Stimulus Onset during 2-back Condition

Effect	<i>F</i>	<i>p</i>	η^2	Post Hoc
Group	2.806	.103	.078	
Region	.106	.747	.003	
Hemisphere	28.052	.000**	.459	<i>M > R & L</i>
Group*Region	.999	.325	.029	
Group*Hemisphere	.358	.701	.011	
Hemisphere*Region	13.736	.000**	.294	<i>Oz > O1 ; Pz > P7</i>
Group*Hemisphere*Region	1.138	.327	.033	

Note. * $p < .05$; ** $p < .01$.

L: Left, R: Right, M : Midline;.

For theta amplitude values at 70 to 130 ms after stimulus onset, the main effect of hemisphere, $F(2, 66) = 4.56, p < .05, n^2 = .12$, and the interaction effect between hemisphere and electrode, $F(2, 66) = 10.25, p < .001, n^2 = .24$, were significant. Follow-up analysis indicated that theta amplitude values were higher in midline hemisphere ($M = -.49, SE = .06$) than left hemisphere ($M = -.68, SE = .05$) at 70 to 130 ms time interval. In addition, theta amplitude at F3 ($M = -.48, SE = .05$) and P7 ($M = -.88, SE = .11$) electrodes were higher than the theta amplitude at F4 ($M = -.50, SE = .06$) and P8 ($M = -.70, SE = .13$) electrodes at 70 to 130 ms time interval, respectively. For theta latency values at 70 to 130 ms after stimulus onset, the main effect of electrode and hemisphere, and, the interaction effect between hemisphere and electrode were significant ($F \geq 11.59, p \leq .001$). Follow-up analysis indicated that, theta latency at 70 to 130 ms time interval were later in parietal electrodes ($M = 108.59, SE = 2.46$) in contrast to frontal electrodes ($M = 95.43, SE = 2.82$). Theta latency at 70 to 130 ms time interval were earlier in midline hemisphere ($M = 90.06, SE = 2.92$) than left ($M = 110.43, SE = 1.79$) and right ($M = 105.53, SE = 2.20$) hemispheres. Theta

latency at F4 ($M = 96.79$, $SE = 3.34$) and P7 ($M = 124.91$, $SE = 2.38$) electrodes were later than the theta latency at F3 ($M = 95.95$, $SE = 3.28$) and P8 ($M = 114.27$, $SE = 4.23$) electrodes at 70 to 130 ms time interval, respectively. However, the main effect of group were not significant for theta latency at 70 to 130 ms time interval ($F = .38$, $p = .55$). ANOVA results for theta latency and amplitude values at 70 to 130 ms time interval onset were reported in Appendix 1.

For theta amplitude values at 180 to 250 ms after stimulus onset, only the main effect of electrode was significant, $F(1, 33) = 4.55$, $p < .05$, $\eta^2 = .12$. Follow-up analysis indicated that, theta amplitude values at 180 to 250 ms time interval were higher in frontal electrodes ($M = -.31$, $SE = .06$) in contrast to parietal electrodes ($M = -.48$, $SE = .06$). No interaction effect for theta amplitude values at 180 to 250 ms time interval was not significant ($F \leq 2.89$, $p \geq .06$). For theta latency values at 180 to 250 ms time interval, only the main effect of electrode was significant, $F(1, 33) = 8.37$, $p < .05$. Follow-up analysis indicated that, theta latency at 180 to 250 ms time interval were later in frontal electrodes ($M = 226.67$, $SE = 3.43$) in contrast to parietal electrodes ($M = 210.17$, $SE = 3.83$). No interaction effect for theta latency values at 180 to 250 ms time interval was not significant ($F \leq 1.53$, $p \geq .22$). ANOVA results for theta amplitude and latency values at 180 to 250 ms after stimulus onset were reported in Appendix 2.

For theta amplitude values 70 to 140 ms after stimulus onset, neither main effects, ($F \leq 3.39$, $p \geq .08$), nor interaction effects, ($F \leq 2.43$, $p \geq .13$), were significant. ANOVA results for theta amplitude values 70 to 140 ms time interval were reported in Appendix 3.

For theta amplitude values at 130 to 240 ms after stimulus onset, neither main effects, ($F \leq 1.99$, $p \geq .15$), nor interaction effects, ($F \leq 8.68$, $p \geq .45$), were significant. For theta latency values at 130 to 240 ms after stimulus onset, only the main effect of electrode was significant, $F(1, 33) = 8.47$, $p < .05$. Follow-up analysis indicated that, theta latency were later in occipital electrodes ($M = 187.02$, $SE = 4.95$) in contrast to frontal electrodes ($M = 167.64$, $SE = 3.79$) at 130 to 240 ms time interval. No interaction effect for theta latency values at 130 to 240 ms time interval was significant ($F \leq 1.06$, $p \geq .35$). ANOVA results for theta amplitude and latency values at 130 to 240 ms time interval were reported in Appendix 4.

For theta amplitude values at 270 to 400 ms after stimulus onset, only the main effect of electrode was significant, $F(1, 33) = 6.44, p < .05, n^2 = .16$. Follow-up analysis indicated that, theta amplitude values were higher in parietal electrodes ($M = .73, SE = .08$), in contrast to occipital ($M = .62, SE = .08$) and frontal ($M = .47, SE = .05$) electrodes at 270 to 400 ms time interval. No significant interaction effect for theta amplitude at 270 to 400 ms time interval was found ($F \leq 1.05, p \geq .39$). For theta latency values at 270 to 400 ms time interval, neither main effects, ($F \leq .41, p \geq .62$), nor interaction effects, ($F \leq 2.17, p \geq .12$), were significant. ANOVA results for theta amplitude and latency values at 270 to 400 ms time interval were reported in Appendix 5.

3.2.2.2 3-back condition. For theta latency values at 70 to 140 ms after stimulus onset, the main effect of group was significant, $F(1, 33) = 6.53, p < .01, n^2 = .17$. Follow-up analyses showed that, theta latency were later in EVGP group ($M = 103.60, SE = 2.67$) when compared to NP group ($M = 94.29, SE = 2.47$) at 70 to 140 ms time interval. The interaction effect between group and electrode were significant, $F(1, 33) = 4.23, p < .05, n^2 = .12$ (Figure 6.). Respectively, theta latency values obtained from the occipital electrodes were later in EVGP group ($M = 107.25, SE = 4.12$) in comparison to the NP group ($M = 91.94, SE = 3.78$) at 70 to 140 ms time interval. For theta latency values at 70 to 140 ms time interval, the main effect of hemisphere was found significant, $F(2, 66) = 34.08, p < .001, n^2 = .51$. Follow-up analyses indicated that, theta latency were later at midline electrodes ($M = 113.72, SE = 2.93$) when compared to right ($M = 92.76, SE = 2.68$) and left ($M = 90.35, SE = 1.97$) hemisphere at 70 to 140 ms time interval. In addition, the interaction effect between region and hemisphere were significant, $F(2, 66) = 10.80, p < .001, n^2 = .25$. Theta latency at Oz ($M = 107.58, SE = 3.76$) and Pz ($M = 119.87, SE = 3.51$) electrodes were later than the theta latency at O1 ($M = 95.51, SE = 3.48$) and P7 ($M = 85.19, SE = 2.00$) electrodes at 70 to 140 ms time interval, respectively (Table 5.).

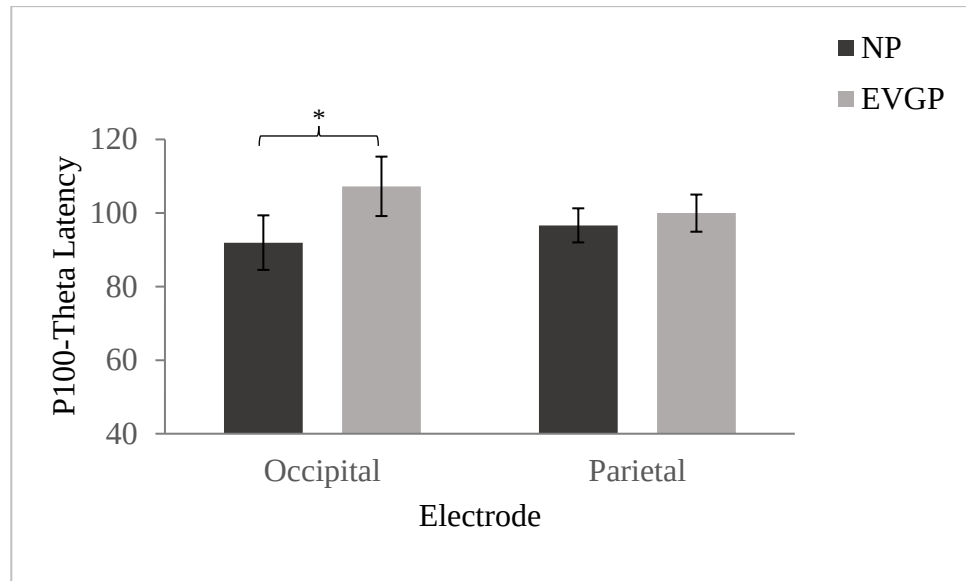


Figure 7 The interaction effect between group and electrode for theta latency at 70 to 140 ms in 3-back condition.

Table 5

A 2x3x2 mixed-design ANOVA results for Theta Latency at 70 to 140 ms After Stimulus Onset during 3-back Condition

Effect	<i>F</i>	<i>p</i>	η^2	<i>Post Hoc</i>
Group	6.527	.015*	.165	EVGP > NP
Region	.199	.658	.006	
Hemisphere	34.075	.000	.508	M > L, R
Group*Region	4.233	.048*	.114	Occipital: EVGP > NP
Group*Hemisphere	.072	.931	.002	
Hemisphere*Region	10.797	.000**	.247	Oz > O1; Pz > P7
Group*Hemisphere*Region	.074	.929	.002	

Note. * $p < .05$; ** $p < .01$.

EVGP: Excessive Video Game Players, NP: Non-players; L: Left, R: Right, M: Midline;.

For theta amplitude values at 70 to 130 ms after stimulus onset, only the main effect of hemisphere, $F(2, 66) = 5.44$, $p < .05$, $\eta^2 = .14$, and the interaction effect between hemisphere and electrode, $F(2, 66) = 11.62$, $p < .001$, $\eta^2 = .26$, were significant. Follow-up analysis indicated that, N100-theta amplitude were higher in midline hemisphere ($M = -.41$, $SE = .06$) than left hemisphere ($M = -.63$, $SE = .06$).

Theta amplitude values at F3 ($M = -.42$, $SE = .06$) and Pz ($M = -.33$, $SE = .07$) electrodes were higher than Fz ($M = -.50$, $SE = .07$) and P7 ($M = -.84$, $SE = .11$) electrodes at 70 to 130 ms time interval, respectively. For theta latency values at 70 to 130 ms time interval, the main effect of hemisphere, and, the interaction effect between hemisphere and electrode were significant ($F \geq 40.60$, $p \leq .01$). Follow-up analysis indicated that, theta latency at midline ($M = 89.58$, $SE = 2.56$) were earlier than left ($M = 111.50$, $SE = 1.73$) and right ($M = 105.92$, $SE = 2.50$) hemispheres at 70 to 130 ms time interval. Moreover, theta latency at F3 ($M = 97.99$, $SE = 3.00$) and P7 ($M = 125.01$, $SE = 2.29$) electrodes were later than the P100-theta latency at F4 ($M = 97.00$, $SE = 3.27$) and Fz ($M = 95.94$, $SE = 2.97$); and, P8 ($M = 114.83$, $SE = 3.99$) and Pz ($M = 83.21$, $SE = 3.45$) electrodes at 70 to 130 ms time interval, respectively. However, the main effect of game group were not significant for theta latency at 70 to 130 ms time interval ($F = .38$, $p = .55$). ANOVA results for theta latency and amplitude values at 70 to 130 ms time interval were reported in Appendix 1.

For theta amplitude values at 180 to 250 ms after stimulus onset, neither main effects, ($F \leq 1.87$, $p \geq .92$), nor interaction effects, ($F \leq .69$, $p \geq .51$), were not significant. For theta latency values at 180 to 250 ms time interval, only the main effect of electrode was significant, $F(1, 33) = 8.37$, $p < .05$. Follow-up analysis indicated that, latency at 180 to 250 ms time interval were later at frontal electrodes ($M = 224.66$, $SE = 3.73$) than parietal electrodes ($M = 210.23$, $SE = 4.07$). No interaction effect for theta latency values was not significant at 180 to 250 ms time interval ($F \leq 1.53$, $p \geq .22$). ANOVA results for theta amplitude and latency values at 180 to 250 ms time interval were reported in Appendix 2.

For theta amplitude values at 70 to 140 ms after stimulus onset, neither main effects, ($F \leq 2.01$, $p \geq .17$), nor interaction effects, ($F \leq 1.66$, $p \geq .21$), were significant. ANOVA results for theta amplitude values at 70 to 140 ms time interval were reported in Appendix 3.

For theta amplitude values at 130 to 240 ms after stimulus onset, neither main effects, ($F \leq 2.85$, $p \geq .08$), nor interaction effects, ($F \leq 22.56$, $p \geq .26$), were significant. For theta latency values at 130 to 240 ms after stimulus onset, only the main effect of electrode was significant, $F(1, 33) = 5.82$, $p < .05$. Follow-up analysis indicated that, theta latency at occipital electrodes ($M = 191.42$, $SE = 4.60$) were later than frontal electrodes ($M = 175.79$, $SE = 5.38$) at 130 to 240 ms time interval. No

interaction effect for theta latency values was not significant at 130 to 240 ms time interval ($F \leq 3.06, p \geq .05$). ANOVA results for theta amplitude and latency values at 130 to 240 ms time interval were reported in Appendix 4.

For theta amplitude values at 270 to 400 ms after stimulus onset, only the main effect of electrode was significant, $F(1, 33) = 14.14, p < .01, \eta^2 = .30$. Follow-up analysis indicated that, theta amplitude values at frontal electrodes ($M = .36, SE = .04$) were lower than parietal ($M = .71, SE = .06$) and occipital ($M = .61, SE = .08$) electrodes at 270 to 400 ms time interval. Interaction effect for N300-theta amplitude values was not significant ($F \leq 1.86, p \geq .12$). For theta latency values, neither main effects, ($F \leq 3.28, p \geq .06$), nor interaction effects, ($F \leq 2.29, p \geq .08$), were significant at 270 to 400 ms time interval. ANOVA results for theta amplitude and latency values at 270 to 400 ms time interval were reported in Appendix 5.

3.2.3 Results for alpha oscillation. In this section, results for theta oscillation were presented for 2- and 3- back conditions, separately.

3.2.3.1 2-back condition. For alpha latency values at 270 to 400 ms after stimulus onset, the interaction effect between hemisphere and electrode were significant, $F(2, 66) = 3.48, p < .05, \eta^2 = .10$. Follow-up analysis indicated that, alpha latency at P8 electrodes ($M = 341.93, SE = 4.24$) were later than the latency at Pz electrodes ($M = 323.00, SE = 5.27$) at 270 to 400 ms time interval (Table 6.).

Table 6

A 2x3x2 mixed design ANOVA results for Alpha Latency at 270 to 400 ms After Stimulus Onset during 2-back Condition

Effect	<i>F</i>	<i>p</i>	η^2	<i>Post Hoc</i>
Group	.134	.716	.004	
Region	2.852	.101	.080	
Hemisphere	3.096	.052	.086	
Group*Region	.025	.876	.001	
Group*Hemisphere	1.509	.229	.044	
Hemisphere*Region	3.871	.026*	.105	P8 > Pz
Group*Hemisphere*Region	1.110	.336	.033	

Note. * $p < .05$; ** $p < .01$.

EVGP: Excessive Video Game Players, NP: Non-players;.

For alpha amplitude values at 70 to 130 ms after stimulus onset, neither the main effects nor the interaction effect was significant, ($F \leq 6.42, p \geq .14$). For alpha latency values at 270 to 400 ms after stimulus onset, neither the main effects nor the interaction effect was significant, ($F \leq 1.51, p \geq .23$). ANOVA results for alpha latency and amplitude values at 270 to 400 ms time interval were reported in Appendix 6.

For alpha amplitude values at 180 to 250 ms after stimulus onset, neither main effects, ($F \leq 2.76, p \geq .08$), nor interaction effects, ($F \leq 1.45, p \geq .24$), were significant. For alpha latency values at 180 to 250 ms after stimulus onset, neither main effects, ($F \leq 2.76, p \geq .08$), nor interaction effects, ($F \leq 1.45, p \geq .24$), were significant. ANOVA results for alpha amplitude and latency values at 180 to 250 ms time interval were reported in Appendix 7.

For alpha amplitude values at 70 to 140 ms after stimulus onset, only the main effect of the hemisphere was significant ($F = 5.35, p = .01$). Follow-up analysis indicated that, alpha amplitude values at midline hemisphere ($M = .50, SE = .06$) were smaller than left ($M = .63, SE = .08$) and right ($M = .62, SE = .07$) hemispheres at 70 to 140 ms time interval. The interaction effect between group and hemisphere was not significant for alpha amplitude values at 70 to 140 ms time interval ($F = .64, p = .53$). For alpha latency values at 70 to 140 ms time interval, neither main effects nor interaction effect were significant ($F \leq 2.70, p \geq .11$). ANOVA results for alpha latency and amplitude values at 70 to 140 ms time interval were reported in Appendix 8.

For alpha amplitude values at 130 to 240 ms after stimulus onset, neither main effects, ($F \leq 2.96, p \geq .06$), nor interaction effect, ($F = .22, p = .80$), were significant. For alpha latency values, neither main effects nor interaction effect were significant at 70 to 140 ms time interval ($F \leq 1.48, p \geq .82$). ANOVA results for alpha amplitude and latency values at 70 to 140 ms time interval were reported in Appendix 9.

For alpha amplitude values at 270 to 400 ms after stimulus onset, neither main effects, ($F \leq 2.59, p \geq .12$), nor interaction effect, ($F \leq 3.79, p \geq .60$), were significant. ANOVA results for alpha amplitude values at 270 to 400 ms time interval were reported in Appendix 10.

3.2.3.2 3-back condition. For alpha latency values at 270 to 400 ms after stimulus onset, the interaction effect between group and electrode was significant, $F(1, 33) = 8.51, p < .05, \eta^2 = .21$ (Figure 7.). Follow up analyses indicated that, alpha

latency values of EVGP group ($M = 333.36$, $SE = 6.89$) were later at occipital electrodes, in comparison to NP group ($M = 315.08$, $SE = 5.97$) at 270 to 400 ms after stimulus onset (Table 7.).

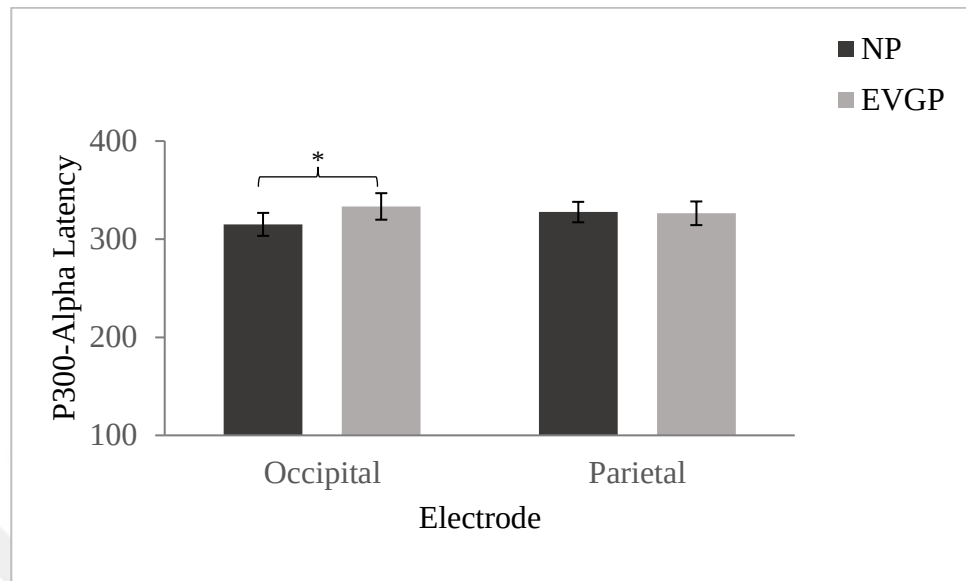


Figure 8. The interaction effect between group and electrode for alpha latency at 270 to 400 ms in 3-back condition.

Table 7

A 2x3x2 mixed design ANOVA results for Alpha Latency at 270 to 400 ms After Stimulus Onset during 3-back Condition

Effect	<i>F</i>	<i>p</i>	η^2	<i>Post Hoc</i>
Group	1.147	.292	.034	
Electrode	.691	.412	.021	
Hemisphere	.073	.929	.002	
Group*Electrode	8.507	.006*	.205	Occipital: EVGP > NP
Group*Hemisphere	.168	.846	.005	
Hemisphere*Electrode	1.145	.324	.034	
Group*Hemisphere*Electrode	.883	.419	.026	

Note. * $p < .05$; ** $p < .01$.

EVGP: Excessive Video Game Players, NP: Non-players.

For alpha amplitude values at 70 to 130 ms after stimulus onset, neither the main effects nor the interaction effect was significant, ($F \leq 3.40$, $p \geq .05$). For alpha latency

values at 70 to 130 ms after stimulus onset, the main effect of the hemisphere was significant, $F(1, 33) = 348.19, p \leq .01$. Follow-up analysis indicated that, alpha latency at midline hemisphere ($M = -.45, SE = .07$) were earlier than left ($M = 107.53, SE = 4.29$) and right ($M = 106.37, SE = 4.21$) hemispheres at 70 to 130 ms time interval. However, the interaction effect between group and hemisphere was not significant for alpha latency at 270 to 400 ms time interval ($F = .27, p = .78$). ANOVA results for alpha latency and amplitude values at 270 to 400 ms time interval were reported in Appendix 6.

For alpha amplitude values at 180 to 250 ms after stimulus onset, neither main effects, ($F \leq 2.44, p \geq .13$), nor interaction effects, ($F \leq 1.80, p \geq .17$), were significant. For alpha latency values, neither main effects, ($F \leq 2.44, p \geq .13$), nor interaction effects, ($F \leq 1.80, p \geq .19$), were not significant. ANOVA results for alpha amplitude and latency values at 180 to 250 ms time interval were reported in Appendix 7.

For alpha amplitude values at 70 to 140 ms after stimulus onset, neither main effects, ($F \leq 1.17, p \geq .32$), nor interaction effect, ($F = 2.07, p = .13$), were significant. For alpha latency values at 70 to 140 ms after stimulus onset, only the main effect of hemisphere was significant ($F = 6.58, p = .02$). Follow-up analysis indicated that, alpha latency at midline hemisphere ($M = 106.08, SE = 3.48$) were later than left ($M = 96.86, SE = 3.02$) and right ($M = 94.64, SE = 3.66$) hemispheres at 70 to 140 ms time interval. The interaction effect between group and hemisphere for P100-alpha latency were not significant ($F = .60, p = .55$). ANOVA results for alpha latency and amplitude values at 70 to 140 ms time interval were reported in Appendix 8.

For alpha amplitude values at 130 to 240 ms after stimulus onset, only the main effect of hemisphere was significant ($F = 3.80, p = .04$). Follow-up analysis indicated that, alpha amplitude values were higher at right hemisphere ($M = .63, SE = .06$) in contrast to midline ($M = .53, SE = .05$) at 130 to 240 ms time interval. The interaction effect between group and hemisphere was not significant for alpha amplitude at 130 to 240 ms time interval, ($F = .07, p = .90$). For alpha latency values at 130 to 240 ms after stimulus onset, neither main effects, ($F \leq 3.47, p \geq .07$), nor interaction effect, ($F = .34, p = .71$), were significant. ANOVA results for alpha amplitude and latency values at 130 to 240 ms time interval were reported in Appendix 9.

For alpha amplitude values at 270 to 400 ms after stimulus onset, neither main effects, ($F \leq 1.45, p \geq .24$), nor interaction effect, ($F \leq 1.53, p \geq .23$), were significant.

ANOVA results for alpha amplitude values at 270 to 400 ms time interval were reported in Appendix 10.

In sum, although there were no group differences in behavioral performance (RTs and accuracy), we found that EVGPs theta latency at 70 to 140 ms after stimulus onset were later than NPs only in the 3-back condition. Furthermore, EVGPs theta latency at 70 to 140 ms time interval and alpha latency at 270 to 400 ms time interval at occipital electrodes were later than NPs in 3-back condition.



Chapter 4

Discussion

4.1 Overview

The present research aimed to investigate the effects of excessive video gaming on WM processes and the neural basis of the WM load. Thus, selected EROs were recorded and analyzed during 2- and 3-back tasks to compare the WM ability of EVGPs and NPs. The EVGP group was selected by looking at their higher violent video gaming hours per week and their Game Addiction Scale and Pathological Game Addiction Symptom List scores in contrast to NPs. In addition, participants' anxiety and depression scores were measured by STAI and BDI-I since anxiety and depression had high comorbidity rates with excessive gaming (Toker & Baturay, 2015; Youh et al., 2017). The number of true and false responses, hits, and false alarms was calculated separately for both 2- and 3-back conditions. Likewise, RTs were calculated for only hits and false alarms. Behavioral analyses were conducted by using ANOVA to compare EVGPs' and NPs' RTs and accuracy. For ERO analyses, theta frequencies were obtained by filtering the data between 4.1 and 8 Hz, and alpha frequencies were obtained by filtering the data between 8.1 and 12 Hz at frontal, central, parietal, and occipital regions. Amplitudes and latency values of at 70 to 140 ms (P100 component) and 270 to 400 ms (P300 component) time windows were calculated in theta and alpha bands. Therefore, statistical analyses were calculated by using mixed-design ANOVA (group x hemisphere x region) for 2- and 3-back conditions, separately. In this section, considering the literature and findings, behavioral and ERO results were evaluated and discussed.

4.2 Discussion for Behavioral Results

Behavioral results indicated that neither true and false responses nor RTs of the EVGP group did not differ from than NP group in 2- and 3-back tasks. In other words, EVGP's accuracy and pace did not change with the increase in WM load and their performance was like NPs, on a behavioral level. These findings contradict our

expectations about EVGP's WM performance since the EVGP group has been expected faster RTs with lower accuracy due to excessive action video-game playing was found to be related to structural and/or functional alterations in the brain in a negative way.

Our results showed that EVGPs were not susceptible to the increase in WM load in contrast to NPs. Contrary to our results, several researchers found that EVGPs had faster RTs and correct responses than NPs with the increase in WM load (Moisala et al., 2016; Colzato et al., 2013). Our results were partly in line with other research which has found that EVGPs and NPs had similar performance in terms of accuracy but not RTs (Irak & Soylu, 2021; Irak, Soylu & Çapan, 2016; McDermott et al., 2014; Boot et al., 2008). Irak et al. (2016) found that, in contrast to NPs, risk group (playing video games 5–16 h a week with 1 or 2 game addiction symptoms) and EVGPs had slower RTs for correct responses without any differences in accuracy in WM task, indicating slower recognition process of previously encoded stimulus. Similar to our findings, Llamas-Alonso et al., (2019) found no behavioral performance differences between action video game players and NPs in the *N*-back task although there were significant changes in rEEG. Authors argued that since only the 1-back task was used in that study participants might find it easier. Other factors may play role in performance differences such in EVGPs had similar performance in the *N*-back task only when they had heavy multitasking habits in contrast to medium multitasking (Cardoso-Leite et al., 2015).

Findings from IA research indicated that people with high IA showed faster RTs than the control group, but the accuracy of both groups was similar against increased WM load (Wang et al., 2020). Pathological gamblers (Yan et al., 2014) and methamphetamine-addicted individuals (Simon et al., 2000) also showed similar behavioral results in terms of RTs and accuracy with healthy controls in the *N*-back task. People with nicotine dependence and excessive marijuana users might have similar neural mechanisms to IGD and had similar behavioral performances with our results (Yan et al., 2021; Ge et al., 2017; Ko et al., 2013; Smith, Hummer & Hulvershorn, 2015). With the increase in WM load, a decrease in alpha (Ilan, Smith & Gevins, 2004) and theta was observed in nicotine-dependent individuals (Fisher et al., 2011) and excessive marijuana users (Ilan et al., 2004) without any significant behavioral differences in RTs and accuracy. One can argue that excessive game

playing does not cause any differences in monitoring skills since none of the research reported different behavioral results in 1-back task, but the differences seen in harder tasks, mostly after the n increased up to three (Moisala et al., 2016; Colzato et al., 2013). Hence, the relationship between excessive gaming activity and maintenance of the information in WM should be in consideration. It also should be noted that N -back task does not include all the components of WM like the phonological loop or visuospatial sketchpad. In our research, we used verbal stimulus (e.g. letters) so we can only contemplate our findings in terms of monitoring and maintenance of verbal stimulus in resistance to WM load. To understand whether excessive game playing affects other components of WM, additional verbal, non-verbal, or digit span tasks can be used.

Different results can be explained by the sample differences. First, other research did not measure the pathological gaming level, and some included professional game players, who might have different brain functions than pathological gamers (Han et al., 2012). Moreover, none of the research did control anxiety and depression levels. As Green et al., (2017) suggested, there are a lot of factors that may affect game players' WM performance like game genre, played hours per week, and playing length. In addition, EVGP's superior performance to NPs might also depend on several individual differences like multitasking ability (Cardoso-Leite et al., 2015) and suitable genetic predisposition on the modulation of the degradation of dopamine (Colzato, Wildenberg & Hommel, 2014). Gender (Jun, Sacco, Bright, & Cunningham-Williams, 2019) and different types of IA like internet pornography usage (Laier, Schulte, & Brand, 2013) and IGD (Du et al., 2017) were also specified as important indicators in N -back performance in people with other behavioral addictions. Since IA and EVGP are usually comorbid, these factors should be taken into consideration. Given that, our EVGP group included participants who are high in-game addiction scores and symptoms, it is reasonable to find contradictory behavioral results with previous research.

Even though we controlled the playing hours, game genre, anxiety, and depression with self-report, another confounding factor onset was not controlled. Such early onset AUD generates more structural changes in the brain and on behavior (De Bellis et al., 2005), the more time a person spends with excessive gaming activity, the more negative effects can be observed. In adults game playing onset and total played

hours of action video games were found to be negatively correlated to the structural grey matter differences in the Entorhinal cortex (Kühn & Gallinat, 2014), which had strong connections with the hippocampus. The entorhinal cortex is associated with the encoding, monitoring, and recognizing of the information in the environment (Eichenbaum & Yonelinas, 2007; Howard, Fotedar, Datey & Hasselmo, 2007), regardless of the place where people locate themselves in space (Jacobs et al., 2010). This finding only pertained to action video games but other game genres that were positively correlated with the GM volume and the reason behind it was not clear. Since the *N*-back task assesses the maintenance and monitoring of the information in the WM and action game playing onset creates structural changes in the related brain area, onset and total played might be crucial factors.

4.3 Discussion for ERO Results

EVGPs showed later theta latency at 70 to 140 ms and alpha latency at 270 to 400 ms in the occipital region compared to NPs, only in the 3-back condition. In addition, only the theta latency values at 70 to 140 ms after the stimulus were modulated by the group in the 3-back condition. The group effect was not significant on the amplitude and latency of alpha and theta responses at 70 to 140 ms, 130 to 240 ms, and 270 to 400 ms time intervals in the 2-back condition. Thus, as the WM load increased, EVGP's theta and alpha latencies were prolonged compared to NPs. Our other results for the theta band indicated that, while theta latency at 70 to 130 ms time interval was later in the parietal region, theta latency at 180 to 250 ms was later in the frontal region than the parietal region only in the 2-back condition. Theta latency at 70 to 130 ms time interval was higher in the midline electrodes than the electrodes in the left hemispheres in both conditions. Theta latency at 70 to 140 ms time interval was observed later at occipital regions in the midline electrodes both in 2- and 3-back conditions. In both conditions, theta latency at 130 to 240 ms time interval was earlier in the frontal region than occipital region. There were no latency differences in terms of theta activity at 270 to 400 ms time interval in neither condition. However, while theta amplitude was bigger in the parietal region in the 2-back condition, theta amplitude was smaller in the frontal region in the 3-back condition at 270 to 400 ms time interval. Other results for the alpha band indicated that there were no alpha

amplitude differences in either condition at 70 to 130 ms and 180 to 250 ms time intervals but alpha midline latency was later than left and right hemispheres in both conditions at 70 to 130 ms time interval. However, alpha midline latency at 70 to 140 ms time interval was later than left and right hemispheres, and alpha amplitude at 130 to 240 ms time interval was bigger in the right hemisphere than midline, only in the 3-back condition. There were no alpha amplitude differences in either condition at 270 to 400 ms time interval. Thus, we can conclude that theta activity at 70 to 130 ms (N100), 180 to 250 ms (N200), 70 to 140 ms (P100) and 270 to 400 ms (P300) time intervals and alpha activity at 70 to 140 ms (P100), 130 to 240 ms (P200) and 270 to 400 ms (P300) time intervals showed differences with the increase in WM load, regardless of the group status.

Previous research examined the neurophysiological changes due to WM load by using the *N*-back task in various subjects and it is proven that it assesses the impact of WM load on the brain effectively (Irak et al., 2020a; Pergher et al., 2019; Chen & Huang, 2016; Brouwer et al., 2012; Palomäki et al., 2012; Başar-Eroğlu et al., 2007; Sauseng et al., 2005; Klimesch et al., 2004) but there is a limited amount of research on the excessive gaming's or other behavioral addictions' reflection on brain oscillations. In general, research with healthy subjects indicated that an increase in WM load can be traced to differences in alpha and theta bands (Pergher et al., 2019; Sauseng et al., 2005; Klimesch, Schack & Lipka, 2004; Klimesch, 1996). In all age groups and healthy subjects, P300 amplitude decrease with the increase in WM load (Käthner et al., 2014) but this decrease is more pronounced in older adults who may show WM deficits (Pergher et al., 2019; Babiloni et al., 2006; Rossini et al., 2007). Our results of later frontal N200-theta activity in 2-back condition and earlier frontal P200-theta activity with an increase in WM load were specified as a task-independent result in our sample's age group (Missonier et al., 2011). Likewise, parietal P300-theta reached its peak in the 2-back condition, but the lowest amplitude values were obtained from frontal P300-theta with the increase in WM load. This was in line with previous research reported that slower RTs were related to smaller parietal and frontal P300 amplitudes with the increase in WM load (Irak et al., 2020a).

Differences in theta and alpha band activities in frontal and parietal regions supported the notion that the fronto-posterior brain network is actively involved in WM processes. Earlier theta and later alpha activities in midline electrodes also

support the fact that WM load has been created effectively, as midline posterior alpha and frontal theta activities are reported to be related to the trying to be vigilant (Caldwell et al., 2003), increased mental effort and the amount of the information required to be manipulated (Deiber et al., 2007; Dai et al., 2017; Maurer et al., 2014; Onton, Delorme & Makeig, 2005; Allison & Polich, 2008; Brouwer et al., 2012). It was argued that increased frontal midline theta activity was located near the dlPFC and ACC, which were reported to be connected to the increase in workload (Ragland et al., 2002; Barch et al., 1997; Gould et al., 2003). This might refer to the more globally efficient network structure in the theta band (Dai et al., 2017) and resulted in an improved attention system for the maintenance of information in the WM to resist more efficiently heightened WM load (Jensen & Tesche, 2002; Hsieh & Ranganath, 2014; Roux & Uhlhaas, 2014). In addition, alpha was reported to be related to the inhibition of other brain regions which did not relate to the current task (Klimesch et al., 2007; Scheeringa et al., 2012; Uusberg et al., 2013) and the trend of weakened alpha response with the increase in WM load was explained with functionally related brain regions in the alpha-band network become weaker during N-back task (Dai et al., 2017). Altogether, our findings were in line with general increasing theta and decreasing alpha activity patterns during the *N*-back task, suggesting that the transition from 2- to 3-back task created WM load successfully in our sample, both in EVGP and NP groups. It should be noted that the lack of theta and alpha activity in other areas did not mean that they are not involved in other WM processes. The *n*-back task can only assess maintenance and monitoring of the information but not the encoding, retention, and recall of the memorized items (Jensen & Tesche, 2002). Thus, it is not possible to fully comprehend the reason behind the increased theta. To solve this problem, another type of WM tasks like the Sternberg Task, which assesses several working memory processes like encoding and retention separately, can be used in further studies.

As mentioned, no research ever investigated the oscillatory activity of the EVGPs due to an increase in WM load by using the N-back task. We could not find any amplitude differences in theta activity at 70 to 140 ms time interval and alpha activity at 270 to 400 ms time interval between the two groups but EVGPs had later theta latency at 70 to 140 ms time interval and alpha latency at 270 to 400 ms in contrast to NPs when the WM load was increased. Significant alterations in latency in

the EVGP group may indicate differences in neural processing such as visual attention and stimulus classification. While amplitude depends on the degree of information processing and relative to the number of attentional resources which was devoted to the task, latency is considered a measure for stimulus classification and information processing speed (Patel and Azzam, 2005). Klimesch et al., (1999) suggested that earlier alpha activity corresponds to the active information processing while later alpha was related to the inhibition of the unwanted stimulus in an *N*-back task that used letters and numbers. Moreover, higher WM load and alpha were positively correlated in the frontoparietal network and are essentially active during memory retention (Palva & Palva, 2007; Jensen et al., 2002). On the other hand, as mentioned theta amplitude, especially in the frontal region, was also found to be associated with an increase in mental effort and task requirement (Jensen et al., 2002). It has been suggested that latency differences at 70 to 140 ms after stimulus onset is mostly related to the stimulus characteristics and increases in the presence of the uncommon stimulus; and, differences at 270 to 400 ms after stimulus onset corresponds to the degree of mismatched stimulus and increases as the uncommon stimulus became similar to a common stimulus (Mehaffey, Seiple & Holopigian, 1993). According to our findings, excessive action video game playing may alter the theta and alpha responses at 70 to 140 ms and 270 to 400 ms after stimulus onset, respectively; however, behavioral reflections of these findings remain unclear.

Amplitude and latency differences at 70 to 140 ms after stimulus onset were most likely refer to the early visual processing and selective attention, localized in the visual cortices (Hillyard & Anllo-Vento, 1998). WM maintenance was linked to early attentional factors (Sreenivasan et al., 2007) and P100 latency during the encoding stage of the selective stimulus was positively correlated with the effect WM maintenance, which was assessed with the successful recognition after the maintenance period (Rutman, Clapp, Chadick & Gazzaley, 2010). ERP modulations (e.g. N170 and P100) at the early stages of the visual processing may be the result of selective attention (Khoe, Mitchell, Reynolds & Hillyard, 2005). It is not clear that whether WM had an essential role in selective attention processes or vice versa (Awh & Jonides, 2001; de Fockert, Rees, Frith, & Lavie, 2001). Selective attention during early visual processing might be utilized through the WM maintenance processes by modulating the distractors (Sreenivasan, Katz, & Jha, 2007; Sreenivasan & Jha, 2007).

Authors argued that this top-down modulation helps us to maintain relevant information to keep in mind for further use. In addition, event-related synchronization (ERS) of theta was found to be related to the memory encoding and retrieval of visual stimulus (Jensen et al., 2002; Karrasch et al., 2004), and its long-lastingness may reflect higher cognitive processing such as maintenance or manipulation of information in *N*-back task (Palomäki et al., 2012). Thus, increased latency in the theta band at 70 to 140 ms after stimulus onset may be explained by EVGP's less efficient WM processes in determining the stimulus characteristic and maintaining it to use later, in contrast to NPs.

In ERP literature latency differences at 270 to 400 ms time window was found as an indicator for the performance of matching the stimulus with the *n* item ago (Watter et al., 2001). Later responses at 270 to 400 ms after stimulus onset were also associated with slowness in the identification and decision-making processes about the stimulus (Luo, 1990). Increased latency at 270 to 400 ms after stimulus onset might also refer to the memory problems since it became later with the additional tasks (Yu et al., 2009), and people with dementia have later P300 responses (Luo, 1990). People with IGD showed later P300 responses in an oddball task, which indicates the reduced speed of stimulus identification and memory decline (Yu et al., 2009). Correspondingly, later responses at 270 to 400 ms time window were linked to the increase in difficulty levels in the categorization of the stimulus (Kok, 2001). An increase in the alpha rhythm was characterized by the encoding phase of WM and top-down attentional mechanisms (Freunberger et al., 2003; Palva & Palva 2007) while earlier and increased posterior alpha were related to the retention of the visually learned material (Jensen et al., 2002). Duration of the alpha also increases with the higher WM load and task difficulty, which can be seen in longer RTs in the 3-back task (Pesonen et al., 2007; Krause et al., 2010). Later alpha response at 270 to 400 ms after stimulus onset might be related to an inefficient cognitive effort to allocate attentional resources during retention of the stimulus and matching period for EVGP individuals. Consequently, the long latency of EVGP's theta responses at 70 to 140 ms and alpha responses at 270 to 400 ms time windows were significant, especially in the occipital region. Given that, EVGPs' successful 3-back performance and coping with WM load may highly depend on neural populations in the occipital cortex but NPs' 3-back performance may depend on the activation of neural populations outside of the

occipital cortex. Wilms et al., (2021) suggested that action video gaming does not have a direct effect on visual skills but with the increase in playing, differences can be seen in the encoding of the visual information in the brain. This finding supported our findings on latency differences in the occipital region without RT differences. Excessive game playing might have created some specific changes in the occipital cortex due to the plasticity of the brain (Gong et al., 2019; Green & Bavelier, 2015) but this significant difference did not reflect on the behavioral performance yet in addition, our results did not support the argument that excessive gaming and its effects are related to the parietal activity (Wu et al., 2012). Longitudinal studies are needed in these regards.

Supporting previous studies (e.g., Irak et al., 2020a; Blacker & Curby; Boot et al., 2008), we concluded that EVGPs are mostly like non-players in *N*-back task, but excessive gaming might negatively affect the processes of encoding and storing visual information when the working memory load increases. Furthermore, excessive game playing, and other behavioral addictions share similar neural mechanisms (Irak & Soylu, 2021). However, unlike previous studies (Wang et al., 2020), it is difficult to conclude that excessive video game playing enhances brain networks responsible for the maintenance of information in WM and visual attention, The exact neurophysiological reason behind significant latency differences without amplitude differences and similar behavioral results between groups remains unclear. However, since the theta response at 70 to 140 ms time interval is related to the visual stimulus characteristics and the alpha response at 270 to 400 ms time interval is related to the attention and retention of stimulus, the reason might be that the letter stimulus used in this study might be too simple, or the 3-back task might not be hard enough for EVGPs. In future research, the increase of the *n* to more than three might help assess the effect of WM load more effectively.

When interpreting the results, it must be kept in mind that frontal midline theta is especially vulnerable to individual differences like neuroticism and avoidance as theta responses in lateral frontal networks are observed in adverse behavioral and emotional reactions to aversive events (Neo & McNaughton, 2011). Moreover, anxiety levels are an important factor since frontal-midline theta activity increased in the midcingulate cortex may be moderated by the level of anxiety and adjusted behavior to uncertainty (Cavanagh & Shackman, 2015). GD and major depressive disorder

(MDD) comorbidity were found to be related to a lower alpha band in frontal electrodes with a higher alpha band in left parietal and occipital electrodes (Youh et al., 2017). Moreover, people with both GD and ADHD had higher intra-hemispheric coherence in theta, alpha, and beta bands, in comparison to people with only ADHD or GD and NPs (Park et al., 2017b). Mental fatigue and decreased arousal may lead to increased alpha activity as a result of attention deficits (Käthner et al 2014) as alpha band increases with the sleepiness (Stampi, Stone & Michimori, 1995), attention lapses (O'Connell et al., 2009) and the old age (Pergher et al., 2009). Thus, attention and WM-related problems due to excessive gaming increase with other psychiatric and neurological problems like depression and ADHD because of the decreased interhemispheric connectivity in frontal areas combined with the increase in alpha in other areas.

4.4 Strengths and Limitations of the Current Study

To the best of our knowledge, the current study is the first study to investigate neural correlates of excessive violent video gamers by examining event-related oscillations during the *N*-back task. Additionally, little research has been done on the ERP responses of EVGPs during WM task (Irak & Soylu, 2021; Irak et al., 2020). Another ERP research investigated the inhibition processes of EVGPs by using Go/NoGo paradigm and found EVGPs had weaker inhibition capacity in contrast to NPs (Irak, Soylu, Tümen & Turan, 2020). Event-related theta and alpha oscillations are being studied widely in various samples and they are mostly associated with WM load. Therefore, it is important to shed light on event-related theta and alpha activities at above mentioned time windows, to understand the neural correlates of EVGPs' WM processes mechanisms. We studied the 70 to 140 ms time interval for P100 peak and 270 to 400 ms time interval for P300 peak as they have mostly been linked to visual and attentional processes in WM, which are closely related to the game playing experience.

Since this study and findings are novel to the literature, there are some limitations we should address and must be considered in future research. First, EVGPs' onset and length of playing violent video games was not controlled, which might influence their pathological game playing level. Moreover, we chose participants who

played violent video games based on the weekly gaming hours and participants rated the level of violence of the games they played. Since there is no index to judge how violent a game is and our method was based on introspection of the participants, this might be a limitation for our sample. In addition, there is no consensus about whether weekly game playing hours can be one of the indicators of pathological game playing. Second, some studies suggested that the *N*-back task is not a suitable measurement for WM processes since it cannot measure individual differences, which might be an important aspect in behavioral addiction research (Jaeggi, Buschkuhl, Perrig & Merrier, 2010).

Moreover, future research may include harder conditions for the *N*-back task by increasing *n*, since research indicated that 3-back might not be that hard for most of the participants. Since different cognitive functions like object recognition and response inhibition are affected differently by excessive gaming (Irak et al., 2016), different tasks should be included in future research. The third, depression level between the two groups was similar but state and trait anxiety levels were higher in the EVGP group. Later alpha and theta activities might be related to the high anxiety levels of the EVGP group. It is not a surprising finding since anxiety and excessive game playing had high comorbidity rates (Toker & Baturay, 2015), but interpreting the results of this study this should be kept in mind as anxiety levels may affect the brain oscillatory responses. Furthermore, to describe and understand how excessive game playing changes the human brain and behavior in the long run, we can investigate our participants' *N*-back performance over time with a longitudinal design, like in a 2 year period. This type of design also helps us to understand the developmental differences behind gaming exposure's effect on the human brain.

It is important to mention that while we found no between-group differences in *N*-back performance on the behavioral level, clear differences in the EEG were detected, suggesting that there are differences in brain functionality between EVGPs and NPs. Given that, game playing might be useful for an individual with its effect on plasticity, distinguishing pathological and healthy game playing will be useful.

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