

Narcissistic disorder and the failure of symbolisation: A Relational Affective Hypothesis



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ABSTRACT

The psychoanalytic concept of narcissistic disorder is broader than that of Narcissistic Personality Disorder (DSM-5 [1]), underlying a range of Personality Disorders (PD) and their co-morbidities. Existing Mentalisation, Psychoanalytic and Cognitive models, fail to account fully for the emerging evidence of biological, developmental, relational and defensive contributions to narcissistic disorder, nor do they account for the common and variant features of co-morbidities namely Anorexia Nervosa, Somatisation, Substance Misuse and Autistic Spectrum Disorder. Alexithymia and concrete modes of relating are common findings in narcissistic disorder and these co-morbid conditions. Current models do not provide a comprehensive account, on the basis of neuro-scientific and developmental evidence, of how affective feelings come to be represented in words and the association between narcissistic disorders and failures of symbolisation. In this paper I propose an empirically based Relational Affective Hypothesis that narcissistic disorder and its comorbidities represent failures at specific points on a representational function pathway through which subcortical affect and visceral feeling in a relational context become the basis for abstraction and language. The elucidation of this pathway allows investigation of the contribution of biological, social and psychogenic factors in narcissistic disorders. It also brings a new understanding of the neurological underpinning of psychodynamic defences in narcissistic disorders. Research and novel treatment implications are briefly considered.

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Introduction

The psychoanalytic concept of Narcissism encompasses a wide range of psychiatric presentations including PD, [2] Anorexia and Bulimia Nervosa, Somatisation and the Autistic Spectrum. This hypothesis proposes these presentations are aetiologically heterogeneous and arise from interruptions at different points along a neurological pathway mediating the representation of bodily feeling states in words. These interruptions may be consequent upon biological or environmental deficit/dysfunction or psychogenic mechanisms arising from psychoanalytic defences.

Cognitive accounts [3] of PD postulate that Cognitive Therapy strengthens Dorsolateral and Ventromesial Prefrontal Cortex regulation of the amygdala improving executive control over dysregulated affect. The amygdala mediates fear and rage responses to threat but not separation distress, motivation and appetite or affective states which are disordered in PD. Transference Focussed Therapy (TFP) [4] a psychoanalytic model, emphasises aggression and failures in the cognitive representation of affect with resultant

loss of 'effortful control'. However the evidence suggests disordered Insular function and disordered perception of relationships in PD [5]. Disorders at the perceptual pole of the mental apparatus are institutionalised in executive function [6] which comprises part of the frontal motor apparatus. TFP does not place emphasis on the projective and introjective processes underlying the claustrophobic presentation and affective lability in these disorders. Mentalisation Based Therapy [7] links the Ventromesial Prefrontal Cortex 'theory of mind' function with attachment and the relational context. This goes some way to explain the link between subcortical mechanisms, relating and social cognition, however it provides no detailed account of the interoceptive and affective inputs to this process, nor the link with the process of representation which explains the clinical association with the major co-morbidities. There is no comprehensive theory which accounts for the complex clinical presentations of these disorders and the emerging neuroscientific evidence.

In this paper I propose a Relational Affective Hypothesis to explain the clinical features of Narcissistic presentations in Personality Disorder, Anorexia Nervosa and Autistic Spectrum Disorder and their association with each other and with failures of symbolisation. I propose a clear neurobiological system to interrogate. I

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believe this hypothesis passes the test of necessity, sufficiency, specificity, empirical derivation and refutability and informs future research and treatment.

A Relational affective hypothesis

Affect and representation

Hypothesis 1

Describes a neurological pathway through which visceral sensation and subcortical affect are translated into verbal language.

'Representation function' is the process through which visceral sensation and affect are translated into emotional feelings which are then available for prosodic interpersonal communication in developmental and social settings as a prerequisite for the development of abstract thinking, symbolic play and language.

Relational disorders in narcissism

Hypothesis 2

Describes disruptions in the experience of what belongs to self and other characteristic of narcissistic disorders. These arise from developmental relational deficits or psychodynamic defences against the reality of dependence upon and separateness from the external world.

Freud, in his theory of primary narcissism (1923) [8] described an inborn hatred of those aspects of bodily and mental life and the external world which, being outside the subject's control, are experienced as alien, disturbing and demanding. Under the influence of libido the unpredictable external world is taken into the ego and so loved narcissistically as part of the self (1923) [9]. Ultimately this egocentric view must be relinquished in favour of recognition of the true state of dependence upon the external world. Secondary narcissism arises as a consequence of the defensive response to environmental failures [10].

The interdependence of representation and relating: the relational affective hypothesis

Hypothesis 3

These two hypotheses lead to a third describing the interface between representation and narcissism.

The embodied affective axis of the pathway and the interpersonal relatedness axis are 'vertical' and 'horizontal' elements of a developmental process. Deficits in the development of symbolic function lead to narcissistic patterns of relating and narcissistic defences lead to the breakdown of symbolisation.

I now briefly consider the empirical support for each of component of these hypotheses (See Fig. 1).

Hypothesis 1: affect and representation

Panksepp (1998): Basic Emotion Command Systems (BECS) [11]

Panksepp identified seven functional, anatomically discrete but interconnected sub-cortical BECS. Two are most relevant here. The SEEKING system is the dopaminergic mesocortical, mesolimbic pathway mediating motivation and exploratory behaviour in pursuit of the satisfaction of homeostatically driven appetite. The PANIC system mediates separation distress and proximity seeking behaviour in maternal and infant mammals through the action of endogenous opiate and oxytocin. The hypothesis proposes that SEEKING and PANIC function reciprocally mediating exploratory and proximity seeking behaviour in securely attached individuals.

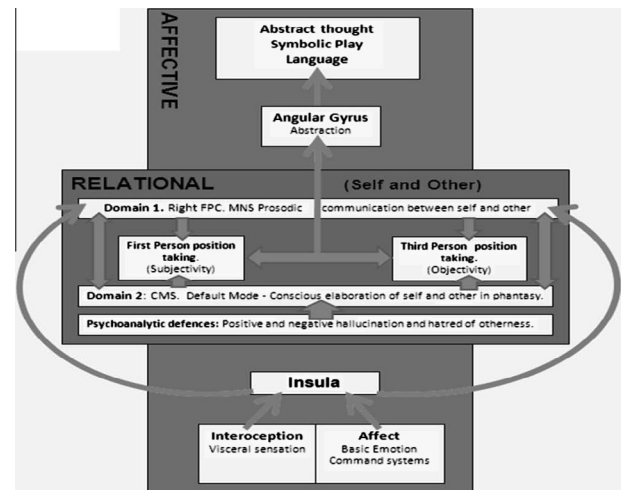


Fig. 1. Outlines the representation function pathway.

Solms (2013): the body as subject and object [12]

Primary and secondary somatotopic maps in the sensory neocortex represent the musculoskeletal and surface body as an object amongst objects. The internal body is the subject of perception and the seat of subjective consciousness. The states of the body as subject are experienced as affects. Conscious feelings tell the subject about its homeostatic state and therefore could be considered an interoceptive sensory modality.

Ramachandran (2001): exteroception [13]

Ramachandran provides an insight into the contribution of exteroception to the representational process through pathways between fusiform gyrus, angular gyrus and Wernicke's area. Although pathways to the Amygdala and Insula are described in this account interoceptive and relational inputs to symbolisation are not specified.

Craig (2009): the role of the Insula [14]

The Insula integrates interoceptive, exteroceptive afferents and affect as impulses progress from posterior to Anterior Insular cortex (AIC). The AIC is the limbic sensory cortex providing a substrate for 'my feeling about that thing at a moment in time'. It is connected with the anterior cingulate cortex (ACC) or limbic motor cortex by Von Economo Neurones. The AIC and ACC together mediate volitional agency. The right AIC is responsible for transmission of arousal and anxiety to higher centres [15] where the left Insula mediates vagal parasympathetic outputs related to restorative experiences following stress or arousal [16].

Uddin (2007): self and other [17]

Uddin et al. described two large scale neural networks representing self and other.

- *The Fronto-Parietal network and the Mirror Neuron System (MNS)*. A lateral network involved with the perception of self and other in the external world (Called here Domain 1).
- *The Cortical Midline Structures (CMS)*. A medial network engaged in internal processing of information about self and other in abstract evaluative terms (Called here Domain 2).

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