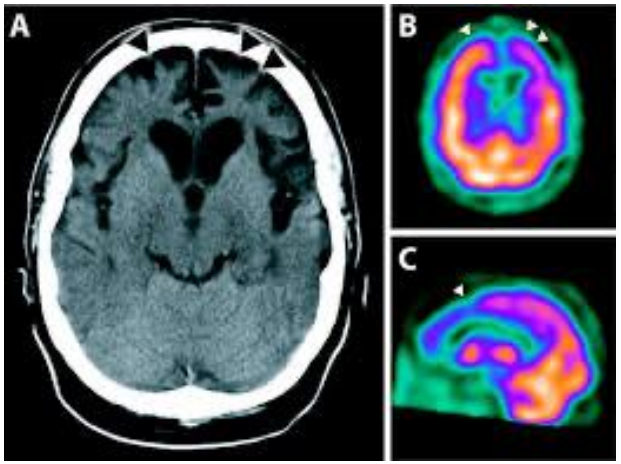
A
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Psychiatric syndromes → motor symptoms ← Neurologic syndromes

The nature of the relationship of psychomotor slowing with negative symptomatology in schizophrenia

Chris Bervoets, Lise Docx, Bernard Sabbe, Sara Vermeylen, Maarten J. Van Den Bossche, Anne Morsel & ...show all

Pages 36-46 | Received 12 Jul 2012, Accepted 20 Feb 2013, Published online: 03 Jun 2013

Motor initiation
Fine motor dysfunction

Acta Psychiatrica Scandinavica

Parsing the components of the psychomotor syndrome in schizophrenia

L. Docx, M. Morrens, C. Bervoets, W. Hulstijn, E. Fransen, M. De Hert, C. Baeken ... See all authors

Psychomotor abnormalities are highly prevalent phenomena & a heterogenous construct

Motor symptoms in psychiatry

Neurol Clin Pract. 2019 Aug;9(4):354-359. doi: 10.1212/CPJ.0000000000000644.

"Pseudo-syndromes" associated with Parkinson disease, dementia, apathy, anxiety, and depression.

Weiss HD[1], Pontone GM[1].

Author information

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Abstract

PURPOSE OF REVIEW: Physicians treating patients with Parkinson disease must evaluate not only motor symptoms but also acquire expertise in assessing the complex behavioral features that often accompany the disease, such as dementia, apathy, anxiety, and depression.

RECENT FINDINGS: There is a risk of diagnostic confusion and error because many of the behavioral and motor symptoms accentuate, overlap, or mimic each other.

SUMMARY: Awareness of potential diagnostic pitfalls and "pseudo-syndromes" should lead to more accurate clinical assessment and better care for our patients.

Obsessive Compulsive disorder and Motor Symptoms

- patients fail to flexibly update fear responses despite normal initial fear conditioning → an absence of ventromedial prefrontal cortex safety signaling inducing cognitive inflexibility, fear, and anxiety

- cortico-striato-thalamo-cortical (CSTC) circuits: involved in diverse computational activities, including reward processing, action selection, habit formation, and motor control

- may explain why compulsive behavior occurs in so many psychiatric syndromes, including OCD

Fineberg et al. 2018

Obsessive-compulsive disorder in the elderly (1)

- OCD first occurring in later life:
  - onset is rare after the age of 50 in clinical populations, but varies with the outpatient setting
  - the Epidemiological Catchment Area study suggests a 6 month prevalence of 1% and an annual incidence of approximately 0.6%
  - Elderly men: decline in incidence with age, elderly women: slight increase
- A relationship between neurological disease and OCD has long been suspected
  - 20% of OCD patients had a history of neurological disease compared to 8% of non-obsessional neurotic 'controls'
    - Sydenham's chorea, lesions in the frontal, temporal and cingulate cortices or the basal ganglia, left sided motor signs in patients with Parkinson's disease, complex partial seizures, Huntington's disease
- The presumed organic aetiology in some late onset cases of OCD does not preclude the possibility of successful treatment

- Reporting and publication bias ?

- N.R. Swerdlow, Serotonin, obsessive compulsive disorder and the basal ganglia, International Review of Psychiatry 7 (1995), 115–129
- R. Tomer, B.E. Levin andW.J.Weiner, Obsessive-compulsive symptoms and motor asymmetries in Parkinson's disease, Neuropsychiatry, Neuropsychology and Behavioural Neurology 1 (1993), 26–39

Obsessive-compulsive disorder in the elderly (2)

- Few differences were found in the symptom presentation of older individuals with OCD, as compared with younger individuals

TABLE 1. Ten most common OCD symptoms in elderly clinic patients

Age	< 60	⩾ 60	*p*
Ritualized hand-washing	46.3	70.0	0.01
Concerns about dirt and germs	48.0	60.0	NS
Use of other means to avoid contamination	42.6	56.7	NS
Excessive cleaning of house	35.0	45.0	NS
Need to tell or confess	40.2	44.0	NS
Fear of having sinned	19.9	42.9	0.02
Fear of harming others	36.2	40.0	NS
Doing and undoing rituals	30.5	36.7	NS
Fear of throwing things out	33.4	35.0	NS
Fear of something going wrong	48.3	32.1	NS

Note: Symptom frequency is given as percentage; contrasted with younger patients by means of Fisher's exact test. OCD = obsessive-compulsive disorder.

- Am J Geriatr Psychiatry 1997; 5:211-215

Obsessive-compulsive disorder in the elderly (3)

Contents lists available at ScienceDirect

European Psychiatry

journal homepage: http://www.europsy-journal.com

first multinational study investigating
clinical differences between G-OCD vs younger patients suffering from OCD

Original article

Obsessive-compulsive disorder in the elderly: A report from the International College of Obsessive-Compulsive Spectrum Disorders (ICOCS)

B. Dell'Osso [a,b], B. Benatti [a,*], C.I. Rodriguez [b], C. Arici [a], C. Palazzo [a], A.C. Altamura [a], E. Hollander [c], N. Fineberg [d], D.J. Stein [e], H. Nicolini [f,g], N. Lanzagorta [g], D. Marazziti [h], S. Pallanti [i], M. Van Ameringen [j], C. Lochner [k], O. Karamustafalioglu [l], L. Hranov [m], M. Figee [n], L. Drummond [o], J. Grant [p], D. Denys [n], D. Cath [q], J.M. Menchon [r], J. Zohar [s]

showed that the age prevalence of OCD and hoarding seem to run a U-shaped curve, with decrease and then increase of OC symptoms after the age of 60, primarily caused by an increase in checking ("not just right") symptoms.

true increase in OC symptom prevalence or a compensation mechanism for decrease in cognitive function with age ?

Obsessive-compulsive disorder in the elderly (4)

Contents lists available at ScienceDirect

European Psychiatry

journal homepage: http://www.europsy-journal.com

Original article

Obsessive-compulsive disorder in the elderly: A report from the International College of Obsessive-Compulsive Spectrum Disorders (ICOCS)

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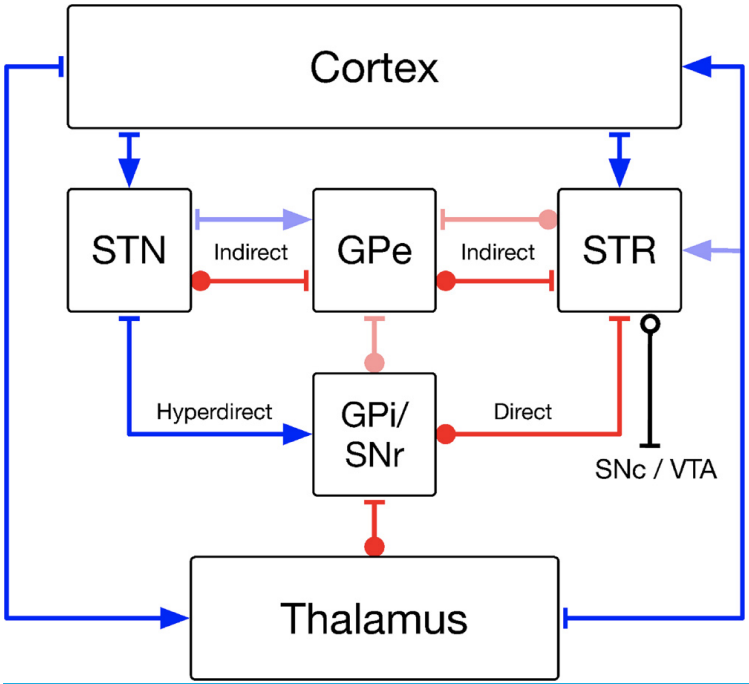
prevalence rate seems low when compared to other psychiatric conditions in geriatric patients, such as depression or anxiety disorders (e.g., generalized anxiety disorder), often comorbid …

Neurobiology (1)

- (obsessions and) compulsions can be considered as "maladaptive skills" resulting from an unappropriated training of the OFC/ACC by the BG
  - During youth (vulnerability?), neurological problem (trigger, re-inforcement?)



cortico - basal ganglia - thalamus network (CBG).

In solid colors (blue: excitatory, red: inhibitory) are showed the connections considered in the "classical" model: the BG works a three layers network - with 2 inputs (STN and STR), 2 outputs (GPi/SNr) and 1 intermediary layer (GPe) –
encompassed in a broad feedback loop to the cortex through the thalamus.

GPe: Globus Pallidus pars Externa; GPi:
Globus Pallidus pars Interna; SNr: Substantia Nigra pars Reticulata;
STN: Subthalamic nucleus; STR: Striatum.

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- Graybiel and Rauch, 2000

Neurobiology (2) Nakao et al.,Psychiatry and Clinical Neurosciences 2014

- Lesions in the cortico-striatothalamic circuit, parietal and temporal cortex, cerebellum and brainstem may induce compulsivity.
- Neuropsychological studies suggest that the persistent and inflexible thought and behavior of OCD might be affected by higher cognitive impairments related to frontal function, such as executive function, spatial cognition, and nonverbal memory.
- Abnormal activity of the frontal-subcortical circuit might cause executive dysfunction and secondary nonverbal disturbances, and result in OC symptoms.

- Sustained OC symptoms enhance abnormal activity of the neurocircuits and neuropsychological disturbance, which resulted in a vicious cycle among brain, cognition, and clinical symptoms.

- Basal ganglia modulate higher cognitive functions, such as behavior planning, attention, social behavior, and decisionmaking, as well as modulate motor function by connecting with the cerebral motor cortex. Impairment of higher cognition in basal ganglia might cause the pathophysiology of OCD

Neurobiology (3)

Table 1. Neuropsychological findings

Neuropsychological function	Authors (date)	Subjects (number)	Tests (and imaging techniques) employed	Findings
Spatial cognition	Hollander *et al.* (1990)[16]	OCD $n = 41$ HC $n = 20$	Cube Copying Test	Declined spatial cognition induced by indominant hemisphere
Memory	Christensen *et al.* (1992)[17]	OCD $n = 18$ HC $n = 18$	WMS-R WCST	Nonverbal memory dysfunction
	Radomsky *et al.* (1999)[19]	OCD $n = 10$ HC $n = 20$	WMS-R/Memory for contaminated stimuli	Memory impairment caused by emotional disturbance
	Savage *et al.* (2000)[23]	OCD $n = 33$ HC $n = 30$	ROCFT/California Verbal Learning Test	Memory impairment secondary to executive function
	Zitterl *et al.* (2001)[20]	OCD $n = 27$ HC $n = 27$	Lern- und Gedachtnistest Corsi Block-Tapping Test	Lower memory score due to lack of confidence, secondary to OCD
Attention	Martinot *et al.* (1990)[24]	OCD $n = 16$ HC $n = 17$	Stroop test (+PET)	Selective attention deficit Negative correlation between frontal metabolism and Stroop test subscores
	Nelson *et al.* (1993)[25]	OCD $n = 15$ HC $n = 15$	Posner task Spatial-linguistic conflict task	Selective attention deficit on visual stimulation
	Schmidtke *et al.* (1998)[26]	OCD $n = 29$ HC $n = 58$	Trail Making Test A	Loss of information processing speed
	Nakao *et al.* (2005)[27]	OCD $n = 24$ HC $n = 14$	Stroop test (+fMRI)	Sustained psychological scores Decreased activation in the ACC and the right caudate
	Gu *et al.* (2008)[28]	OCD $n = 21$ HC $n = 21$	Task-switching paradigm (+fMRI)	Higher error rate in the trial Decreased activation in the dorsal frontal-striatal regions, ventromedial prefrontal and right OFC
Executive function	Head *et al.* (1989)[29]	OCD $n = 19$ HC $n = 19$	WCST Block Design (WAIS-R)	Lower WCST scores
	Abbruzzese *et al.* (1995)[30]	OCD $n = 33$ HC $n = 33$	WCST	No significant impairment
	Gross-Isseroff *et al.* (1996)[31]	OCD $n = 15$ HC $n = 15$	WCST Object Alternation Test	Set-shifting disability caused by OFC dysfunction
	Lucey *et al.* (1997)[32]	OCD $n = 15$ HC $n = 15$	WCST (+SPECT)	Correlation between cerebral blood flow in the caudate and inferior frontal and number of errors in WCST
	Cavedini *et al.* (1998)[33]	OCD $n = 28$ HC $n = 29$	WCST Object Alternation Test	Set-shifting disability due to OFC dysfunction
	Pujol *et al.* (1999)[34]	OCD $n = 20$ HC $n = 20$	Word generation test (+fMRI)	Stronger activation of left frontal cortex
Working memory	Purcell *et al.* (1998)[35]	OCD $n = 23$ HC $n = 23$	Cambridge Neuropsychological Test Automated Battery	Decreased spatial working memory
	Mataix-Cols *et al.* (1999)[36]	OCD $n = 35$ HC $n = 35$	Tower of Hanoi	Decreased working memory
	van der Wee *et al.* (2003)[37]	OCD $n = 11$ HC $n = 11$	Spatial n-back task (+fMRI)	Poor performance at highest level of task difficulty Same set of brain regions as HC in areas of medial, prefrontal, and parietal cortices
	Nakao *et al.* (2009)[38]	OCD $n = 40$ HC $n = 25$	n-back task (+fMRI)	Greater activation in right DLPFC and left STG and insula

ACC, anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; fMRI, fuctional magnetic resonance imaging; HC, healthy controls; OCD, obsessive–compulsive disorder; OFC, orbitofrontal cortex; PET, positron emission tomography; ROCFT, Rey–Osterrieth Complex Figure Test; STG, superior temporal gyrus; SPECT, single-photon emission computed tomography; WAIS-R, Wechsler Adult Intelligence Scale – Revised; WCST, Wisconsin Card Sorting Test; WMS-R, Wechsler Memory Scale-Revised.

Neurobiology (4)

Table 2. Findings of structural neuroimaging

Technique/ Design	Authors (date)	Subjects (number)	Findings
MRI ROI	Rosenberg *et al.* (1997)[41]	OCD $n = 19$ HC $n = 19$	Smaller striatal volumes and significantly larger third ventricle volumes
	Szeszko *et al.* (1999)[42]	OCD $n = 26$ HC $n = 26$	Reduced bilateral OFC and amygdala volumes
	Gilbert *et al.* (2000)[43]	OCD $n = 21$ HC $n = 21$	Greater thalamic volumes Declined significantly after paroxetine monotherapy
	Kwon *et al.* (2003)[44]	OCD $n = 22$ HC $n = 22$ Sc $n = 22$	Reduced bil. hippocampal volume in both OCD and Sc Enlarged left amygdala volume in OCD
MRI VBM	Kim *et al.* (2001)[45]	OCD $n = 25$ HC $n = 25$	Increased GM density in left OFC and thalamus Reduced GM density in left cuneus and left cerebellum
	Pujol *et al.* (2004)[46]	OCD $n = 72$ HC $n = 72$	Reduced GM volume in medial frontal gyrus, medial OFC, and left insulo-opercular region Increased GM volume in ventral part of the putamen and in anterior cerebellum
	Valente *et al.* (2005)[47]	OCD $n = 19$ HC $n = 15$	Increased GM in posterior OFC and parahippocampal regions Decreased GM in left ACC in OCD
	van den Heuvel *et al.* (2009)[48]	OCD $n = 55$ HC $n = 50$	Decreased GM volume in left lateral OFC, left inferior frontal, left DLPFC and right medial prefrontal cortices Decreased bilateral prefrontal WM volume
	Togao *et al.* (2010)[49]	OCD $n = 23$ HC $n = 26$	Significant reduction of GM volume in bilateral medial prefrontal cortex, right premotor area, right OFC, right DLPFC, and bilateral temporal and occipital regions. Significant WM volume increase in right anterior limb of internal capsule, right orbitofrontal region, and significant WM volume reduction in left anterior cingulate gyrus
	Okada (submitted)	OCD $n = 37$ HC $n = 37$	Specific negative correlations between symptomatic dimension scores and regional GM volumes, mainly as decreased right cerebellum in 'aggression' and decreased right insula in 'contamination'
MRI DTI	Szeszko *et al.* (2005)[50]	OCD $n = 15$ HC $n = 15$	Significant lower FA bilaterally in ACC white matter in OCD
	Nakamae *et al.* (2008)[51]	OCD $n = 15$ HC $n = 15$	Higher FA in bilateral semioval center extending to subinsular white matter
MRI DTI + VBM	Zarei *et al.* (2011)[52]	OCD $n = 26$ HC $n = 26$	Increased GM volume in caudate bilaterally and right putamen. Higher FA values in regions including left inferior longitudinal fasciculus, bilateral superior longitudinal fasciculus, right inferior fronto-occipital fasciculus, and bilateral corticospinal tract

ACC, anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; DTI, diffusion tensor imaging; FA, fractional anisotropy; GM, grey matter; HC, healthy controls; MRI, magnetic resonance imaging; OCD, obsessive–compulsive disorder; OFC, orbitofrontal cortex; ROI, region of interest; VBM, voxel-based morphometry; WM, white matter.

Neurobiology (5)

Table 3. Functional neuroimaging (PET/SPECT) findings

Technique	Authors (year)	Subjects (number)	Design/task	Findings
PET ^{18}F-FDG	Baxter *et al.* (1987)[7]	OCD $n = 14$ Depression $n = 14$ HC $n = 14$	Resting state	Elevated metabolic rates in OFC and caudate nucleus
			Resting state	Elevated metabolic rates in right PFC and left ACC
			Before and after CBT and fluoxetine	Correlation between OCD symptom improvement and reduction of metabolism in right caudate
				Metabolic correlation between right OFC, caudate, and thalamus disappeared after treatment
			Resting state	Increased in ACC, thalamus and pallidum/putamen complex
			Before and after BT and fluoxetine	Higher normalized metabolism in left OFC was associated with greater improvement with BT treatment, but worse outcome with fluoxetine
			Before and after taking paroxetine	Lower pretreatment metabolism in bilateral OFC predicted greater improvement of clinical severity after treatment
			Resting state, before fluvoxamine treatment	Lower rCBF values in OFC and higher rCBF values in posterior cingulate cortex predicted better treatment response
			Resting state, before paroxetine treatment	Clinical improvement was correlated to higher pretreatment glucose metabolism in right caudate nucleus in OCD, while improvement of MDD symptoms were correlated with pretreatment metabolism in amygdala, thalamus, and medial prefrontal cortex
			Before and after treatment	Increased [^{11}C]Rac binding potential in basal ganglia corresponding to clinical improvement
			Symptom provocation task	Increasing anxiety correlated with hyper-perfusion in caudate nucleus and OFC
			Resting state	Medial prefrontal hyperperfusion
			Resting state	Decreased perfusion in right OFC and left ACC
			Resting state	Decreased availability of dopamine transporter in striatum and decreased availability of serotonin transporter in thalamus and midbrain
			Before and after treatment	Decreased rCBF in right head of caudate nucleus after treatment
			Before and after treatment	Decreased N-acetyl aspartate concentration in ACC corresponding to augmentation of SRI with atypical neuroleptics

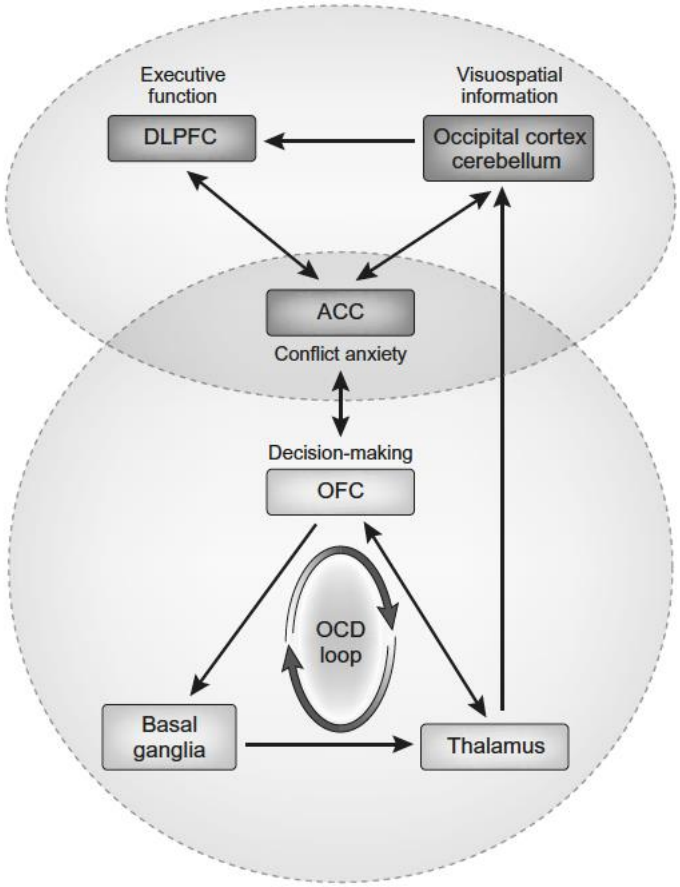
CBT, cognitive behavioral therapy; HC, healthy controls; MDD, major depressive disorder; OCD, obsessive–compulsive disorder; OFC, orbitofrontal cortex; PET, positron emission tomography; SPECT, single-photon emission computed tomography.

Table 4. Functional neuroimaging (fMRI) findings

Technique	Authors (date)	Subjects (number)	Design/task	Findings
fMRI	Breiter *et al.* (1996)[76]	OCD $n = 10$ HC $n = 5$	Symptom provocation task	Increased activation in OFC, ACC and caudate
fMRI	Adler *et al.* (2000)[77]	OCD $n = 7$	Symptom provocation task	Increased activation in OFC, DLPFC, temporal, and ACC
fMRI	Shapira *et al.* (2003)[78]	OCD $n = 8$ HC $n = 8$	Symptom provocation task	Increased activation in insula, parahippocampus, and inferior frontal cortex
fMRI	Cannistraro *et al.* (2004)[79]	OCD $n = 10$ HC $n = 10$	Provocation task for recognition of human faces	Weak response in bilateral amygdala
fMRI	Nakao *et al.* (2005)[80]	OCD $n = 10$	Symptom provocation task and neurocognitive task before and after treatment	Decreased activation in bilateral OFC, DLPFC and ACC during symptom provocation and increased activation in parietal and cerebellum during Stroop task after symptom improvement
fMRI	Nabeyama *et al.* (2008)[81]	OCD $n = 11$	Neurocognitive task before and after BT	Increased activation in cerebellum and parietal and decreased activation in the OFC, middle frontal gyrus, and temporal regions after symptom improvement
fMRI	Harrison *et al.* (2009)[82]	OCD $n = 21$ HC $n = 21$	Resting state	Increased functional connectivity along a ventral corticostriatal axis, implicating the OFC and surrounding areas
fMRI	Sanematsu *et al* (2010)[83]	OCD $n = 17$	Symptom provocation task before taking fluvoxamine	Correlation between pre-treatment activation in right cerebellum and left STG and Y-BOCS %improvement
fMRI	Murayama *et al.* (2013)[84]	OCD/check $n = 10$ OCD/wash $n = 12$ HC/check $n = 10$ HC/wash $n = 9$	Symptom provocation task	Hypoactivation in left caudate and left ACC in Checkers. Hyperactivation in several bilateral cortico-cerebellar regions in Checkers

ACC, anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; fMRI, functional magnetic resonance imaging; HC, healthy controls; OCD, obsessive–compulsive disorder; OFC, orbitofrontal cortex; STG, superior temporal gyrus; Y-BOCS, Yale–Brown Obsessive Compulsive Scale.

Neurobiology (6)

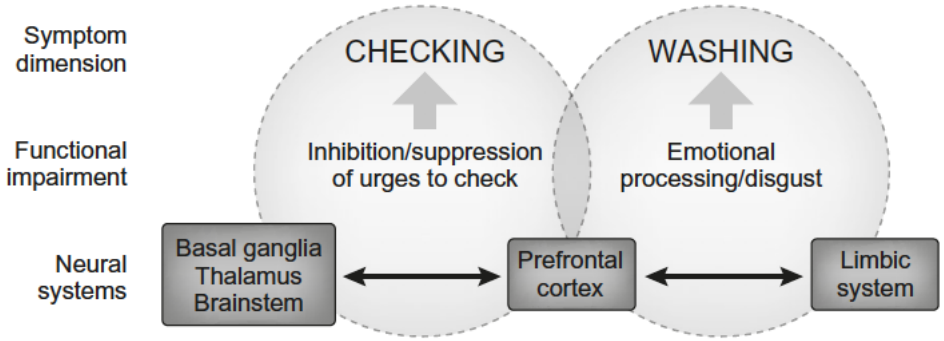


Functional neuroanatomy in obsessive–compulsive disorder (OCD) brain:

A network including the dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC) and posterior regions may be related to cognitive processes in OCD, while orbitofronto-striatal regions (OCD-loop) may be involved with OCD symptomatology.

Clinical improvement of OCD might accompany functional improvement of brain and cognitive improvement, such as visuospatial information, decision-making, working memory, and executive function.

Neurobiology (7)



Multidimensional model of obsessive–compulsive disorder (OCD).
Different obsessive–compulsive symptom dimensions may be mediated by relatively distinct components of neural circuits. Aberration of the basal ganglia system may be attributed to difficulty in inhibition of unwanted impulses, such as urges to check, while neural components of the limbic system are likely to process emotional dimensions, such as disgust for uncleanness. OCD thus seems best conceptualized as a spectrum of multiple, overlapping syndromes rather than a unitary disease entity.

Obsessive Compulsive Personality Disorder and Parkinson's Disease (1)

- personality traits and Parkinson's disease
  - a personality profile characterized by industriousness, inflexibility, punctuality, cautiousness and lack of novelty seeking
  - Cloninger's model:
    - temperament traits (novelty seeking, harm avoidance and reward dependence) →brain systems modulated by dopamine, serotonin, and noradrenaline.

- ''premorbid'' parkinsonian personality?

Obsessive Compulsive Personality Disorder and Parkinson's Disease (2)

Table 1 Major non-motor symptoms in Parkinson disease

Autonomic dysfunction	Orthostatic hypotension Urogenital dysfunction Constipation Heat or cold intolerance Hypo- or hyperhidrosis
Sleep disorders	Sleep fragmentation Insomnia Excessive daytime somnolence (EDS) Sleep attacks REM sleep behaviour disorder (RBD) Periodic limb movements in sleep (PLMS) Restless legs syndrome (RLS)
Neuropsychiatric disorders	Fatigue Apathy Anhedonia Depression and anxiety Impulsive-compulsive disorders Mild cognitive impairment (frontal executive dysfunction) Dementia Psychosis
Sensory disorders	Hyposmia Colourvision deficits Vestibular deficits Abnormal sensations Pain

Compulsive/impulsive disorders can be described as behavioural addictions, lying along an impulsive-compulsive spectrum with mild dopamine deficiency-related behavioural disorders at one end and obsessive-compulsive disorders at the other

Obsessive Compulsive Personality Disorder and Parkinson's Disease (3)

Table 1. Frequency of PeDs among PD patients and controls.

PeDs	PD (n = 100)	Controls (n = 100)	*p-value*
Avoidant	6	1	**0.05**
Dependent	3	/	0.08
Obsessive Compulsive	32	7	**<0.0001**
Passive aggressive	1	1	1.0
Depressive	11	4	**0.003**
Paranoid	4	5	0.7
Schizotypal	1	1	1.0
Schizoid	1	/	0.3
Histrionic	3	/	0.08
Narcissistic	2	/	0.1
Borderline	3	/	0.08
Antisocial	/	1	0.1
More than one	13*	4**	0.02

PeDs = Personality Disorders;
* = 8 were affected by obsessive-compulsive PeD and 3 by Depressive PeD;
**3 were affected by obsessive-compulsive PeD.

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Obsessive Compulsive Personality Disorder and Parkinson's Disease (4)

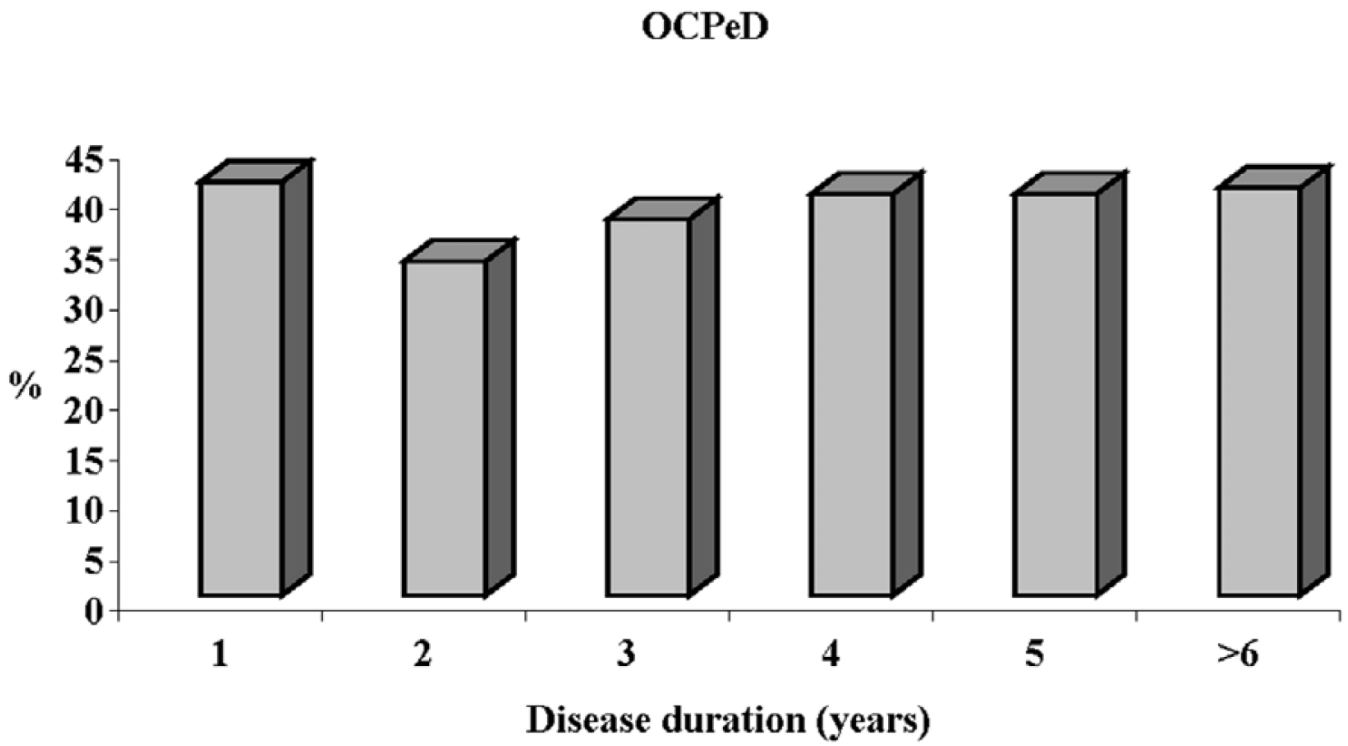

Figure 1. Frequency OCPeD by disease duration. OCPeD = Obsessive-compulsive Personality Disorder.
doi:10.1371/journal.pone.0054822.g001

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Obsessive Compulsive Disorder and Parkinson's Disease (5)

- PD is characterized by dysfunction in the frontobasal ganglia circuitry and a similar circuitry has also been implicated in the pathophysiology of OCD.
- The higher incidence of obsessive-compulsive symptoms in PD might be caused by the involvement of a shared circuitry ?

- Many non-motor symptoms occur early in PD and some of them, such as olfactory deficit, REM behaviour disorder, depression, constipation, may even predate also of many decades the diagnosis of PD which is based on motor signs

Comparison between mild Parkinson's disease (PD), severe PD, and control groups in MOCI and LOI questionnaires

	Mild PD	*Severe PD*	*Controls*	*F Value*	*Post hoc*
Group 1:	(n=25)	(n=47)	(n=72)	(2,141)	
MOCI total	4.12 (3.15)	7.62 (4.19)	4.83 (3.57)	10.412[1-165]	1-151 1-152
MOCI checking	1.08 (1.29)	2.23 (2.01)	1.26 (1.74)	5.326[1-160]	1-152
MOCI cleaning	1.28 (1.43)	1.87 (1.41)	1.21 (1.37)	3.417[1-150]	1-151 1-152
MOCI doubting	1.48 (1.36)	3.09 (1.60)	2.18 (1.48)	10.331[1-165]	1-151 1-152
MOCI slowness	2.28 (0.61)	2.32 (1.07)	2.14 (0.74)	0.724	
Group 2:	(n=23)	(n=31)	(n=54)	(2,105)	
LOI	48.04 (29.04)	62.03 (28.18)	48.41 (22.75)	3.14[1-150]	1-151 1-152
LOI TS items	1.30 (2.18)	2.03 (3.05)	1.44 (1.79)	0.86	
LOI OCD items	16.43 (11.55)	21.06 (9.27)	18.31 (8.37)	1.70	

- Values are means (SD).
- ↵1-150 p<0.05;
- ↵1-160 p<0.01;
- ↵1-165 p<0.001 from controls.
- ↵1-151 Significant differences between mild PD and severe PD.
- ↵1-152 Significant differences between severe PD and normal controls.
- TS=Tourette's syndrome; OCD=obsessive-compulsive disorder.

- Bower JH, Grossardt BR, Maraganore DM, Ahlskog JE, Colligan RC et al. (2010) Anxious personality predicts an increased risk of Parkinson's disease. Mov Disord 25: 2105–13.
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Obsessive Compulsive Disorder and Parkinson's Disease (6)

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- Many non-motor symptoms occur early in PD and some of them, such as olfactory deficit, REM behaviour disorder, depression, constipation, may even predate also of many decades the diagnosis of PD which is based on motor signs

Comparison between mild Parkinson's disease (PD), severe PD, and control groups in MOCI and LOI questionnaires

	Mild PD	*Severe PD*	*Controls*	*F Value*	*Post hoc*
Group 1:	(n=25)	(n=47)	(n=72)	(2,141)	
MOCI total	4.12 (3.15)	7.62 (4.19)	4.83 (3.57)	10.412[1-165]	1-151 1-152
MOCI checking	1.08 (1.29)	2.23 (2.01)	1.26 (1.74)	5.326[1-160]	1-152
MOCI cleaning	1.28 (1.43)	1.87 (1.41)	1.21 (1.37)	3.417[1-150]	1-151 1-152
MOCI doubting	1.48 (1.36)	3.09 (1.60)	2.18 (1.48)	10.331[1-165]	1-151 1-152

The obsessional scores for patients with severe Parkinson's disease were higher than those for patients with mild Parkinson's disease and controls

- ↵1-165 p<0.001 from controls.
- ↵1-151 Significant differences between mild PD and severe PD.
- ↵1-152 Significant differences between severe PD and normal controls.
- TS=Tourette's syndrome; OCD=obsessive-compulsive disorder.

- Bower JH, Grossardt BR, Maraganore DM, Ahlskog JE, Colligan RC et al. (2010) Anxious personality predicts an increased risk of Parkinson's disease. Mov Disord 25: 2105–13.
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- Alegret M, Junqué C, Valldeoriola F, et al Obsessive-compulsive symptoms in Parkinson's disease Journal of Neurology, Neurosurgery & Psychiatry 2001;70:394-396.

Obsessive Compulsive disorder and frontotemporal dementia (1)

Table 1. Symptom overlap between FTD and psychiatric disorders.

Syndrome	Symptoms commonly shared with FTD	Symptoms rarely shared with FTD
Major depressive disorder	Anhedonia, psychomotor agitation or retardation, decreased motivation and energy, decreased concentration and focus	Depressed mood, weight loss, feelings of worthlessness, poor self-esteem, suicidal ideation
Mania	Logorrhoea, flight of ideas, distractibility, increase in goal-directed activities, impulsive behaviour	Elevated mood, decreased need for sleep
Obsessive–compulsive disorder	Compulsions (repetitive behaviours the patient feels compelled to perform)	Obsessions (unwanted, recurrent intrusive thoughts that cause anxiety)
Schizophrenia	Disorganized speech and behaviour, affective flattening, alogia, avolition	Complex delusions, auditory hallucinations (more common in C9ORF72 mutation carriers)

Huey et al., 2008

Obsessive Compulsive disorder and frontotemporal dementia (2)

- The typical psychiatric misdiagnoses of FTD are MDD, bipolar affective disorder (BAD), obsessive – compulsive disorder (OCD) and schizophrenia
  - shared neuroanatomic substrates
    - Orbitofrontal and anterior cingulate cortices, basal ganglia, and thalamus are involved in the pathogenesis of OCD and FTD
      - stereotyped behaviours = compulsions of OCD, absent obsessions →differential contribution of specific frontal cortical and subcortical structures to the OCD phenotype?
    - MDD: two separate factors, ‘ cognitive/affective ’ (e.g. sadness, poor self-esteem, suicidal state) and ‘ somatic ’ (e.g. decreased energy, appetite changes, poor concentration (= FTD)

Huey et al., 2008, Beck et al., 1996; Steer et al., 1999, Koenigs et al., 2008

Obsessive Compulsive disorder and frontotemporal dementia (3)… Red Flags for FTD…

- cognitive dysfunction, especially aphasia or executive dysfunction
- a lack of distress
- progressive and refractory to treatment
- an unusual psychiatric presentation, such as sustained manic-like states without grandiosity or euphoria, compulsions without obsessions, schizophrenia lacking hallucinations or complex delusions

What is the role of psychiatry in FTD (4)?

- FTD = disorders of emotion, behaviour and cognition
- Psychiatric medicines are commonly used to treat behavioural features of FTD
- Strengths of psychiatry:
  - the integration of pharmacological and behavioural treatments
  - involvement of the family in the treatment process
- Psychiatry clearly benefits from examining the neuroanatomy and genetics of psychiatric symptoms (in FTD and in PD)

The case of Hoarding (1)

- a behavioural phenomenon characterized by the excessive collection and failure of discard of poorly useable objects

- The mental disorders reported in association with the condition cover almost the whole range of psychiatric diseases.
  - hoarding is either a very unspecific symptom or a collective name for various similar, but distinct symptoms
  - hoarding behaviors can also be viewed as an expression of the need to make the environment “feel right”
  - Or mnestic problem ?

- The controversy about the nosological status of hoarding is still unresolved (Diogenes syndrome)

- Clark AN, Mankikar GD, Gray I. Diogenes syndrome. A clinical study of gross neglect in old age. Lancet 1975;1:366–368.

The case of Hoarding (2)

- Relationships between OCS (total, and each of the six contributing domains (i.e., hoarding, ordering, obsessing, neutralizing, washing, and checking)) and both hoarding behaviors and hoarding cognitions were generally strong. This suggests some overlap between the two concepts

  - Reid et al 2011

Table 4
Partial correlations between hoarding behaviors, hoarding cognition, and obsessive–compulsive symptoms controlling for depressive symptoms.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. SI-R total	1.00											
2. SI-R-DD	.87**	1.00										
3. SI-R-C	.81**	.50**	1.00									
4. SI-R-AP	.83**	.68**	.48**	1.00								
5. SCI-R	.48**	.51**	.28**	.42**	1.00							
6. OCI-R total	.45**	.40**	.30**	.44**	.60**	1.00						
7. OCI-hoarding	.68**	.67**	.50**	.53**	.60**	.69**	1.00					
8. OCI-ordering	.28**	.29**	.14	.30**	.50**	.81**	.45**	1.00				
9. OCI-obsessing	.29**	.22**	.24**	.30**	.39**	.74**	.44**	.49**	1.00			
10. OCI-neutralize	.39**	.34**	.27**	.38**	.56**	.83**	.53**	.62**	.62**	1.00		
11. OCI-washing	.14	.10	.10	.17*	.34**	.71**	.28**	.52**	.52**	.61**	1.00	
12. OCI-checking	.23**	.18*	.13	.31**	.35**	.75**	.41**	.52**	.38**	.48**	.39**	1.00

Note: SI-R = Savings Inventory-Revised; DD = Difficulty Discarding; C = Clutter; AP = Acquisition Problems; SCI-R = Savings Cognitions Inventory-Revised; OCI-R = Obsessive–compulsive Inventory-Revised.

* $p < .05$.
** $p < .01$.