**Unpacking the role of self-reported compulsivity and impulsivity in obsessive-compulsive disorder**

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**Abstract**

**Objective:**

We aimed to determine whether individuals with obsessive-compulsive disorder (OCD) and demographically-matched healthy individuals can be clustered into distinct clinical subtypes based on dimensional measures of their self-reported compulsivity (OBQ-44 and IUS-12) and impulsivity (UPPS-P).

**Methods:**

Participants (*n* = 217) were 103patients with a clinical diagnosis of OCD; 79 individuals from the community who were ‘OCD-likely’ according to self-report (Obsessive-Compulsive Inventory-Revised scores equal or greater than 21); and 35 healthy controls. All data were collected between 2013-2015 using self-report measures that assessed different aspects of compulsivity and impulsivity. Principal component analysis revealed two components broadly representing an individual’s level of compulsivity and impulsivity. Unsupervised clustering grouped participants into four subgroups, each representing one part of an orthogonal compulsive-impulsive phenotype.

**Results:**

Clustering converged to yield four subgroups: one group low on both compulsivity and impulsivity, comprised mostly of healthy controls and demonstrating the lowest OCD symptom severity; two groups showing roughly equal clinical severity, but with opposing drivers (i.e., high compulsivity and low impulsivity and vice-versa); and a final group high on both compulsivity and impulsivity and recording the highest clinical severity. Notably, the largest cluster of individuals with OCD were characterised by high impulsivity and low compulsivity. Our results suggest that *both* impulsivity and compulsivity mediate obsessive-compulsive symptomatology.

**Conclusion:**

Individuals with OCD can be clustered into distinct subtypes based on measures of compulsivity and impulsivity, with the latter being found to be one of the more defining characteristics of the disorder These dimensions may serve as viable and novel treatment targets.

**Introduction**

Obsessive-compulsive disorder (OCD) is a neuropsychiatric disorder affecting between 0.3 to 3.1% of the general population 1, and places enormous personal, social and economic burden on society 2. Clinically, OCD can present with a variety of unwanted obsessions, compulsions, or an idiosyncratic mix of both. In addition, OCD can also be associated with other compulsive and impulsive tendencies, such as substance and gambling addiction 3. Historically, there has been a strong focus on the symptom phenotypes of OCD, however, given its heterogeneous presentation and high rates of relapse, it is important to understand OCD from the perspective of motivational phenotypes that may underlie the disorder. That is, to understand the precise processes and traits that mediate the dysfunctional neural circuits implicated in OCD (for a review, see Pauls, Abramovitch, Rauch, & Geller, 2014) and its superficial symptomology 5. This approach may offer new avenues for progress with regard to intervention and prevention 6–9.

OCD has traditionally been viewed as a disorder driven primarily by anxiety-avoidance and excessive self-control 10. In recent years, however, a competing perspective has emerged that sees two phenotypes driving the behavioural features of OCD: compulsivity and impulsivity 7,9,11. Impulsivity is commonly defined as a predisposition to reacting to stimuli in a rapid and unplanned fashion with reduced concern of the potential consequences 7,12. In contrast, current conceptualisations suggest that compulsivity reflects the repetitive performance of behaviours without adaptive function that carried out habitually or in response to avoidance and fear of potential negative outcomes 7. Traditional views posit that these phenotypes are diametrically opposed, whereas contemporary views suggest that compulsivity and impulsivity may be more overlapping in nature 9,13, suggesting that disorders typically characterised by clinically significant expression of one may also show maladaptive expression of the other.

OCD has been labelled the archetypal compulsive disorder 14. It is, therefore, unsurprising that individuals with OCD differ from healthy controls (HCs) by showing compulsive responses across a number of tasks 7. However, a growing body of evidence highlights the potential implication of impulsivity in OCD. For example, a number of studies examining trait impulsivity in OCD have found that OCD patients show higher ‘attentional’, ‘motor’ and ‘nonplanning’ impulsivity than HCs 10,15–19, as well as increased ‘negative urgency’ 20. Individuals with OCD, relative to HCs, tend to make risky decisions, favouring options that provide large initial rewards but ultimately lead to a disadvantageous outcome 10,21–28. This consistent pattern of irrational responding might reflect an exacerbated anticipation for reward 29, or failure of a somatic marker to signal differences between advantageous and disadvantageous outcomes 30 or both. Furthermore, OCD patients show a robust impairment in response inhibition 31–34. Most strikingly, Figee et al35 found increased reward anticipation in the nucleus accumbens - a key region subserving reward, motivation and impulsivity 36 - in individuals with OCD, compared to HCs37. This growing body of evidence notwithstanding, a number of issues remain. First, some scattered studies have not found differences between individuals with (clinical or subclinical levels of) OCD and HCs on some measures of impulsivity 14,15. Evidence from our large sample should assist in interpreting whether these few studies are exceptional. Secondly, and more importantly, it is also unclear whether a potential impulsive subgroup would show more or less severe clinical outcomes than other more compulsively-driven subgroups. Impulsive tendencies could plausibly compound or protect against compulsive tendencies. The scant evidence that speaks to this issue suggests a potential impulsive subgroup might be especially vulnerable to worse prognosis 24, but further investigation is needed.

In this study, we apply k-means clustering to trait measures of compulsivity and impulsivity to regroup a large sample of individuals with OCD. Our aims were to determine whether (i) orthogonally related components of compulsivity and impulsivity could be used to separate groups of OCD patients and (ii) these phenotypes are linked to clinical severity. To address the first aim we separated our sample into four clusters using self-report measures of compulsivity and impulsivity. Specifically, we expected to find a cluster of OCD individuals who score low on both compulsivity and impulsivity, a cluster who score high on compulsivity but low on impulsivity, a cluster who score low on compulsivity but high on impulsivity (i.e., the reverse of the previous cluster), and a cluster who score high on both. With regard to the traditional anxiety-avoidance model in OCD 10, we expected to find the largest proportion of individuals with OCD falling into the high compulsivity and low impulsivity cluster. Furthermore, we predict that the cluster scoring high in both clinical phenotypes should demonstrate the highest clinical severity 24.

**Methods**

**Subjects**

The total sample consisted of 217 age- and sex-matched participants (female = 108; mean age = 35.70 years; age range = 18-77): 103 individuals clinically diagnosed with OCD; 79 ‘OCD-likely’ individuals; and 35 HCs. All participants gave written informed consent to participate in study protocols approved by the relevant Australian and Brazilian ethics committees. Clinically-diagnosed individuals participated in the present study at one of two clinical institutes as part of larger studies: Monash Institute of Cognitive and Clinical Neurosciences, Melbourne (n = 36) and the Institute of Psychiatry of the Federal University of Rio de Janeiro (n = 67). Recruitment for both sites was primarily from patient clinics local to each site, as well as media advertisements placed in the broader community. A primary diagnosis of OCD was determined by the patient version of the *Structured Clinical Interview for the DSM-IV (SCID-IV)*. If patients had other comorbid diagnoses thought to be more severe or to underpin their OCD they were excluded from the study. OCD-likely individuals were recruited online through the Amazon Mechanical Turk community. OCD-likely was defined as a score of 21 or higher on the *Obsessive Compulsive Inventory-Revised (OCI-R),* an 18-item self-report instrument that determines the severity of OCD symptoms in sub-clinical populations 38. HCs were part of a larger study at the Melbourne site. HCs were screened using the Mini International Neuropsychiatric Interview (MINI; DSM-IV) as well as an in-house structured interview designed to assess lifetime history of neurological illness, and were excluded if they presented with any past or present psychiatric or neurological illness.

The definitions of impulsivity and compulsivity outlined in the introduction have a strong focus on behaviour, however, here we were interested in examining the underlying motivational phenotypes that drive these behaviours. As such, all participants completed a 30-minute computerised battery of self-report questionnaires that putatively assessed compulsivity and impulsivity. To assess compulsivity, we used *Intolerance of Uncertainty Scale-Short Form* *(IUS-12)* 39 and *Obsessive Beliefs Questionnaire-44* (*OBQ-44)*40. The OBQ-44 measures belief domains linked to OCD using four sub-scales: perfectionism, importance and control of thoughts, responsibility and overestimation of threat. The IUS-12 measures responses to uncertainty, ambiguous situations and the future using two sub-scales: prospective intolerance to uncertainty, and inhibitory response to uncertainty. Obsessive beliefs and intolerance to uncertainty are hallmarks of OCD and are predictive of symptomatology 41,42. Thus, the IUS-12 and OBQ-44 likely capture critical motivational elements of compulsivity in OCD. To assess impulsivity, we used the well-validated *UPPS-P* 43,44. The UPPS-P measures several dimensions of impulsivity: negative urgency, lack of premeditation, lack of perseverance, sensation seeking, and positive urgency. Extended descriptions of the compulsive/impulsive measures can be found in supplementary material. Finally, the *OCI-R* 38 and the *Beck Depression Inventory (BDI)* 45 were included to measure symptom severity.

**Data analysis**

For assumption checks please refer to supplementary material.

**Compulsive and impulsive phenotypes of OCD**

To investigate whether participants with OCD could be regrouped using the constructs of compulsivity and impulsivity, we performed unsupervised clustering on the subscales of the *UPPS-P*, *OBQ-44,* and *IUS-12*. Principal component analysis (PCA) was first used to reduce the questionnaire data down to two leading principal components, which explained 52.7% of the variance in the data. A two component (i.e., a compulsivity component and an impulsivity component) solution was selected primarily to be consistent with the theoretical framework of the study. These two components carried Eigenvalues of 4.21 and 2.20, respectively. While a third PC may also have been retained due to having an Eigenvalue greater than 1 (Eigenvalue = 1.10), it explained only an additional 9% of variance and contained loadings from only two subscales of the OBQ. Thus, to facilitate interpretability, we only retained two components. Additionally, as some subscales showed variance inflation factors of greater than 3, PCA also served to reduce multicollinearity, and subsequent overrepresentation, during clustering.

**Primary analysis: Compulsive and impulsive clustering of individuals with OCD**

Using the PCA results from the above step, we clustered the clinical and OCD-likely participants by grouping individuals together with similar scores on our compulsive and impulsive phenotypes using *k*-means clustering 46. *K*-means requires that the number of clusters drawn from the data be defined *a priori*. Given our primary aim to investigate the overlapping constructs of compulsivity and impulsivity, we extracted four clusters from the data. Participants within each *k*-means cluster should exhibit homogenous scores on the compulsive and impulsive phenotypes. To examine whether these groupings provide clinical utility in OCD, we compared the clusters’ OCD severity using the *OCI-R*. See supplementary material for validation against different clustering methods and of the optimal number of clusters.

**Secondary analysis: Compulsive and impulsive clustering of individuals with OCD and HCs**

To capture a broad spectrum of OCD severity, we included OCD-likely participants in our primary analysis, which introduced the influence of subclinical OCD tendencies into our sample. To extend the spectrum of severity even further toward normality 6,47, we repeated our PCA and clustering analyses including 35 HC participants and examined the proportions of HCs and OCDs (both clinical and likely) that fell into each of the four clusters extracted from the data using *k*-means clustering.

**Results**

**Sample characteristics**

OCD and OCD-likely Individuals had significantly higher (*p* < 0.001) *OCI-R* scores (*M* = 31.1) than HC participants (*M* = 4.82). The OCD, OCD-likely and HC samples did not differ on age or gender (*p* > .05). Pearson correlations between total OCI-R scores and the questionnaire measures can be found in Supplementary Table 1.

**Compulsive and impulsive phenotypes of OCD**

Our primary goal was to evaluate whether orthogonally related constructs of compulsivity and impulsivity, measured using the OBQ-44 and IUS-12, as well as the UPPS-P, respectively, could distinguish symptom severity in a sample of OCD and OCD-likely participants. PCA revealed two components that putatively represent an individual’s level of compulsivity and impulsivity (Table 1). Internal consistency for the compulsivity and impulsivity components were α = 0.76 and 0.70, respectively. Table 1 illustrates how the measures of compulsivity and impulsivity used loaded onto respective compulsive and impulsive components.

**Compulsive and impulsive clustering of OCD**

We used unsupervised clustering methods to identify subgroups of OCD participants based upon their levels of compulsivity and impulsivity. When extracting four clusters using *k*-means, the results identify four groups of OCD participants that mapped onto our predicted compulsive/impulsive phenotype (Figure 1). Cluster 1 contained 30 participants scoring low on both compulsivity and impulsivity (hereafter referred to as low/low); cluster 2 had 41 participants scoring moderately on compulsivity and low on impulsivity (hereafter referred to as high/low); cluster 3 contained 71 participants low on compulsivity and high on impulsivity (hereafter referred to as low/high); and cluster 4 contained 40 participants high on both compulsivity and impulsivity (hereafter referred to as high/high). It is important to note that low and high scores on the PCs represents a score relative to average of the sample. Thus, ‘low’ on compulsivity (or impulsivity) may not represent qualitatively ‘normal’ levels of compulsivity, rather lower compared to other OCDs in this sample.

Assessing the robustness of our clustering solution using the OCD and OCD-likely groups, we also found that: (i) two-step cluster analysis confirmed the four cluster solution as optimal, with the Silhouette measure of cohesion and separation equal to 0.5 48; (ii) a four cluster solution was also supported by convergence between Ward’s and k-means methods (Cramer’s V = 0.74, *p* < .001); (iii) cluster membership was not driven by whether participants were diagnosed with OCD or whether they were OCD-likely (Cramer’s V = 0.10, *p* = .065); and (iv) clusters did not differ in terms of gender (χ2 = 2.11; *p* = .065), age (*F*(3, 178) = 1.380, *p* = .250), or depression (Cramer’s V= .185; p=.105).

**OCD severity across subgroups**

There was a significant effect of cluster membership on OCD severity (F (3, 178) = 19.91, *p* < .001). Post-hoc t-tests indicated that the high/high OCD cluster had higher scores on the OCI-R (*M* = 39.23, *SD* = 12.27) than all other clusters (*p*s < .05; Figure 2), whereas the low/low OCD cluster had lower scores on the OCI-R than all the other clusters (*M* = 21.60, *SD* = 8.90, *p*s < .001). There was no difference between the high/low OCD cluster (*M* = 32.88, *SD* = 9.84) and the low/high OCD cluster (*M* = 30.04, *SD* = 9.48, *p* = 0.937). Interestingly, apart from the obsessing subscale which was significantly higher in the high/high OCD group compared to the other three clusters (p < .008), there were no significant differences in OCI-R subtypes across the four clusters, suggesting that the clusters were not driven by classical OCD subtypes.

**Compulsive and impulsive clustering of individuals with OCD and HCs**

Having established that a self-reported compulsive/impulsive phenotype can be used to separate OCD participants to reveal significant differences in disorder severity, we wanted to explore what influence introducing a HC group had on the clustering solution. The above PCA and *k*-means analyses were repeated, this time including 35 HCs. This time the compulsivity and impulsivity components accounted for 58.2% of the overall variance. Again, a clear compulsive/impulsive phenotype emerged. Table 2 illustrates how specific compulsive and impulsive measures loaded onto the principal components.

Results of *k*-means clustering revealed a qualitatively similar pattern of compulsive/impulsive clusters in the data (Figure 2). Specifically, the low/low, high/low, low/high and high/high clusters were all recaptured when adding the HC participants. Notably, 83.3% of the healthy controls fell into the low/low cluster, making up approximately half of the participants in that cluster (Figure 3). The high/high cluster was made up exclusively of OCDs and the high/low and low/high clusters were predominantly composed of OCDs. Again, as before, multiple clustering techniques converged on a four-cluster solution and clusters did not differ in terms of gender (χ2 = 2.78; *p* = .42). There was an effect of age (*F* (3,213) = 2.75, *p* = .043) but post-hoc analyses showed no age differences between any two clusters (all *p*s < 0.524).

There was a significant effect of cluster membership on OCD severity (F (3, 213) = 55, *p* < .001). Post-hoc t-tests indicated that the high/high cluster showed the highest OCI-R score (*M* = 38.39, *SD* = 11.74) compared to the other groups (all *p*s < .001). Again, the low/low cluster recorded the lowest OCI-R score (*M* = 11.51, *SD* = 11.30) (all *p*s <.001). There was no difference between the high/low OCD cluster (*M* = 28.26, *SD* = 11.50) and the low/high OCD cluster (*M* = 29.59, *SD* = 9.72, *p* = .45).

**Discussion**

Using unsupervised clustering methods applied to questionnaire data, we grouped participants with OCD into 4 distinct subgroups based on their levels of impulsivity and compulsivity. We expected that the constructs of impulsivity and compulsivity would relate orthogonally to one another. Accordingly, OCD participants were delineated on the basis of high levels of inter-individual compulsivity (i.e., high/low), impulsivity (low/high), or both (high/high), as well as low levels of both (low/low). These phenotypic differences in OCD participants were also linked to differences in OCD severity.

Traditionally OCD has been viewed as the archetypal compulsive disorder. Contemporary views have argued that OCD may also encompass issues with impulsivity and may therefore be alternatively conceptualised as a behavioural addiction 10,11,49. However, this hypothesis remains controversial 50, with some researchers reporting higher impulsivity in OCDs relative to controls 10,24 while others report no difference 13 or lower impulsivity in OCD 51. Rather than argue for one driver over another, our results suggest that *both* impulsivity and compulsivity mediate obsessive-compulsive symptomatology. Within our sample of OCD participants, there are subsets of individuals characterised by elevated levels of impulsivity with or without elevated compulsivity. Notably, the largest cluster of participants in our results were characterised by high impulsivity and low compulsivity relative to the sample mean. This suggests that a ‘one size fits all’ approach to understanding OCD phenomenology (e.g., OCD as an anxiety-avoidance disorder vs. a behavioural addiction) is likely to be overly simplistic.

Fontenelle 11 postulated that OCD behaviours may become more ‘impulsive’ in nature with chronicity, suggesting impulsivity as a potential end point for some OCD patients. While chronicity has been linked to increased severity of OCD 52, our results suggest that the relationship with impulsivity is not so clear. We found that OCD severity differed across our clusters, with the low/low cluster showing the lowest OCD severity and the high/high cluster showing the highest OCD severity, suggesting that an interaction between the phenotypes may be linked to worse prognosis. However, we also found that while OCD severity was higher for the high/low and the low/high clusters relative to the low/low cluster, severity did not differ between the two. More comprehensive understanding of the role of impulsivity in relation to OCD severity requires longitudinal data not present in the current study. Nevertheless, our results point towards the clinical importance of understanding an OCD patient’s impulsivity and demonstrate how traditional diagnostic categories can mask the underlying motivations driving OCD behaviours.

 Our finding that OCD severity links to a compulsive/impulsive phenotype may have potential implications for the management of OCD treatment in clinical practice. For instance, specific psychotherapeutic strategies to manage impulsivity may be added to exposure and response prevention of OCD patients. We have previously suggested that medication targeting impulsive behaviours (such as naltrexone, topiramate, and methylphenidate, among others) may be of theoretical help to some OCD patients 11. However, there is mixed data in relation to the efficacy of these drugs in OCD, with some reports or improvement 53,54, no change 55, or even worsening of OCD symptoms 56. These heterogeneous outcomes maybe ascribed to specific patient profiles and assessment of baseline impulsive features [e.g. severity of compulsive symptoms 54] may help determining the phenotype that would be best targeted by an anti-impulsive medication in OCD.

**Limitations**

A limitation of this study is the inclusion of only self-report measures of compulsivity and impulsivity. Compulsivity and impulsivity are both heterogeneous constructs 9,57 and are unlikely to be fully covered by self-report alone. Thus, future work should investigate whether our findings replicate when including behavioural tasks either instead of self-report measures or alongside. However, our results demonstrate that self-report measures of the compulsivity and impulsivity phenotypes are sensitive enough to distinguish clinically relevant differences in OCD subgroups. This has strong clinical utility given the convenient and cost-effective nature of self-report data compared to behavioural tasks, which often require training and practice to be administered successfully. Finally, the fact that approximately half of the low/low cluster was composed OCD participants when including healthy control participants may be ascribed to the fact that our OCD sample includes patients who were in different stages of treatment.

**Conclusions**

In summary, we used objective data-driven methods applied to self-report data to show that individuals with OCD can be sub grouped based on their levels of compulsivity (OBQ-44 and IUS-12) and impulsivity (UPPS-P), which, in turn links to differences in clinical severity. We found that individuals with high levels of impulsivity (and relatively lower compulsivity) have comparable clinical severity to those with high compulsivity (i.e., the stereotypical OCD presentation). Furthermore, we showed that the combination of high levels of compulsivity and impulsivity is linked to the highest clinical severity.

**References**

1. Fontenelle LF, Mendlowicz M V., Versiani M. The descriptive epidemiology of obsessive–compulsive disorder. *Prog Neuro-Psychopharmacology Biol Psychiatry [Internet].* 2006 May;30(3):327–37. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0278584605003465

2. Hollander E, Doernberg E, Shavitt R, Waterman RJ, Soreni N, Veltman DJ, et al. The cost and impact of compulsivity: A research perspective. *Eur Neuropsychopharmacol [Internet].* 2016 May;26(5):800–9. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0924977X16000602

3. Fineberg N a, Brown A, Reghunandanan S, Pampaloni I. Evidence-based pharmacotherapy of obsessive-compulsive disorder. *Int J Neuropsychopharmacol [Internet].* 2012 Sep 9;15(8):1173–91. Available from: http://ijnp.oxfordjournals.org/cgi/doi/10.1017/S1461145711001829

4. Pauls DL, Abramovitch A, Rauch SL, Geller DA. Obsessive–compulsive disorder: an integrative genetic and neurobiological perspective. *Nat Rev Neurosci [Internet].* 2014 May 20;15(6):410–24. Available from: http://www.nature.com/doifinder/10.1038/nrn3746

5. Gottesman II, Gould TD. The Endophenotype Concept in Psychiatry: Etymology and Strategic Intentions. *Am J Psychiatry [Internet].* 2003 Apr;160(4):636–45. Available from: http://psychiatryonline.org/doi/abs/10.1176/appi.ajp.160.4.636

6. Cuthbert BN, Insel TR. Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Med [Internet].* 2013;11(1):126. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3653747&tool=pmcentrez&rendertype=abstract

7. Fineberg N a, Chamberlain SR, Goudriaan AE, Stein DJ, Vanderschuren LJMJ, Gillan CM, et al. New developments in human neurocognition: clinical, genetic, and brain imaging correlates of impulsivity and compulsivity. *CNS Spectr [Internet].* 2014 Feb [cited 2014 May 5];19(1):69–89. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24512640

8. Gillan CM, Kosinski M, Whelan R, Phelps EA, Daw ND. Characterizing a psychiatric symptom dimension related to deficits in goal-directed control. *Elife [Internet].* 2016 Mar 1;5:1–24. Available from: http://elifesciences.org/lookup/doi/10.7554/eLife.11305

9. Robbins TW, Gillan CM, Smith DG, de Wit S, Ersche KD. Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. *Trends Cogn Sci [Internet].* 2012 Jan [cited 2014 Mar 20];16(1):81–91. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22155014

10. Grassi G, Pallanti S, Righi L, Figee M, Mantione M, Denys D, et al. Think twice: Impulsivity and decision making in obsessive–compulsive disorder. *J Behav Addict [Internet].* 2015 Dec;4(4):263–72. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=4712760&tool=pmcentrez&rendertype=abstract

11. Fontenelle LF, Oostermeijer S, Harrison BJ, Pantelis C, Yücel M. Obsessive-compulsive disorder, impulse control disorders and drug addiction: common features and potential treatments. *Drugs [Internet].* 2011 May 7;71(7):827–40. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21568361

12. Chamberlain SR, Sahakian BJ. The neuropsychiatry of impulsivity. *Curr Opin Psychiatry [Internet].* 2007 May;20(3):255–61. Available from: http://content.wkhealth.com/linkback/openurl?sid=WKPTLP:landingpage&an=00001504-200705000-00015

13. Tavares H, Gentil V. Pathological gambling and obsessive-compulsive disorder : towards a spectrum of disorders of volition. *Rev Bras Psiquiatr.* 2007;29(55 11):107–17.

14. Chamberlain SR, Leppink EW, Redden SA, Grant JE. Are obsessive–compulsive symptoms impulsive, compulsive or both? *Compr Psychiatry [Internet].* 2016 Jul;68:111–8. Available from: http://dx.doi.org/10.1016/j.comppsych.2016.04.010

15. Benatti B, Dell’Osso B, Arici C, Hollander E, Altamura AC. Characterizing impulsivity profile in patients with obsessive-compulsive disorder. *Int J Psychiatry Clin Pract [Internet].* 2014 Aug;18(3):156–60. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24151922

16. Boisseau CL, Thompson-Brenner H, Caldwell-Harris C, Pratt E, Farchione T, Barlow DH. Behavioral and cognitive impulsivity in obsessive-compulsive disorder and eating disorders. *Psychiatry Res [Internet].* 2012 Dec 30;200(2–3):1062–6. Available from: http://dx.doi.org/10.1016/j.psychres.2012.06.010

17. Ettelt S, Ruhrmann S, Barnow S, Buthz F, Hochrein A, Meyer K, et al. Impulsiveness in obsessive-compulsive disorder: results from a family study. *Acta Psychiatr Scand [Internet].* 2007 Jan [cited 2014 Mar 18];115(1):41–7. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17201865

18. Sohn SY, Kang JI, Namkoong K, Kim SJ. Multidimensional Measures of Impulsivity in Obsessive-Compulsive Disorder: Cannot Wait and Stop. *Soriano-Mas C, editor. PLoS One [Internet].* 2014 Nov 5;9(11):e111739. Available from: http://dx.plos.org/10.1371/journal.pone.0111739

19. Summerfeldt LJ, Hood K, Antony MM, Richter M a, Swinson RP. Impulsivity in obsessive-compulsive disorder: comparisons with other anxiety disorders and within tic-related subgroups. *Pers Individ Dif [Internet].* 2004 Feb [cited 2014 May 1];36(3):539–53. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0191886903001132

20. Zermatten A, Van der Linden M. Impulsivity in non-clinical persons with obsessive-compulsive symptoms. *Pers Individ Dif [Internet].* 2008 Jun;44(8):1824–30. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0191886908000469

21. Cavedini P, Riboldi G, D’Annucci A, Belotti P, Cisima M, Bellodi L. Decision-making heterogeneity in obsessive-compulsive disorder: ventromedial prefrontal cortex function predicts different treatment outcomes. *Neuropsychologia [Internet].* 2002;40(2):205–11. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11640942

22. Cavedini P, Zorzi C, Piccinni M, Cavallini MC, Bellodi L. Executive dysfunctions in obsessive-compulsive patients and unaffected relatives: searching for a new intermediate phenotype. *Biol Psychiatry [Internet].* 2010 Jun 15;67(12):1178–84. Available from: http://dx.doi.org/10.1016/j.biopsych.2010.02.012

23. da Rocha FF, Alvarenga NB, Malloy-Diniz L, Corrêa H. Decision-making impairment in obsessive-compulsive disorder as measured by the Iowa Gambling Task. *Arq Neuropsiquiatr [Internet].* 2011 Aug;69(4):642–7. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21877034

24. Kashyap H, Fontenelle LF, Miguel EC, Ferrão Y a, Torres AR, Shavitt RG, et al. “Impulsive compulsivity” in obsessive-compulsive disorder: a phenotypic marker of patients with poor clinical outcome. *J Psychiatr Res [Internet].* 2012 Sep [cited 2014 Feb 22];46(9):1146–52. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22647523

25. Kim HW, Kang JI, Namkoong K, Jhung K, Ha RY, Kim SJ. Further evidence of a dissociation between decision-making under ambiguity and decision-making under risk in obsessive-compulsive disorder. *J Affect Disord [Internet].* 2015 May 1;176:118–24. Available from: http://dx.doi.org/10.1016/j.jad.2015.01.060

26. Kodaira M, Iwadare Y, Ushijima H, Oiji A, Kato M, Sugiyama N, et al. Poor performance on the Iowa gambling task in children with obsessive-compulsive disorder. *Ann Gen Psychiatry [Internet].* 2012;11(1):25. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3508952&tool=pmcentrez&rendertype=abstract

27. Starcke K, Tuschen-Caffier B, Markowitsch HJ, Brand M. Dissociation of decisions in ambiguous and risky situations in obsessive-compulsive disorder. *Psychiatry Res [Internet].* 2010 Jan 30;175(1–2):114–20. Available from: http://www.ncbi.nlm.nih.gov/pubmed/20004479

28. Zhang L, Dong Y, Ji Y, Tao R, Chen X, Ye J, et al. Trait-related decision making impairment in obsessive-compulsive disorder: evidence from decision making under ambiguity but not decision making under risk. *Sci Rep [Internet].* 2015;5(February):17312. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=4658550&tool=pmcentrez&rendertype=abstract

29. Fontenelle LF, Oostermeijer S, Ferreira GM, Lorenzetti V, Luigjes J, Yücel M. Anticipated Reward in Obsessive-Compulsive Disorder. *J Clin Psychiatry [Internet].* 2015 Sep 23;76(9):e1134–5. Available from: http://www.psychiatrist.com/jcp/article/pages/2015/v76n09/v76n0908.aspx

30. Cavedini P, Zorzi C, Baraldi C, Patrini S, Salomoni G, Bellodi L, et al. The somatic marker affecting decisional processes in obsessive-compulsive disorder. *Cogn Neuropsychiatry [Internet].* 2012;17(2):177–90. Available from: http://www.psypress.com/cogneuropsychiatry%5Cnhttp://dx.doi.org/10.1080/13546805.2011.614152

31. Wright L, Lipszyc J, Dupuis A, Thayapararajah SW, Schachar R. Response inhibition and psychopathology: A meta-analysis of go/no-go task performance. *J Abnorm Psychol [Internet].* 2014;123(2):429–39. Available from: http://doi.apa.org/getdoi.cfm?doi=10.1037/a0036295

32. Lei H, Zhu X, Fan J, Dong J, Zhou C, Zhang X, et al. Is impaired response inhibition independent of symptom dimensions in obsessive-compulsive disorder? Evidence from ERPs. *Sci Rep [Internet].* 2015 May 20;5(October 2014):10413. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=4438428&tool=pmcentrez&rendertype=abstract

33. Bannon S, Gonsalvez CJ, Croft RJ, Boyce PM. Response inhibition deficits in obsessive–compulsive disorder. *Psychiatry Res [Internet].* 2002 Jun;110(2):165–74. Available from: http://linkinghub.elsevier.com/retrieve/pii/S016517810200104X

34. Chamberlain SR, Fineberg NA, Blackwell AD, Robbins TW, Sahakian BJ. Motor inhibition and cognitive flexibility in obsessive-compulsive disorder and trichotillomania. *Am J Psychiatry [Internet].* 2006 Jul 1;163(7):1282–4. Available from: http://psychiatryonline.org/article.aspx?doi=10.1176/appi.ajp.163.7.1282

35. Figee M, van den Munckhof P, Schuurman R, Denys D. Neuroimaging deep brain stimulation in psychiatric disorders. [Internet]. *Abelson Axer, Belin, Bewemick, Bingley, Bloch, Bohlhalter, Bora, Coenen, Coenen, de Koning, Draganski, Drevets, Figee, Greenberg, Harrison, Hasler, Hassler, Hommer, Jimenez-Ponce, Knutson, Koob, Kuhn, Le Jeune, Lehman, Luigjes, Mailer, Mallet, Malone, Ma A, editor. Deep brain stimulation: A new frontier in psychiatry.* Figee, Martijn: Department of Psychiatry and Neurosurgery, Academic Medical Center, Amsterdam, Netherlands, m.figee@amc.uva.nl: Springer Science + Business Media; 2012. p. 225–36. Available from: http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=psyc8&NEWS=N&AN=2012-27788-021

36. Basar K, Sesia T, Groenewegen H, Steinbusch HWM, Visser-Vandewalle V, Temel Y. Nucleus accumbens and impulsivity. *Prog Neurobiol [Internet].* 2010 Dec;92(4):533–57. Available from: http://dx.doi.org/10.1016/j.pneurobio.2010.08.007

37. Knutson B, Adams CM, Fong GW, Hommer D. Anticipation of Increasing Monetary Reward Selectively Recruits Nucleus Accumbens. 2001;21:1–5.

38. Foa EB, Huppert JD, Leiberg S, Langner R, Kichic R, Hajcak G, et al. The Obsessive-Complusive Inventory: Development and validation of a short version. *Psychol Assess [Internet].* 2002;14(4):485–95. Available from: http://doi.apa.org/getdoi.cfm?doi=10.1037/1040-3590.14.4.485

39. Carleton RN, Norton M a PJ, Asmundson GJG. Fearing the unknown: A short version of the Intolerance of Uncertainty Scale. *J Anxiety Disord.* 2007 Jan;21(1):105–17.

40. Obsessive Compulsive Cognitions Working Group. Psychometric validation of the obsessive belief questionnaire and interpretation of intrusions inventory—Part 2: Factor analyses and testing of a brief version. *Behav Res Ther [Internet].* 2005 Nov;43(11):1527–42. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0005796705001300

41. Tolin DF, Abramowitz JS, Brigidi BD, Foa EB. Intolerance of uncertainty in obsessive-compulsive disorder. *J Anxiety Disord [Internet].* 2003 Jan;17(2):233–42. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0887618502001822

42. Gentes EL, Ruscio AM. A meta-analysis of the relation of intolerance of uncertainty to symptoms of generalized anxiety disorder, major depressive disorder, and obsessive-compulsive disorder. *Clin Psychol Rev [Internet].* 2011;31(6):923–33. Available from: http://dx.doi.org/10.1016/j.cpr.2011.05.001

43. Cyders M a, Smith GT, Spillane NS, Fischer S, Annus AM, Peterson C. Integration of impulsivity and positive mood to predict risky behavior: development and validation of a measure of positive urgency. *Psychol Assess.* 2007 Mar;19(1):107–18.

44. Whiteside SP, Lynam DR, Miller JD, Reynolds SK. Validation of the UPPS impulsive behaviour scale: a four-factor model of impulsivity. *Eur J Pers [Internet].* 2005 Dec;19(7):559–74. Available from: http://search.proquest.com/docview/304699541?accountid=14553%5Cnhttp://openurl.library.uiuc.edu/sfxlcl3?url\_ver=Z39.88-2004&rft\_val\_fmt=info:ofi/fmt:kev:mtx:dissertation&genre=dissertations+&+theses&sid=ProQ:ProQuest+Dissertations+&+Theses+Full+Text&atitle=

45. Beck A, Steer R, Brown G. Beck Depression Inventory-II. *San Antonio.* 1996;12–5.

46. Hastie T, Tibshirani R, Friedman J. The Elements of Statistical Learning [Internet]. New York, NY: Springer New York; 2009. (Springer Series in Statistics). Available from: http://link.springer.com/10.1007/978-0-387-84858-7

47. Cuthbert BN, Kozak MJ. Constructing constructs for psychopathology: The NIMH research domain criteria. *J Abnorm Psychol [Internet].* 2013;122(3):928–37. Available from: http://doi.apa.org/getdoi.cfm?doi=10.1037/a0034028

48. Zhang T, Ramakrishnan R, Livny M. BIRCH: An Efficient Data Clustering Method for Very Large Databases. *Proc 1996 ACM SIGMOD Int Conf Manag Data [Internet].* 1996;1:103–114. Available from: http://doi.acm.org/10.1145/233269.233324

49. Fineberg N a, Potenza MN, Chamberlain SR, Berlin H a, Menzies L, Bechara A, et al. Probing compulsive and impulsive behaviors, from animal models to endophenotypes: a narrative review. *Neuropsychopharmacology [Internet].* 2010 Feb [cited 2014 Mar 20];35(3):591–604. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3055606&tool=pmcentrez&rendertype=abstract

50. Abramovitch A, McKay D. Behavioral Impulsivity in Obsessive–Compulsive Disorder. *J Behav Addict [Internet].* 2016 May 9;1–3. Available from: http://www.akademiai.com/doi/abs/10.1556/2006.5.2016.029

51. Stein DJ, Hollander E, Simeon D, Cohen L. Impulsivity scores in patients with obsessive-compulsive disorder. *Vol. 182, The Journal of nervous and mental disease.* 1994. p. 240–1.

52. Visser HA, van Oppen P, van Megen HJ, Eikelenboom M, van Balkom AJ. Obsessive-compulsive disorder; chronic versus non-chronic symptoms. *J Affect Disord [Internet].* 2014 Jan;152–154(1):169–74. Available from: http://dx.doi.org/10.1016/j.jad.2013.09.004

53. Sandyk R. Naloxone Abolishes Obsessive-Compulsive Behavior in Tourette’s Syndrome. *Int J Neurosci [Internet].* 1987 Jan 7;35(1–2):93–4. Available from: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list\_uids=3476477

54. Berlin HA, Koran LM, Jenike MA, Shapira NA, Chaplin W, Pallanti S, et al. Double-Blind, Placebo-Controlled Trial of Topiramate Augmentation in Treatment-Resistant Obsessive-Compulsive Disorder. *J Clin Psychiatry [Internet].* 2011 May 15;72(5):716–21. Available from: http://article.psychiatrist.com/?ContentType=START&ID=10006998

55. Amiaz R, Fostick L, Gershon A, Zohar J. Naltrexone augmentation in OCD: A double-blind placebo-controlled cross-over study. *Eur Neuropsychopharmacol [Internet].* 2008 Jun;18(6):455–61. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0924977X08000230

56. Insel TR, Pickar D. Naloxone administration in obsessive-compulsive disorder: report of two cases. *Am J Psychiatry [Internet].* 1983 Sep;140(9):1219–20. Available from: http://www.ncbi.nlm.nih.gov/pubmed/6614234

57. Grant JE, Kim SW. Brain circuitry of compulsivity and impulsivity. *CNS Spectr [Internet].* 2014 Feb [cited 2014 May 8];19(1):21–7. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23659364