Towards improving treatment for childhood OCD: Analyzing mediating mechanisms & non-response

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Citation for published version (APA):
Summary & General discussion
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What are mediating mechanisms in CBT for childhood OCD? For whom does CBT work and for whom is CBT less effective? What should be recommended for those children who do not sufficiently benefit from CBT? The studies described in the present thesis were aimed to answer these questions. In this section the findings are summarized followed by clinical implications and a general discussion.

Summary

Part I. Candidate mediating mechanisms:
Dysfunctional cognition and selective attention for threat

Before we could focus on our research questions, some preparatory work had to be done. Chapter 1 and 2 are about two questionnaires that we had selected to examine dysfunctional beliefs in childhood OCD. Dysfunctional beliefs are assumed to play a key role in the development, maintenance, and treatment of OCD.

The aim of the first study, described in Chapter 1, was to evaluate the psychometric properties of the Dutch version of the child version of the Obsessive Beliefs Questionnaire (OBQ-CV; Coles et al., 2010), and to examine whether children with OCD reported more dysfunctional beliefs than typically developing children. For this purpose, we translated the OBQ-CV and conducted a validation study in a community sample of 8-to-18-years-old children ($N = 547$) and a clinical sample of children with OCD ($N = 67$). Overall, results supported the reliability and validity of the Dutch version of the OBQ-CV. The internal consistency was good to excellent. Test-retest reliability was acceptable to good. Results of a confirmatory factor analysis suggest that the OBQ-CV consists of four factors representing perfectionism/intolerance of uncertainty, overimportance and control of thoughts, inflated responsibility, and overestimation of threat. These factors were highly related and one higher order factor representing the total amount of obsessive beliefs explained the correlations between the four lower order factors to a large extent. This raises questions about the validity of separate belief domains. Furthermore, children with OCD reported more obsessive beliefs than non-clinical children. They
reported both a wider variety of obsessive beliefs and a higher frequency of these beliefs. Obsessive beliefs were related to self-reported obsessive-compulsive (OC) symptoms. There was, however, no significant relation between obsessive beliefs and clinician-rated severity of OCD. In addition, obsessive beliefs may not be specific for OCD, as they were also related to symptoms of anxiety and depression.

In the second study, described in Chapter 2, we examined the psychometric properties of the Dutch version of the Meta-Cognitions Questionnaire for adolescents (MCQ-A; Cartwright-Hatton et al., 2004), and whether children with OCD reported more meta-cognitions than typically developing children. The MCQ-A was administered in a non-clinical sample (N = 317; 12–18 years), and a clinical sample of adolescents with OCD (N = 40, 12–18 years). The results support the five-factor structure of the MCQ-A consisting of positive beliefs about worry (PB); beliefs about uncontrollability of worrying and about the dangers of failing to control worrying (UD); cognitive confidence (CC); beliefs about superstition, punishment and responsibility related to harmful consequences of failing to control worry (SPR); and cognitive self-consciousness (i.e., preoccupation with one's own thought processes; CSC). The MCQ-A showed fair to good internal consistency and overall good test-retest reliability, but results were equivocal with regard to the test-retest reliability of the SPR subscale. Adolescents with OCD reported more meta-cognitive beliefs than non-clinical adolescents, although the ranges of the scores of both groups showed considerable overlap. Cognitive confidence, that is the lack of confidence in one's own memory and attentional capabilities, did not discriminate between adolescents with OCD and non-clinical adolescents. This finding was unexpected as a lack of cognitive confidence is expected to play a role in OCD according to the meta-cognitive model, especially with regard to checking (e.g., Van den Hout & Kindt, 2003a, 2003b, 2004). Results concerning the relation between meta-cognitive beliefs and OC symptoms provided equivocal evidence for the meta-cognitive model of OCD. There was no relation between meta-cognitive beliefs and clinician-rated severity of OCD. Only the subscales UD, CSC, and SPR were related to self-reported OC-symptoms. The relation between meta-cognitive beliefs (UD, CSC, and SPR) and symptomatology was not specific for OCD, these beliefs were also related to symptoms of anxiety and depression. This may be not surprising, as the meta-cognitive model has been developed as a general model concerning worry (e.g., Papageorgiou & Wells, 2003; Wells, 1997).
Towards improving treatment for childhood OCD

To summarize, results of these two studies provide some evidence for cognitive models of the development and maintenance of OCD. Children with OCD reported more dysfunctional beliefs and with a higher frequency than non-clinical children. Furthermore, most belief domains were related to self-reported OC symptoms, independent of anxiety and depression. However, in contrast to what has been predicted from cognitive models, dysfunctional beliefs were not significantly related to clinician-rated severity of OCD, were not specific for OCD, and differences in dysfunctional beliefs between clinical and non-clinical children were small.

The studies described in Chapter 3 and 4 were aimed to examine whether increased selective attention for (OCD-related) threat is an underlying mechanism in childhood OCD. We developed an adapted version of the dot probe task and conducted a pilot study in a community sample of 8-to-18-years-old children ($N = 33$). The aim of this study, described in Chapter 3, was to examine whether typically developing children show an attentional bias for threatening information and whether this bias can be controlled over time. During the dot probe task threatening and neutral pictorial stimuli were shown for several exposure durations (17 ms masked, 500 ms, and 1250 ms). We expected all children to automatically orient attention towards threat (17 ms), and still orient towards threat after 500 ms stimulus exposure. In the 1250 ms condition we expected a decrease of attentional bias for older children (from 12 years and over) as children of this age may have acquired the ability to inhibit the tendency to selectively attend to threat and 1250 ms may provide sufficient time to redirect attention.

In line with these hypotheses, results revealed an automatic attentional bias for threat (17 ms condition) that persisted during later, more controlled stages of information processing (500 ms and 1250 ms condition). Contrary to our expectations, no effects of age and anxiety on attentional processes were found. This suggests that attentional bias is not dependent on anxiety level nor age. However, the absence of a relation with age and anxiety could also be due to insufficient power caused by the small sample size in this study. Furthermore, participants showed a delayed response to threat-containing trials relative to neutral trials in the 500 and 1250 ms condition, indicating interference by threat. Together, these results suggest that children may selectively direct attention towards threat, and that an attentional bias for threat precedes
behavioral interference in children.

We conducted a second dot probe study, described in Chapter 4, to examine whether children with OCD show increased selective attention for (OCD-related) threat compared to anxious and typically developing children. Data were collected in a sample of children with OCD (8–18 years, $N = 58$), a control group of children with other anxiety disorders ($N = 58$), and a non-clinical control group (community sample; $N = 58$). Participants were matched on age and gender. We hypothesized that children with OCD would show an increased attentional bias for OCD-related threat compared to the community sample, which could be explained by increased vigilance for threat initially (500 ms stimulus exposure), followed by difficulty to disengage from threat (1250 ms stimulus exposure). Similar results were expected for the anxiety sample regarding general threat stimuli.

Contrary to our expectations, no increased selective attention for disorder-specific threat was found for children with OCD and for children with other anxiety disorders compared to the community sample. All children showed a bias away from OCD-related threat initially (stimuli visible for 500 ms), which disappeared over time (no bias for OCD stimuli visible for 1250 ms). In addition, all children showed a bias towards general threat stimuli initially (500 ms), which also disappeared over time (1250 ms). When we distinguished between vigilance and difficulty to disengage, we found a negative vigilance effect (i.e., a slower response to congruent threat trials than to neutral trials, indicating that children directed attention away from threat) for OCD-related and general threat stimuli visible for 500 ms, and difficulty to disengage from OCD-related and general threat stimuli visible for 1250 ms. Further analyses revealed that response times for trials containing (OCD-related) threatening information were slowed down in all children, suggesting interference by threat. Because behavioral interference confounds vigilance and difficulty to disengage analyses (vigilance effects can be masked by interference, and difficulty to disengage effects can also be explained by a more general interference effect), results regarding vigilance and difficulty to disengage cannot be unambiguously interpreted.

In conclusion, our results do not support the hypothesis that increased selective attention for OCD-specific threatening information is an underlying mechanism in childhood OCD. Although children with OCD directed attention away from OCD-related threat initially, they did not differ in this respect
Towards improving treatment for childhood OCD

from typically developing children. Similar, although children with OCD and children with other anxiety disorders initially showed a bias towards threat (general threat stimuli), this bias did not deviate from the bias in typically developing children. Furthermore, behavioral responses were interrupted by the processing of threatening information for all children.

Part II. Mediating mechanisms and non-response
Next, our main research questions needed to be answered: What are mediating mechanisms in CBT for childhood OCD?; For whom does CBT work and for whom is CBT less effective?; and, What should be recommended for children who do not sufficiently benefit from CBT? Chapters 5 and 6 address these questions.

Knowledge about mechanisms of therapeutic change may provide clues for improving treatment. Based on the extant literature, we selected two potential mediating mechanisms: change in OCD-related dysfunctional beliefs and decrease of selective attention for OCD-related threat. In Part I of this thesis we concluded that dysfunctional beliefs may be related to OC symptoms in childhood OCD, although results were not unequivocally supporting cognitive models of OCD. An important note is that results from the studies described in Part I cannot reveal whether dysfunctional beliefs cause OC symptoms, whether they are the result of OC symptoms, or whether they are only co-occurring. Further, the study described in Chapter 4 raised questions about increased selective attention for OCD-related threat in childhood OCD. Based on the present results we rejected our hypothesis concerning increased selective attention for (OCD-related) threat in childhood OCD. Consequently, selective attention for threat was not further tested as a potential mediator of treatment outcome. We proceeded the study on mediating mechanisms in CBT for OCD with one potential mediator: changing OCD-related dysfunctional beliefs. This study is described in Chapter 5. Fifty-eight children with OCD (8–18 years) received 16 weekly sessions CBT. Dysfunctional beliefs and OCD severity were measured pre-treatment, mid-treatment, post-treatment, and at 16-week follow-up. Based on the studies on the psychometric properties of the OBQ-CV and the MCQ-A, we decided to leave out the MCQ-A from the mediation study. The MCQ-A showed less consistent results regarding the relation between meta-cognitive beliefs (especially with regard to cognitive confidence) and
OC symptoms. Furthermore, the MCQ-A was administered in children from 12 years and older, whereas the OBQ-CV covered a broader age range (8–18 years) and was therefore available for a larger sample. Several Latent Different Score models were computed to examine whether changes in dysfunctional beliefs preceded changes in OCD severity, were a consequence of changes in OCD severity, or whether the relation was bidirectional. According to cognitive models, changes in beliefs were expected to precede changes in OCD severity.

Results showed that OCD severity and dysfunctional beliefs decreased during CBT. We found, however, no support for the hypothesis that changing dysfunctional beliefs is a mediating mechanism in CBT for childhood OCD. Instead, changes in beliefs were better explained by changes in OCD severity that occurred within the same time interval. Noteworthy, our results do not allow for the conclusion that changes in dysfunctional beliefs are the effect of changes in OC symptoms, as a relation over time was not demonstrated. That does not alter the fact however, that he present finding that changes in dysfunctional beliefs were explained by changes in OCD severity rather than the reverse, cast doubt on cognitive models for OCD.

The aim of the last study, described in Chapter 6, was twofold: we examined whether severity of OCD, the rate of improvement during the first eight treatment sessions, and co-morbid autistic traits were predictive of treatment effect in CBT. We further examined whether patients with moderate to severe OCD can be effectively treated with CBT monotherapy and whether continuation of CBT monotherapy was an effective strategy for partial and non-responders to first-line CBT. The first part of the study was a randomized controlled trial on the effect of CBT monotherapy versus a waitlist condition. In the second part of the study we examined the effect of continuation of CBT for partial and non-responders. Originally we had planned a controlled trial to examine the effects of continuation of CBT versus the combined treatment of CBT and an SSRI. However, we changed the design into an open study since almost all partial and non-responders preferred to continue the CBT and refused medication. In the open design, the clinician in consultation with the patient and the patient’s parents decided about further treatment policy after 16 sessions of CBT. When more treatment was needed based on clinical judgment, CBT was continued. Only when complaints were untenable for the patient or the family, or in case of deterioration of symptoms or severe depression, the combination of CBT
Towards improving treatment for childhood OCD

and an SSRI was indicated. Participants were 58 children (8–18 years) with a primary diagnosis of OCD.

Results showed that CBT was associated with a significant decrease in OCD severity. More severe OCD at baseline and autistic traits predicted poorer treatment outcome. More specifically, children with severe OCD and high levels of autistic traits ended treatment with more residual complaints, but OCD severity and autistic traits were not predictive for the rate of improvement during CBT and follow-up (both groups showed comparable symptom reduction). Although low initial treatment response after eight sessions of CBT was related with poorer treatment outcome after 16 sessions of CBT, after one-year follow-up the low and high response group showed comparable levels of OC symptoms. Furthermore, continuing CBT after the protocol (16 sessions of CBT) had been completed, proved to be effective. These results implicate that CBT is an effective treatment for childhood OCD, also for patients with moderate to severe OCD and for patients with autistic traits. Furthermore, low response at the start of CBT does not necessarily predict low or non-response in the end. Finally, continuing CBT after the protocol can lead to further improvement, at least for a significant number of patients.

Clinical implications
What should we say to the boy who concluded in surprise that treatment is just arbitrary? Fortunately, CBT is not just arbitrary. It is based on strong theoretical assumptions. We found that one of these assumptions – changing dysfunctional beliefs is essential for treating OCD – may be incorrect. Restructuring dysfunctional cognitions may not be an essential component in treatment for OCD in children. This does not imply that cognitive interventions are irrelevant. They may be very useful to support exposure-based treatment.

Based on the present findings, we conclude that when cognitive interventions do not appeal to an individual patient, therapists can focus solely on exposure with response prevention.

A second implication derived from the present study is that although attentional bias training procedures have been developed for OCD and anxiety (e.g., Cowart & Ollendick, 2011; Najmi & Amir, 2010), our results do not support the addition of attentional bias training to regular, evidence-based treatment for children with OCD. As a first step, we recommend further research to shed more light on attentional processes in childhood OCD.
Third, results indicate that CBT monotherapy is an effective treatment for childhood OCD, also in cases of severe OCD and for patients with co-morbid autistic traits, although these patients may end with more residual complaints. In addition, results suggest that therapists should not give up too early when patients show only small improvement in OC symptoms during the first treatment phase. Improvement may increase when the therapist carries on. For patients with severe OCD, patients with autistic traits, and patients who show small initial improvement, treatment may take more time. (Noteworthy, we do not argue that therapists should carry on when treatment is not effective at all.) The present results indicate that adding medication to CBT may be less often needed than recommended by the AACAP treatment guidelines (Geller et al., 2012). This is important as medication can have adverse effects, and moreover in the long term adverse effects of medication are unknown. However, CBT monotherapy may not be a sufficient treatment for all patients with OCD. Presently, for whom CBT will be effective and for whom additional treatment modalities are needed, is still unknown. Empirical support for the decision to add medication to CBT is lacking, and by now adding medication for children with OCD is a matter of trial and error.
Towards improving treatment for childhood OCD

**General discussion**

Now that we have reached the final stage of our study, we realize in looking back that some parts of the study could have been conducted differently and perhaps better. This section contains a discussion of issues that set us thinking during the process of the study.

When we started our study we were quite ambitious. This became clear in our dot probe study. A literature search revealed several issues that had to be taken into account, such as the selection of the paradigm, the type of stimuli, the combination of stimulus pairs within trials (to measure different aspects of selective attention), and the exposure duration of the stimuli. We selected stimuli containing OCD-related threat, general threat, and neutral scenes. Because an attentional bias can operate for emotional stimuli in general and may not be specific for threat (the emotionality hypothesis; e.g., Martin, Williams, & Clark, 1991), we also included positive stimuli. Stimuli were displayed for several exposure durations including automatic as well as controlled conditions (17 ms masked, 500 ms, 1250 ms) to examine the time-course of attentional processes. We collected a wealth of data addressing several aspects of selective attention. This would, however, have resulted in too many analyses based on a too small sample. We decided to concentrate on hypotheses concerning selective attention for threat over time. We left out several variables, such as the positive stimuli and the trials displayed for 17 ms as this exposure duration was quite experimental and complicated the design. However, still interpreting the findings turned out to be challenging. Unexpected findings with regard to the analyses of vigilance and difficulty to disengage from threat raised questions on how best to analyze the data and resulted in a reconsideration of the analyses.

Koster and colleagues (2004) have noted that an attentional bias towards threat can be explained by either vigilance for threat or by difficulty to disengage attention from threat, and they developed an alternative way of analyzing dot probe results to discriminate between these two processes. In these analyses neutral trials (neutral-neutral stimulus pairs) were added as a baseline. A faster response to congruent trials than neutral trials indicates vigilance for threat. A slower response to incongruent trials than neutral trials indicates difficulty to disengage from threat. Figure 1 shows the procedure for congruent and incongruent trials in the dot probe task.
The question whether an attentional bias is due to increased vigilance for threat or difficulty disengaging from threat is clinically relevant. Can anxiety be understood as the consequence of a quick orientation by anxious individuals towards threat (vigilance), which may lead to the impression that the environment is quite threatening? Or do all individuals tend to allocate attention preferentially to threat, but do anxious individuals differ from non-anxious individuals in their ability to redirect attention away from threat when no real threat exists (difficulty to disengage from threat)? We analyzed vigilance and difficulty to disengage in our dot probe study and encountered a remarkable finding. Results indicated a negative vigilance effect (children showed a slower response to congruent trials than to neutral trials, indicating that they directed attention away from threat stimuli), and difficulty disengaging from threat (they also showed a slower response to incongruent trials than neutral trials, indicating that their attention was stuck to the threat stimuli). These results seem contradictory and cannot be explained by either vigilance or difficulty to disengage from threat.

We concluded that vigilance and difficulty to disengage cannot be completely distinguished with the dot probe task. If participants show vigilance for threat, they preferentially allocate attention towards threat. Thus, when the dot appears at the threatening location in a congruent trial, it would be easily detected by the participant resulting in a fast response for congruent trials (interpreted as vigilance). However, if the dot had appeared at the neutral location (incongruent trial), the participant would have focused at the wrong location resulting in a slower response (interpreted as difficulty to disengage). Thus, vigilance always results in both a faster response to congruent trials and
Towards improving treatment for childhood OCD

a delayed response to incongruent trials. The inverse is also true, if participants show difficulty to disengage, attention is focused at the threatening location and stays there for a while. This will result in a slower response to incongruent trials and a faster response to congruent trials. Therefore, it seems impossible to detect pure vigilance without difficulty to disengage, or the reverse. Noteworthy, vigilance and difficulty to disengage may not be completely indistinguishable. The more trouble a participant has with disengaging from threat, the longer it will take to detect the probe at the neutral location in an incongruent trial, without becoming equally faster for congruent trials. In addition, in our opinion the exposure duration of the stimuli should also be taken into account by analyzing vigilance versus difficulty to disengage. Vigilance for threat may occur in an early stage of information processing and can be followed by disengaging from threat. Therefore, it seems more relevant to examine vigilance for stimuli displayed for short durations, and difficulty to disengage for stimuli displayed for longer durations. However, up to now it is still unknown which exposure duration captures the crucial moments of specific attentional processes.

Following the above reasoning, we expected a slower response to incongruent trials combined with a faster response to congruent trials in our dot probe study. However, results showed a slower response to both incongruent and congruent trials, a pattern that had also been reported in several previous studies. For example, Koster et al. (2004) reported a delayed response to both congruent and incongruent trials using highly threatening stimuli in a student sample. Legerstee et al. (2009) reported that clinically anxious children (8–16 years; N = 131) showed a delayed response to incongruent trials using severely threatening pictures, but no difference between congruent threat and neutral trials. How could we explain this?

Behavioral responses (pressing the response key) may have been interrupted by the processing of threatening information. Indeed, in our sample response times were slowed down in all threat-containing trials, which suggests an interference effect of threat. Interference effects can also explain the above-mentioned inconsistencies in other studies. Although interference of threat has been mentioned by some other authors (e.g., Koster et al., 2004; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996; and by now Mogg, Holmes, Garner, & Bradley, 2008), it still receives little attention in studies on selective attention. That is remarkable, as interference may mask vigilance and incorrectly suggests difficulty to disengage.
What has resulted from our studies on selective attention for threat? Besides our comments on the analyses concerning vigilance and difficulty to disengage and the interference effects of threat in dot probe studies, our primary aim was to examine the role of selective attention for (OCD-related) threat in childhood OCD. We found no evidence for increased selective attention for threat in children with OCD. These results fit into new developments concerning the Diagnostic and Statistical Manual of Mental Disorders (DSM). In the fifth edition of the DSM OCD will not any longer be classified as an anxiety disorder, but a new category of obsessive-compulsive spectrum disorders has been proposed (e.g., Hollander, Kim, Braun, Simeon, & Zohar, 2009). This implicates that anxiety is not considered a core phenomenon in OCD (e.g., Mataix-Cols, Pertusa, & Leckman, 2007), which, however, does not mean that anxiety does not play a role in OCD.

The study described in Chapter 5 yielded no support for the hypothesis that changing dysfunctional beliefs is a mediating mechanism in CBT for childhood OCD. So, our results cast doubt on cognitive models of the development and maintenance of OCD. Cognitive models have dominated the OCD literature for decades, but critics are arising (e.g., Julien, O’Connor, & Aardema, 2007; Longmore & Worrell, 2007). For example, not all patients with OCD may have more dysfunctional beliefs than non-clinical individuals (e.g., Calamari et al., 2006; Taylor et al., 2006), and dysfunctional beliefs may not be exclusively related to OC symptoms but also to anxiety and depression (e.g., Julien et al., 2007; Chapter 1 and 2 of this thesis). This implicates that these beliefs may be neither a sufficient nor a necessary condition for the development and maintenance of OCD. Furthermore, although several studies have reported a relation between dysfunctional beliefs and OC symptoms, thus far no study convincingly has demonstrated that such beliefs cause OC symptoms. In addition, there hardly is any empirical support for the assumption that cognitive changes are a mediating mechanism in CBT for OCD patients. The few studies that have examined this question, almost all in adult patients, yielded inconsistent results (e.g., Anholt et al., 2008; Polman, Bouman, Van Geert, de Jong, & Den Boer, 2011; Rheaume & Ladouceur, 2000; Storchheim & O’Mahony, 2006; Williams, Salkovskis, Forrester, & Allsopp, 2002). In our study, it has also not been demonstrated that changing dysfunctional cognitions is a mediating mechanism. This may be related to the difficulty of measuring thoughts. For example, a questionnaire provides only
Towards improving treatment for childhood OCD

information about explicit cognitions. Implicit measures are needed to examine whether cognitions play a role at the unconscious level. We developed a measure to examine OCD-related dysfunctional cognitions at the implicit level, but during the process of the study some serious concerns were raised about the validity of this task. We decided that first more research was needed before we could use this task. In addition, it may be rather complex to disentangle a relation over time between beliefs and OC symptoms. In cognitive models it is assumed that patients with OCD ascribe too much meaning to unwanted intrusive thoughts, which raises anxiety. Compulsions are then performed to neutralize or prevent anxious feelings. In treatment patients learn to substitute the dysfunctional interpretations of intrusions with functional ones that do not raise anxiety. Consequently, patients will not experience the urge to perform compulsions anymore, and it seems unlikely that a patient persists in compulsive behavior while there is no urge to do so. As a result, no temporal lag will exist between cognitive changes and changes in OC symptoms. However, this still cannot explain the findings of some previous studies that changes in dysfunctional beliefs sometimes followed changes in compulsions (Anholt et al., 2008; Rheaume & Ladouceur, 2000), or that beliefs and OC symptoms changed in opposite directions (Polman et al., 2011).

It may be that cognition and behavior are causally related, but in the opposite direction than what generally is assumed. This is put forward by Lamme (2011). In his book ‘De vrije wil bestaat niet’ (‘Free will does not exist’), Lamme describes an experiment with a split brain patient (i.e., the corpus callosum connecting the left and right cerebral hemispheres is split which hinders any interhemispheric communication). Two pictures were presented to the patient. The first picture, presented at the left side, showed a house in the snow. The other picture, presented at the right side, showed a bird’s foot. Information entering the eyes via the left side (the house in the snow) is processed by the right hemisphere; information entering the eyes via the right side (the bird’s foot) is processed by the left hemisphere. Because for this patient hemispheres could not exchange information, the left hemisphere did not know about the house in the snow, and the right hemisphere did not know about the bird’s foot. Some other pictures were presented and the patient was instructed to select a picture that was associated with the previous picture. With his left hand, coordinated by the right hemisphere (that saw the house in the snow), he selected a shovel. With his right hand, coordinated by the left hemisphere
General discussion

(that saw the bird’s foot), he selected a chicken. Next, the patient was asked to explain his choices, a task performed by the left hemisphere as this part usually produces language and interprets behavior. The choice of the chicken was easy to explain, as this was clearly associated with the bird’s foot. However, the left part could not know about the house in the snow. Confidentially the patient explained that the shovel was chosen to clean out a chicken shed (Gazzaniga, 1998; Lamme, 2011). This experiment shows that our brain may, without being aware of it, interpret and reconstruct behavior afterwards rather than initiate behavior. The patient was convinced that he selected the shovel to clean the chicken shed. The ‘chatter box’ (kwebbeldoos) is what Lamme called this brain function. Obsessions in patients with OCD often provide an explanation for their compulsive behavior: they clean to prevent sickness, they check to prevent fire or burglars, and they repeat rituals to exorcise harm and misfortune. Do these explanatory thoughts indeed cause compulsive behavior (as supposed by cognitive theorists), or are they resulting from post-hoc interpretations of behavior? In the latter case, obsessions are explanations afterwards instead of the cause of OC symptoms. This does, however, not imply that obsessions are irrelevant. Explanations afterwards may be bothersome too.

At the very end of this thesis, I would like to discuss two additional issues that I have noticed during this project.

As in all studies many choices have been made during the process of the study, concerning the measurement methods, the design of the study, the analyses, etc. Preferably, decisions are based on empirical evidence. We searched the literature, discussed issues in our research group, and consulted colleagues. Of course, we also had to deal with practical limitations. After all arguments had been weighted, a decision was made. However, usually many alternatives exist which also have valid arguments. Beforehand it is often impossible to know which option will be the right choice. In addition, you never know what has been missed. Once published, the findings become part of our scientific knowledge. Part of this knowledge depends on the decisions that the researcher has made.

It is not my intention to cast doubt on the value of our scientific knowledge. Rather, I have learned that it is important to interpret results with an open mind. During our research project I noticed that some researchers attempt to interpret results according to their hypotheses. This may be justified, but not in
Towards improving treatment for childhood OCD

all cases. When results do not confirm the expectations of the researchers this is often explained by limitations of the study. Sometimes the option of rejecting the hypothesis is not even mentioned. In contrast, when the hypothesis is confirmed by the data, in several cases limitations of the study are only mentioned in passing. This is unfortunate as results that are not expected, are often the ones that create new insights.

Not any study stands alone. In addition, it would be impossible for a single study to address all issues that may arise. Future studies are needed to examine alternative choices and test newly raised hypotheses. Furthermore, only future research can demonstrate whether a conclusion is justified, by replicating results and extending on previous studies. This way our scientific knowledge is extended and improved. Our study is part of this process. I enjoyed working on it, and look forward to going on!