Psychiatr. Pol. ONLINE FIRST Nr 322: 1–20
Published ahead of print 1 August 2023
www.psychiatriapolska.pl
ISSN 0033-2674 (PRINT), ISSN 2391-5854 (ONLINE)
DOI: https://doi.org/10.12740/PP/OnlineFirst/167394

Changes in brain structure in people with gaming disorder. A review of neuroimaging studies

Mateusz Wojtczak, Anna Walenda, Katarzyna Kucharska

Institute of Psychology, Cardinal Stefan Wyszynski University in Warsaw

Summary

This review aims to summarise the current knowledge on structural brain changes among people diagnosed with gaming disorder and the resulting clinical implications. The review will show the theoretical psychological and neurobiological models of computer gaming disorder in conjunction with the results of structural neuroimaging studies. Previous epidemiological studies indicate that the prevalence of gaming disorder in the population may reach approximately 2%. Researchers indicate that the aetiopathogenesis of computer game use disorder is complex and includes psychological, social, as well as neurological and hormonal factors. From the perspective of psychological research exploring gaming disorder, it can be concluded that a person has certain specific psychopathological features and/or symptoms, which, through mediating factors, such as the inability to cope with stress or negative emotions, influence the formation of the symptoms of the disorder. In the context of the neurobiology of behavioural addictions, researchers point to disorders in the mesocorticolimbic reward system, which is influenced by dysfunctional neuronal mechanisms of emotion and stress regulation. When describing structural changes in the brain, researchers most often report differences in the volume of grey matter, which include areas of the dorsolateral prefrontal cortex, temporo-occipital cortex, superior and posterior parietal cortex, anterior cingulate cortex, cerebellum, insular cortex, limbic system, and basal ganglia.

Key words: gaming disorder, MRI, structural brain changes

Disorders of game use in DSM-5 and ICD-11

The appearance of the latest version of the International Classification of Diseases and Health Problems – ICD-11 (International Statistical Classification of Diseases and Related Health Problems, 11th Edition) has brought significant changes in the context of understanding addictions, as evidenced by, among others, the emergence of a category such as disorders due to addictive behaviours, to which a new disease entity was included – gaming disorder (GD; code: 6C51) [1]. In turn, in the American

psychiatric classification DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5th Edition), the above nosological unit does not occur, and the subcategory of non-substance-related disorders includes only gambling disorder [2]. On the other hand, internet gaming disorder (IGD), which is the name used in DSM-5, has been assigned to section 3 as requiring further research. The prevalence rate of GD in the world population is estimated at about 2%, and it is the highest in Asian countries [3]. The ratio of men to women with this disorder is approximately 2.5:1 [3], and the most susceptible period in males is adolescence and early adulthood [1].

GD and IGD can be included in the so-called disorders due to addictive behaviours. By this term we understand all mental disorders characterised by a permanent or recurrent loss of control over the intensity of performing a given activity (e.g. eating, working, shopping, having sex, exercising or gambling), despite the negative consequences resulting from this behaviour [4]. Disorders due to addictive behaviours are not associated with the use of psychoactive substances, although their clinical picture and mechanisms seem to be like those in substance addictions [4].

In ICD-11, GD is defined as a continuous or episodic and recurrent pattern of behaviour associated with playing digital or video games (online or offline), characterised by: (1) loss of control over gaming (e.g. frequency, duration, context), (2) prioritising gaming over other interests and daily activities, (3) continuing or intensifying this behaviour despite its negative consequences (e.g. conflicts in the family, worse performance at work or school). To make this diagnosis, the maladaptive gaming pattern must typically have lasted for at least 12 months and cause marked distress or impairment in the individual's personal, family, social, educational, professional, or other important areas of functioning. However, the time criterion may be shortened if all the above conditions are met and the symptoms of the disorder are severe. It should be noted that GD, in accordance with the ICD-11 and DSM-5 classifications, does not apply to online gambling.

In contrast, the DSM-5 proposes nine diagnostic criteria for IGD, of which the individual must meet at least five over a 12-month period to make this diagnosis. However, there is no consensus among researchers as to which of them are necessary and which are sufficient to make this diagnosis [4]. The discussed criteria were developed based on those applicable to addiction to psychoactive substances. In DSM-5, the following is listed: (1) preoccupation with gaming; (2) withdrawal symptoms when unable to play, such as difficulty concentrating, irritability, anxiety or sadness; (3) tolerance defined as the need to engage more and more in gaming to achieve the same effect; (4) unsuccessful attempts to take control over gaming, i.e. its cessation or limitation; (5) loss of previous interests, hobbies other than those related to gaming; (6) continuing a self-destructive gaming pattern despite its negative consequences; (7) underestimating in conversations with family, therapist and other people the time spent on gaming; (8) gaming as a way of coping with problems and as a method of

regulating emotions; (9) difficulties in interpersonal relations (conflicts, resignation from contacts with friends, resulting in social isolation), problems at work and school (e.g. absenteeism, neglect of duties) due to excessive gaming [2].

GD is also associated with somatic complications resulting from the deprivation of basic physiological needs, such as: eating, sleeping or personal hygiene, as well as from the lack of physical activity [1, 5]. People with this disorder often suffer from vision and/or hearing problems, migraine headaches, back pain, carpal tunnel syndrome, as well as other pains or changes in the musculoskeletal system [1, 5]. Studies have shown that excessive, self-destructive gaming may be associated with depression [6], ADHD [6, 7], anxiety [6], autism spectrum disorder [7, 8], and aggressive behaviour [9, 10]. For example, Coyne et al. [11] in a 6-year longitudinal study observed that young adults who initially showed a moderate amount of symptoms of pathological video game playing, and then intensification of these symptoms over a 6-year period also reported higher levels of depression, anxiety and aggression than their peers who did not play pathologically during this period. On the other hand, Han et al. [12] showed that the lower initial level of depression and attention deficits in the clinical group of people with GD were predictors of recovery from this disorder. However, the number of longitudinal studies that would enable the identification of a cause-and-effect relationship is still not sufficient.

Theoretical models of gaming disorder

Aetiopathogenesis and risk factors

As in the case of addiction to psychoactive substances, there is no unequivocal answer to the question of what is the basis and mechanisms of the formation and maintenance of GD. One of the models that seems to integrate the existing aetiological concepts of substance addiction and disorders due to addictive behaviours, including GD, is the biopsychosocial model [13]. It assumes that the above-mentioned disorders, including GD, are the result of interactions between many factors, such as biological susceptibility (e.g. genetic predisposition or neurobiology), psychological factors (e.g. personality) and socio-cultural context (e.g. the influence of family/peer environment or culture). In the research literature, an important place is also occupied by the concept of risk factors and protective factors in the context of disorders such as GD [13], but much less is known about protective factors than about risk factors [4].

Factors motivating to play

The literature on the subject indicates that the motives for playing computer games play an important role in GD [14], and in recent years they have been studied on a large scale. Game developers try hard to satisfy various psychological needs of players,

and thus encourage them to play [13]. The author of one of the most widespread and researched models identifying motivating factors for playing massively multiplayer online role-playing games, the so-called MMORPGs (massively multiplayer online role-playing games) is Nick Yee [15]. In reference to the concept of Bartle's player types [16], he created his own model using factor analysis, in which he distinguished 3 basic factors, composed of a total of 10 sub-factors: (1) achievement (advancement, mechanics, competition), (2) immersion (discovery, role-playing, customisation, escapism) and (3) social motivation (socialising, relationships, teamwork). Motivation for advancement shows the desire to achieve the goals intended by the player and fast progress in the game, resulting in the accumulation of resources. Mechanics is about the satisfaction of learning and understanding the rules of the game in order to use them optimally. Competition refers to enjoying competing with other players, and socialising refers to helping and getting to know them. Relationship motivation shows a willingness to form meaningful and lasting relationships with others, while teamwork is about the joy of working together with others. Discovery is the satisfaction of exploring the game world and gaining information unknown to other players. Role-playing is about immersing oneself in the story as seen through the eyes of the created character, and customisation is about the joy of designing their appearance and adjusting it to a specific game convention. Escapist motivation, on the other hand, indicates the use of the game for relaxation, relief from stress and problems of the outside world.

In turn, Demetrovics [17], based on a review of the literature and a cross-sectional study, created a 27-item Motives for Online Gaming Questionnaire (MOGQ), in which he distinguished seven types of factors motivating to play online games: (1) social, (2) escape, (3) competition, (4) coping (playing to reduce negative affect and tension), (5) skill development (the desire to increase concentration, coordination, and other abilities through gaming), (6) fantasy (the desire to experience being someone else and part of another reality) and (7) recreation.

Personality and motivating factors – empirical research

Psychological factors associated with GD include introversion [18], shyness [11], neuroticism, anxiety as a trait [19] and low emotional intelligence [20]. Gentile et al. [21] conducted a 2-year longitudinal study involving 3,034 children and adolescents. The authors concluded that lower social competence and increased impulsiveness were risk factors for the development of GD, while anxiety, social phobia, depression and poorer school performance were consequences of the disorder's persistence. In an extensive systematic review of the literature, Şalvarlı and Griffiths [22] identified 32 studies that showed a positive relationship between impulsivity and GD. Research results also indicate that individuals with this disorder experience a sense of loneliness [9]. In the study by T'ng et al. [9], GD symptoms were a mediating factor between

loneliness and the four components of aggression (i.e. anger, hostility, physical and verbal aggression). Other authors also indicate a higher prevalence of hostility and aggressiveness as traits in people who play computer games pathologically [19]. Researchers mention the possibility of establishing social relationships as one of the factors motivating individuals to engage in online gaming [23]. Ultimately, however, this disorder results in a weakening of the relationship between the person with GD and other people in real life, thus constituting a feedback loop.

Researchers observed a relationship between GD and the tendency to experience boredom [24], sensation/novelty seeking [19] and narcissism [25]. Individuals with GD are also characterised by low self-esteem [10] and low self-efficacy in real life [26], as opposed to high self-efficacy in virtual reality. Kwon et al. [27] showed a positive relationship between GD symptoms and the discrepancy between the 'real self' and the 'ideal self' in adolescents. According to the authors, this discrepancy leads to negative affect, which in turn arouses the desire to 'escape from the self' (understood according to Baumeister's theory), which ultimately leads to the symptoms of GD. Baumeister's concept [28] assumes that individuals engage in self-destructive behaviour in order to temporarily free themselves from negative self-perception and related difficult emotions. Some researchers suggest that individuals engage in online gaming in order to experience being someone else, i.e. a character they create [23] (fantasy [17], role-playing [15]). Other factors motivating to play are achievements, advancement or competition [29, 30], which, to generalise, prove the desire to achieve the goals intended in the game, and thus the sense of agency, one's own competence and recognition among other players.

Bäcklund et al. [14] conducted the most comprehensive systematic review and meta-analysis to date (46 studies), involving 49,192 participants, on the motivation of people with GD to play. These researchers identified a statistically significant relationship between GD and 23 motivational factors, among which escapist motivation, i.e. the desire to escape from the problems of everyday life, turned out to be the most strongly associated with the symptoms of this disorder. This type of motivation may probably result from deficits in regulating emotions by people with GD, which have been proven in empirical research. For example, Yen et al. [31] assessed 87 young adults diagnosed with IGD, showing that these people more often suppressed their emotions, and less often used cognitive reinterpretation. In turn, Amendola et al. [32] observed a relationship between the symptoms of problematic video game playing and difficulties in accepting emotions, engaging in purposeful behaviour, impulse control when experiencing negative emotions, and limited access to emotion regulation strategies.

Theoretical models of gaming disorder

The model of pathological Internet use in the cognitive-behavioural approach by Davis [33] distinguishes two forms – specific (e.g. excessive playing of Internet

games) and generalised of this disorder. According to the author [33], specific forms of pathological Internet use, including GD, result from the psychopathology of the individual, while the generalised form – from social isolation and the need for social relations related to it. Davis [33] emphasises the role of the susceptibility-stress model, which means that an individual with certain personal characteristics in contact with stress may develop symptoms. The cognitive-behavioural approach of this model, in turn, emphasises the importance of cognitive distortions in the pathological use of the Internet, including GD.

Dong and Potenza [34], based on models of addiction to psychoactive substances, developed a cognitive-behavioural model of GD with a proposal of therapeutic interventions. The authors indicate the motivational factor related to reward-seeking, i.e. pleasure and stress reduction, as one of the three basic elements involved in all types of addiction, including GD. Furthermore, they cite the concept of 'reward deficiency syndrome', suggesting that individuals with this disorder engage in addictive behaviours to compensate for underactive reward signals in the mesolimbic dopaminergic pathway. Moreover, the results of studies using neuroimaging techniques indicate deficits in executive functions, including impaired inhibitory and cognitive control in people with GD [35]. Executive functions control engaging in reward-seeking behaviours, so their deficits may make it difficult for an individual to resist the temptation to engage in compulsive activity, i.e. gaming. According to the authors [34], the discussed dysfunctional pattern of gaming may further weaken executive functions, leading to disturbances in decision-making processes – i.e. to choosing immediate, short-term pleasure, without taking into account the long-term, negative consequences of a given behaviour.

It seems that the only model in the literature that comprehensively captures the mechanisms of developing and maintaining symptoms of general and specific forms of dysfunctional use of the Internet is the I-PACE model (Interaction of Person-Affect-Cognition-Execution model) by Brand et al. [36, 37]. Its first version was created in 2014 and assumes that specific forms of maladaptive use of the Internet, including GD, are the result of interactions between an individual's psychopathological susceptibility, specific motives for using games, and mediating factors in the form of dysfunctional styles of coping with stress and an individual's expectations towards a given activity on the Internet – e.g. gaming (i.e. expecting an improvement in mood and/or reduction of negative affect). In other words, this model postulates that an individual has certain specific characteristics and/or psychopathological symptoms that, through mediating factors such as the inability to cope with stress, affect the development of the symptoms of the disorder. These risk factors (e.g. psychopathology) predispose to the development of many mental disorders, not only GD, while the motivational factors, as emphasised by Brand et al. [37], are specific ones, involved in the development of GD specifically, and not another disorder. This seems to be in line with the concept of

Kardefelt-Winther [38], who postulates that the diagnostic criteria in DSM-5 do not reflect the specificity of GD, and he proposes to extend them with motivational factors, primarily achievement and escapist motivation, in order to distinguish GD from other disorders due to addictive behaviours.

In the revised version of the I-PACE model from 2016, the authors suggest that specific forms of dysfunctional use of the Internet arise as a result of the relationship between an individual's susceptibility (neurobiological, psychological, personality, motivational, psychopathological factors), moderators (coping style, cognitive biases and attentional bias towards gaming/Internet-related stimuli), mediators (affective and cognitive responses to given situations), and impaired inhibitory control [37]. Affective reactions to specific events include the desire to experience pleasure or reduce negative affect, while cognitive reactions include positive associations with a given behaviour. Brand et al. [37] mention factors moderating the relationship between predisposing factors and the disorder, and only in the presence of the so-called moderators, the disorder can develop. These moderators include low social competences (maladaptive strategies for coping with stress, deficits in emotion regulation skills) and cognitive distortions specific to dysfunctional use of the Internet. Moderating and mediating factors are dynamic in nature, which means that they can develop because of the ongoing GD. It also proves that they are susceptible to change under the influence of pharmacological or psychological interventions, while the characteristics of the individual related to their susceptibility (e.g. personality, genetics) are relatively constant [36, 37].

If a person with a vulnerable mental structure comes into contact with an addictive stimulus, i.e. gaming, and this activity causes, for example, an improvement in mood or a decrease in tension – through positive reinforcement, he or she becomes convinced that playing helps to cope with emotions, and during the next similar situation feels a greater desire to engage in this activity [36, 37]. According to Brand et al. [37], this may result in ignoring other, potentially adaptive ways of dealing with negative mood in the future. The literature on the subject, in addition to reports on the primary occurrence of deficits in inhibitory control and cognitive control in individuals with GD [35], also indicates that these deficits may progress as the disorder persists, which is emphasised by the I-PACE model [37]. The development of attentional bias, focused on game-related stimuli, and cue-reactivity [37] related to games are also characteristic of the further stage of GD. Initially, gaming may bring the effect intended by the player, but in the long-term perspective it leads to negative consequences in the life of an individual, both somatic, social, educational and professional, as well as to other mental disorders [1, 2].

Neurobiological model of behavioural addictions

Most of the available research on the neurobiological basis of addiction relates to psychoactive substance use disorders (substance dependence) [4]. Thanks to neuroimaging studies of the brain in addicted people, more and more scientific studies have been published in recent years indicating the neuroanatomical and neurohormonal basis of disorders due to addictive behaviours [39]. Advances in neuroscience have begun to elucidate the mechanisms underlying profound disorders of decision-making ability and emotional balance in people displaying compulsive behavioural behaviours. These findings provide insight into how basic neurobiological structures, when altered, can disrupt voluntary behavioural control and emotional self-regulation contributing to development of these disorders [40].

In the context of the neurobiology of the above-mentioned disorders, researchers point to dysfunctions in the mesocorticolimbic reward system, which is influenced by dysfunctional neuronal mechanisms of emotion and stress regulation [41, 42]. The stress axis (hypothalamic-pituitary-adrenal axis, HPA) is activated by stress factors and experiencing negative emotions [42]. Along the sensory networks, information about the stressor reaches the paraventricular nucleus of the hypothalamus, where corticoliberin is released. This hormone is then transported to a specific area of the pituitary gland (i.e. the anterior pituitary gland) where it interacts with specific proteins on the surface of pituitary cells (i.e. CRH-1 receptors). This interaction stimulates the anterior pituitary gland to produce a molecule called pro-opiomelanocortin (POMC), which is then converted into smaller, biologically active peptides, including β-endorphin and adrenocorticotropic hormone (ACTH). ACTH is then transported by the blood to the adrenal glands, where it induces the secretion of glucocorticoids, including cortisol [42].

The cause of emotional dysregulation in gaming disorder and other disorders due to addictive behaviours is the altered activity of the mesocorticolimbic dopamine system [40, 41]. This brain pathway includes several interconnected structural areas. Their action involves the signalling molecule (i.e. neurotransmitter) dopamine. The central components of the mesocorticolimbic dopamine system are neurons whose cell bodies are in an area called the ventral tegmental area (VTA). Long projections (i.e. axons) from these cell bodies reach various other areas of the brain, most notably the nucleus accumbens (NAC), which is located in an area of the brain called the ventral striatum and is part of the limbic system [40]. The nucleus accumbens assigns meaning (i.e. value) to the addictive experience. In the NAC and prefrontal cortex, VTA neurons release the neurotransmitter dopamine from the axon terminals into the space that separates the axon from its neighbouring cell (i.e. the synapse). The released dopamine then interacts with specific receptors on adjacent neurons in these areas of the brain and alters their activity in specific ways [39]. A characteristic property of the mesocorticolimbic dopamine system is its ability to generate signals that represent the relationship between a rewarding and expected experience (gaming disorder) and

the actual reward. A reward that is greater than predicted causes increased activity of dopamine-releasing (i.e. dopaminergic) neurons. A reward that is less than predicted results in decreased activity of these neurons [40].

The combination of the reward system and the corticotropic system occurs when glucocorticoids (induced by negative emotions and stress) alter the mesocorticolimbic dopamine system through two separate actions: first, they activate the cell bodies of dopaminergic neurons in the VTA [43], primarily by stimulating glutamate-mediated signalling [40]. This indirectly leads to increased dopamine release in the NAC. Secondly, they directly affect the axons of dopaminergic neurons in the NAC [43]. Then glucocorticoids increase mesocorticolimbic dopamine accumulation in response to disorders due to addictive behaviours, enhancing the rewarding experiences of these disorders [40]. However, over the long term, excessive levels of glucocorticoids, along with changes in glutamate and corticotropin-releasing factor activity, reduce dopamine signalling and result in decreased reward. This increases the frequency and intensity of addictive behaviours. This area certainly requires further research [43].

Structural brain changes in GD – a review of neuroimaging studies

The aim of this review is to discuss the empirical studies to date that have used neuroimaging techniques to explore the emerging problem of computer game use disorder (IGD) from a neuroscience perspective. In connection with the above, a review of the literature on the above-mentioned research issues published in Polish and English between January 2010 and December 2022 was carried out. Research literature was searched using the following databases: MEDLINE, PsycINFO, PubMed, Science Direct, Web of Knowledge and Scopus. Based on the searches, 13 publications were identified and included in the review. The classification of mental disorders was based on ICD-11 and DSM-5.

Thanks to the use of structural magnetic resonance imaging (MRI), many techniques for imaging the morphology of the brain can be used. The most frequently used measures in MRI studies include: the volume or thickness of the cerebral cortex, as well as the density of the white matter microstructure, which can be measured for the entire brain or specific regions [44]. Structural changes in the brain can be studied using voxel-based morphometry (VBM). VBM is used to compare the volume of brain regions and the density of grey and white matter [44]. Another MRI technique is diffusion tensor imaging (DTI). DTI is a white matter imaging method that assesses the diffusion of water molecules in the brain, which helps identify interconnected brain structures using fractional anisotrophy (FA). This measure is an indicator of fibre density, axonal diameter, and white matter myelination [44]. Researchers using the above neuroimaging methods showed that GD is associated with structural changes in the brain, which in turn entail changes in various mental functions (see Table 1).

Behavioural control

Neuroimaging exploration of problematic gaming suggests that computer game use disorder is associated with structural abnormalities in the grey matter of the brain that affect behavioural control and impulsivity [45]. The results showed a negative correlation between the mean impulsivity scale score and reduced grey matter volume in the areas of the right dorsomedial prefrontal cortex, bilateral insula, orbitofrontal cortex, right amygdala and left fusiform gyrus in the GD group compared to healthy controls. Changes in the right dorsal-medial prefrontal cortex are associated with impaired top-down control of behaviour, including inhibition of initiated actions [45]. Structural changes in the bilateral insula, in turn, lead to dysregulation of cognitive control and attention processes. The altered structure of the amygdala is of key importance in the context of emotional memory, which is responsible for the assimilation of pleasant affective experiences in addictive behaviours [40]. In addition, changes in the structure of the fusiform gyrus negatively affect the processing of emotions accompanying facial perception and multi-element structure-based stimuli. The results of the analysis showed that the dysfunction of these areas of the brain involved in inhibiting behaviour, regulating attention processes and processing emotions may contribute to problems with impulse control in adolescents with GD and thus contribute to its development [45].

Cognitive control

VBM studies have also identified a potentially harmful effect of GD on the functioning of cognitive control. In comparison to a healthy control group, the GD group showed an increased volume of grey matter in the left thalamus [46]. The thalamus is a key target for dopaminergic pathways and plays a major role in conditioned reinforcement and reward anticipation [47]. The increased thalamus volume has a positive relationship with the increased rewarding effect when playing games. In addition, the results of the study report morphometric abnormalities in the grey matter volume of the left anterior cingulate gyrus, both inferior temporal gyri and both occipital gyri in patients addicted to online games [46]. Disrupted functions in the anterior cingulate gyrus in people with GD may impair the individual's ability to cognitively monitor and inhibit inappropriate behaviour. In addition, changes in both grey and white cingulate matter correlated with measures of aggression, hostility, self-esteem, and the degree of computer game addiction [48]. On the other hand, the reduced volume of both gyri of the occipital cortex (primary visual cortex) may be associated with excessive exposure to visual stimulation (computer monitor) among people with GD.

Motivational processes

Another study shows that the severity of GD was positively correlated with grey matter volume in the left caudate nucleus and negatively related to the functional connectivity between the left caudate nucleus and the right middle frontal gyrus [49]. Structural changes in the caudate nucleus, which is part of the striatum, affect the neural processes in the reward system, which is associated with perceived pleasure. Therefore, it has a significant impact on the motivation to reproduce addictive behaviours. Dysfunction of the right middle frontal gyrus may affect the persistence of addiction by reducing the ability to regulate the integration and selection of cognitive-motivational processes [43]. This study shows that GD involves changes in frontostriatal circuits that dysregulate affect, motivation, and cognitive control, and structural and functional abnormalities in these regions have been reported in other addictions such as substance abuse and pathological gambling [49].

Executive control

Individuals with GD showed less grey matter volume in brain areas involved in executive control, such as the anterior cingulate cortex and the supplementary motor area [50]. Structural changes in the cingulate gyrus affect executive functions such as: attention allocation, reward anticipation, decision-making, morality, performance monitoring, and error detection. In turn, neuronal changes in the supplementary motor area affect the initiation and planning of movements in the context of the perception of visual, auditory and tactile stimuli when these stimuli are used as signals to initiate a movement or a series of movements [51]. When the signal indicates that the movement must be delayed, the neurons of the supplementary motor area react strongly during the delay period. The dysfunction of this area among people with GD may be the reason for the inability to stop the started episode of gaming [51].

Memory and learning

People with GD also showed a smaller volume of the left ventrolateral prefrontal cortex and left inferior parietal lobe compared to the control group of healthy people [52]. Structural changes in the area of the left ventrolateral prefrontal cortex may affect the cognitive control of memory and thus prevent access to the knowledge contained in memory pathways, which is important for given goals and tasks [52]. In turn, the altered volume of the left inferior parietal lobe may dysregulate the neural processes responsible for language processing, episodic memory as well as the processing of spatial stimuli [53]. Compared to controls, gaming addicts showed significantly lower grey matter density in the bilateral inferior frontal gyrus, right precuneus, and right hippocampus [54]. Changes in the volume of the inferior fron-

tal gyrus may involve Broca's area and thus affect language processing and speech production, as well as cause dysfunctions in the inhibition of the activity performed [55]. The precuneus is an area of the brain involved in a variety of complex mental functions such as recalling episodic memory and remembering, integrating information related to the perception of the environment, self-awareness, and affective responses to pain. Therefore, a larger volume of the precuneus in GD may be associated with incorrect orientation of attention to stimuli and memories associated with computer games [55]. The hippocampus is the main centre of semantic and spatial memory. Research suggests that changes in the hippocampus play a key role in the acquisition, retention, and learning of addiction-related stimuli that lead to compulsive and addictive behaviour [40].

Discussion

So far, the literature on the subject lacks a consistent definition of the disorder in question. On the one hand, this makes comparisons between studies in this area difficult. Thus, distinguishing GD as a disease entity in ICD-11 is a breakthrough not only for clinicians, but also for researchers. On the other hand, the aforementioned inconsistency of the definition is related to the diversity of the concepts of GD among scientists, which may be the basis for creating complex models of this disorder. It seems that the most comprehensive of them, covering the mechanisms of shaping and maintaining symptoms, e.g. GD, is the I-PACE model by Brand et al. [36, 37]. The authors of this model postulate that factors mediating or moderating the relationship between the individual's predispositions and the disorder should be sought (e.g. deficits in emotion regulation or coping with stress). It is recommended to continue research in the area of GD mediators/moderators, taking into account the fact that they are susceptible to therapeutic effects.

The neuroimaging results described above are ambiguous and do not show specific patterns of structural changes due to gaming disorder. Researchers most often report differences in the volume of grey matter, which include areas of the dorsolateral prefrontal cortex, temporo-occipital cortex, superior and posterior parietal cortex, anterior cingulate cortex, cerebellum, insular cortex, limbic system, and basal ganglia [51]. It should be emphasised that VBM studies are helpful in demonstrating potential structural changes in the brain of people with GD. Many areas of the brain that have been altered in people with GD have been associated with functions contributing to the development of addictive or compulsive behaviours [41]. The included studies suggest that people with GD show weaker inhibition processes and dysregulation of emotions, impaired functioning of the prefrontal cortex and cognitive control, worse working memory and decision-making abilities, reduced visual and auditory functioning, and impaired mechanisms of the neural reward system [51]. These deficiencies are similar to those found in substance addicts, suggesting that both substance addic-

tions and disorders due to addictive behaviours share common predisposing factors and may be part of an addiction syndrome. Although some of the studies have shown conflicting changes in different brain regions, these discrepancies help illustrate the different ways in which GD can affect overall brain function and the changes it can cause at the behavioural and cognitive levels, further highlighting the complexity of the phenomenon [41]. It should be considered that many of the analysed VBM studies were conducted on samples of adolescents, and their brains are still undergoing developmental changes [51].

The results presented may not be generalisable to all age groups. One potential way to control this would be to conduct similar studies on samples of children and adults to compare the results obtained. First, it is difficult to exclude the heterogeneity of methodologies between studies (including MRI type, patch thickness, pretreatment protocol, and statistical threshold) that may affect generalised conclusions [51]. Second, this review did not address neurological differences in patients with GD of different sexes. Since most of these studies are cross-sectional, it is not possible to establish causal relationships between GD and altered structures in the brain reported in these studies, particularly in VBM studies. Future studies should adopt other methodological strategies to help overcome these shortcomings [41]. Further prospective and ecological momentary assessment (EMA) studies are required to understand the role of altered brain structures in the mechanism of GD. In addition, further studies should be characterised by larger sample sizes, as the currently evaluated studies were conducted on small clinical samples [51].

Table 1. **Overview of neuroimaging studies on structural brain changes in GD**

Study	Method	Study (M = age)		Results for people with GD
		GD	Control group	
1. Sun et al. 2014 [56]	Diffusion imaging of kurtosis and voxel-based morphometry.	18 (20.5)	21 (21.95)	↑ Increased volume of grey matter in the areas of: right inferior and middle temporal gyrus, right parahippocampal gyrus.
2. Wang et al. 2015 [57]	Optimised voxel-based morphometry technique, psychological Stroop test.	28 (18.8)	28 (19.3)	↓ Smaller grey matter volume in areas: bilateral anterior cingulate cortex (ACC), precuneus, supplementary motor area (SMA), superior parietal cortex, left dorsolateral prefrontal cortex (DLPFC), left insula, bilateral cerebellum.

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3. Lin et al. 2015 [54]	Voxel-based morphometric analysis.	35 (22.20)	36 (22.28)	↓ Significantly lower grey matter density in bilateral inferior frontal gyrus, left cingulate gyrus, insula, right precuneus and right hippocampus. ↓ Significantly lower white matter density in areas: inferior frontal gyrus, insula, amygdala, anterior cingulate gyrus.
4. Du et al. 2016 [45]	Barratt Impulsiveness Scale (BIS-11), voxel-based morphometric correlation (VBM).	25 (17.28)	27 (17.48)	↓ Decreased correlations between impulsiveness scale score and grey matter volume of right dorsomedial prefrontal cortex (dmPFC), bilateral insula and the orbitofrontal cortex (OFC), right amygdala and left fusiform gyrus in the GD group compared to the control group.
5. Han et al. 2012 [46]	VBM using a two-sample t-test with statistical parametric mapping (SPM5).	20 (20.90)	18 (20.90)	↑ Volume in left thalamic grey matter. ↓ Reduced grey matter volume in inferior temporal gyrus, right middle occipital gyrus, and left inferior occipital gyrus.
6. Jin et al. 2016 [50]	Voxel-based morphometric analysis (VBM) and functional connectivity (FA).	25 (19.12)	21 (18.78)	↓ Significantly reduced grey matter volume in prefrontal cortex regions including bilateral dorsolateral prefrontal cortex (DLPFC), orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), and right supplementary motor area (SMA).
7. Ko et al. 2015 [58]	Measurement of grey matter density (GMD) and functional connectivity using resting fMR.	30 (21.70)	30 (22.40)	↓ Lower grey matter density of bilateral amygdala.

table continued on the next page

8. Lee et al. 2018 [52]	Voxel-based morphometric analysis with diffeomorphic anatomical registration through exponentiated Lie algebra algorithm (DARTEL).	31 (23.57)	30 (24.23)	↓ Grey matter volume in anterior cingulate cortex and supplementary motor area. ↓ Grey matter volume in lateral prefrontal and parietal cortex including left ventrolateral prefrontal cortex and left inferior parietal lobe.
9. Lee et al. 2019 [59]	Whole brain voxel-based morphometry with diffeomorphic anatomical registration through exponentiated Lie algebra algorithm, voxel-level unadjusted threshold for multiple comparisons.	20 (23.90)	20 (22.70)	↓ Less grey matter volume in right anterior cingulate cortex, left inferior frontal gyrus and left insula. ↑ Greater volume of grey matter in the right angular gyrus.
10. Mohammadi et al. 2020 [48]	Diffusion tensor and T1-weighted 3D MR images were acquired to investigate grey (via voxel-based morphometry) and white (via tract-based spatial statistics) matter.	29 (22.20)	29 (22.28)	↓ Grey matter density in right posterior cingulate, left precentral and postcentral gyrus, right thalamus. ↓ Fractional anisotropy, a marker of white matter structure, was reduced in the left and right cingulate bundles.
11. Seok and Sohn 2018 [49]	Voxel-based morphometry and connectivity analysis at rest.	20 (23.60)	20 (22.70)	↑ Grey matter volume in left caudate nucleus. ↓ Volume of grey matter in the middle frontal gyrus.
12. Yoon et al. 2017 [55]	Voxel-based morphometry, psychological scales.	19 (22.90)	25 (25.40)	↑ Greater volume in hippocampus/amygdala and precuneus ↑ Greater volume in the hippocampus positively correlated with the severity of GD symptoms

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13. Weng et al. 2013 [60]	Voxel-based morphometry (VBM) analysis and tract-based spatial statistics (TBSS), Young's Addiction Internet Scale (YIAS).	17 (16.25)	17 (15.54)	↓ Significant grey matter atrophy in the right orbitofrontal cortex, bilateral insula, and right supplementary motor area. ↓ Decreased FA in the right genu of the corpus callosum, bilateral white matter of the frontal lobe and the right external capsule. ↑ YIAS positive correlation with the grey matter volume (GMV) of the right orbitofrontal cortex, the bilateral insula and the FA value of the right external capsule.

Diagnostic implications

In the context of the discussed issue of GD, it is worth noting that the diagnostic procedure will be based on the ICD-11 criteria. In addition, the following screening tools can be helpful in the diagnosis (all available in Polish): Internet Gaming Disorder-20 (IGD-20) [61], Internet Gaming Disorder Scale – Short-Form (IGDS9-SF) [62], which were developed on the basis of DSM-5 criteria, as well as the Gaming Disorder Test (GDT) [63] questionnaire developed on the basis of ICD-11 criteria. These tools allow one to assess the severity of the symptomatology of GD and can be an invaluable help for specialists dealing with the diagnosis of this mental disorder.

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Address: Mateusz Wojtczak
Cardinal Stefan Wyszynski University in Warsaw
Institute of Psychology
01-938 Warszawa, Wóycickiego Street 1/3, build. 14
e-mail: mt.wojtczak@student.uksw.edu.pl