Cognitive theory in anorexia nervosa and bulimia nervosa: Progress, development and future directions

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Abstract

Important developments have taken place in cognitive theory of eating disorders (EDs) (and also in other disorders) since the review paper published by M.J. Cooper in 1997. The relevant empirical database has also expanded. Nevertheless, cognitive therapy for anorexia nervosa and bulimia nervosa, although helpful to many patients, leaves much to be desired. The current paper reviews the relevant empirical evidence collected, and the theoretical revisions that have been made to cognitive models of eating disorders, since 1997. The status and limitations of these developments are considered, including whether or not they meet the criteria for “good” theory. New theoretical developments relevant to cognitive explanations of eating disorders (second generation theories) are then presented, and the preliminary evidence that supports these is briefly reviewed. The lack of integration between cognitive theories of EDs and risk (vulnerability) factor research is noted, and a potential model that unites the two is noted. The implications of the review for future research and the development of cognitive theory in eating disorders are then discussed. These include the need for study of cognitive constructs not yet fully integrated (or indeed not yet applied clinically) into current theories and the need for cognitive theories of eating disorders to continue to evolve (as they have indeed done since 1997) in order to fully integrate such constructs. Treatment studies incorporating these new developments also urgently need to be undertaken.

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1. Introduction

A paper has previously reviewed cognitive theories of eating disorders (EDs), their status, and limitations (Cooper, 1997). Developments in theory that might improve and extend our understanding of cognition and its role in eating disorders were suggested and the implications of these for clinical (specifically cognitive therapy) practice with eating disorder patients were outlined.

Important developments have taken place in cognitive theory of eating disorders, particularly anorexia nervosa (AN) and bulimia nervosa (BN) (and also in other disorders) since the 1997 paper was published. The relevant empirical database has also expanded. The aim of the current paper is to review the theoretical developments that have taken place in our cognitive understanding of eating disorders since 1997 and consider the relevant empirical evidence. The status and limitations of these developments will be assessed, including whether or not they meet the criteria for “good” theory. The relationship between (EDs) and risk (vulnerability) factor research is briefly considered, and a unifying model is noted. Implications for the future, including clinical practice and research, will then be discussed. This will include suggestions for the study of cognitive constructs not yet fully integrated into current theories.

As in the 1997 paper the current review will focus on primarily on AN and BN, and not binge eating disorder (BED) or Eating Disorder Not Otherwise Specified (ED-NOS). Although researchers are becoming increasingly interested in BED, a cognitive theory of BED is still lacking. Moreover, there is some evidence to suggest that BED differs in important demographic, symptom and psychological characteristics from AN and BN, even though it would appear at first glance to have much in common with the latter (for a brief discussion of the differences between BN and BED see Cooper, 2003). Eating Disorder Not Otherwise Specified (ED-NOS), although it appears to be very common in clinical practice (Turner & Bryant-Waugh, 2004), is also currently poorly understood in cognitive terms, and the relevant evidence has not yet been collected or presented in the published literature.

Nine hypotheses derived from four theoretical contributions were considered in the 1997 paper. These are reproduced in Table 1. As in the 1997 paper, and as has been indicated for the reasons outlined above, the discussion will be limited to the two classical eating disorders, i.e. anorexia nervosa (AN) and bulimia nervosa (BN).

The nine hypotheses identified in the 1997 paper were based on four theoretical contributions (Fairburn, Cooper, & Cooper, 1986; Garner & Bemis, 1982; Guidano & Liotti, 1983; Vitousek &

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Table 1
Hypotheses derived from early cognitive theories

<table>
<thead>
<tr>
<th>Hypothesis</th>
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<tr>
<td>1. Treatment based on the models, i.e. cognitive therapy, will be effective;</td>
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<tr>
<td>2. self statements or automatic thoughts will reflect concern with food and eating, weight and shape;</td>
</tr>
<tr>
<td>3. underlying assumptions reflecting concern with food and eating, weight and shape will be strongly endorsed;</td>
</tr>
<tr>
<td>4. core beliefs will reflect global negative evaluations of the self;</td>
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<tr>
<td>5. dysfunctional styles of reasoning or information processing errors and biases will be found in food and eating and in weight and shape concerns;</td>
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<tr>
<td>6. there will be a causal relationship between underlying assumptions and self-statements and eating behaviour, particularly dietary restraint;</td>
</tr>
<tr>
<td>7. dietary restraint, mediated by dichotomous thinking, will result in episodes of binge-eating;</td>
</tr>
<tr>
<td>8. schema driven processes will be evident in areas of core belief concerns;</td>
</tr>
<tr>
<td>9. early experience will be important in the formation of core beliefs.</td>
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