## Examiner's commentary

Video game addiction has recently become a central topic of interest for students. It may be difficult for students to be objective about the effects of the excessive use of technology, but it is interesting to find out more about the effects it may have on their brain. This Extended Essay takes a novel approach to the exploration by wondering if there are neurobiological factors that may contribute to turn use into addiction. The student provides research evidence that supports a neural mechanism behind VGA. Research on psychosocial mechanisms seems to suggest that environmental and social pressures promote addiction. This research is presented as an alternative argument rather than as a counterpoint to the research on neurobiological factors. The student includes complex research material. Studies are evaluated with emphasis on the limitations of conclusions based on correlational research. The essay includes other methodological limitations that are clearly but succinctly explained. This Extended Essay demonstrates some level of risk taking on the part of the student since the complexity of the material required analytical skills. The novelty of the approach will probably be appealing to students interested in a further understanding of the nature of gaming.

**Extended Essay: Psychology**

**Title:**

# Neurobiological and Psychosocial Factors in Video Game Addiction

**Research Question:**

To What Extent Do Neurobiological Factors Increase Disposition Towards Video Game Addiction (VGA)?

**May 2018**

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## Introduction:

Video gaming (VG) is becoming increasingly prevalent worldwide, and along with it are prevalence rates of video game addiction (VGA), with estimates as high as 27.5% of the global population (Mihara & Higuchi, 2017). Similarly, research into VGA has increased (Kuss & Lopez-Fernandez, 2016; Cash, D. Rae, H. Steel & Winkler, 2012), yet as of writing, there is no scientific consensus on what constitutes as VGA. In 2018 the WHO announced the inclusion of *gaming disorder* in the next ICD-11 manual to be published (World Health Organization, 2018); similarly, the APA has listed Internet Gaming Disorder (IGD) as a "Condition for Further Study" in the upcoming DSM-5 ("Internet Gaming Disorder in DSM-5", 2019). According to the ICD-11 (World Health Organization, 2018), *gaming disorder* is a disorder arising from addictive behaviour, with the definition:

"1) impaired control over gaming 2) increasing priority given to gaming to the extent that gaming takes precedence over other life interests and daily activities; and 3) continuation or escalation of gaming despite the occurrence of negative consequences".

For the purposes of this essay, VGA/*gaming disorder*/IGD will be used interchangeably, all referring to the ICD-11's definition of *gaming disorder*.

Given the increases in VG and VGA prevalence worldwide, there exists a need to understand the mechanism behind VGA, such that diagnosis and treatments can be properly administered. Yet there is no definitive understanding of VGA's mechanism. However, scientific consensus has recognised VGA as a behavioural addiction, which is "characterized by the compulsive, repetitive involvement in a rewarding non-substance-related behaviour, despite consistent adverse consequences." (Naim-Fell, 2013). As such, one can turn to well-established models of behavioural addiction in order to explain the causes of VGA. There are two main arguments in understanding the causes of behavioural addiction: the

neurobiological perspective suggests that abnormalities within neural circuitry would increase disposition towards behavioural addiction; the psychosocial approach suggests that environmental and social pressures would promote addictive behaviour. While both models have merit, more research has been done under the neurobiological approach. Thus, in this essay, I will mainly investigate the neurobiological approach, with the psychosocial approach serving as an alternative explanation. Hence, the research question: **To what extent do neurobiological factors increase disposition towards VGA** will be used.

## 1. The Neurobiological Approach towards VGA

Evidence obtained through brain-imaging techniques has supported the notion that behavioural addiction would arise due to abnormalities within the reward system of the brain (Brewer & Potenza, 2008). The reward system consists of brain structures that govern reward cognitions, which include associative learning (through operant and classical conditioning) between a reward and its corresponding stimulus, incentive salience (the motivation for a reward), and positive emotions such as pleasure (Schultz, 2015). In order to communicate between such structures, the neurotransmitter dopamine (DA) is used. DA's functions include reward processing (Schultz, 2000), reward expectation (Volkow, Fowler & Wang, 2003), producing pleasurable feelings (Volkow, 2010) for motivation and reinforcement of behaviour (Schultz et al., 1993).

According to Volkow et al. (Volkow et al., 2010), behavioural addictions would arise due to the combined dysfunctions in different brain structures that compose of the interrelated components of the reward circuit. The components include: "reward", consisting of the nucleus accumbens (NA); "motivation", consisting of the orbitofrontal cortex (OFC); "learning", consisting of the amygdala and hippocampus; "control", consisting of the pre-frontal cortex (PFC) and the anterior cingulate cortex (ACC). Increasing disposition towards addictive behaviour would occur under the interaction between two conditions: 1. Enhanced "reward-motivation-learning" (RML) pathways increasing motivational salience for behaviour; 2. "Control" structures are dysfunctional and thus unable to inhibit the urges arising from the enhanced RML pathways.

The enhancement of the RML components in Volkow's model can be further explained through Brewer's model (Brewer & Potenza, 2008). In this model, enhancement refers to an increased association between a stimulus and its reward, also referred to as

conditioning. Conditioning of behaviour would occur first when the nucleus accumbens shell (NAs) has assigned a "want" to specific behaviour (Nestler et al., 2015). Next, decreased inhibitory signals from the PFC and increased excitatory signals from the amygdala reach the nucleus accumbens core (NAcc), which correspondingly increase behavioural salience until behaviour is conditioned to become habitual. When there is decreased inhibition from the "control" structures, such habitual behaviour would eventually form addictive behaviour, as predicted by Volkow's model.

Hence, these two models suggest that abnormalities within the "control" and RML pathway structures would increase disposition towards VGA. To evaluate this claim, we will first examine the evidence behind RML abnormalities and VGA.

## 1.1 Regarding the RML pathway and the Function of Dopamine

The conditioning of behaviour is maintained through the dopaminergic mesolimbocortical pathway (Vousooghi, Zarei, Sadat-Shirazi, Eghbali & Zarrindast, 2015). This pathway originates in the ventral tegmental area (VTA) and goes primarily to the ventral striatum (VS) and the PFC, but also extends to structures within the RML pathway such as the OFC, amygdala and hippocampus. The VS contains the nucleus accumbens (NA), which is further separated into the shell (NAs) and core (NAcc). Dopamine, which is secreted in the VTA after receiving certain stimuli, is transmitted along the structures in the pathway, acting as a signaller. Structures in the pathway would contain dopamine $D_2$ receptors (D2R) in order to receive the dopaminergic signals. Higher levels of DA secretion would increase the signals sent. In turn, this would could produce more rewarding feelings, increasing motivation and reinforcement for the DA stimulus, which also includes behaviour. Behavioural conditioning would occur when DA levels are high enough to increase salience towards specific behaviour (Vousooghi et al., 2015).

For VGA, it appears that the conditioning of VG behaviour also occurs through this pathway, evidenced by Koepp et al.'s study (Koepp et al., 1998). The study measured dopamine released from participants' VS when they played VGs, achieved through usage of the molecule raclopride, which can bind to D2R. Reduction in the binding of raclopride to the VS would signal the increased release and binding of dopamine to D2R. As raclopride is radioactive, positron emission tomography (PET) is utilised to see the full extent of dopaminergic changes. The conclusion that dopamine is released as a result from VG play is supported by Weinstein's study (Weinstein, 2010), where healthy participants were shown to have produced 10.5% more dopamine after playing VGs, using the same measurement method as Koepp's study. If conditioning of VG behaviour occurs along the

mesolimbocortical pathway, it suggests that VGs are comparable to addictive stimulants that are also conditioned along this pathway. The results from Weinstein's study are indeed comparable to the dopamine levels produced from amphetamine (Farde, 1992) intake. This further suggests that VG use may also produce similar dopaminergic effects found in substance addicts, for example reductions in D2R, lower amounts of DA released, lower metabolic activity in "control" structures and higher metabolic activity in RML structures (Volkow et al., 2010).

Such effects have been observed in VGA players, lending support towards Volkow's model in explaining VGA. Kim et al.'s study (Kim et al., 2011) used raclopride and PET to show that individuals with VGA, determined using Young's Online Internet Addiction Test (IAT) (Young, 1998), had reduced D2R availability in the striatum. Hou et al.'s study (Hou et al., 2012) also used PET to determine that VGA individuals had lower numbers of DA transporters. Such neurological effects could increase disposition towards VGA, as hypothesised by Blum et al.'s (Blum et al., 2000). He suggested that with decreased DA transporter and receptor numbers, specific stimuli (e.g. VG play) would release lower levels of DA, thus there would be increased craving towards the stimuli and increasing disposition towards addiction (i.e. VGA).

Furthermore, abnormalities in metabolic activity have been observed VGA players. Tian et al.'s study (Tian et al., 2014) had IGD participants and healthy controls undergo PET while playing VGs. The results showed that IGD subjects had decreased glucose metabolism in the PFC, which is part of the "control" structure. Park et al.'s study (Park et al., 2010) showed increased glucose metabolism in the orbitofrontal cortex (OFC) for IGD individuals when compared to healthy controls. These results are in line with Volkow's model. As glucose is the main energy source for brain structures (Mergenthaler et al., 2013), lower

metabolism suggests decreased function of the structure and vice versa. The PFC is responsible for inhibiting highly salient behaviours (Brewer and Potenza, 2008). With decreased function, there will be increased salience towards VGA, leading to increased likelihood of VGA.

The OFC, along with the thalamus, hippocampus and NA, have been suggested to be associated with increased desire for VG play. This is shown in Han et al.'s study (Han et al., 2011), where non-IGD participants were asked to play a VG for 60 minutes/day for 10 days. At the end of the 10 days, participants were assessed with fMRI scans while watching gameplay footage. Desire to play the game was assessed by self-report. It was observed that some of the participants played more of the video game (MVG) than the general player group (GP). The results showed that those in the MVG group had increased activity in the OFC, hippocampus and thalamus when exposed to video-gaming cues (i.e. the gameplay footage), more so than the GP. Increased activation of these areas is also correlated with increased VG desire, which is predicted by Volkow's model. Both the OFC and thalamus are responsible for encoding the expectancies for stimuli (Parkinson, Cardinal & Everett, 2000), while the OFC modulates the saliency of different stimuli, thus providing the motivation to do addictive behaviour (Rolls, 2009). The hippocampus is responsible for storing the saliency values for a stimulus, which changes depending on whether the stimulus produced a positive/negative experience (Volkow et al., 2003), thus it helps modulate the desire for specific behaviour. The activation and interaction between these areas would then increase the desire for VG play. Such findings are further supported by Ko et al.'s study (Ko et al. 2009), regarding fMRI scans of IGD players viewing VG images. The study's results also showed activation in the aforementioned structures, along with the nucleus accumbens. As explained before, the NA is essential in behaviour conditioning and can lead to VG play

being a habitual behaviour. Through the enhancement of such brain structures, they will increase the desire for VG play, thus increasing disposition towards VGA.

In conclusion, there is a large amount of empirical evidence supporting how the salience of VGA can be enhanced, thus increasing disposition towards VGA. Next, we will examine how abnormalities within the "control' structures would increase disposition towards VGA.

## 1.2 Regarding the Control structures (ACC and PFC).

Structural differences in "control" brain structures seem to increase VGA disposition. In Jin et al.'s study (Jin et al., 2015), the grey matter volumes of VGA and healthy controls were compared through voxel-based morphometry (VBM). Compared to the control group, VGA players had decreased grey matter in the ACC and dorsal-lateral PFC (DLPFC). The ACC is associated with error detection and urgent inhibition control, while the DLPFC is involved in cognitive inhibitory tasks (Blasi et. al, 2006). The correlation between decreased ACC and DLPFC volumes, which can be considered as "control" structures, and VGA players is in line with Volkow's model. It can be suggested that decreased volumes in these "control" structures also decrease inhibition towards conditioned VG play, thus increasing disposition towards VGA. However, structural differences do not necessitate differences in brain functions, especially inhibition. Hence, we should examine how differences in inhibitive behaviour and cognition could increase disposition towards VGA.

In Ding et al.'s study (Ding et al., 2014), inhibition of impulsive behaviour between IGD and healthy controls was measured. This was achieved by having participants engage in Go/No-go tests, which are robust measures of impulsivity (Gomez, Ratcliff, Perea, 2009), while under a fMRI machine. Results showed that IGD participants had higher levels of activation during response inhibition within the PFC and the ACC than non-IGD participants, however there was no significant difference in Go/No-Go performance between the groups. Similar results were also obtained in Dong et al.'s study (Dong et al., 2012), testing the impulsivity of IGD and non-IGD participants using a Stroop task (MacLeod, 2015), which measures impulsivity based on test performance and participant reaction time. Results showed that while IGD participants were able to obtain similar results compared to controls, they had greater levels of ACC activity and slower reaction times. These results suggest that

the cognitive process of inhibition was harder for IGD individuals, hence requiring increased brain activity in the "control" centres to produce inhibitive behaviour, and thus leading to the performance results observed in the Stroop and Go/No-go tasks. Thus, these results also suggest that lowered inhibition towards impulsive behaviour could increase disposition towards VGA.

Furthermore, there also seems to be a link between increased impulsivity and decrease in "control" structure volumes. In Han et al.'s study (Han et al., 2012), the grey matter volumes between those with VGA, e-sport professionals and healthy control groups were compared through MRI scans. Impulsivity was measured using the Barratt Impulsiveness Scale ("BIS11", 2019). The results found that VGA players had decreased grey matter volumes in the right posterior cingulate gyrus (CG), which is part of the ACC and thus a "control" structure. However, compared with VGA players, e-sport professionals had higher volumes in the CG. The results also found that increases in CG volumes were correlated with higher levels of impulse control. These results suggest that impulsivity control and "control" structure volumes are interlinked and provide further evidence that inhibition of impulsive behaviour is correlated with decreased addictive behaviour. However, it should be noted that with brain-imaging studies, all conclusions obtained are correlational. It cannot be determined whether VGA is resulted from decreased inhibition, or excessive VG leads to decreases in inhibition and "control" structure volume. But, assuming that e-sports professionals and VGA players have similar levels of VG and e-sports professionals have higher impulsivity control, it seems that high levels of VG do not necessitate deterioration in inhibition nor "control" structure volumes.

Yet, there is evidence suggesting the contrary. In Han et al.'s study, (Han et al., 2010), a 6-week study was conducted between players who played VGs for more than 60

minutes/day (PVM) and those who played less (PVL). All participants were asked to play a VG for 60 minutes/day for 6 weeks, however, play time was not strictly controlled. Initial fMRI and post-play fMRI scans were taken of the participants. Those who were classified as PVM also tended to play more than those in the PVL group. The results were that for PVM players, there was significant increase in ACC activity between pre and post-play fMRI scans, while no change was observed in PVL groups. While no test for impulsivity was conducted, it can be inferred from previous studies that higher ACC activity could be a sign of requiring increased inhibition on conditioned impulses (in this case VG images). This seems to suggest that prolonged VG could decrease inhibition of VG impulse behaviour. This is further supported through Lee et al.'s VBM study (Lee, Namkoong, Lee & Jung, 2017) on long-term IGD players and healthy controls. The results were that long-term IGD players had less grey matter volume in the ACC and left ventro-lateral PFC (VLPFC). As described in Han et al.'s 2012 study, there is a correlation between decreased volumes in "control" structures and increases in impulsivity. Through these studies, it appears that decreases in impulsivity and "control" centre volumes would be correlated with increased disposition towards IGD. However, there are problems with this conclusion, which will be discussed in the next section.

## 1.3 Conclusion to the Neurobiological Approach

To conclude, most studies used in the neurobiological approach make use of brain-imaging studies, thus the conclusion that faulty "control" centres and RML structures would increase disposition towards VGA can only be considered as correlational. The root cause of VGA cannot be adequately identified as a result, even considering the retrospective studies of Han et al. 2010 and 2011 and Lee et al. 2017. In these studies, some of the participants inherently played more VG than other participants, and it is unknown whether their motivation stems from pre-existing neural abnormalities. Hence, there is bidirectional ambiguity with regards to VGA/excessive VG and neural abnormalities, suggesting other factors that can affect disposition towards VGA, such as psychosocial factors.

With regards to the participants used in the studies, abnormalities and their resulting effects appeared consistent throughout cultures, yet more research regarding IGD has been conducted in East Asian countries. This could be due to cultural norms favouring people to play VGs, increasing the likelihood of people developing VGA and hence more research would be conducted there due to large number of samples being available. There is also a heavy bias towards male samples. This is perhaps due to how global gender roles bias males in playing VG, as such there would be less representation of VGA females. However, in the studies examined, there appears to be no gender differences in the neural abnormalities of IGD individuals. Overall, there is sufficient empirical evidence to support the neurobiological models of Volkow and Brewer.

## 2. The Psychosocial approach towards VGA

In the psychosocial approach towards addiction, one of the most comprehensive theories would be Orford's *Excessive appetites* theory (Orford, 2001). Orford's theory proposes that addiction, or "appetitive consumption", would arise due to psychosocial factors triggering people's certain degree of need for addictive stimuli. Orford also notes that most behaviours have the potential to be addictive. Orford suggests that as different behaviours would produce different levels of pleasure, each behaviour would have unique psychosocial mechanisms behind them. However, the strongest and most common predictors behind each addiction are ecological, socio-economic, cultural in nature. These include the ease in doing the behaviour and the normative influence of friends (Orford, 2001). Furthermore, it suggests that associative learning would enhance the development of attachment towards addictive behaviour, and without environmental and physiological deterrence, appetitive consumption would increase till excess (i.e. addictive behaviour develops).

With this viewpoint in mind, Orford's theory can be adapted to VGA in that risk factors lie more on the person's situational, social and cultural context. A person could develop VGA as a result of normative influence from other problematic players, thus compromising a person's cognitive ability to discriminate between normal and excessive behaviour. Forrest's (Forrest, 2016) study investigates this effect, by investigating the association between VG play time and social factors. Normative influence was determined by surveying Australian adult participants' problematic gaming levels, perceived social support and the makeup of participants' friends and family members. The results showed that problematic gaming positively correlated with frequency of play alone, looking for social interaction through VG, and percentage of time spent playing online with strangers.

Problematic gaming was negatively correlated with perceived support from significant others and friends. The results of the study support Orford's theory on how normative influence would be important in the maintenance of excessive consumption. However, owing to the self-report and correlational nature of the study, it is not known whether the correlation discussed above would be an effect or cause of VGA. Thus, to improve upon this limitation a longitudinal study can be considered to narrow down the social factors behind VGA.

A 5-year longitudinal study conducted by Rehbein and Baler (Rehbein & Baler, 2014) is able to show social factors for VGA. The 5-year study was first conducted on young German children aged 10 years old, measuring their VGA levels through the Video Game Addiction scale (Lemmens, Valkenberg and Peter, 2009) and their family backgrounds, school life and media usage. The study's results showed that single-parent backgrounds and high media usage (including gaming time and problematic gaming behaviour) would lead to increased VGA scores; high levels of social integration within school, heightened parental devotion and supervision in childhood are associated with lower VGA scores. The first group of associations are as predicted in Orford's model, where uninhibited VG play (due to general inefficiency of supervision from a single parent) lead to an increased likelihood of VGA. This is due to the reduced inhibition on children's VG play, which led them to accept problematic VG play as normal. Also, the increased social support in the second group of associations reduced the likelihoods of VGA, as these groups of children are able to recognise the limits in VG play and thus would not increase their excessive consumption of VGs. This is supported by similar results in Bonnaire & Phan's study (Bonnaire & Phan, 2017), where non-problematic gamers are associated with better parental monitoring and family cohesion.

Finally, psychological factors should be considered to obtain a holistic outlook of the psychosocial factors in VGA. Gentile et al.'s 2-year longitudinal study (Gentile et al., 2011) on primary and secondary-level school children in Singapore aimed to investigate such factors. The participants answered questionnaires regarding the participants' weekly amount of game play, impulsivity, social competence, depression, social phobia, anxiety, and school performance. The results found that low social competence (low empathy and emotional regulation) and high impulsivity would result as psychological risk factors for VGA; it was also found that factors such as depression, anxiety, social phobias would actually be outcomes of pathological gaming. The second conclusion appears to refute previous correlations observed in IGD-afflicted individuals, where they were suggested to be factors for VGA (Bargeron and Holmes, 2017; Wartberg et al., 2017; Rho et al., 2017). The psychosocial factors of low social competence and high impulsivity also fit within Orford's excessive appetites model, as lower social competence could decrease the normative influence of friends, which is stated to be a deterrence in excessive consumption of an addictive behaviour. It is also interested to note that impulsivity is correlated with abnormalities in the "control" structures as stated in the neurobiological approach, and this will be further discussed in the section below.

## 2.1 Conclusion to the Psychosocial Approach

While it seems that the psychosocial factors of the normative influence of friends, social competence, family cohesion and impulsivity are convincing psychosocial predictors of VGA, there are limitations using this approach. For one, the predictive validity of the factors can be affected by the methodology used to determine them. As seen in the above studies, these factors were determined by survey methods or interviews. Such methods have biases including social desirability and interviewer bias, which may lead to participants decrease in reporting of VG play or increase reporting of negative social experiences such as lack of family support. Furthermore, with impulsivity also being correlated with neurobiological structures, it brings into question as to whether psychosocial or neurobiological factors would be the major factor towards VGA. Finally, the conclusions obtained are still correlational in nature due to the nature of survey methods and longitudinal studies. Hence, no specific cause can be identified for VGA.

The sample sizes in the studies used are all very large (>500) and come from a wide range of cultural backgrounds. Thus, the results can be generalised to a wider population that has similar access to VGs as the countries discussed above. Gender seemed to be less of a predictor of VGA than psychosocial factors, suggesting that the skewed gender bias observed in the neurobiological approach is due to societal gender roles instead, and not reflective of the differences in prevalence between genders. Overall, the psychosocial approach has produced descriptive predictors of VGA, but does not fully explain why VGA could occur from these predictors.

## 3. Conclusion

With regards to the research question:

**To what extent do neurobiological factors increase disposition towards VGA?**

This question was answered through heavy examination of the neurobiological approach, particularly within the areas of neural abnormalities and the role of dopamine and supplemented with an alternative explanation through the psychosocial approach.

As the neurobiological approach provided large amounts of empirical evidence that supported a neural mechanism behind VGA, while the psychosocial approach was only able to provide predictors of VGA and not a precise mechanism, it can be argued that neurobiological factors increase disposition towards VGA to a large extent.

However, it must be noted that neither approach was able to provide a definitive cause for VGA. This is due to how the methodology in both approaches all led to correlational conclusions, as experimental methodologies would be impossible for ethical reasons. It should also be noted that the neurobiological approach may be reductionist in that there are other factors that would promote VGA, such as psychosocial factors. Hence, the neurobiological approach should not be taken as the definitive approach. Regarding the psychosocial approach, there are even more factors that would affect VGA predictors such as personality disposition or socioeconomic backgrounds. If further research is to be conducted within the psychosocial approach, these factors must be considered.

Research from this essay may not only promote understanding into the neural circuitry behind VGA, but also suggest a direction for VGA treatment. With VGA being shown to have similarities with substance addictions in producing neural abnormalities, treatment options for VGA can be modelled after treatment for substance addictions. For example, medication targeting the mesocorticolimbic pathway or cognitive behavioural

therapy can be used in the treatment for VGA. Following along this line, further research could also be conducted regarding treatment methods that employ the psychosocial approach in treating VGA.

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# EE/RPPF

**For use from May/November 2018**



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## Extended essay - Reflections on planning and progress form

***Candidate:*** *This form is to be completed by the candidate during the course and completion of their EE. This document records reflections on your planning and progress, and the nature of your discussions with your supervisor. You must undertake three formal reflection sessions with your supervisor: The first formal reflection session should focus on your initial ideas and how you plan to undertake your research; the interim reflection session is once a significant amount of your research has been completed, and the final session will be in the form of a viva voce once you have completed and handed in your EE. This document acts as a record in supporting the authenticity of your work. The three reflections combined must amount to no more than 500 words.*

**The completion of this form is a mandatory requirement of the EE for first assessment May 2018. It must be submitted together with the completed EE for assessment under Criterion E.**

***Supervisor:*** *You must have three reflection sessions with each candidate, one early on in the process, an interim meeting and then the final viva voce. Other check-in sessions are permitted but do not need to be recorded on this sheet. After each reflection session candidates must record their reflections and as the supervisor you must sign and date this form.*

## First reflection session

Candidate comments:

Video games (VG) play a large role in entertainment, and there are inherent risks attached to its use, such as developing video game addiction (VGA). Being an avid gamer myself, I would like to investigate the factors behind why VGA would occur, and therefore learn how to recognise and prevent it. As VGA can be explained by a large number of factors, at first I narrowed down my research solely towards Mark Griffiths' studies on the design characteristics of VGs (i.e. the structure and elements of VGs). However, after my initial reflection session, I realised that such studies take less into account the psychological approaches taught in class (e.g. the sociocultural approach), leading to a very reductionist conclusion. Hence, I plan to incorporate the biological approach in my research to serve as an alternative explanation, using studies that investigate the correlation between brain activity and VGA.

Date: March 8, 2018

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## Interim reflection

Candidate comments:

It was disheartening to disregard my research on VG design characteristics. There isn't much research in this area, and most of it was theoretical with little supporting evidence. Hence, I have switched my research focus onto the neurobiological approach, with the psycho-social approach as the alternative explanation. However, my understanding on addiction neurobiology is limited, as we did not specifically study addiction in IB Psychology. I had to learn the neural circuitry behind addiction through online textbooks and research papers, then verified my understanding through my supervisor. I was glad to have found lots of research through the neurobiological and psychosocial approach. Yet, I found it difficult to decide which research to use, especially when figuring out which social factors would affect VGA. Writing the outline and discussing with my supervisor really helped me in prioritising which research to use, and how to move on from deleting my previous work.

Date: March 16, 2018

## Final reflection - Viva voce

Candidate comments:

I am proud to be able to realise my research interests. I believe this came about due to my choice in switching my focus to neurobiology, which was better researched than the factor of VG design characteristics in VGA. However, I was disappointed in being unable to find significant research into the genetic factors of VGA, which are often cited as causes for other behavioural addictions such as gambling. In hindsight, not employing genetic factors in my argument may have strengthened it instead, with how flimsy the genetic evidence for VGA is. In doing so, I learnt much about how to prioritise the evidence I should use and how to build a strong argument.
I very much enjoyed researching this topic, and in the process I developed a new appreciation for neurobiology. It is amazing to discover how our reward circuitry can have such a large effect on our behaviour, and I now believe that the supposed factor of VG design characteristics on promoting VGA is essentially due to such characteristics being able to overload our reward circuitries instead. Over the course of my research, I feel that my information synthesis and self-learning skills have improved, leading me to become a better researcher overall.

Date: February 1, 2019