

The common neuronal basis to drug taking and gambling behaviour

[Health & Medicine](#), [Addiction](#)



INTRODUCTION

The objective of this review is to find a research publication that addresses the question, 'is there a common neuronal basis to drug taking and gambling behaviours?'. The publication that addresses this question and demonstrates an advancement in research findings is, 'Shared microstructural features of behavioural and substance addictions revealed in areas of crossing fibres' by Yip, Morie, Xu, Constable, Malison, Carroll, & Potenza (2017). The understanding that gambling behaviours and substance use share common clinical features is demonstrated by Diagnostic and Statistical Manual of Mental Disorders (DMS-IV) in 2013, renaming disordered gambling to pathological gambling and reclassifying it under a new classification titled 'Substance related and addictive disorders' (Christensen, Jackson, Dowling, Volberg, & Thomas, 2015).

Christensen et al., (2015. p. 788), identify that gambling had previously been classified as 'Impulse-Control Disorders Not Elsewhere Classified', along with compulsive stealing (kleptomania), fire starting (pyromania) and hair pulling (trichotillomania). Disordered gambling, which is the sole behavioral addiction in the 'Substance related and addictive disorders' group, is thought to have similarities to cognitive, neurological, genetic, and behavioral relationships found in substance dependence, as well as high rates of substance dependence comorbidity (Christensen et al., 2015). Many clinical studies have researched the shared clinical characteristics, such as loss of control and cravings between behavioral addiction and substances

use disorders and proved similarities (Romanczuk-Seiferth, Koehler, Dreesen, Wüstenberg, & Heinz, 2015).

Other studies, such as Leeman & Potenza (2012, p. 469-490), have focused on impulsivity and compulsivity reviewing neurocognitive tasks, brain function and neurochemistry to determine similarities and differences between substance use disorders and gambling. There have been many neuroimaging studies that have collected data suggesting that there are similarities between behavioural and substance addictions that may relate to disease etiology (Potenza, 2008). Changes within white-matter (WM) tracts that have been assessed using diffusion-weighted magnetic resonance imaging (dMRI) have been documented among subjects with substance addictions, such as cocaine use disorder (CUD) and with those with behavioural addictions such as gambling disorder (GD), but lack a study of comparison. Other studies have shown neural functional similarities between CUD and GD, despite these findings the extent of neural structural changes within addictive disorders has not been determined (Potenza, 2008).

Yip et al., (2017, p. 188), believe that the ability to identify common and distinct neural structural changes in individuals with addiction subtypes could be used to further new interventions based on known brain features. Notably dMRI measures have demonstrated links to neurocognition and behaviour and may also be receptive to behavioural and pharmacologic interventions. The publication by Yip et al., (2017), is the first research to test the hypothesis of shared WM tissue alterations between behavioural and substance addiction via comparison of dMRI measures from individuals with

GD, individuals with CUD, and healthy comparison (HC) individuals. The reviewed study concludes that anisotropy reduction of secondary fibres within the forceps major, corona radiata, the left internal capsule and posterior thalamic radiation among individuals with GD and CUD compared to HC. Also, no changes in anisotropy measures between GD and CUD participants, confirming their hypothesis.

The study conducted by Yip et al., (2017), included 38 individuals with GD, 38 with CUD and 38 HC. Yip et al., (2017. p. 188), recognised the need for their study to assess the more complex WM architecture i. e. voxel which contains fibres of different orientations, they were able to complete the study using the crossing fibre model proposed by Behrens et al. Data from dMRI was able to calculate the partial volume estimates (PVEs) corresponding to the primary or secondary fibre orientations (PVE1 and PVE2). Yip et al., (2017. p. 188), predicted that individuals with CUD and individuals with GD would display reductions in anisotropy measures when compared with HC individuals, however they would not alter from each other. Limitations to this study highlighted by Yip et al., (2017. P. 193), included control groups not being well matched for tobacco use and years of education. They also did not have a biological measure of drug use whilst scanning individuals and there is an absence of common measure of addiction severity which could have allowed for further exploration on neuroimaging measures and illness severity.

Yip et al., (2017. p. 189), anticipated specifically that GD and CUD participants would have reduced anisotropy within WM tracts such as the

genu, splenium, internal capsules, thalamic radiations, corona radiata and superior longitudinal fasciculus. Included in this study were GD and CUD individuals with alcohol use disorder (AUD), as other studies like this have eliminated AUD from their study or others tried to control AUD effects. Not limiting themselves in their research Yip et al., (2017, p. 189), has added a third aim to assess the relationship between impulsivity and WM characteristics across addictive disorder diagnosis. By the end of the study Yip et al., (2017), had demonstrated that CUD and GD individuals had decreased anisotropy with corticolimbic tracts compared to HC individuals but did not alter from one another, as consistent with their primary hypothesis.

Areas implicated in reward processing and addiction vulnerability: the internal capsule, corona radiata and forceps major were areas identified as separating GD and CUD from HC participants. This reduced internal capsule anisotropy could possibly be a neural structural mechanism for blunted ventral striatal activity or for changes in ventral striatal functional connectivity of which both have been reported in individuals with GD and/or CUD. The forceps major shows altered network activity among people with attention-deficit/hyperactivity disorder. Yip et al., (2017, p. 193), having shown decreased anisotropy to the forceps major; thus, may relate to alterations in these network activities. Not being able to compare these results with related research as this is the first of its kind provides motivation for further research with transdiagnostic research to test this hypothesis and

also to determine whether there are link between functional MRI and dMRI findings associated with behaviours and substance addiction.

To ensure their study was not limited Yip et al., (2017), included participants with AUD due to the high co-occurrence rates of AUD in GD and CUD, this is demonstrated by approximately 50% of GD and CUD study participants. As others had excluded AUD in other studies this is an enhancement in their research field. Yip et al., (2017. p. 189), hypothesised that within the genu there would be reductions in anisotropy measures in individuals with a history of AUDs as opposed to in those without. Among GD individuals with a history of AUDs, evidence showed primary fibre PVEs were decreased and secondary fibres PVEs were increased compared to those GD without a history of AUDs. CUD participants with and without AUDs showed nil differences in anisotropy. Anisotropy estimates (FA, PVE1, PVE2) for the corpus genu did not confirm Yip et al., (2017), hypothesis as the results did not differ across participants. As the genu is made up of mostly uniformly oriented, densely packed fibres; the reductions in primary fibre anisotropy to this area would most likely reflect the reduced fibre density. Secondary fibre PVEs within the genu that showed increases is yet to be determined. One hypothesis suggest that this may be due to aberrant compensatory development of non-dominant fibres, future research could be directed to confirm this. Increase in impulsivity have been reported in individuals within the range of addiction and thought of as a shared vulnerability marker of addictions and other disorders.

Yip et al., (2017, p. 189), hypothesised they would be able to replicate previous results of negative associations between frontal WM and the anterior corpus callosum of individuals with GD and CUD who self-reported impulsivity. Self-reported impulsivity was assessed using the Barrat impulsivity scale version II (BIS-II) which when examined separately within GD, CUD and HC groups, it found it was not related with anisotropy measures. Combined analyses of all diagnostic groups combined indicated a negative result of anisotropy within the internal capsule and forceps major clusters, this is consistent with transdiagnostic dimensional concepts of impulsivity. This does not support Yip et al., (2017), hypothesis that micro-structural WM aspects are related to individual fluctuations in impulsivity of those diagnosed with GD and CUD. As there are no anisotropy differences in the forceps major or internal capsule of GD and CUD groups, it would suggest that alterations in these regions could be a common element of substance and behavioural addictions. Following this, participants with GD and CUD with/without AUD did not differ in anisotropy in these regions. GD individuals with/without substance abuse/dependence also did not differ in anisotropy of these regions.

These findings then suggest that reduced secondary fibre anisotropy within the parietal-occipital and striatal regions is a mutual feature of addiction subtypes possibly related to disease etiology. Again, as this is the first study of its kind Yip et al., (2017), suggests that further research be done to determine accurate function significance of these results. CONCLUSIONThe overall goal of this review was to answer the question, ' is there a common

neuronal basis to drug taking and gambling behaviours?'. As Yip et al., (2017), could demonstrate that anisotropy reduction of secondary fibres among individuals with GD and CUD compared to HC, and that there were no changes in anisotropy measures between GD and CUD participants. They were able to confirm their original hypothesis and able to begin to answer the overall question. This is the first study to compare diffusion indices directly between substance addictions and behaviour addictions and is also the largest dMRI study of GD. Findings of the study conducted by Yip et al., (2017), indicate similar WM microstructural alterations across addictions that can't be attributed entirely to exposure to drugs or alcohol and therefore must be a vulnerability mechanism of addictive disorders. To further the work done by Yip et al., (2017), to answer the question, 'is there a common neuronal basis to drug taking and gambling behaviours?', a large-scale longitudinal research study is needed. This would then also include greater social economic factors, comparing WM changes with grey matter changes in GD and CUD, common measures of addiction severity and the effects of treatment outcomes on WM pathways in these individuals.

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