# An Integrative Bio-Psycho-Social Theory of Anorexia Nervosa

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**The need for novel approaches to understanding and treating anorexia nervosa (AN) is well recognized. The aim of this paper is to describe an integrative bio-psycho-social theory of maintaining factors in AN.**

**We took a triangulation approach to develop a clinically relevant theory with face validity and internal consistency. We developed theoretical ideas from our clinical practice and reviewed theoretical ideas within the eating disorders and wider bio-psycho-social literature. The synthesis of these ideas and concepts into a clinically meaningful framework is described here.**

**We suggest eight key factors central to understanding the maintenance and treatment resistance of anorexia nervosa: genetic or experiential predisposing factors; dysfunctional feelings processing and regulation systems; excessive vulnerable feelings; 'feared self' beliefs; starvation as a maladaptive physiological feelings regulation mechanism; maladaptive psychological coping modes; maladaptive social behaviour; and unmet physical and psychological core needs. Each of these factors serves to maintain the disorder.**

**The concept of universal physical and psychological core needs can provide an underpinning integrative framework for working with this distinctly physical and psychological disorder. This framework could be used within any treatment model. We suggest that treatments which help address the profound lack of trust, emotional security and self-acceptance in this patient group will in turn address unmet needs and improve well-being. Copyright © 2016 John Wiley & Sons, Ltd.**

**Key Practitioner Message**
- The concept of unmet physical and psychological needs can be used as an underlying integrative framework for understanding and working with this patient group, alongside any treatment model.
- A functional understanding of the neuro-biological, physiological and psychological mechanisms involved in anorexia nervosa can help patients reduce self-criticism and shame.
- Fears about being or becoming fat, greedy, needy, selfish and unacceptable ('Feared Self') drive over-compensatory self-depriving behaviour ('Anorexic Self').
- Psychological treatment for anorexia nervosa should emphasize a focus on feelings and fostering experiences of acceptance and trust.
- Treatment for patients with anorexia nervosa needs to be longer than current clinical practice.

**Keywords:** Anorexia Nervosa, Bio-Psycho-Social, Theory, Core Needs, Emotions, Trust

## Understanding Anorexia Nervosa

There is limited understanding of the processes involved in the development and maintenance of anorexia nervosa (AN) (Strober & Johnson, 2012). Existing treatments show disappointing outcomes and no clearly superior model of treatment (Hay, Claudino, Touyz, & Abd Elbaky, 2015). There is therefore an urgent need for novel approaches to understanding and treating AN (Agras *et al.*, 2004; Strober, 2005). It has been proposed that more complex theoretical models are needed as current models lack sufficient explanatory power (Fox & Power, 2009; Strober & Johnson, 2012). There is a place therefore for new theoretical approaches which seek to explain causation, development and maintenance of the disorder.

## Neuro-Biological Models

An underlying genetic contribution to AN is well established (Trace, Baker, Penas-Lledo & Bulik, 2013), yet

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the epigenetic mechanisms of relevance in AN are only beginning to be explored (Kim, Kim, Kim & Treasure, 2014). Hatch and colleagues proposed a disturbance of emotional processing at the non-conscious level with consequent down-stream effects on thinking, feeling and self-regulation (Hatch *et al.*, 2010). Nunn and colleagues describe a noradrenergic dysregulation theory, generating raised anxiety levels and cerebral regional hypo-perfusion. Their focus is on hypo-perfusion of the Insula and likely consequent effects on body-image (Nunn, Frampton & Lask, 2012). Keating (2010) proposed a model of hypoactivity in the anterior cingulate cortex as the basis for altered reward experiences. Lipsman and colleagues subsequently described a broader neuroanatomical model emphasizing dysfunction in key limbic modulatory structures, the insula and the subcallosal cingulate, as the driver of dysregulated emotional processing (Lipsman, Woodside & Lozano, 2015). There is considerable evidence of dysfunction in dopaminergic, serotonergic and noradrenergic systems in the AN population (Kaye, Wierenga, Bailer, Simmons & Bischoff-Grethe, 2013). Other new areas of interest are emerging, such as the role of the oxytocinergic system (Maguire, O'Dell, Touyz & Russell, 2013). However, neuro-biological theories alone are not sufficient to capture the full breadth of this complex disorder.

## Socio-Cultural Models

There are socio-cultural theories for understanding the apparently increasing problem of body dissatisfaction. Objectification theory posits that the prevailing societal ideal of thinness as desirable, conveyed through a variety of socio-cultural means, is internalized and results in dissatisfaction with appearance (Fredrickson & Roberts, 1997). The tripartite influence model proposes that alongside internalization of a thin ideal, appearance comparison is a further key mechanism that can result in appearance dissatisfaction. The three socio-cultural mechanisms of greatest import in these processes are identified as peers, parents and the media (Keery, van den Berg & Thompson, 2004). Riva (2014) integrates objectification theory with an understanding of embodied cognition and social neuroscience, proposing that people who develop eating disorders may become unable to alter and update the misleading way their body is experienced. Further work on integrating socio-cultural theories relating to eating and body dissatisfaction, with psycho-social and neuro-biological models, would appear to be needed.

## Prominent Psycho-Social Models

Psycho-social models have largely focused on approaches within cognitive-behavioural or inter-personal frameworks. Fairburn and colleagues developed their trans-diagnostic model, describing maintaining factors

such as perfectionism, low self-esteem, mood intolerance and interpersonal difficulties as key obstacles to change in any eating disorder (Fairburn, Cooper, & Shafran, 2003). Schmidt & Treasure described a cognitive-interpersonal model, emphasizing perfectionism, experiential avoidance, beliefs about the value of the illness and interpersonal difficulties, as the central maintaining factors (Schmidt & Treasure, 2006). In a subsequent update, they highlight the evidence for obsessive–compulsive traits, underpinned by the cognitive processing styles of poor set-shifting and central coherence; social–emotional processing difficulties producing anxiety and avoidance; and interpersonal difficulties perpetuating the illness (Treasure & Schmidt, 2013). The model described by Corstorphine (2006) places greater importance on secondary emotions such as anger, guilt and shame, yet the treatment interventions proposed within this model remain predominantly cognitive and behavioural, rather than emotion-focused. Wildes and colleagues pursue a similar direction, highlighting emotional avoidance as a central factor in maintaining anxious and depressive symptoms. They emphasize awareness and acceptance of distressing emotions and work on social behaviour as a mechanism for change (Wildes, Ringham, & Marcus, 2010).

## Novel Psycho-Social Models

Those psycho-social theories expanding beyond narrow cognitive-behavioural or inter-personal frameworks could be considered third generation psycho-social models. Fox & Power (2009) take a fundamentally emotion-focused approach, applying a multi-level model of emotions to eating disorders. Their model emphasizes the centrality of secondary emotions such as disgust and anger, and the role of association in emotion generation. When emotion is produced without effortful cognition, this is considered the associative route. This can occur in two ways: evolutionary preparedness for specific stimuli to trigger emotion; or events becoming automatically linked with an emotion through a learning process. They propose increased use in treatment of exposure-driven behavioural experiments alongside work on beliefs at the schema level. The concreteness of the mental lives of people with AN is viewed by Skårderud (2007) as an impairment of reflective function, resulting in a very close relationship between emotional and physical experiences. This theory underpins the mentalization-based therapy model for the treatment of AN, with a focus on improving the ability to understand feelings, cognitions, intentions and meanings in oneself and others. Goss & Allan (2010) emphasize work on emotions, particularly highlighting the shame arising from self-criticism and addressing this by cultivating compassion to access the affiliative soothing system. Their model is rooted in the work of Paul Gilbert in

describing an evolutionary neuroscience model of affect regulation (Gilbert, 2009). Similarly, Pinto-Gouveia and colleagues have proposed a specific model linking the social ranking theory concepts of external shame, insecure striving and social comparison, mediated by self-critical emotion regulation strategies, to produce body dissatisfaction and drive for thinness (Pinto-Gouveia, Ferreira & Daurte, 2014). The radically open dialectical behaviour therapy model for AN also appears to draw on many of the foundations described by Paul Gilbert. Over-controlling behaviour is understood in the context of neurological, physiological and psycho-social factors. This model also posits the centrality of emotional inhibition and control in disturbing the formation of close social bonds (Lynch, Hempel & Dunkley, 2015). Park and colleagues have applied the interacting cognitive subsystems paradigm to generate a maintenance theoretical model emphasizing dysfunctional processing of emotional and bodily experiences. They describe unhelpful modes of mind (Park, Dunn & Barnard, 2012). These broader largely integrative models appear to have some overlapping and common features.

*Schema Therapy Model*

Schema Therapy offers a theoretical understanding of AN rooted in the concept of universal core needs. It is also an integrative model, built on cognitive-behavioural, attachment, object relations, gestalt and mindfulness theories and techniques. There is a focus on working with vulnerable emotional states, underlying schema's and psychological coping modes, and building a trusting therapeutic relationship as the primary means of effecting change. The Schema Therapy model has been shown to be highly effective in large clinical trials amongst both Borderline and Cluster C Personality Disorders (Bamelis, Evers, Spindhoven & Arntz, 2014; Giesen-Bloo *et al.*, 2006), both highly prevalent co-morbid conditions amongst patients with AN. We have adapted the Schema Therapy model for work with patients with severe AN (sAN) over the last 8 years, integrating further ideas from neuroscience, physiology and theories of emotion (Munro *et al.*, 2014). Simpson and colleagues have developed and are testing further a group Schema Therapy model (Simpson, Morrow, van Vreeswijk & Reid, 2010). A review identified the potential for use of the Schema Therapy model in the treatment of eating disorders (Pugh, 2015), and a recent RCT of a form of Schema Therapy demonstrated equivalent efficacy to CBT amongst sufferers of binge eating problems (McIntosh *et al.*, 2016).

*The Case for Integrative Models*

In clinical practice, a relatively narrow range of largely cognitive-behavioural models of therapy dominate. Even those models which have incorporated aspects of the cognitive neuroscience to their therapy models have shown disappointing results in recent RCTs (Schmidt *et al.*, 2015). Whilst there is increasing integration of ideas from across the traditional bio-psycho-social boundaries, our review of the literature found no fully integrative bio-psycho-social model of AN. Given the apparently limited benefit of the current models of treatment for people with AN, we suggest there is a case for re-examining biological, emotional and psycho-social theoretical concepts of potential relevance to AN. The aim of this paper is to describe theoretical ideas and concepts of relevance to the maintenance and treatment resistance of AN, and the bio-psycho-social model arising from the integration of these ideas with the clinical presentations of our patients, with the intention of further developing our clinical model. Although we will explore ideas of relevance to the development of AN, our primary focus is on understanding maintenance and treatment resistance.

## METHOD

The methodology reflects a broad and inclusive approach to exploring and integrating biological, social and psychological ideas and concepts. We used a triangulation approach (Denzin, 1978), seeking multiple perspectives and convergence of ideas relating to the understanding of AN, to lend strength to the validity of a final model. Our methodology was mixed qualitative and quantitative.

We carried out an initial systematic search of the eating disorders literature. Electronic searches of PsycINFO, MEDLINE and EMBASE databases, for the years 1990 onward, were undertaken using the searches: *anorexia nervosa OR eating disorders AND theory OR model.* We generated clinical hypotheses from our qualitative clinical experience, about the functions of the illness and the common maintenance factors. This informed further targeted searching of relevant areas in the wider bio-psycho-social literature. Additional papers were sourced from prior reviews and hand searching of key eating disorders journals. A systematic search of the eating disorders literature was last repeated August 2015. We synthesized those ideas and concepts which were commonly reflected amongst our patient group and for which there was strong supporting evidence within the literature.

## RESULTS

The key ideas and concepts arising from this methodology and their development and synthesis into a clinically meaningful framework are described below.

## Core Needs

### The Development of Core Needs Research

Maslow (1962) described a hierarchy of human needs as a means to describe human motivation and behaviour. This hierarchy included physical and psychological needs, and he asserted that all human beings are intrinsically motivated to meet these needs. Subsequent needs and motivation research has questioned the hierarchical structure proposed by Maslow but developed needs concepts further.

Ryff and Keyes (1995) described a model of universal psychological needs with six concepts: autonomy; environmental mastery; personal growth; positive relations with others; purpose in life and self-acceptance. Three key psychological needs for competence, autonomy and relatedness, and their relationship with motivation, were described by Deci and Ryan (2008). Tay and Diener (2011) recognized the substantial commonality in the concepts described by these theorists and others (Baumeister & Leary, 1995; Csikszentmihalyi & Csikszentmihalyi, 1988). They described six universal needs concepts: basic needs for food and shelter; safety and security; social support and love; feeling respected and pride in activities; mastery; self-direction and autonomy. This model returns to Maslow's recognition of both physical and psychological needs. They examined evidence of the relationship between these needs and subjective well-being within the Gallup World Poll of 60 865 individuals. Their analysis provides cross-cultural empirical evidence for universal core needs that relate directly to wellbeing.

Universal psychological needs are the cornerstone of the Schema Therapy psychotherapeutic model developed by Young and colleagues (Rafaeli, Bernstein & Young, 2011). They describe an expanded set of 10 core needs, for use in a psychotherapeutic context. These are safety, stability, nurturance, acceptance, autonomy, competence, sense of identity, freedom to express feelings, spontaneity and play, and realistic limits. They argue that individuals will vary in terms of the strength of each need, but, that all needs must be met to a minimum level to achieve psychological health.

As AN is a severe psychological and physical illness, to understand it we must consider physical needs. Maslow acknowledged physical needs as the base of his hierarchy. Tay and Diener (2011) describe that basic physical needs tend to be prioritized over other needs. This happens almost automatically for most people, yet in people with AN physical needs are neglected to an extreme degree. This form of self-neglect is a distinctive feature of the illness. As is only too apparent to those who work with patients with sAN, psychological needs can only be addressed if at least the bare minimum of physical need can be maintained to avoid death.

A needs-based understanding of human motivation and behaviour has been applied to other psychiatric populations from forensic to depressed patients, with recognition that behaviour motivated by needs can be aberrant, anti-social or destructive (Roychowdury, 2011; Zuroff et al., 2007). Self-destructive behaviour, which appears to go against basic needs, is a central feature of AN.

### A Needs Framework for Severe Anorexia Nervosa

We therefore developed a basic needs framework, encompassing both physical and psychological needs, to apply a needs-based understanding of behaviour to patients with AN. This represents a composite of the needs research described above, expressed in terminology relevant to patients with AN, and similar in conceptualization to that described by Tay and Diener (2011) and Young and colleagues (Rafaeli et al., 2011). The four core psychological needs in our framework are: *Emotional Safety, Control and Competence, Nurturance and Acceptance.* The four physical needs are: *Physical Safety, Activity, Nutrition and Rest* (Table 1). To understand how the needs system fails to operate adequately in patients with AN, we

Table 1.  Core Needs Framework in Anorexia Nervosa.

| Psychological Core Needs | Physical Core Needs |
|---|---|
| *Emotional Safety*[1]: The need to *feel* safe and secure enough | *Physical Safety*[3]: The need for enough warmth, shelter and protection from physical harm |
| *Control & Competence*[2]: The need to feel 'in control' enough, have a sense of autonomy and competence | *Activity*[2]: The need for enough movement and activity |
| *Nurturance*[3]: The need to *feel* loved and cared for enough, by others and by yourself | *Nutrition*[3]: The need for enough food and fluids |
| *Acceptance*[2]: The need to *feel* accepted enough, for your strengths and fallibility, by others and by yourself | *Rest*[3]: The need for enough rest and sleep |

[1]Excessive prioritisation of the need to feel safe above all other needs.
[2]The partial, dysfunctional and inadequate meeting of needs, in the pursuit of feeling safe enough.
[3]The marked neglect or denial of some needs.

must first consider the neurobiological, physiological and psychological systems that normally drive and motivate humans to behave healthily and adaptively to meet their core needs.

### The Function of Feelings

*Theory of Emotions*

Despite a multiplicity of theoretical models for understanding emotions and emotion regulation, there is some broad agreement amongst emotion researchers. Emotions are functional, focussing attention or motivation to generate intra-personal or inter-personal responses and consequent behaviour (Izard, 2010). A group of theoretical models relating to emotions offer conceptual ideas or frameworks which resonated with our clinical work and are described in the following sections.

*Feelings as Indicators of Needs*

We adopt a broad concept of 'feelings' as physical or emotional feelings. These feelings are the responses to internal and external triggers, operating as crude 'signals' reflecting underlying needs. This conceptualization is consistent with the accepted view of emotions as functional. Many theories also acknowledge the physical or physiological component of emotions (Porges, 2007; Gross & Jazaieri, 2014). The Somatic Marker Hypothesis of Damasio (1996) in particular emphasizes that somatic 'signals' arising within the body or the brain help generate dispositional states including emotions, which facilitate decision making, based on past experience and the present situation. The Communicative Theory of Emotions similarly describes the idea of signalling, in defining emotions as '*signals that set body and mind into modes that have been shaped by evolution and individual experience to prompt a person towards certain types of action*' (Oatley & Johnson-Laird, 2011). Prevailing models of emotion acknowledge both fast or slow and unconscious or conscious processing, referred to as 'dual-process' theories, i.e., fast, crude, largely unconscious 'rapid response' signals and relatively slow, largely conscious, more considered signalling activity (Oatley & Johnson-Laird, 2011).

There is therefore acceptance that emotions are operating at an unconscious and a conscious level. This can be defined within a neuro-biological or a psychological framework in various ways. Unconscious can be described as automatic mental activity, stimulated by external environmental or internal visceral cues, via the brainstem, occurring in less than 200 ms (Williams *et al.*, 2008). Alternatively, an enduring psychological definition of the unconscious, as outside of awareness but influencing the construction of conscious content, remains valid (Helmholtz, 1962/1866). From a clinical perspective, an acknowledgement of unconscious processes driving

aberrant behaviour can temper self-criticism and shame, so prominent in this population. As such, with patients, we use the labels of 'drives' to describe the unconscious emotional processes and 'feelings' to denote the emotion-related mental activity available for conscious reflection. So 'feelings', be they physical or emotional, are the conscious awareness of underlying 'drives', triggered by the internal or external environment, reflecting needs and resulting in motivation for action.

As an individual's experience of their feelings may be relatively rapid and habitual, to understand the function of them within a verbal psychotherapeutic relationship, patients need a simple enough model of feelings and core needs to aid reflection. Without reflection on outcomes, the patient struggles to identify how their feelings may be misleading them. The exploration of the origins and function of aberrant feelings and drives during earlier life experiences can facilitate an acceptance of the 'good reasons' why certain coping strategies emerged, but have become maladaptive. Without this functional reflection on feelings, or with an overly cognitive or 'rational' approach, patients with AN often conclude that they are simply inadequate and deficient because they are acting irrationally.

What we seek to add for clinical use to existing theory is an understanding of the functional connection between feelings and core needs. For example, a feeling of fear in the presence of another person threatening violence is an emotional indicator of both the need for physical safety and the need for emotional safety, i.e., to *be* safe and to *feel* safe. A feeling of hunger is usually a physical signal of the need for nutrition but can be an emotional indicator of the need for emotional safety through self-soothing, i.e., 'comfort eating'. Vulnerable or negative physical feelings (e.g., pain, cold, hunger) and emotional feelings (e.g., anxiety, anger, fear) reflect unmet core needs. These usually motivate an individual to act to try to meet that need. Safe or positive physical feelings (e.g., fullness, comfort, warmth) and emotional feelings (e.g., calm, confidence, excitement) reflect met needs. These usually motivate efforts to retain the met need, although in the context of psychopathology, this may become distorted and a motivation to perpetuate or amplify a negative feeling may also occur (Fernandez, Jazaieri & Gross, 2016). We will explore further below an understanding of the systems that process physical and emotional feelings and result in motivation for particular behaviour.

### Current Evidence of Unhealthy Feelings Processing in AN

*Healthy and Unhealthy Processing of Feelings*

Regardless of which theories of feelings we adopt, we suggest that there is sufficient agreement about the

functional role of feelings to distil a broad description of healthy and unhealthy processing of feelings, in relation to met or unmet core needs.

Healthy functioning of the feelings processing systems leads to the meeting of core needs and a prospering, healthy individual. This occurs with the development of a repertoire of behavioural responses, and therefore the ability to adapt to changing circumstances. Feelings are unconsciously and consciously processed, resulting in motivation to behave in adaptive ways. An individual learns with experience how best to relieve vulnerable feelings and achieve or retain positive rewarding feelings. In a healthy person, even the conscious processing occurs in a semi-automatic way as they can trust their feelings to guide their behaviour in most circumstances and achieve a 'good enough' outcome. Conversely, unhealthy functioning of the feelings processing systems leads to unmet core needs and an unhealthy individual struggling to cope.

## Unhealthy Processing of Feelings in Anorexia Nervosa

There is abundant evidence of problems with feelings and feelings processing from the eating disorders literature. Negative emotional states such as anxiety, fear, shame, guilt, disgust, anger and depression are dominant in AN (Bruch, 1973; Engel et al., 2005; Fox et al., 2013; Geller, Cockell, Hewitt, Goldner & Flett, 2000; Goss & Gilbert, 2002; Keith, Gillanders & Simpson, 2009; Kelly, Carter & Borairi, 2014; Waller et al., 2003). Hatch and colleagues (2010) described disturbance of the neural processing of emotions in patients with AN, both before and after weight gain, suggesting that this is not simply secondary to starvation. Oldershaw and colleagues (2011) reviewed studies of socio-emotional processing in AN. They described heightened sensitivity to and attention bias towards threatening stimuli, such as critical and dominant facial expressions. They also described poor recognition of emotional states in others and of emotional recognition within the self. Three studies have specifically tested patients weight restored after in-patient treatment (Haynos, Roberto, Martinez, Attia & Fruzzetti, 2014; Tchanturia et al., 2004; Moser et al., 2003) with no association found between weight gain and improved deficits relating to emotional recognition or regulation. Two studies have specifically shown that lower BMI was actually related to better emotion regulation (Brockmeyer et al., 2012; Racine & Wildes, 2013). Taken together, these studies describe emotional processing and regulation deficits and that starvation is not the central cause. Improvement in cognitive, perceptual or socio-emotional deficits in recovered patients, therefore, seems likely to largely reflect a psychological process of change rather than simply a physical recovery. There is also physiological evidence suggesting unhealthy processing and regulation of feelings, with hypothalamic–pituitary–adrenal axis overactivity in the AN population, resulting in persistently raised Cortisol levels and reflecting an overactive stress response (Connan, Campbell, Katzman, Lightman & Treasure, 2003; Kaye, 2008; Lawson et al., 2012; Monteleone, Scognamiglio, Monteleone, Perillo & Maj, 2014).

## Psychological Systems of Feelings Processing and Regulation

To help illucidate potential mechanisms underlying this dysfunctional processing and regulation of feelings in AN, we will explore the experience of our AN patients through the lens of three key models of psychological processing (Fox & Power, 2009; Gilbert, 2009; Sheppes, Suri & Gross, 2015).

### Gilbert Model

The psychological model of processing of feelings, described by Paul Gilbert (2009), informed by widely accepted theories of emotions (LeDoux, 1998; Panksepp, 1998) provides a functional and evolutionary framework. The three systems described are the threat and self-protection system; the incentive and resource seeking, drive–excitement system; and the soothing, contentment and safeness system. The threat and self-protection system detects dangers and responds by motivating 'fight', 'flight' or 'freeze' (submission) responses. From an evolutionary perspective, the brain prioritizes response to danger over other things, with complex thinking switched off in favour of rapid action (Liotti & Gilbert, 2011). This is a combination of unconscious response and sub-conscious semi-automatic responses, if we become aware of feelings indicating an unmet need for safety. For convenience, we will refer to this as the 'threat–protection' system. The incentive, resource seeking, drive–excitement system functions to respond to unmet needs by activating a desire to strive and seek rewards. It is a 'doing' and energizing system that rewards through feelings of achievement and pride. It has less evolutionary priority than the threat–protection system. We will refer to this as the 'drive–achieve' system. The third system is the soothing, contentment and safeness system. It has a crucial function in positive socialization and connection with others, motivating behaviour that supports the development of relationships for mutual benefit. It generates an experience of contentment, calm and self-acceptance. We will call this the 'calm–connect' system. The social and inter-personal aspects of emotion regulation, including the Social Mentality Theory (Liotti & Gilbert, 2011), will be explored further in the social behaviour section.

Adopting the lens of the Gilbert model, what do we find in AN? The 'threat–protection' system and the 'drive–achieve' system dominate, and the 'calm–connect'

system is rarely accessed. This is reflected in vulnerable negative emotional states, driven behaviour, pride in the achievement of thinness and a lack of calm, secure or safe feelings (Aldao, Nolen-Hoeksema & Schweizer 2010; Brockmeyer *et al.*, 2012; Connan *et al.*, 2003; Fox & Power, 2009; Harrison, Sullivan, Tchanturia & Treasure, 2010; Haynos & Fruzzetti, 2011; Lawson *et al.*, 2012; Nunn, Frampton & Lask, 2012; Oldershaw *et al.*, 2011; Park, Dunn & Barnard, 2012; Racine & Wildes, 2013; Svaldi, Griepenstroh, Tuschen-Caffier & Ehring, 2012). The negative emotional states such as anxiety, fear, shame, guilt, disgust, anger and depression are largely a product of the 'threat–protection' system. The driven perfectionistic, controlling behaviour, associated with pride in achievement is a product of the 'drive–achieve' system (Goss & Allan, 2009; Skårderud, 2007b). The marked difficulties in achieving states of calm and in developing trust in relationships with others reflect a lack of access to the 'calm–connect' system (Ringer & Crittenden, 2007; Treasure & Schmidt, 2013; Pinto-Gouveia *et al.*, 2014).

### Extended Process Model of Emotion Regulation

The Extended Process Model of Emotion Regulation offers a cognitive and behavioural framework for the conscious regulation of feelings (Sheppes, Suri & Gross, 2015). This defines emotion dysregulation as failure to engage in regulation or mis-regulation. Using this model, we can identify the stages in conscious emotion regulation which go awry amongst AN sufferers. Patients with AN rarely have difficulties engaging in regulation, being highly attuned to negative feelings, but mis-regulate them in many ways. The model describes four stages where mis-regulation may arise: stage 1, identifying feelings that need regulation; stage 2, selecting a strategy; stage 3, implementing the strategy; stage 4, monitoring the implemented strategy over time and adjusting if necessary. Dysfunction occurs at each stage in AN.

Poor recognition of emotional states within the self and in others is well described (Oldershaw *et al.*, 2011) reflecting an identification problem at stage 1. This is reflected in physical feelings such as hunger as much as emotional feelings. For example, our patients often express doubt and uncertainty about whether they feel hungry or not. The spontaneous selection of an appropriate strategy for managing feelings is profoundly impaired at stage 2. The habitual AN responses to negative feelings are over-control or emotional detachment. With high levels of anxiety, the instinctive drive to meet the short-term need to feel safe is not entirely maladaptive, but is maladaptive in the magnitude of response. At stage 3, a lack of cognitive flexibility certainly impairs implementation. We experience patients who at a cognitive 'rational' level have made a decision to change, learn how to respond differently to their feelings, but apparently cannot

implement this regularly enough to establish new habitual patterns of response. In patients trying hard to recover this can generate intense self-criticism about being 'irrational'. At the final stage, difficulty in monitoring, reflecting and adjusting maladaptive mechanisms is a clear problem amongst AN patients. The short-term prioritization of the need to feel safe is intense, despite clear negative medium and long-term consequences such as physical problems relating to starvation, loss of social and occupational roles, persisting anxiety and fear. The hyper-emotion theory (Johnson-Laird, Mancini & Gangemi, 2006) and the evolutionary prioritization of threat response (Liotti & Gilbert, 2011) offer explanations for why over-whelming anxiety may block the conscious reflective monitoring processes.

### SPAARS Model

The SPAARS multi-level model of emotion, applied to eating disorders, has been specifically described (Fox & Power, 2009). We have explored this with reference to our clinical experiences with AN and the concept of core needs.

Fox and Power (2009) hypothesized that feelings can become aberrantly 'coupled' by becoming linked and working in a facilitatory or inhibitory way. They describe how the basic emotion of disgust can become aberrantly coupled with the basic emotions of anger, fear and sadness and generate secondary emotions of shame and guilt. They hypothesize that anger is coupled with disgust, as a dysfunctional means of managing this emotion by converting it into disgust located in the body, which in turn is perceived as more controllable (Fox *et al.*, 2013). They describe a concept of 'locking' which biases incoming information and keeps the sufferer locked in a default mode of experiencing and dysfunctionally regulating their emotions.

This idea can also be applied to the coupling of physical and emotional feelings. If for example the physical feeling of hunger is coupled with a feeling of anticipation of pleasure, the nutritional need will be met by motivating eating behaviour. However, if the feeling of hunger becomes linked with feelings of shame, because the hunger has been labelled 'unacceptable', then the motivated behaviour is directed towards relieving the shame associated with feeling hungry rather than the hunger itself. The hungry feeling is ignored, suppressed or denied as it is too shameful to accept. The consequence is that the true underlying need for nutrition is not met. The vulnerable feeling may then be partially offset by the striving to control that feeling, giving some sense of satisfaction or pride, through the 'drive–achieve' system. So, the final linkage of feelings and behaviour may be: hunger – shame – striving to control feelings – pride or relief; yet the underlying need for nutrition remains unmet and inevitably the hunger returns.

Calm, secure, restful feelings may also become an aversive experience, through an aberrant coupling with secondary feelings of shame. If for example the physical feeling of muscle relaxation is linked with feelings of calm or security, then the physical core need for rest can be met. If, however, the feeling of muscle relaxation is labelled at a cognitive level as 'lazy' and secondarily linked with shame, then the drive is for activity instead of rest. So the unhealthy linkage may be: muscle relaxation – 'lazy' – shame – motivation to do something – pride or relief, yet the underlying need for rest is not met. We suggest that this aberrant coupling of vulnerable, calm or angry feelings, with shame, guilt or disgust, results in failure of the feelings processing system to produce behaviour that meets core needs. We hypothesize that this occurs, at least in part, through the substitution of the 'calm–connect' system with the 'drive–achieve' system. This provides only a partial response to the underlying needs, yet it is partially rewarding, satisfying the need to feel safe and acceptable in the short term. This is understandably valued and crucially contributes to the maintenance of the disorder.

## Physiological Systems of Feelings Processing and Regulation

The physiological systems for processing feelings are closely integrated with the psychological systems (Uvnäs-Moberg, Arn & Magnusson, 2005). To explore physiological mechanisms of potential relevance, we will explore three theories and relate these to our clinical experience.

### Polyvagal Theory

The Polyvagal Theory describes how the three circuits of the autonomic nervous system control physiological drives and consequent behaviour, and are ordered in terms of evolutionary emergence (Porges, 2007). The most primitive circuit is that of the unmyelinated vagus nerve, associated with a physiological drive resulting in immobilization behaviours, e.g., playing dead, vaso-vagal syncope or behavioural shut-down. This is associated with the 'freeze' response of the psychological 'threat–protection' system. The next circuit to evolve was that of the hypothalamic–pituitary–adrenal (HPA) axis, central to the Sympathetic Nervous System (SNS), and associated with a physiological state supporting mobilization behaviours of 'fight–flight' or activity. This is similarly associated with the 'threat–protection' system but also the 'drive–achieve' system. The third circuit is that of the myelinated vagus nerve, the Parasympathetic Nervous System (PNS), associated with a very different physiological state supporting rest, recovery and social communication behaviours (e.g., engaging facial expressions, calm responses, listening). This is directly linked with

the psychological 'calm–connect' system. The polyvagal theory proposes that these three systems operate in an evolutionary hierarchical manner. The newer myelinated vagus inhibits the older circuits, acting as the 'vagal brake' inhibiting SNS activity. If the myelinated vagus of the parasympathetic circuit is malfunctioning, the older circuits automatically become more active. So, if the calm–connect system is failing, as we suggest it does in AN, a physiological shift occurs with increased activation of the SNS, the threat–protection and drive–achieve systems. In other words, a greater sense of threat leads to increased vigilance and activity with less rest and socializing.

We certainly experience patients unable to access states of rest and calmness, associated with myelinated vagus nerve activity. The dominance of activity in the HPA axis, reflected in raised cortisol levels and excessive arousal in response to circumstances, reflects dysfunctional physiological processing. Clinically, some patients present with tachycardia and raised blood pressure, despite the homeostatic down-regulation in starvation operating in the opposite direction, which can be best explained by persistent dominance of SNS activity.

### Other Models of Emotion Processing Relating to Physiology

Reflecting back to the somatic marker hypothesis (Damasio, 1996), the somatic experience of an overactive SNS reflects a 'dispositional state' anticipating threat when there is none, reflecting a failure of the physiological processing of feelings, as Porges suggests. We may also draw on the Hyper-Emotion Theory of psychological illness proposed by Johnson-Laird *et al.* (2006). This proposes that beyond problems in emotion regulation, psychological illness may be caused by hyper-intense emotion. The somatic experience of an overactive stress response in AN may reflect a causal excess of negative emotion generation, as the hyper-emotion theory would suggest (Johnson-Laird *et al.*, 2006), or a failure in processing or regulation, as the Polyvagal theory (Porges, 2007) and Somatic Marker Hypothesis (Damasio, 1996) hypothesize.

### Maladaptive Physiological Mechanism of Feelings Regulation

We also propose that rather than being the central cause of the emotion regulation problems seen in AN, as is frequently assumed, starvation and over-activity, may in fact represent maladaptive physiological mechanisms for regulating a dysfunctional autonomic nervous system, explaining the findings of Brockmeyer and colleagues (2012) and Racine & Wildes (2013). A homeostatic down-regulation occurs in persistent starvation in order to conserve energy (Keys, Brozek, Henschel, Michelson & Taylor, 1950). This blunts some elements of the SNS

response by reducing heart rate, blood pressure and body temperature and may therefore reduce the somatic aspects of anxiety. Intermittent hypoglycaemia due to fasting may facilitate a temporary but rewarding experience of detachment or depersonalization. Protein deficiency in persistent starvation can cause myelin sheath damage and neuropathy (McLoughlin *et al.*, 1998) reducing bodily sensory feedback and adding to a desired experience of 'numbness'. Nandrino and colleagues found a dissociation between cognitive and physiological response to emotional stimuli in patients with AN and suggest that this may be a mechanism for dampening psychological stress responses (Nandrino *et al.*, 2012). Nunn and colleagues proposed a mechanism for the short-term reduction in cortisol production and SNS activity during periods of intense food restriction (Nunn *et al.*, 2012), again potentially dampening the stress response. Furthermore, adaptation to a persistently reduced body temperature can reduce SNS activity and stimulate PNS activity (Mäkinen *et al.,* 2008). We also know that exercise can directly increase PNS activity (Hamer & Steptoe, 2007) which may account in part for the drive for over-activity in AN patients.

So considering these physiological starvation related mechanisms as a whole, there may be a reduction in SNS effects and an increase in PNS activity due to starvation and over-activity. We hypothesize that the dangerous maladaptive drives for both starvation and over-exercise temporarily alter the balance in the autonomic nervous system in a maladaptive drive to counteract the underlying excess of SNS activity, providing relief from anxiety, uncomfortable physical feelings and a sense of threat. This maladaptive physiological feelings regulation hypothesis is consistent with the experience of our patients with sAN who universally describe a desired experience of reduced anxiety, through numbness and detachment, due to food restriction and over-activity.

## Neurobiological Dysfunction

People who develop AN may have predisposing genetic or environmental vulnerability factors contributing to the maintenance of their illness. These vulnerabilities may be specific genetic profiles (Trace *et al.*, 2013), epigenetic phenomenon reflecting the effects of adverse experience on genes (Kim *et al.*, 2014) or simply experiences leading to dysfunctional learning. Given the limits of our current knowledge, a diathesis-stress model seems a reasonable way to understand the interaction of predisposing vulnerability with subsequent life experience. There is certainly good evidence of disruption to serotonergic, dopaminergic and noradrenergic neurotransmitter systems. The specifics of this dysfunction are not well delineated, yet effects on mood, impulse control, satiety, reward,

obsessionality, stress response and body-image have all been attributed to specific neurotransmitter systems (Kaye, Fudge, & Paulus, 2009; Kaye *et al.*, 2013; Nunn *et al.*, 2012). Similarly, there is clear evidence of changes in various central and peripheral appetite modulators, particularly leptin, ghrelin, brain derived neurotrophic factor and endocannabinoids. There is significant evidence these modulators have homeostatic effects in relation to starvation (Monteleone & Maj, 2013).

Functional abnormalities are also evident from fMRI studies. Dorsal corticostriatal circuits, involved in executive control (including the dorsal caudate, dorsolateral prefrontal cortex and parietal cortex), have been shown to be overactive in fMRI studies amongst sufferers of AN. Within limbic ventral corticostriatal circuits, involved in processing the reward value of emotionally significant stimuli (including the amygdala, anterior insula, parietal lobe, anterior ventral striatum, ventral anterior cingulate cortex and orbitofrontal cortex), there is also significant dysfunction, such as hypo-activation of the ventral anterior cingulate-striato-thalamic loop involved in motivation-related behaviour (Zastrow *et al.*, 2009; Kaye *et al.*, 2009). However there is also evidence of differential activation of these circuits, with increased reward related activation in response to starvation-related stimuli (Fladung *et al.*, 2010). Appetite modulators such as leptin and ghrelin may also have an important role in reward processing (Monteleone & Maj, 2013).

The fMRI evidence has led to a hypothesized inhibitory function for the executive control circuits, as a means of modulating the excessive anxiety generated by dysfunction in limbic circuits amongst sufferers of AN (Kaye *et al.*, 2013). Lipsman and colleague's review and model of AN neurocircuitry place emphasis on the key limbic modulatory functions of the insula and the subcallosal cingulum (Lipsman *et al.*, 2015). They focus on dysfunctional integration by these key modulatory structures, of the 'top-down' inputs from cortical structures and the 'bottom-up' inputs from sub-cortical structures, resulting in disrupted emotional processing and, in consequence, the symptoms and behaviour of AN.

This evidence of genetic, neurotransmitter, appetite modulation and neurocircuitry dysfunction in AN is consistent with the psychological and physiological ideas and concepts we have described so far in this paper. The key executive control and limbic circuits, including the modulatory regions of the insula and subcallosal cingulum, highlighted by Lipsman and colleagues, can usefully be considered alongside the psychological and physiological systems, as alternative ways of understanding and describing the unbalanced feelings processing that leads to the maladaptive behaviour seen in AN.

## Oxytocinergic System in AN

Multiple neurotransmitter systems and neural circuits are clearly implicated in generating the symptoms and experiences of AN. Some of the processes involved are emerging, as described above. We propose that adding an understanding of the oxytocinergic system has the potential to enhance existing models.

Oxytocinergic dysfunction has been shown in the AN population, including reduced CSF oxytocin levels (Demitrack *et al.* 1990); reduced peripheral nocturnal oxytocin secretion (Lawson *et al.*, 2011); hypoactivation of the insula and food motivation neurocircuitry (Holsen *et al.*, 2012) and increased methylation of the oxytocin receptor gene (Kim *et al.*, 2014). It is well established that the hypothalamic–pituitary–adrenal axis is overactive in the AN population, resulting in persistently raised Cortisol levels (Connan *et al.* 2003; Kaye, 2008; Lawson *et al.*, 2012; Monteleone *et al.* 2014). Nunn and colleagues (2012) described the dysregulation in the noradrenergic system amongst patients with AN as an explanation for this. We hypothesize that the abnormal development of the oxytocinergic system may also be important, as has been demonstrated in children with high levels of stress and unmet emotional needs (Wismer Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005) This could result in social experiences with others becoming aversive from an early age. For example, interactions with others would generate vulnerable emotions, be more likely to be experienced as stressful or even threatening and less likely to be experienced as calming or soothing. In health, higher levels of oxytocin are associated with decreases in stress hormones such as cortisol and increases in trust, positive social interactions and attachment (Grippo, Trahanas, Zimmerman, Porges & Carter, 2009). Oxytocin also appears to promote plasticity with new learning (Dölen, 2015) or conversely, a central oxytocin deficit would contribute to rigid behaviour and impaired ability to change, as we see in AN. Persistent raised cortisol levels also have the effect of impairing plasticity, social adaptation and emotional regulation (Sapolsky, Romero & Munck, 2000).

Nunn and colleagues (2012) in addition to describing the adverse effects of noradrenergic dysregulation highlighted the potential role of Insula hypo-activity in AN. This may disrupt the integration of feelings and thoughts, as well as the internal experience of self-awareness, the automatic awareness and interpretation of one's bodily feelings, including appetite. The oxytocinergic system has an important role in the Insula (Leng *et al.*, 2008). Such Insula dysfunction may result in an experience of one's physical and emotional internal world as at best uncertain or confusing, and at worst actively threatening. Although the Insula seems likely to be of greater import, the Anterior Cingulate Cortex (ACC) is also amongst the limbic structures of potential importance in AN (Lipsman *et al.*, 2015) and is a region of high oxytocin receptor density (Boccia, Petrusz, Suzuki, Marson, & Pedersen 2013). It has a role in decision making. Moral hypersensitivity and an obsession with 'doing the right thing' and avoiding mistakes are commonly seen in patients with AN. Preckel and colleagues (2015) demonstrated that oxytocin reduced activation in the ACC, reduced arousal and increased acceptance, during a moral decision-making task.

Oxytocin appears to function centrally largely as a neuro-hormone, but also acts peripherally with a wide range of effects (Veening, de Jong & Barendregt, 2010). The effects of oxytocin in healthy populations of direct relevance to AN include: increased ability to infer the mental states of others from facial expressions; willingness to accept social risks; enhancing PNS modulation of sympathetic arousal; reduced anxiety; a complex central and peripheral role in satiety; effects on pain modulation; stimulating of sexually intimate behaviour and promoting of social affiliative behaviour (Veening *et al.*, 2010; Maguire, O'Dell, Touyz & Russell, 2013; Gamer & Büchel, 2012). These are all areas of known difficulty amongst the AN population.

The use of oxytocin has been shown to reduce anxiety (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). There is also a growing experimental literature in clinical populations, particularly amongst the autistic spectrum population amongst whom socio-emotional deficits have been described which have similarities with the AN population. Two small experimental studies in AN patients have explored the effect of Oxytocin on attention biases and response to facial expressions in patients with AN (Kim, Kim, Park, Pyo & Treasure, 2014; Kim *et al.*, 2014b). A single dose of Oxytocin did reduce selective attention towards food and fat body shape images in the AN group and reduced selective attention to disgust facial expressions. Two studies have also now found a correlation between the oxytocinergic dysfunction and severity of illness in AN. An association was demonstrated between peripheral oxytocin levels and severity of disordered eating psychopathology (Lawson *et al.*, 2012), and a similar association was found between severity of eating disorder psychopathology, anxiety, BMI and increased methylation of the Oxytocin receptor gene (Kim *et al.*, 2014).

We propose therefore that oxytocinergic dysfunction is a possible underlying neurobiological mechanism that could account for some of the symptomatic patterns and psycho-social dysfunction seen in AN, including the overactive stress response, the distorted body-image and the difficulties in developing trusting relationships. Much of this may relate to dysfunction in the limbic ventral cortico-striatal pathway, such that sufferers of AN misperceive their internal world (bodily and emotionally) and the external world, as threatening. The overactivity in the dorsal cortico-striatal pathways can be seen as a

compensatory over-controlling executive function response, employing the cognitive strategies of planning and worrying to overcome fear or uncertainty. This acts as a self-protective response to the experience of threat and insecurity generated by the limbic system. This initially partially succeeds with a temporary reduction in anxiety, but ultimately fails with a reinforcement of fears and therefore an increase in anxiety. Existing data suggests an association between oxytocinergic dysfunction and the severity of clinical presentation. This is consistent with a diathesis-stress model and suggests that if it was possible to correct a central oxytocin deficit this may have the greatest impact on those patients with the most severe disorders.

### Neurobiological Dysfunction, Feelings Processing and Core Needs

The unhealthy psychological and physiological processing of feelings in patients with sAN are consistent with the neurobiological dysfunction described above. For example, the dominance of vulnerable feelings, the difficulty in accessing calm and soothing states, and the over-active stress response are all consistent with evidence of limbic system dysfunction, an overactive HPA axis and an underactive oxytocinergic system. The overactive 'drive–achieve' system is also consistent with evidence of over-active compensatory executive function activity. The possible rewards of physiological adaptation to starvation are also consistent with an oxytocinergic deficit. With reduced central oxytocin to activate the PNS, the myelinated vagus nerve fails to act as a 'vagal break' inhibiting the SNS; hence, alternative mechanisms emerge to dampen the excessive stress response.

Our experiences with sAN patients fit with the neuro-biological dysfunction described. For example, patients often describe the experience of being driven to be active or to eat less, even when they appear to genuinely want to change. The experience of dampened anxiety or 'numbness' from a period of calorie restriction appears universally rewarding amongst people who develop AN. Their difficulties in accessing states of calm or relaxation, except through food restriction or over-activity, are often striking.

### Feelings Processing Conclusions and the 'Feared Self'

#### Feelings Processing Conclusions

We have explored existing evidence of dysfunction in the systems for generating, processing and regulating psychological and physical feelings in patients with AN. Theoretical models and concepts from psychological, physiological and neuro-biological approaches have been considered in relation to this evidence and our clinical experience, to help understand this dysfunctional feelings processing. We conclude that dysfunctional feelings

processing and regulation systems, whether considered in psychological, physiological or neuro-biological terms, generate a misleading experience of internal and external threat, reflected in an excess of vulnerable feelings.

#### Feared Self-Concept

What is the outcome of this experience of excessive vulnerability at the cognitive level? Our experience of exploring early life emotional histories amongst our patients is that the vulnerable feelings become labelled as 'unacceptable' from an early age. As such feelings themselves become a threat to be managed. They are misinterpreted as dangerous, wrong and shameful, and individuals begin to label themselves highly negatively. The cognitive end-point of this unhealthy processing of feelings is a potent set of self-critical shame-based core beliefs, which lie central to the disorder. These are core beliefs about themselves as a person: *I am unacceptable*; *I am too needy* and *I am lazy & greedy*. Also, core beliefs about their emotional and physical feelings: *My feelings are unacceptable; My body is unacceptable.* This view of self becomes accepted as true, or inevitable unless they exercise extreme self-control and hide their 'true' self from others. We conceptualize this as a potent sense of a shameful *Feared Self*, which is so disgusting and unacceptable that it must be controlled and hidden at all costs. We believe such distorted beliefs and sense of self, drive maladaptive physical, psychological and relational coping behaviour. We have described our hypothesis that starvation and over-activity represent a maladaptive feelings regulation mechanism. We describe in the following section our hypothesis that maladaptive psychological coping-modes generate the intra-personal and inter-personal behaviour that constitute and maintain the psycho-social presentation of AN.

### Maladaptive Social Behaviour

#### Motivated Social Behaviour and Attachment

The result of the processing of physical and emotional feelings is motivation to behave in a particular way. The initial response can be sub-conscious and semi-automatic or a conscious choice. Broadly, the motivated behaviour can be: external, inter-personal and social; or internal, intra-personal and individual. This can be considered within the basic behavioural framework of approach and avoidance (Elliot & Thrash, 2002) and has been expressed in the eating disorders literature as 'Push–Pull' (Ward, Ramsay, Turnbull, Benedettini & Treasure, 2000).

Attachment theory provides us with a framework for understanding inter-personal behaviour (Bowlby, 1973). The Social Brain Hypothesis (Hamilton, 1975) suggests higher order social cognition abilities evolved to confer advantage to operating in social groups. The Social

Mentality Theory (Liotti & Gilbert, 2011) describes how competitive, caring, care-seeking, cooperative and mating mentalities motivate us to achieve particular social goals. How such theories inform our understanding of AN is explored below.

To successfully meet needs through inter-personal and social behaviour requires a secure and safe enough connection with others. Early experience with an attachment figure directly managing external threats and modelling healthy internal responses to threat provides a secure safe base from which curiosity and exploration can foster healthy development. Without safeness, complex social cognition such as the ability to infer and think about the mental states of others may not develop well as it is overtaken by rapid 'better safe than sorry' responses (Liotti & Gilbert, 2011). This kind of habitual excessive response to perceived but often absent social threat is important in AN. Avoidance and over-control dampen curiosity and exploration in sufferers of AN, limiting their experience. Fear and insecure attachment amongst AN sufferers impinges on meeting the social goals described in social mentality theory. For example, the competitive mentality becomes maladaptive and dominant, e.g., obsessively seeking to create a good impression of themselves in the minds of others, driven by fear of inadequacy. A failure to establish an internalized secure and safe base from a good enough early attachment experience leaves individuals unable to establish safe enough affiliative non-attachment relationships, key to achieving the social goals of alliance building and cooperation with others.

Attachment is therefore important in understanding the development and maintenance of social inter-personal difficulties in AN. It is an area that has been widely explored in eating disorders, with secure attachment rarely described (Bamford & Halliwell, 2009; O'Kearney, 1996; Ramacciotti et al., 2001; Ward et al., 2000). A review of the attachment literature relating to eating disorders confirms the association with insecure attachment and suggests there may be a relationship between the severity of eating disorder symptoms and attachment insecurity (Tasca & Balfour, 2014). Ringer and Crittenden (2007) describe a rich analysis of attachment in a sample of eating disordered individuals. They described three clusters of insecure attachment styles amongst their AN group. Broadly, cluster 1 involves intra-personal, internally focused behaviour, in order to avoid attachment or reliance on others to meet needs; cluster 2 involves dysfunctional interpersonal, approach behaviour, with desperate efforts to make others pay attention to their unmet needs; cluster 3 involves a mixture of both attachment styles, in other words a complex and seemingly contradictory mix of avoidant and approach behaviour, push and pull.

Ringer and Crittenden's clusters reflect our clinical experience with the majority of patients with sAN. None are securely attached and whilst often one attachment style dominates, both dysfunctional approach and avoidance behaviours are apparent. Our experience is that more severe cases tend to display dominance of the avoidant, internalized, self-reliant attachment style. Another way of conceptualizing this is that the safest attachment is with the internal 'relationship' with their eating disorder rather than interpersonal attachments to others. Hence, the valuing and defending of their eating disorder (Schmidt & Treasure, 2006). As the Social Brain Hypothesis implies (Hamilton, 1975), healthy functioning necessitates the ability to meet needs 'externally' in relationships with others, exercising the higher functions of the social brain, and 'internally' in relation to different aspects of the self. Such disruption of interpersonal attachment and interpersonal affiliative relationships in general, therefore, inevitably leads to unmet needs.

### Dysfunctional Modes and Maladaptive Behaviour in Anorexia Nervosa

The failure of behaviour which adequately satisfies needs leaves the individual needing to find a way to manage their threatening experience of the world. We conceptualize this psychologically in the development of dysfunctional psychological coping modes.

The mode concept is that of different 'parts' of the self reflected in persistent patterns of thoughts, feelings and behaviours (Rafaeli et al., 2011). Maladaptive coping modes develop in an attempt to respond to the core experience of vulnerability. If individuals have a persistent internal experience of vulnerability, which is not soothed and relieved enough through relationships with others, they will inevitably seek other means to feel safe. Some AN sufferers develop modes driving unhelpful 'external' inter-personal behaviour, seeking to get their needs met more effectively by others. Such 'external' focussed modes when active are typically reflected in anger and other-critical cognitions, driving behaviour to force others to treat them differently, to punish them or to push others away. After engaging in these 'angry' modes, there is often shame about behaving unreasonably. If such external inter-personal focussed modes fail, they fall back on 'internal' behaviour, using coping modes in attempts to control or suppress vulnerable feelings. This turn inwards is the dominant coping style in patients with AN. Broadly, there are three types of 'internal' maladaptive coping modes: self-critical and self-punitive modes, associated with feelings of shame about vulnerable feelings, and usually functioning to motivate behaviour to try harder to control or suppress these vulnerable feelings; over-controlling modes which seek to control vulnerable feelings or triggers; and avoidant modes which seek to suppress or self-soothe in response to vulnerable feelings.

We believe that such maladaptive coping modes result in a distorted prioritization of core needs. Consequently, there is an almost complete focus on trying to feel safe,

in control or acceptable, at the cost of other core needs (Table 1). The dysfunctional coping modes often lead to temporary and partial, but inadequate, meeting of needs. Modes which involve the 'drive–achieve' system give a temporary sense of competence and acceptability. For example, attempts to be thinner may temporarily lead to a sense of being more competent and acceptable. This, however, is a partial and highly conditional self-acceptance, which remains only whilst one continues to lose weight. As we have already described, the physiological effects of starvation may also temporarily contribute to a sense of feeling safe. The irony is that the extreme efforts to try and feel safe, in-control, competent and acceptable, result in the opposite outcome. The consequence is a barren life of psychological and physiological self-deprivation, harsh self-criticism, extreme self-control, social isolation and mistrust of others.

## Synthesis and Conclusions

Our aim was to describe ideas and concepts of relevance to understanding the maintenance and treatment resistance of AN. We suggest that the concept of universal core needs, allied with a functional understanding of physical and emotional feelings operating as crude signals of underlying needs, is a valuable framework within which to understand this. There is abundant evidence of dysfunctional feelings processing and regulating systems amongst sufferers of AN. Whether these are conceptualized in neuro-biological, physiological or psychological

terms, this is a key factor. The consequence is an experience of excessive negative vulnerable feelings and of threat, including a felt sense of being too big. Hence, the feelings signalling systems are misleading the individual about their underlying needs. We suggest this experience fosters beliefs about being inadequate, disgusting, needy, greedy and shameful. In short, of being or becoming an unacceptable 'feared self' who will be criticized by others.

We describe maladaptive ways of coping with this experience of vulnerability and inadequacy, in both physiological and psychological terms. We hypothesize that starvation and over-activity function as a maladaptive physiological feelings regulation mechanism. We also hypothesize that maladaptive psychological coping modes emerge to manage uncomfortable emotional and physical feelings. Both physiological and psychological mechanisms provide short-term reward by prioritizing the need to feel safe and acceptable in the short-term, over adequately meeting core needs in the medium to long-term. The maladaptive social behaviour results in insecure inter-personal relationships with a lack of trust, the sufferer increasingly turning to the internal 'relationship' with their eating disorder, social isolation and adoption of anorexia as an identity.

We have therefore described eight key factors in understanding the maintenance and treatment resistance of AN (Figure 1):

1. Genetic or experiential predisposing factors
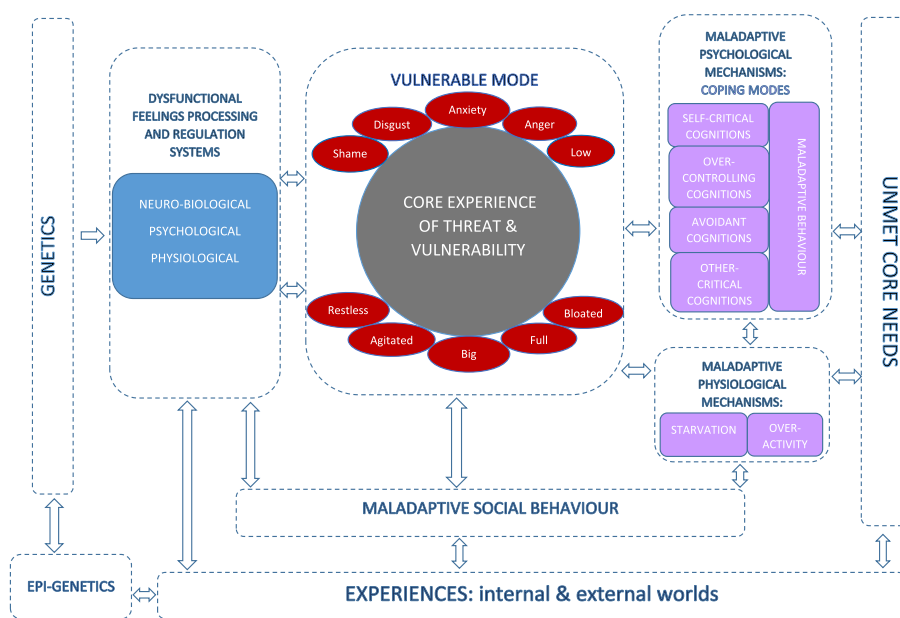2. Dysfunctional feelings processing and regulation systems



Figure 1. Bio-psycho-social maintenance model of anorexia nervosa. [Colour figure can be viewed at wileyonlinelibrary.com]

    a. Neurobiological (oxytocinergic system potentially important)
    b. Psychological
    c. Physiological

3. Excessive vulnerable feelings
4. 'Feared Self' Beliefs
5. Maladaptive physiological feelings regulation mechanism: starvation and over-activity
6. Maladaptive psychological feelings regulation mechanisms: coping modes
7. Maladaptive social behaviour
8. Unmet Core Needs

Below is an example of how this may be reflected in an individual. The core psychological needs to feel safe enough and competent enough are not being adequately met. This generates instinctive vulnerable drives to motivate action. The feelings processing systems generate a misleading experience of excessive vulnerability and threat, with a number of consequences. The powerful need to feel safe in the short-term is prioritized over other needs. An experience of one's vulnerability as meaning I am too needy, greedy, big, disgusting and inadequate generates a belief in an unacceptable 'feared self'. Consequent maladaptive behaviour may be: criticizing oneself about being fat and greedy (Self-Critical Mode) to drive restrictive eating and over-exercising (Over-Controller Mode and physiological starvation effects); avoiding other people and sticking to rigid routines, to cut off from feelings (Detached Protector mode, physiological starvation effects and maladaptive social behaviour); or being dismissive or angry with others expressing concern or encouraging eating (Angry Protector mode & maladaptive social behaviour). The consequence is that maladaptive modes provide short-term relief, temporarily and partially meeting the need to feel safe, competent and 'in control'. However in the medium term emotionally meaningful relationships are disrupted, increasing time is spent on food and weight control and physical health deteriorates. Ultimately, there is a failure to meet the basic psychological and physical needs. Suffering increases and reward decreases. The detailed description of how these elements are conceptualized within a Schema Therapy clinical treatment model, the specific maladaptive coping modes, the numerous maintenance cycles involved and how these are addressed using ST techniques is beyond the scope of this paper and will be published separately.

## DISCUSSION

### The Case for Integration

Our understanding of AN and therefore our treatments for it are not good enough. We suggest that to improve treatments, reductionist research exploring components of the illness must be combined with investigation of integrative models explaining the illness as a whole. Such models should be influenced from a greater understanding of the patient's experience, combined with understanding of relevant biological, psychological and social components and processes. The field often appears stuck in a straight-jacket of reductionist dualities, such as 'brain' versus 'mind' (Harris & Steele, 2014; Schmidt & Campbell, 2013). Understanding AN should not be about brain versus mind, or mind versus body, or cognitive versus emotional, or individual versus societal, but about all of these. This after all, is how the illness is presented to us in the whole form of each individual who arrives in front of us needing help.

### Existing and Novel Ideas

Dysfunctional processing of feelings is well described in the AN literature. It can be understood within the various theoretical models we have described. The physiological understanding of feelings processing has been relatively neglected in relation to AN. The Polyvagal theory offers a useful way to understand this (Porges, 2007). The evidence of an excessive stress response and over-active SNS in AN is clear. This is also consistent with the Hyper-Emotion Theory (Johnson-Laird *et al.* 2006). Similarly, the maladaptive social inter-personal behaviour of AN sufferers is well-evidenced and undoubtedly disrupts affiliative and attachment relationships. These well-established ideas of dysfunctional feelings processing, an excessive stress response and maladaptive inter-personal behaviour, are central to our maintenance model of AN.

We have also described in this paper novel or further developed neuro-biological, physiological and psychological ideas to add to current conceptualizations of AN and generate an integrated bio-psycho-social maintenance model: the physical and psychological core needs framework; the widespread potential effects of a dysfunctional oxytocinergic system; the maladaptive role of physiological mechanisms in starvation; the concept of the shameful 'feared self' and the role of maladaptive coping modes. We suggest that these ideas offer potential for new or adapted means of working with the AN population.

### Developmental Factors

We have not directly addressed an understanding of the development of AN, as this task is beyond the scope of this paper. We hypothesize that an imbalance in the feelings processing systems, resulting in an excess of vulnerable feelings such as hunger and anxiety, could be present from birth. Equally, the imbalance in the feelings processing systems could arise neuro-developmentally as a consequence of adverse or insufficiently emotionally validating interpersonal experiences during childhood

(Anda *et al.*, 2005). Genetic and neurobiological factors alone are insufficient explanation for the disorder and the interaction with socio-cultural and psychological factors, perhaps through epigenetic mechanisms (Kim *et al.* 2014), must also be understood. We suggest that there is likely to be a spectrum of predisposition to the development of AN. Predisposition may in some cases almost inevitably result in the disorder, whilst other cases may arise predominantly because of adverse interpersonal or socio-cultural experiences. A greater understanding of the processes involved in the development of AN is required to aid prevention and early intervention.

*Clinical Implications of our Ideas*

The concept of universal core needs provides an underpinning structure which we have adapted for clinical use with AN. Van der Kaap-Deeder and colleagues (2014) have shown a relationship between improved psychological needs satisfaction and increases in BMI amongst patients with AN. This study did not use Schema Therapy and therefore highlights that the concept of core needs could be integrated into any treatment approach. We suggest the use of the core needs concept with AN patients is strengthened by integrating physical and psychological needs into one framework and the explicit conceptualization of physical and emotional 'feelings' as the conscious although often misleading signals of unmet underlying needs.

Neurobiological interventions such as deep brain stimulation (DBS) and trans-cranial magnetic stimulation (TMS) (Lipsman *et al.* 2013; McClelland, Kekic, Campbell & Schmidt, 2016) from limited pilot outcome data are now undergoing further clinical testing. We have outlined the widespread potential impact of an oxytocin deficit on development and maintenance of AN across a broad range of symptomatic areas relevant to AN. Given the evidence of oxytocin system dysfunction in AN, if oxytocin could be safely and effectively delivered as a treatment, it may hold as much potential as neuro-biological treatments currently being tested.

Our hypothesis regarding starvation functioning as a maladaptive physiological mechanism for feelings regulation can be used to provide a somatic and functional framework for patients to understand their problems and therefore reduce shame. Consistent with the Polyvagal Theory (Porges, 2007), this can be seen as a substitute for insufficient PNS activity. Starvation and over-activity may therefore operate physiologically as mechanisms to dampen the excessive stress response of an overactive SNS. Such a functional explanation for why a person may have potent drives for starvation and over-activity can reduce self-blame and shame about persisting with behaviour that is so clearly physically harmful. It also provides an explanation for why active

weight loss only temporarily and partially meets the needs to feel safe and in-control, as the underlying needs for physical safety, nutrition and rest are increasingly neglected. If the physiological process of continued weight loss and over-activity is a key reward mechanism, this also offers a rational for why no weight is ever low enough. Any clinician who like me has witnessed a patient continue to be driven to exercise and seek further weight loss at a BMI less than 9, even when they are exhausted and fully aware they are dying, cannot doubt there are potent and highly maladaptive drives at work.

The psychological concept of a 'feared self' can help patients understand how their behaviour is driven by fear not reality. The excess of vulnerable feelings generates fears about being too needy and therefore unacceptable. Body-image disturbance, potentially driven by hypo-activity in the insula and reinforced by socio-cultural influences about body-shape ideals, feeds into these fears, generating beliefs that their body is unacceptable. The cognitive end-point for patients with AN, are beliefs about being fat, greedy, needy, selfish, disgusting and unacceptable—a 'feared self'. The fear of being or becoming like this drives excessive over-compensatory behaviour, to be controlled, self-depriving, selfless and thin. We use this clinically to discuss spectrums of behaviour, identifying unhealthy behaviour at either end of a continuum, and a wide range of healthy enough behaviour that meets core needs in the middle. For example, highly self-critical and self-depriving behaviour is set at one end of a spectrum; balanced self-monitoring, self-acceptance and 'good enough' healthy behaviour found in the wide range in the middle of the spectrum; and self-indulgent, selfish and entitled behaviour at the other extreme. This describes the reality of an over-compensatory 'anorexic self' at one extreme of a spectrum, driven by the terror of becoming an unacceptable 'feared self' at the opposite extreme. We use these concepts to gradually nudge our patients towards the wide range of 'normal' in the middle of the spectrum, where their needs are met.

The mode conceptualization of different parts of the self that individuals inhabit in response to circumstances is central to the Schema Therapy model we use. The core experience of excessive vulnerable feelings is largely captured in the vulnerable mode. The maladaptive self-critical, other-critical, over-controlling and avoidant coping modes emerge in response to the excessive vulnerable feelings. This functional conceptualization can serve to both validate and highlight the maladaptive nature of these modes, reducing shame about their behaviour, yet providing a rationale for change. The development of a trusting therapeutic relationship, combined with the mode conceptualization, can open the door to change. A therapeutic experience is generated which is fundamentally emotional and inter-personal. This occurs

through direct reflection on emotional experiences and reflection on mode activation within therapy and in the rest of life. Experiential techniques such as imagery and chair-work help deepen the emotional connection to modes and allow development and practice of healthy mode responses. Our experience is that cognitive-behavioural treatments can lack sufficient emotional focus and fail to acknowledge the emotional drives to persist with learnt patterns of behaviour. Often this includes externalizing of 'the anorexia' as an external disconnected 'bad' self, which for some patients generates increased self-criticism and shame. If one learns that 'thinking errors' and 'anorexic thinking' are wrong and then learn the 'correct' responses yet continue to be powerfully driven to behave in the same way and are unable to change, the experience is of failing to act logically or rationally. This increases self-criticism, reducing self-acceptance, strengthening feelings of shame and beliefs about inadequacy. In contrast, the mode concepts help validate the function, albeit largely maladaptive, of the particular parts of a complex self, some of which produce the AN illness. In a study of preferred therapist character-istics, the most common preference amongst AN sufferers was for a therapist who helped them feel acceptable (Gulliksen et al., 2012), which we suggest the Schema-mode approach inherently addresses.

### Third Generation Models

As previously noted, a number of more integrative con-ceptual models for AN have been described, with which our model has some features in common (Fox & Power, 2009; Goss & Alan, 2010; Park et al., 2012; Pinto-Gouveia et al., 2014; Lynch et al., 2015; Skårderud, 2007). Conver-gence of ideas is welcome and encouraging as it may sug-gest some new directions from which novel clinical models can emerge. To date we are aware of two clinical models emerging from these ideas for which there is limited published preliminary outcomes evidence—Compassion-focussed therapy for Eating Disorders (CFT-E) and Radically Open Dialectical Behaviour Therapy (RO-DBT). The CFT-E model was tested in a service eval-uation study with a sample of $n = 19$ AN patients, selected from 99 AN patients entering the service. Reliable and clinically significant change on the EDE-Q global score was shown in 21% (4 of 19) AN patients (Gale, Gilbert, Read & Goss, 2014). A further pilot study of CFT-E has been published recently but only three patients with AN were included in the CFT treatment arm (Kelly, Wisniewski, Martin-Wagar & Hoffman, 2016). RO-DBT has also been tested in a service evaluation of 47 patients entering an inpatient treatment programme. Outcomes for 20 treatment completers show promising outcomes, with 30% (6 of 20) within 1 standard deviation of the community norms on the EDE-Q global (Lynch et al. 2013).

There is preliminary evidence on the use of ST in group therapy for mixed diagnosis eating disorders (Simpson, Morrow, van Vreeswijk & Reid, 2010), and a further clinical trial of group treatment is underway. We will shortly publish promising service evaluation out-comes for a cohort of $n = 28$ patients with sAN, treated in a community team using Schema Therapy. A pilot case se-ries is underway with moderate–severe patients, to test and develop the schema therapy treatment model further (clinicaltrials.gov NCT02666495).

### Limitations

As our secondary literature searches have been in part guided by our qualitative experience of patients with AN, we have not gathered all the literature of possible relevance to the disorder. Our qualitative experiential influence has come from work with patients suffering sAN, so this may have introduced selection bias into our secondary literature searches, rendering our model less generalizable to the population of AN sufferers across the severity spectrum. These can be considered weak-nesses of our methodology. In our view, reductionist and quantitative approaches to understanding this illness have to date shown limited return. We suggest that the mixed quantitative and qualitative approach we have taken, using clinical experiences from patient and clinician per-spectives, despite some inevitable limitations, may gener-ate a more holistic integrated understanding of the illness.

### Conclusion

For our theory to be of any value, it must have face validity, be internally consistent and generate testable hypotheses (Shaw & Costanza, 1982). We believe we have produced an internally consistent, theoretical framework with clinical face validity. We contend that therapy approaches centred on feelings, functional understanding and fostering trust and self-acceptance will reduce drop-out and produce outcomes superior to current usual prac-tice. Repairing the consequences of prolonged unmet core needs and insecure attachment are major psycho-social tasks. Physical recovery from starvation is a major physi-ological task. As such, we believe that psychological ther-apies for this complex illness must be longer than current usual practice.

A recently updated Cochrane review concluded that current evidence is insufficient to recommend the superiority of any specific model of psychotherapy tested to date (Hay et al., 2015). There is also no clear evidence of efficacious socio-cultural interventions or neurobiological treatments, including psychotropic medication. This disappointing evidence of limited efficacy for models of treatment tested to date suggests that it is important that

the eating disorders field is open to ideas and thinking that can inform a new generation of treatments. We suggest, as have others, that overly reductionist approaches to understanding this severe and complex illness may be limiting our ability to produce effective integrated treatments. Whether the ideas we have presented in this paper prove to have value in the treatment of AN, or not, we agree with those who argue for the importance of complex integrative models to understand better this severe multifaceted disorder (Fox & Power, 2009; Nunn *et al.*, 2012; Strober & Johnson, 2012).

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