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**Disordered gambling and the effects of the two major comorbidities
in decision-making, brain function and structure: substance use
disorder & depression risk**

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Disordered gambling is suggested to be a suitable model to investigate addiction in general, because no toxicity from drugs of abuse is involved. Therefore alterations or differences found in disordered gambling patients will be due to the behavior or a pre-existing vulnerability. Recognizing heterogeneity in disordered gambling, this thesis dealt with comorbidity and its effect in rational decision making, effort related monetary reward processing and brain gray matter volume. A large cohort of patients diagnosed with disordered gambling was included in the investigation, matched to an equally large cohort of healthy controls. Decision-making abilities in both disordered gamblers with and without substance use disorder resemble that of substance use disorder patients. Deficits in that domain of cognition characterise all disordered gambling subgroups relative to controls. What is more, risk taking was the element that differentiated the groups (with vs. without substance use disorder). Alcohol and nicotine dependent disordered gamblers proved risk takers. However, disordered gamblers free of substance use disorder comorbidities avoided risk taking tendencies. Likewise, gray matter tissue volume differentiated patients from healthy controls not only as a whole group but also on the basis of their substance use disorder. Specifically, gray matter volume reductions were found in disordered gambling alcohol dependent participants mainly in the frontal cortex and insula similar to gray matter tissue reductions often seen in alcohol dependent subjects. However, disordered gamblers without substance use disorder comorbidity exhibited gray matter volume alterations in the form of striatal volume increase compared to healthy controls and in line with previous findings. Regarding results from the effort related monetary reward processing; gamblers do not necessarily suffer from either blunted or hyperactive brain reward function. They seemed to exhibit similar brain activation to healthy controls. Interestingly though depression symptomatology distinguished brain activity. Disordered gamblers at risk for depression hyper-activated compared to those disordered gamblers with lower risk for depression. Differences were found only during feedback phase and involved increased blood oxygen level dependent response in the insula and striatum of disordered gamblers with high scores as suggested by the Beck depression inventory. Disordered gamblers show similar deficit as patients with substance abuse problems, despite the lack of drug of abuse toxicity in their brain. Current findings support the view that disordered gambling stands as an addiction facet and justify the recent reclassification of disordered gambling from an impulse control disorder to addictive disorder in the 5th edition of the diagnostic and statistical manual of mental disorders.