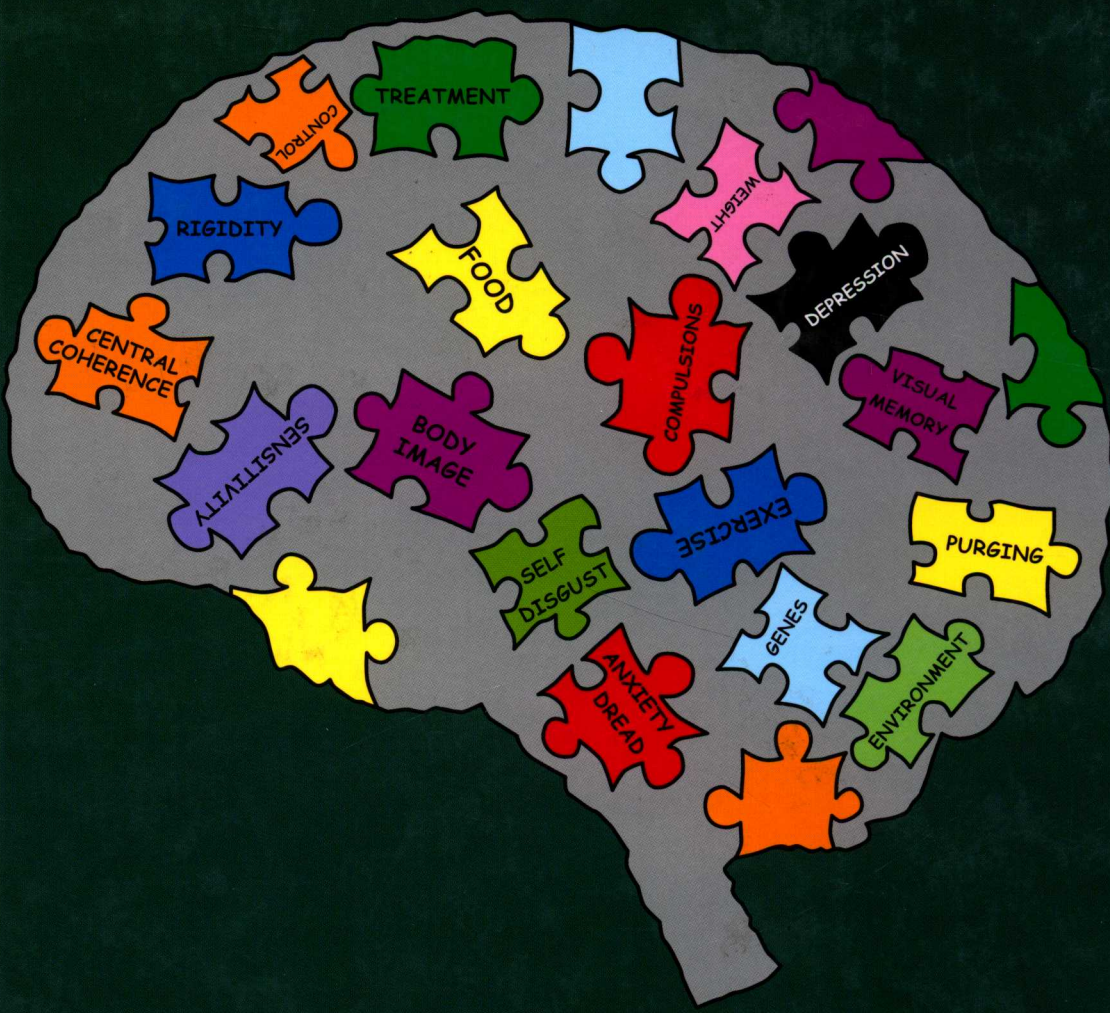
Eating Disorders and the Brain

Edited by Bryan Lask and Ian Frampton



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Eating Disorders and the Brain

Bryan dedicates this book to Ana.
Ian dedicates this book to Katie, Ellie and Merryn.

Preface

Bryan Lask and Ian Frampton

The human brain is often described as 'the most complex structure in the universe, too complex for the human brain to understand' – a most delightful paradox. Eating disorders too are extraordinarily complex, difficult to understand, demanding to treat, have a poor prognosis and are themselves full of paradox. Surely then a union between these two unlikely bedfellows, as attempted in this book, cannot be an easy task? And perhaps this is why there have been relatively few such attempts to date. Yet, despite decades of effort, enormous amounts of research money and well over 100 000 publications, we still do not have an adequate understanding of the pathogenesis of eating disorders! We are still unable to explain why any one individual develops an eating disorder, while the majority do not, and we still do not have effective treatments. Until relatively recently, sociocultural attempts to elucidate the development of eating disorders have been the most influential. However, while offering a part-explanation, they have proved insufficient in accounting for the very specific phenomena so characteristic of these conditions.

In recent years, to an extent perhaps inspired by the 'decade of the brain', there has been more of a focus on what neuroscience might contribute to our understanding. And the results have been enlightening. For example, neuropsychological studies have shown impairments in different cognitive functions, especially executive and visuospatial skills, which appear to be trait- rather than state-related; in other words, they seem to predate the onset and therefore may actually be risk factors for the development of an eating disorder. Structural (anatomical) neuroimaging studies show cortical atrophy and ventricular enlargement, which do indeed reverse with refeeding and are therefore likely to be secondary to inadequate nutrition. However, functional neuroimaging consistently reveals regional and asymmetrical reduction in blood flow, suggesting dysfunction in specific brain regions, which is unlikely to be due simply to starvation and suggests regional dysfunction. This too may be a predisposing factor or a reflection of one. Neurochemistry studies show dysregulation within neurotransmitter

systems, with effects upon the modulation of feeding, mood, anxiety, neuroendocrine control, metabolic rate, sympathetic tone and temperature. These studies indicate that neural mechanisms have a fundamental role in the origin and maintenance of the disorders. Thus we now have far more detailed information on the effects on the brain of starvation, overeating, chaotic eating and dehydration. Even more importantly, we now understand far more about the underlying brain abnormalities and dysfunctions that may contribute to the development of these serious disorders.

In this book we explore in depth how neuroscience knowledge informs our understanding of eating disorders and how it may be applied in clinical practice. We may have erred by focusing rather more on anorexia nervosa (AN) than the other eating disorders, but hope to be forgiven, because there is more neuroscience-based information available on this condition. The first chapter, by clinician David Wood, offers an invitation to fellow clinicians to become acquainted with the seemingly scary, but actually fascinating, world of neuroscience, as applied to eating disorders. In Chapter 2, Beth Watkins provides a meticulous review of the eating disorders – most of the information anyone is likely to need is contained therein. The next three chapters review in detail contemporary knowledge of neuroimaging (Tone Fugslet and Ian Frampton), neuropsychology (Joanna Steinglass and Deborah Glasofer) and neurochemistry and genetics (Ken Nunn). In Chapter 6, Maria Øverås explores how neuroscience contributes to our understanding of one of the core features of eating disorders, body-image disturbance. Mark Rose and Ian Frampton, in Chapter 7, explore and comment upon a number of neuroscience-based conceptual models of eating disorders. Each advances our understanding to some extent but none offers a full explanation of the pathogenesis, phenomenology and maintenance of any of the eating disorders. In Chapter 8, Ken Nunn, Ian Frampton and Bryan Lask attempt the seemingly impossible and propose just such a model for AN. It is for the reader, and subsequent testing of the model, to decide whether or not they have succeeded. The next two chapters explore the clinical relevance of neuroscience knowledge: Ilina Singh and Alina Wengaard (Chapter 9) consider the consequences of the development of neurobiological models for the understanding of eating disorders by patients and their families and their receptivity to treatment. Camilla Lindvall and Bryan Lask (Chapter 10) explore how this empirical knowledge can be converted into practice, with emphasis on its application in both an educational and a clinical context. In relation to the latter they offer a specific focus on cognitive remediation therapy. In the final chapter, the editors offer a summary of the contents of the previous chapters and explore how these findings might be investigated in the future, considering in turn each of the US National Institute of Mental Health Research Domain Criteria (RDoC) Project classes: genes, molecules, cells, neural circuits, behaviours and self-reports.

We hope that this volume will take us a small step forward in our understanding of the neuroscience of eating disorders and will open up an exciting and relevant avenue for all of its readers, regardless of their previous knowledge in the field.

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Bryan Lask and Ian Frampton
January 2011

Contents

1 Why clinicians should love neuroscience: the clinical relevance of contemporary knowledge

David Wood

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1.1 Introduction

Clinicians at times appear to have an uneasy relationship with neuroscience. At a superficial level it may seem that there might be little need to question whether the relationship between neuroscience and clinical work is problematic. However, despite their now reasonably lengthy coexistence, there still exists a tension between these two fields of endeavour. This leads to misunderstanding, and even distrust, which inhibits the undoubted opportunities – if not necessity – for creative and fruitful interaction. Questions are still asked within the clinical domain about the relevance of neuroscientific study, and neuroscientists can become so absorbed and fascinated with their subject that they lose sight of the clinical relevance of what they are studying. It is the contention of this chapter that the relationship between neuroscience and clinical work should not be problematic, and that those on both sides of the divide can learn, not only to live together, but also to admire each other's concepts.

Why should we love neuroscience? Of course, telling someone what he or she should love is a supremely arrogant and rather fruitless enterprise, as anyone who has tried to get their child to love eating, say, oysters will know. But neuroscience is not just an acquired taste; it does not require great familiarity to appreciate its qualities. It is certainly possible to comprehend the wonder, awe and excitement that this field of endeavour can evoke without having to fully understand its every detail. And without some appreciation of the currently available knowledge about the brain, clinicians are in danger of setting off down many a blind alley in carrying out clinical practice.

In order to support this argument, it is first necessary to review some fundamental problems. This will be followed by a brief, and highly condensed, overview of some

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current neuroscience facts, which will then be reviewed within the context of current developments within the field of eating disorders.

1.2 The legacy of mind–body dualism

The tension between clinical work and neuroscience would seem to be supported by the continuing predominance of dualistic thinking, not only within scientific discourse, but in postmodern culture more generally. Given the lengthy history of dualism, from Plato, down through Descartes, to William James and beyond, it is not surprising that it does not easily throw in the towel. The fundamental problem with which Homo sapiens has wrestled for so long is how can we reconcile our sense of ourselves as free agents, capable of choosing our path through life, with a notion of our bodies (including our brains) being constructed of physical stuff that obeys the deterministic laws of nature.

Plato considered that humans had earthly bodies and ethereal souls, and put the mental properties of reason, desire and appetite firmly in the domain of the soul. Indeed, Aristotle thought that the brain was merely an organ for cooling the blood and that the heart was really where the passions lay. Continuing in the Platonic tradition, Descartes, in his pamphlet 'On the Passions of the Soul' [1], decided that bodies were made up of stuff such as blood, muscles, nerves and so on, and were controlled by 'bodily spirits', whereas our thoughts and our passions belonged to the soul, and our mental experiences were instances of awareness of the movements of the bodily spirits via contact between soul and body in the pineal gland. It is hard to know what Plato would have made of someone whom we might now diagnose as suffering from anorexia nervosa (AN). It is reasonable to surmise that he would probably not have considered them to be suffering from an illness. More likely he would have marvelled at the way in which they were able, with so much stoicism, to conquer their appetites and disentangle themselves from the world of the senses, thus liberating their ethereal soul from the constraints of the material body. For Plato and his successors, the passions were seen as something that needed to be subjugated, brought under control, an idea that presages the current interest in emotion regulation and AN.

Dualist accounts, particularly of emotion, have been hard to shake off, and continue beyond Descartes, through Locke and Hume, to William James, Popper and Eccles [2], and even perhaps to some elements of modern emotion theory such as the somatic marker hypothesis [3–6]. They remain alive and well in some clinicians' apparently unshakeable belief that AN is a 'brain' disease, just as in others' similarly unshakeable beliefs that it is a 'mental' illness without physical correlates. But to argue either way implies a distinction between brain and mind that really can no longer be justified.

In essence, all dualist accounts come up against the difficulty that there is no convincing explanation of how, if brain and mind are of different stuff, they can interact, and how mental events can have a causative role in behaviour. There would seem to be little doubt that despite Cartesian dualism's refusal to go quietly, the general direction of neuroscientific endeavour has been inexorably towards a monist[1] position. However, this has brought with it new difficulties.

[1] The term 'monist' here refers to the view that the mind and the body are of one substance, as opposed to the two of the dualist position.