

Neuroimaging and Attachment in Eating Disorders: Parental Love Nurtures the Brain

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Editorial

Eating Disorders (EDs) are mental disorders characterized by specific brain dysfunctions which may affect body perception, fine tuning of emotions and food appetite, and cognitive impairment. Biological, psychological, socio-cultural and environmental factors interact in the pathogenesis of Anorexia (AN) and Bulimia (BN) and coexist in different stages of their development [1-3]. For this reason Eating Disorders (EDs) are complex psychiatric disorders which represent a crossroad between the suffering of mind and body, and may be considered prototypical psychosomatic disease [4, 5]. They still display an unsatisfactory response to treatments [6]. Modern neuroimaging techniques and attachment theory have been jointly applied to produce new insight in the pathogenesis of these disorders. This may permit to approach new therapeutic interventions.

Research exploring the neurobiological basis of EDs searched for diagnostic biomarkers to help the classification of these disorders characterized by a high discrepancy between diagnostic criteria and clinical experience [7]. Many researches applied neuroimaging structural and functional techniques to study the brain regions involved in pathophysiology of EDs [3]. Neuroimaging techniques lead to interesting results which may be relevant to the understanding of pathogenesis of EDs. Global cerebral and cerebellar grey matter (GM), white matter (WM) atrophy and ventricular enlargement has been found in AN [8-11]. Some cerebral areas such as thalamus, midbrain [12], para central lobule [13] hippocampus-amygdala complex [14] and anterior cingulate cortex [15] are particularly involved by AN malnutrition effects. Not only grey matter but also white matter is damaged by anorexia. Recent VBM studies on AN patients have found significant reduction of total white matter (WM) volume and focal GM atrophy in cerebellum, hypothalamus, caudate nucleus and frontal, parietal and temporal areas [16]. As a fact, the onset of EDs during adolescence [17] when brain maturation is still incomplete [18] implies a high vulnerability to short and long-term brain alterations due to malnutrition.

From a psychological and developmental point of view, a strong protective factor with respect to development during adolescence is represented by the attachment to parents and family [19]. Possibly for this reason Dozier and coworkers [20] showed a strong link between attachment style and the risk of developing eating disorders during adolescence and early

adulthood. Winnicott [21] underlined that only when the relationship of the adolescent with caregivers is solid and stable he/she is able to detach from family and face with adult identity. In fact the attachment behavior system is a motivational inborn system, biologically evolved, activated by perceived threats and dangers [22]. At the moment the attachment theory represents one of the most important frameworks for understanding affect regulation and human relationships [23]. The attachment function is strictly and stably represented by neurobiological substrates and brain organization during child and adolescent development. Interpersonal relationships between child and caregivers are encoded in the implicit memory system and represent internal working models of attachment for future relationships [24, 25]. Since attachment plays a role in the onset and the development of these disorders [19, 26] the integration of attachment theory with neuroimaging may be of relevance to produce a better insight in the mechanisms which produce brain alteration.

Few studies investigated the neural correlates of attachment [27-29] using fMRI. Some studies [27, 29] indicated a possible network of cortical and subcortical areas involved in the attachment system including the amygdalae, the thalamus, the frontal cortex. In particular Buchheim and coworkers (2006) examined the neural correlates of attachment applying the Adult Attachment Projective (AAP) in adults affected by borderline personality disorders during an fMRI scan assessing attachment status (organized versus disorganized) with respect to attachment

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trauma. The participants with unresolved attachment showed increased activation of medial temporal regions during AAP administration. This phenomenon involved also the amygdala and the hippocampus which are fundamental components of the limbic system. The role of these structures in the attachment circuits is underlined by the evidence that levels of activity within the amygdalae are related to attachment insecurity [30].

As concerns the eating disorders a recent study by Cicereale and coworkers (2013) [31] used the AAI to assess mental representations of attachment in AN subjects. This study was the first that attempted to analyze together attachment, brain anatomy and functionality in AN women. According to previous literature, they found that non-secure attachment is prevalent in AN with similar patterns of those already found [32, 33]. The role of parents in the onset and the development of AN is controversial (Amianto et al., 2013, 2015a; Fassino et al., 2009) [34, 35], but it is supported by these results. In particular AN subjects experienced higher *pressure to achieve* and lower *love* received from the mothers. These results confirm the findings of Witkowska and coworkers [36] and other studies on parenting [37] sustaining that young women with AN perceive their mothers as more demanding and less caring than subjects from general population. More in general these results support the clinical experience and the early theories about eating disorders that postulate those whole family systems and their rules may have a great effect on the creation and maintenance of eating disorders [34, 35, 38].

Moreover in the study of Cicereale and coworkers [31] GM volume of different brain areas correlates with several AAI subscales. In particular, the AAI coding system considers the Coherence of Mind and the Coherence of Transcript as the most accurate indicator of the state of mind with respect to attachment [39]. The poor CoM which has been found in AN patients suggests difficulty in creating a narrative process on psychological development, self-awareness and incoherencies on the report of their own experiences [40, 41]. CoM and CoT positively correlated with the volume of GM in a network of brain areas comprising the temporal poles, both amygdalae, the midbrain, the thalamus and the anterior and middle sections of cingulate cortex [27, 30]. Finally, two different brain networks were linked to passivity and anger. Higher passivity correlated with lower GM volumes in mesial areas, such as the precuneus which are involved in autobiographical memory and auto-referential processes [42]. This lowered functionality can explain the behavioral aspects typical of passivity interpreted as the difficulty to access to clear and coherent autobiographical memories, as it was seen in people with entangled attachment organization [27, 29].

In the AN sample two scales pertaining the experienced relationship with the caregivers during childhood strongly related to the overall state of mind as assessed by the AAI [31]. The maternal love (LM) positively correlated with a network of brain areas which overlap to those linked to CoM and CoT, while Anger anti-correlated with parts of the same network (for instance, the precuneus and part of the limbic system). It is thus possible to hypothesize that childhood loving experiences and anger

feelings perceived towards the caregivers had opposite effects on brain areas belonging to attachment circuits. The experience of love seems to be protective with positive effects on the brain trophism. While anger may have some role in producing GM atrophy in specific areas.

As a final consideration, substantial overlapping exists between the areas that Cicereale and coworkers have found to be linked to attachment and the areas reported in the literature as markedly atrophic in anorectic subjects [42] which are the thalamus, the cingulate cortex and the amygdalae. It is thus possible that atrophic areas in AN were already hypofunctional before the onset of AN because of the impairment of attachment functions and of anger management.

Brain imaging in eating disorders has shown several findings that seem to reliably describe alterations in anorexia and bulimia nervosa. Nevertheless the direction of their correlation between brain alterations and the eating disorder, e.g., if they are involved in the pathogenesis or consequent to malnutrition, is still uncertain. Recent studies on neuroimaging of attachment, coupled with growing evidences about the pathogenic role of attachment on eating disorders [19, 43], and with some new evidences about the characteristics of the parents of ED subjects [34, 37, 44-46] suggest that the influence of parental cares on the outburst and maintenance of EDs is not eminently operating eating psychopathology [5], but mostly on the attachment substrate on which psychopathology is expressed. Moreover, due to the eminently structural correlates of the attachment, the influences of parenting may produce specific alterations on brain trophism which may represent the original wound from which the eating psychopathology sprouts.

Similarly to what has been shown in rodents and other mammals [47, 48] receiving parental cares during the early development is crucial for the healthy development of human brain. The love received from the mother is a good indicator of secure attachment and may represent an essential protective factor for anorexia nervosa during the period of adolescent development of the brain. On the other hand, the anger experienced towards the caregivers is a potential risk factor which reduces brain trophism, and may foster those brain malfunctions which facilitate the development of eating psychopathology. The positive effects of growing up in a caring environment may thus have a protective and neurotrophic effect on brain areas shown to be linked to AN. It is thus possible that the re-activation of parental cares and/or the reduction of anger feelings towards caregivers may represent rehabilitative tools to modify the functioning of some brain areas which are involved in the maintenance of the eating disorders.

The reactivation of the circuits related to attachment is generally performed indirectly by emotional corrective experiences such as individual psychotherapy, family therapy or other family approaches (like counseling) acting with the reactivation of attachment functions [34, 44]. On the other hand the evidences concerning the specific alteration of brain circuits may suggest that therapy may also target a direct stimulation of malfunctioning brain areas applying techniques such as Repetitive Transcranial Magnetic Stimulation (r-TMS) or Deep Brain Stimulation (DBS).

The current findings join two areas of interest for the research in EDs. The therapeutic strategies aimed at correcting anomalies in the functioning of some specific brain areas related to attachment and the experiences of new patterns of attachment within therapeutic relationships permit to program jointed or sequential therapeutic interventions on brain circuitry related

to attachment. Moreover, the current joint evidences about attachment and brain imaging also suggest a reflection on possible preventive interventions on eating disorders in children and adolescents. In fact the education to a correct and healthy eating may be of lesser relevance for brain development than the promotion of the emotional and affective nurture of a loving parenting [46].

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