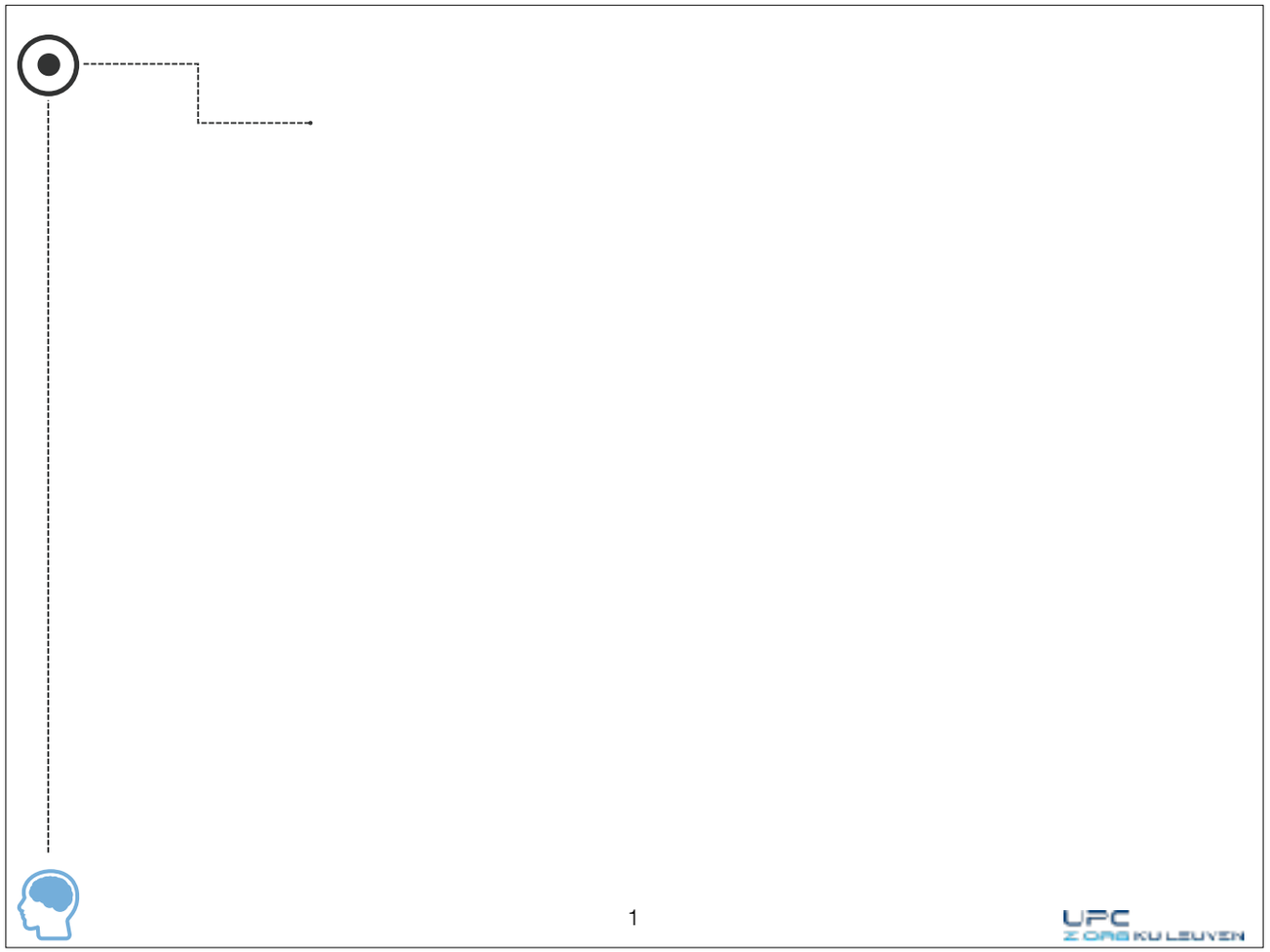


OCD epidemiologie

- 1-jaar prevalentie: 1.6 % (SE 0.2%)*° & levenslange prevalentie: 1.7 % (SE 0.1%)*
- maar gecontesteerd door anderen;
- 1- jaar prevalentie: 0.095 %** (uit verzekeringsdatabase, behandelde casus..)
- verschil wellicht te verklaren door het hoge aantal onbehandelde patiënten in de maatschappij!

*NIMH-ECA study, 1982-1984, °Weissman et al. 1994, **Koran, Leventhal, Fireman and Jocobson (unpublished data)1998

zelfs tot lifetime 2.3 % (Ruscio 2010)

*These estimates may be high, since both studies utilized trained lay interviewers to administer a structured diagnostic interview. Studies involving psychiatrists have reported lower prevalence rates.
**clinically recognized OCD in a large prepaid health plan, the Kaiser Northern California Health Plan, which has more than 1.8 million members. Chart reviews on all cases with an OCD diagnosis in the Plan's computerized data base produced a one year treated prevalence rate of 0.095% in adults aged 18 years or older. This is less than the 10% of Karno et al. and Weissman et al.'s reported rates, and only 15% of the more conservative rate reported by Stein et al..

OCD kliniek: screening

- heb je onaangename gedachten die aanwezig blijven?
- vrees je iemand impulsief pijn te doen?
- moet je dingen tellen, handen wassen, zaken controleren, altijd opnieuw?
- pieker je of je religieuze rituelen correct uitvoert of immoreel bent?
- heb je zorgen over seksuele zaken?
- ben je bezig met symmetrie en/of exacte orde?
- heb je er moeite mee om dingen weg te gooien?
- tot slot, storen deze zaken je in het werk, school, familie, vrienden…?

Although an individual's symptoms often change over time, symptoms present at a given time exhibit certain understandable patterns. In a study conducted by Baer in 1994, current symptoms drawn from 107 patients' Y-Bocs symptoms checklist clustered into three groups:

1 symmetry and exactness obsessions strongly correlated with ordering compulsions and mildly with repeating and hoarding rituals -- yet hoarding obsessions, which were weakly associated with symmetry obsession, were strongly correlated with hoarding compulsions and mildly with ordering rituals;

2 Contamination obsessions were strongly correlated with cleaning compulsions, as would be expected, but surprisingly, given the clinical distinction often made between cleaners and checkers, these obsessions were also mildly correlated with checking rituals;

3 Sexual and religious obsessions were mildly correlated, and clustered with aggressive obsessions.

A study using similar methods by Leckman et al. in 1997, but examining lifetime experience of symptoms, generally confirmed these relationships, but found a fourth cluster: in this analysis aggressive, religious and sexual obsessions clustered with checking compulsions, confirming the longstanding clinical impression that patients check to be sure they have not harmed others or exposed them to risk. In 1992, Rasmussen and Eisen suggested that three core features may be more fundamental that these symptom groups: abnormal risk assessment, pathological doubt and incompleteness. To date, no phenomenological sub-grouping of OCD symptoms has been found to confer clinically important prognostic information although the individual symptoms of harding and obsessional slowness seem particularly difficult to treat.

Frequency of Obsessional Themes

Theme	Percent of Patients: Study A*	Study B**
Contamination	50	35
Pathologic Doubt***	42	–
Symmetry	33	7
Symmetry	32	10
Aggressive	31	24
Sexual	24	6
Multiple Obsessions	12	–
Religious	–	6
Hoarding	–	5
Unacceptable Urges	–	4

Notes:
* Data were derived from a study conducted by Rasmussen and Eisen in 1992. N=60 Patients meeting DSM-III or DSM-III-R criteria.
** Data were derived from a study conducted by Foa and Kozak in 1995. N=425 patients meeting DSM-IV criteria.
*** Fear or doubt regarding responsibility for a terrible event.

Frequency of Compulsive Behaviors

Compulsions	Percent of Patients: Study A*	Study B**
Checking	61	28
Cleaning-washing	50	27
Counting	36	2
Need to Ask/Confess	34	–
Symmetry/Exactness	28	–
Multiple compulsions	58	–
Ordering	–	6
Hoarding	18	4
Repeating	–	11
Mental Rituals	–	11

Notes:
* Data were derived from a study conducted by Rasmussen and Eisen in 1992. N=560 Patients meeting DSM-III or DSM-III-R criteria.
** Data were derived from a study conducted by Foa and Kozak in 1995. N=425 patients meeting DSM-IV criteria.

The American Psychiatric Association's Diagnostic and Statistical Manual (Fourth Edition) describes obsessions as recurrent, persistent ideas, thoughts, images or impulses that are experienced at some time during the illness as ego-dystonic, i.e., intrusive, senseless, excessive, repugnant, or absurd. The obsessions are not simply worries about real-life problems. Common morbid themes are contamination, aggression, harm avoidance, distasteful or excessive sexual ideas, religious concerns, fears of offending others, a need to know, orderliness and perfection. The person recognizes these ideas as products of his or her own mind and tries to suppress or ignore them, without much success.Compulsions are repetitive, seemingly purposeful behaviors or mental acts performed according to rigid rules. The acts are designed to prevent a future feared event, but are not realistically connected to the event, or are excessive. They carry a sense of subjective compulsion and bring no pleasure. Common compulsions are washing, checking, a need to ask or to confess, arranging, repeating, hoarding, and mental compulsions such as counting or praying.

Symptoms of Other Psychiatric Disorders to be Differentiated From the Obsessions, Compulsions, and Rituals of Obsessive-Compulsive Disorder (OCD)

Disorder	Symptom	How the Symptom Differs from Symptoms of OCD
Obsessive-compulsive personality disorder (OCPD)	Hoarding, scrupulosity, perfectionism, preoccupation with rules and order	In OCD, obsessions and compulsions usually focus on specific feared events; in OCPD, thoughts and behaviors are globally colored by traits, such as perfectionism and preoccupation with rules.
Necrophilias	Intrusive sexual thoughts and urges	OCD obsessions are resisted, are morally dissonant to the individual, and lead to avoidance.
Postpartum depression	Urges to harm an infant	OCD thoughts and urges do not emerge from depressed mood or psychosis and are resisted.
Posttraumatic stress disorder	Intrusive thoughts and images	The thoughts replay actual events rather than anticipate future events as in OCD.
Schizophrenia	Schizophrenic delusions	The content is usually bizarre or related to persecution, grandiosity, passivity experiences, or ideas of reference.
Tourette's disorder	Complex vocal or motor tics	Tics, unlike compulsions, are not preceded by thoughts nor aimed at relieving anxiety or preventing or undoing an event.

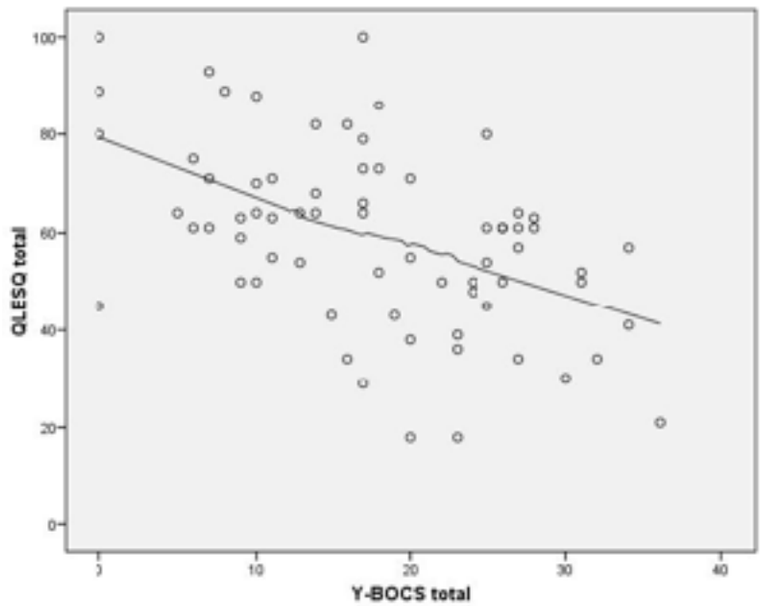
Disorder	Symptom	How the Symptom Differs from Symptoms of OCD
Bipolar disorder	Manic delusions	The content of the delusions is usually related to grandiosity.
Body dysmorphic disorder	Recurrent and intrusive preoccupation with a perceived bodily defect	The preoccupation is limited to the body.
Depressive disorders	Depressive ruminations	Unlike OCD obsessions, depressive ruminations are experienced as consistent with one's self-image and usually concern self-criticism, failure, guilt, regret, or pessimism about the future. Unlike obsessions, depressive ruminations do not lead to compulsive rituals.
Eating disorders	Intrusive thoughts and unwanted behaviors regarding weight and eating	The thoughts and behaviors are limited to weight and eating.
Generalized anxiety disorder	Worry	Unlike with OCD, worry does not lead to compulsive rituals.
Hypochondriasis	Fear or belief regarding serious disease	In OCD, such fears arise from an external stimuli (e.g., that causes contamination) rather than misinterpretation of an ordinary bodily sign or symptom.

Y-BOCS

0-7	subclinical
8-15	mild
16-23	moderate
24-31	severe
32-40	extreme

- 10-item, door arts
- niet voor diagnose maar voor de ernst van de symptomen!
- vijf dimensies voor obsessies en compulsies:
  - „verloren” tijd
  - interferentie met werk/relaties
  - graad van ongemak en stress
  - weerstand tegen symptomen
  - succes in het uitoefenen van weerstand

Y-BOCS & QOL = lineair



Y-BOCS severity and QLESQ showing a linear relationship across the severity spectrum

Depress Anxiety. 2009; 26(1): 39–45.
doi: 10.1002/da.20506

patients experience a 25% decrease in a Y-BOCS score as mild to moderate improvement, and decrease of 35-50% as moderate to marked. In controlled treatment trials, a decrease of greater than or equal to 35% is widely accepted as indicating a clinically meaningful response and translates into a global improvement rating of much or very much improved; many studies, however, have accepted a lower criterion of greater than or equal to a 25% decrease.
Lastly, it is worth noting that in Goodman et al.'s report in 1989, the Y-BOCS' reliability, validity and sensitivity to change are well established. However, extensive reviews of alternative rating scales are available.
-we also examined the relationship between Y-BOCS severity and QOL to determine if the relationship was linear by using LOESS curves (see Figure 1). In contrast to Eisen et al., we found a linear relationship best fit the data. When controlling for depression using the BDI (Table 4), many of the relationships between OCD severity and QOL or functional impairment became non-significant, except impairment in work, social life, and family life as measured with the SDS3.

OCD: stagering

TABLE	Obsessive-compulsive disorder stages of response
Recovery	Not at all ill; Y-BOCS < 8
Remission	Y-BOCS < 16
Full response	≥ 35% Y-BOCS reduction and CGI 1 or 2
Partial response	Between 25% and 35% Y-BCCS reduction
Nonresponse	< 25% Y-BOCS reduction, CGI 4
Relapse	Symptoms return (CGI 6, or 25% Y-BOCS increase from remission score) after 3+ months of treatment at adequate dosage
Refractory	No change or worsening of symptoms with all available therapies

Y-BOCS, Yale-Brown Obsessive Compulsive Scale; CGI, Clinical Global Impressions.
Adapted from Pallanti S et al. *Int J Neuropsychopharmacol*. 2002.[35]

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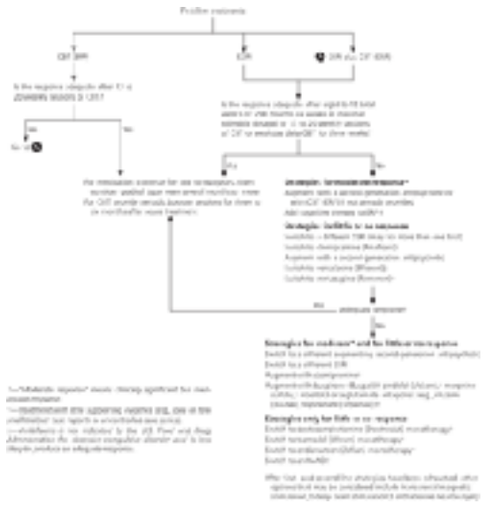
OCD:behandeldoelen

- frequentie symptoom = < 1 uur per dag dwangen en handelen (Y-BOCS < 8)
- ernst symptoom = lineariteit Y-BOCS met QoL!
- ondanks alle inspanningen:
  - 1/4 geen enkele verbetering op eerste SSRI*
  - van deze non-responders zal 1/3 responderen na switch naar een tweede verschillende SSRI*

**J Clin Psychiatry,* 1997

therapie initiëren?



geen echte keuze meer, start met CBT

•

CBT yielded very large effect sizes compared to wait list and placebo.
CBT was significantly better than antidepressants.
The addition of antidepressants did not potentiate the effect of CBT.
There was no significant difference between ERP and cognitive therapy.
There was no significant difference between individual and group treatment.

EERSTE LIJNS BEHANDELING

- CBT(+ ERP): (1X week, 25 weken) of R/ (dosis, tijd)?

Effect sizes (Hedges' *g*) on Y-BOCS for all OCD RCTs and divided on comparison conditions for post-treatment assessments.

Comparison	*k*	*g*-Value	95% CI	*z*-Value	*Q*-value	I^2
All studies	62	0.57	0.39–0.75	6.20[c]	306.4[c]	80
CBT vs. WLC	15	1.31	1.08–1.55	10.85[c]	22.3	37
CBT vs. placebo: all	8	1.33	0.91–1.76	6.16[c]	24.7[b]	72
CBT vs. placebo: psychological	6	1.29	0.76–1.81	4.81[c]	21.3[b]	77
CBT vs. all active Tx	37	0.09	−0.05–0.22	1.19	70.5[b]	49
Individual vs. Group Tx	5	0.17	−0.05–0.40	1.45	2.6	0
ERP vs. CT	7	0.07	−0.15–0.30	0.64	5.5	0
ERP/CBT vs. Medication	4	0.55	0.05–1.04	2.17[a]	9.7[a]	69
ERP/ERP + Pla. vs. ERP + Med	6	−0.25	−0.46–0.03	1.71	5.1	0

Note: *k* = number of comparisons. A positive *g*-value means that the first treatment in the comparison is better and a negative *g*-value means that the second treatment is better.

a $p < 0.05$.

b $p < 0.001$.

c $p < 0.0001$.

Cognitive behavioral treatments of obsessive–compulsive disorder. A systematic review and meta-analysis of studies published 1993–2014 Öst et al, 2015

shows the results of the meta-analysis at post-treatment for all comparisons and divided on the different types of comparison conditions at post-treatment. The overall Hedges's g was moderate (0.53) but significantly different from zero. Both indices of heterogeneity were also significant. The effect sizes for CBT compared with waiting-list (1.31), all placebo conditions (1.33), and psychological placebo only (1.29) were very large and also significantly heterogeneous, except for WLC. The ES for comparisons between individual and group treatment, and ERP with CT were less than small and non-significant. ERP/CBT was significantly better than ADM (0.55) with significant heterogeneity, whereas ERP + Placebo was nonsignificantly worse (-0.25) than ERP + medications.

EERSTE LIJNS BEHANDELING

- CBT+ ERP: (1X week, 25 weken) of R/ (dosis, tijd) ?

Attrition in the OCD-studies.

Condition	k	Dropout	95% CI	z-Value	Q-value	I^2
ERP[a]	28	19.1%	16.1–22.7%	13.22[b]	27.7	2.6
CT	8	11.4%	7.4–17.0%	6.55[b]	6.9	0
CBT	18	15.5%	12.5–18.2%	12.92[b]	22.8	21.0
Antidepressants	4	30.3%	23.5–38.3%	4.62[b]	9.0[a]	66.8
ERP/CBT + ADM	7	32.0%	24.2–40.9%	3.83[b]	14.5[a]	57.1
Placebo	6	18.8%	9.3–33.8%	4.58[b]	3.8	0
WLC	8	13.7%	7.9–22.6%	5.88[b]	4.9	0

a $p < .05$.

b $p < .0001$.

Cognitive behavioral treatments of obsessive–compulsive disorder.
A systematic review and meta-analysis of studies published 1993–2014
Öst et al, 2015

shows the dropout rate for the different treatments used in this meta-analysis. A subgroup analysis yielded a significant Qbetween (df 6) = 35.58, $p < 0.0001$. The dropout rates of antidepressants (30.3%) and ERP + antidepressants (32.0%) were significantly higher than for cognitive therapy (11.4%), CBT (15.5%), ERP (19.1%), placebo (18.8%) and waitlist control (13.7%). There were no significant differences between the other cognitive-behavioral treatments or the control conditions.

EERSTE LIJNS BEHANDELING

- CBT+ ERP: (1X week, 25 weken) of R/ (dosis, tijd)?

Effect sizes (Hedges' g) on Y-BOCS for all OCD RCTs and divided on comparison conditions for follow-up assessments.

Comparison	k	g-Value	95% CI	z-Value	Q-value	I^2
All studies	27	0.06	− 0.13–0.24	0.60	55.4[b]	53
CBT vs. active Tx	25	0.04	− 0.16–0.24	0.41	54.3[b]	56
Individual vs. Group Tx	8	0.21	− 0.03–0.45	1.75	1.7	0
ERP vs. CT	4	0.07	− 0.27–0.41	0.39	3.7	20
ERP/CBT vs. Medication	2	0.38	− 0.81–1.57	0.62	6.1[a]	84
ERP/ERP + Pla. vs. ERP + Med	3	− 0.06	− 0.68–0.55	0.18	3.3	39

Note: *k* = number of comparisons. A positive *g*-value means that the first treatment in the comparison is better and a negative *g*-value means that the second treatment is better.

a $p < 0.05$.

b $p < 0.001$.

c $p < 0.0001$.

Cognitive behavioral treatments of obsessive–compulsive disorder. A systematic review and meta-analysis of studies published 1993–2014 Öst et al, 2015

shows the effect sizes at follow-up assessment, on average 15.1 months after the end of treatment (for the 30 studies reporting follow-up data). The overall ES (0.06) was less than small, which is understandable since WLC conditions (and often placebo conditions) do not continue to follow-up. The individual versus group ES was small (0.21) and of the same magnitude as at post-treatment, which was also the case for the ERP versus CT effect size (0.07). However, the ERP/CBT vs. medication ES (0.38) was reduced from post-treatment and no longer significant, and so was the case for the ERP + Placebo vs. ERP + medication ES (– 0.06).

**********Regarding the overall ES the trim-and-fill method suggested that 9 studies should be trimmed which would reduce the mean ES from 0.57 to 0.31. Concerning the Placebo-, Individual vs. group-, and ERP/CBT vs. medication two studies each should be trimmed, leading to marked reductions of the ES. However, for the WLC-, ERP vs. CT-, and ERP + Placebo vs. ERP + medication comparisons no studies should be trimmed.

„trade off”

- type en ernst van symptomen
- co-morbiditeit
- beschikbaarheid van CBT en positieve attitude tov OCD!
- hoog drop-out risico

(S)SRI: vuistregels

- optitreren to max getolereerde dosis (QTc!)
- vroegste effect na 4 weken
- 12 weken, waarvan min 6 op max getolereerde dosis
- beheer van neveneffecten

TABLE 3. Dosing of Serotonin Reuptake Inhibitors (SRIs) in Obsessive-Compulsive Disorder

SRI	Starting Dose and Incremental Dose (mg/day)[a]	Usual Target Dose (mg/day)	Usual Maximum Dose (mg/day)	Occasionally Prescribed Maximum Dose (mg/day)[b]
Citalopram	20	40–60	80	120
Clomipramine	25	100–250	250	—[c]
Escitalopram	10	20	40	60
Fluoxetine	20	40–60	80	120
Fluvoxamine	50	200	300	450
Paroxetine	20	40–60	60	100
Sertraline[d]	50	200	200	400

[a]Some patients may need to start at half this dose or less to minimize undesired side effects such as nausea or to accommodate anxiety about taking medications.
[b]These doses are sometimes used for rapid metabolizers or for patients with no or mild side effects and inadequate therapeutic response after 9 weeks or more at the usual maximum dose.
[c]Combined plasma levels of clomipramine plus desmethylclomipramine 12 hours after the dose should be kept below 500 ng/mL to minimize risk of seizures and cardiac conduction delay.
[d]Sertraline, alone among the selective serotonin reuptake inhibitors, is better absorbed with food.

therapie wijzigen?

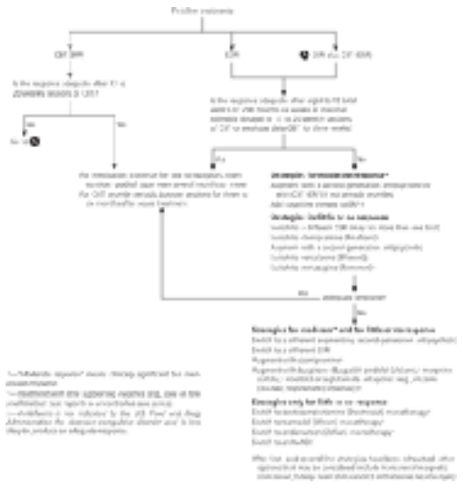


TABLE	Obsessive-compulsive disorder stages of response
Recovery	Not at all ill; Y-BOCS < 8
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Relapse	Symptoms return (CGI 6, or 25% Y-BOCS increase from remission score) after 3+ months of treatment at adequate dosage
Refractory	No change or worsening of symptoms with all available therapies

Y-BOCS, Yale-Brown Obsessive Compulsive Scale; CGI, Clinical Global Impressions. Adaptsd from Pallanti S et al. *Int J Neuropsychopharmacol*. 2002.[10]

casus opnieuw evalueren
eerste lijn zelden volledige remissie: er is altijd ruimte voor verbetering!
de beslissing hangt (ook) af van wat patiënt als comfortabel ziet!
welke wijzigingen?

AUGMENTATIE: SSRI, ERP, AP
hoge dosis SSRI = off label, informed consent
cardiaal, stolling, ionogram opvolgen
farmacokinetiek: fluoxetine/clomipramine vs risperidone

SWITCH: (S)SRI, venlafaxine, remergon
zwakke evidentie, geen dose finding studies

8.8.7. Novel agents, as monotherapy or augmentation strategies
The following compounds are under investigation for OCD and have already shown some evidence of efficacy, but because they so far lack convincing validation in controlled studies, they cannot at present be judged to be effective. The glutamatergic compound, memantine has appeared helpful as an adjunct to an SSRI in a few open-label trials and two small randomized placebo-controlled trials (Ghaleiha et al., 2013 and Haghighi et al., 2013). Preliminary results from open-label studies suggesting efficacy for riluzole (Coric et al., 2005), another glutamate modulating agent, have so far not been substantiated. In a placebo controlled trial of riluzole in children with refractory OCD, no significant difference was noted on any of the primary or secondary outcome measures (Grant et al., 2014). The glutamatergic hypothesis has been further explored through investigations of ketamine in OCD. A randomized controlled cross-over trial of ketamine versus placebo infusion led to >35% reduction in YBOCS score in 50% of those infused with ketamine (n=8) (Rodriguez et al., 2013). However, in another 3-day open label trial of ketamine in 10 subjects with refractory OCD and depression there were no OCD responders and although depressive symptoms improved, the post-baseline improvement in Y–BOCS amounted to <12% (Bloch et al., 2012). Further, ketamine has to be used cautiously, given its association with lower urinary tract (bladder) damage (Winstock et al., 2012). The 5-HT3 receptor antagonist ondansetron, administered in combination with fluoxetine, demonstrated a superior effect over placebo plus fluoxetine on the Y–BOCS in a randomized controlled pilot study in treatment-resistant patients (Soltani et al., 2010). However, the results of an as yet unpublished multicentre trial did not meet the primary efficacy endpoint to demonstrate an improvement in OCD symptoms versus placebo (Biotechnologyevents.com 2013). Mirtazapine as monotherapy has been reported to significantly improve outcomes in a placebo-controlled discontinuation study of 15 open-label mirtazapine responders. In the 8-week, double-blind, placebo-controlled discontinuation phase, the mirtazapine group’s mean Y–BOCS score fell a mean±S.D. of 2.6±8.7 points while the placebo group’s mean score rose a mean±S.D. of 9.1±7.5 points (Koran et al., 2005). Clonazepam, as an adjunctive to an SRI, may produce symptomatic benefit (Hewlett et al., 1992), possibly through improving associated anxiety. It is less suitable in those with previous history of benzodiazepine or other substance abuse or dependence.
VALIDHTML
It has been suggested that antiepileptic mood stabilizers may, in combination with an SSRI, have a role in the treatment of OCD, but the supporting evidence at present is not strong and further placebo-controlled trials are necessary. Positive results were obtained in a small randomized controlled trial of lamotrigine (Bruno et al.,

(niet-) invasieve neuromodulatie?

ECT

te weinig evidentie
(geen DB RCT)

Fontenelle, 2015
doi:10.1038/mp.2016.124

DBS

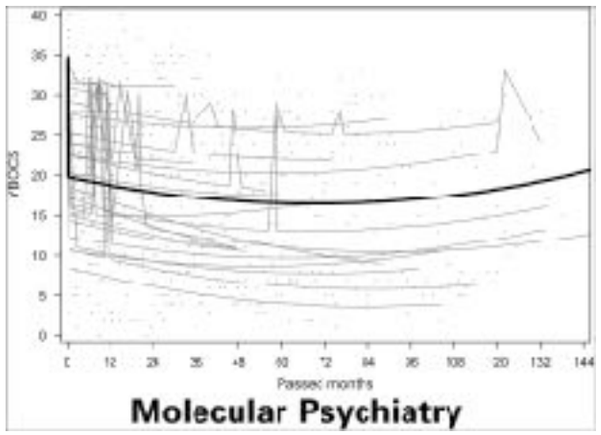
case series
semi-gecontroleerde
studies

ABLATIEVE NC

case series
semi-gecontroleerde
studies

TMS

OFC-SMA
power!



Raymaekers et al., Molecular Psychiatry advance online publication 02 August 2016.
doi:10.1038/mp.2016.124

9.1. Electroconvulsive therapy (ECT)
Evidence to support the use of ECT in OCD is limited due to sample size and study design issues, with an absence of blinded controlled trials. The UK National Institute for Health and Clinical Excellence (NICE 2006) and the APA Practice Guidelines on OCD (Koran et al., 2007) concluded that there is insufficient evidence on which to base a recommendation for the use of ECT in the treatment of OCD, especially given potential associated risks.

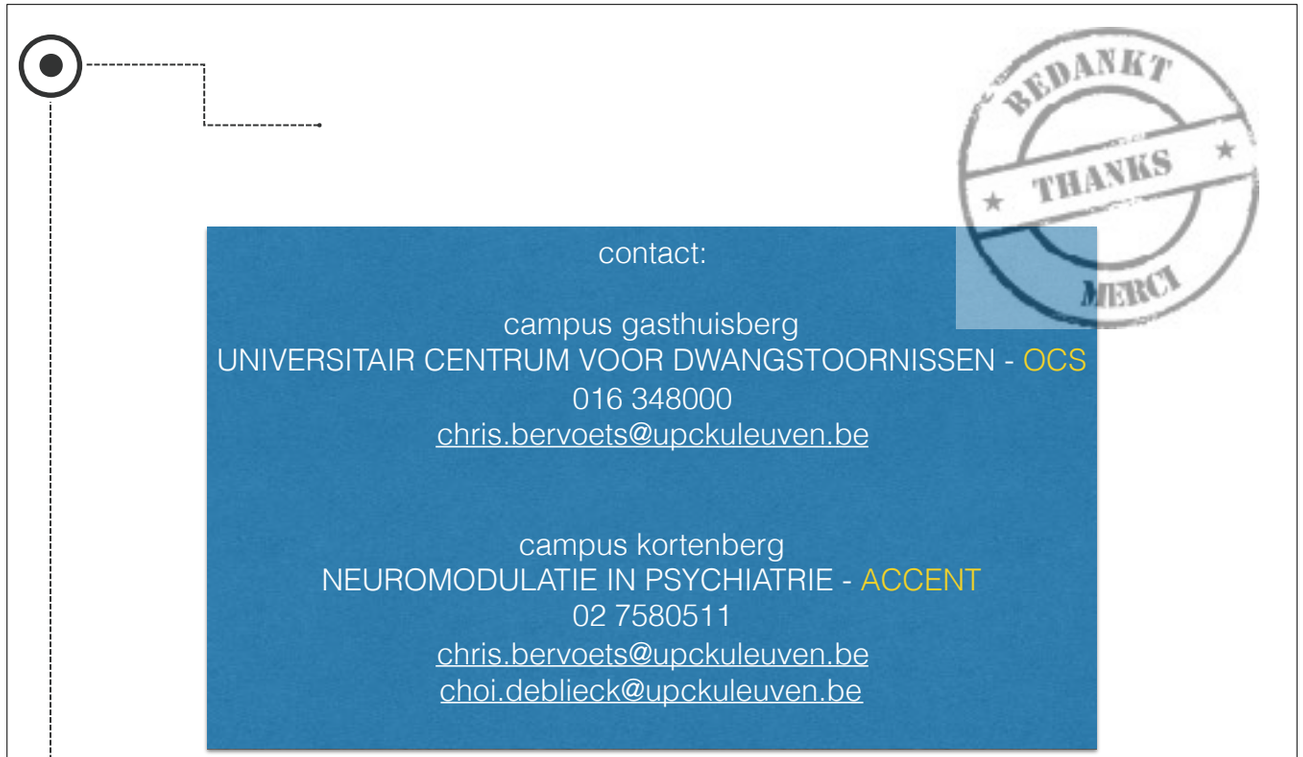
9.3. Deep brain stimulation (DBS)
Deep brain stimulation (DBS) is a neurosurgical treatment that involves the implantation of electrodes that send electrical impulses to specific locations in the brain, with areas selected according to the type of symptoms to be addressed. This approach permits focal, relatively low risk, and relatively reversible modulation of brain circuitry. DBS may bring about therapeutic effects in OCD by modulating the cortico–striatal neurocircuitry that is widely proposed to mediate OCD (Bourne et al., 2012). Stimulation of the ventral capsule/ventral striatum appears to improve mood, obsessions, and compulsions, whereas stimulation of the sub-thalamic nucleus may selectively improve compulsions (Milan et al., 2010). Small studies with at best partially controlled designs have reported significant overall average Y–BOCS decreases ranging from 6.8 to 31 points (in severely ill patients with baseline Y–BOCS scores usually exceeding 30), and the average overall responder rate is 50%. The procedure is reported to be ‘relatively safe’ with limited side effects (de Koning et al., 2011). However, adverse events have been reported. In a study (Greenberg et al., 2006) that followed up the 3-year outcomes following bilateral stimulation of ventral capsule/ventral striatum areas in 10 adult OCD patients meeting stringent criteria for severity and treatment resistance, the following surgical adverse effects were reported: asymptomatic hemorrhage, a single seizure, and superficial infection. Psychiatric adverse effects included transient hypomanic symptoms as well as worsening of depression and OCD when DBS was interrupted. Acute adverse effects of DBS included transient sadness, anxiety, and euphoria or giddiness. Anxiety was more frequent with monopolar than with bipolar stimulation. Suicide events were not noted when DBS was interrupted, and cognitive events were described as relatively benign. At the present time, DBS remains a highly experimental treatment, with evidence largely based on case series.

(niet-) invasieve neuromodulatie: toekomst

therapeutisch alternatief
predictief instrument
(criteria DBS aanvullen)
(therapeutische) biomarkers

BESLUIT

- groot „treatment gap”
- weinig CBT/ERP therapeuten met OCD „bias”
- richtlijn: meer gewicht gegeven aan CBT (+ ERP)
- augmentatie met AP is belangrijkste 2de stap
- hoog doseren, (zeer) lang doorgaan
- belang van neuromodulatie



9.1. Electroconvulsive therapy (ECT)

Evidence to support the use of ECT in OCD is limited due to sample size and study design issues, with an absence of blinded controlled trials. The UK National Institute for Health and Clinical Excellence (NICE 2006) and the APA Practice Guidelines on OCD (Koran et al., 2007) concluded that there is insufficient evidence on which to base a recommendation for the use of ECT in the treatment of OCD, especially given potential associated risks.

9.3. Deep brain stimulation (DBS)

Deep brain stimulation (DBS) is a neurosurgical treatment that involves the implantation of electrodes that send electrical impulses to specific locations in the brain, with areas selected according to the type of symptoms to be addressed. This approach permits focal, relatively low risk, and relatively reversible modulation of brain circuitry. DBS may bring about therapeutic effects in OCD by modulating the cortico–striatal neurocircuitry that is widely proposed to mediate OCD (Bourne et al., 2012). Stimulation of the ventral capsule/ventral striatum appears to improve mood, obsessions, and compulsions, whereas stimulation of the sub-thalamic nucleus may selectively improve compulsions (Milan et al., 2010). Small studies with at best partially controlled designs have reported significant overall average Y–BOCS decreases ranging from 6.8 to 31 points (in severely ill patients with baseline Y–BOCS scores usually exceeding 30), and the average overall responder rate is 50%. The procedure is reported to be ‘relatively safe’ with limited side effects (de Koning et al., 2011). However, adverse events have been reported. In a study (Greenberg et al., 2006) that followed up the 3-year outcomes following bilateral stimulation of ventral capsule/ventral striatum areas in 10 adult OCD patients meeting stringent criteria for severity and treatment resistance, the following surgical adverse effects were reported: asymptomatic hemorrhage, a single seizure, and superficial infection. Psychiatric adverse effects included transient hypomanic symptoms as well as worsening of depression and OCD when DBS was interrupted. Acute adverse effects of DBS included transient sadness, anxiety, and euphoria or giddiness. Anxiety was more frequent with monopolar than with bipolar stimulation. Suicide events were not noted when DBS was interrupted, and cognitive events were described as relatively benign. At the present time, DBS remains a highly experimental treatment, with evidence largely based on case series.