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Bisogna pur mangiare: i DCA nella dimensione clinica residenziale

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Therapeutic Advances in Psychopharmacology Review

Genetic risk factors for eating disorders: an update and insights into pathophysiology

Hubertus Himmerich, Jessica Bentley, Carol Kan and Janet Treasure

Ther Adv Psychopharmacol
2019, Vol. 9: 1–20
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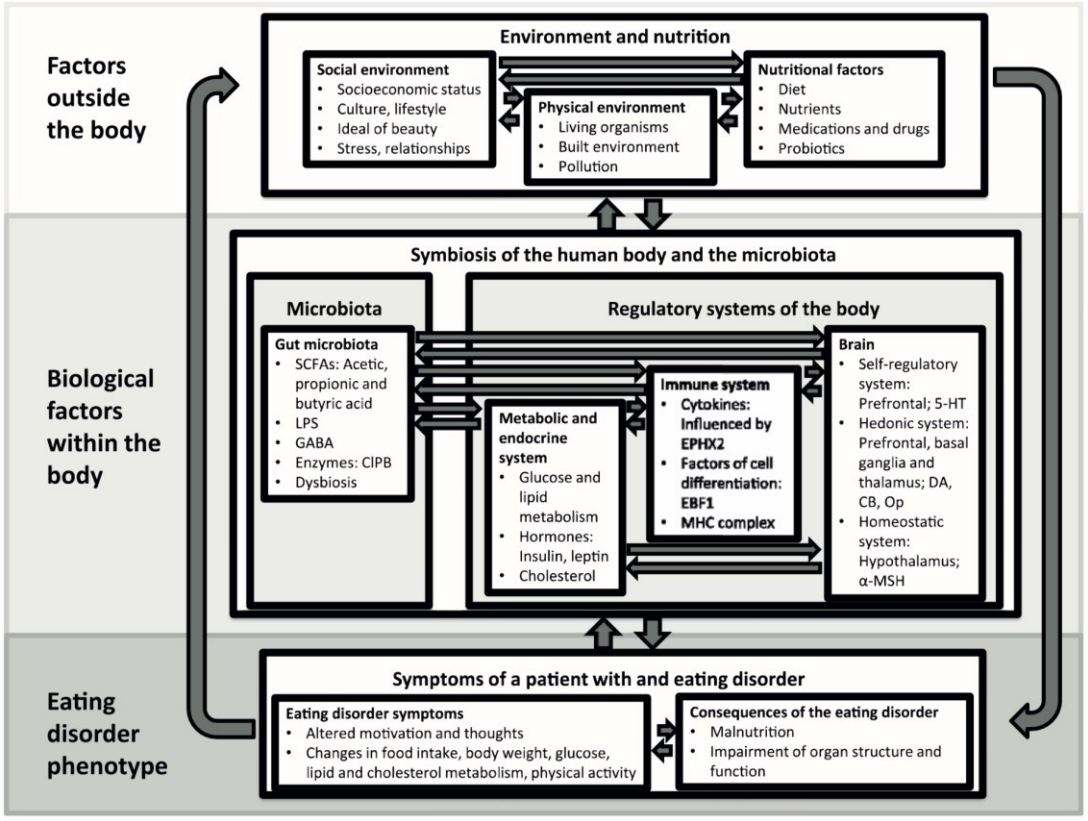

Figure 1. Schematic and simplified depiction of a pathophysiological model of eating disorders based on genetic findings.
SCFAs, short chain fatty acids; LPS, lipopolysaccharides; GABA, gamma-aminobutyric acid; B ClpB, caseinolytic protease; EBF1, early B-cell factor 1; EPHX2, epoxide hydrolase 2; MHC, major histocompatibility complex; 5-HT, serotonin; DA, dopamine; CB, cannabinoid; Op, opioids; α-MSH, melanocyte-stimulating hormone.

BMC Psychiatry

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A debate on current eating disorder diagnoses in light of neurobiological findings: is it time for a spectrum model?

BMC Psychiatry 2012, **12**:76 doi:10.1186/1471-244X-12-76

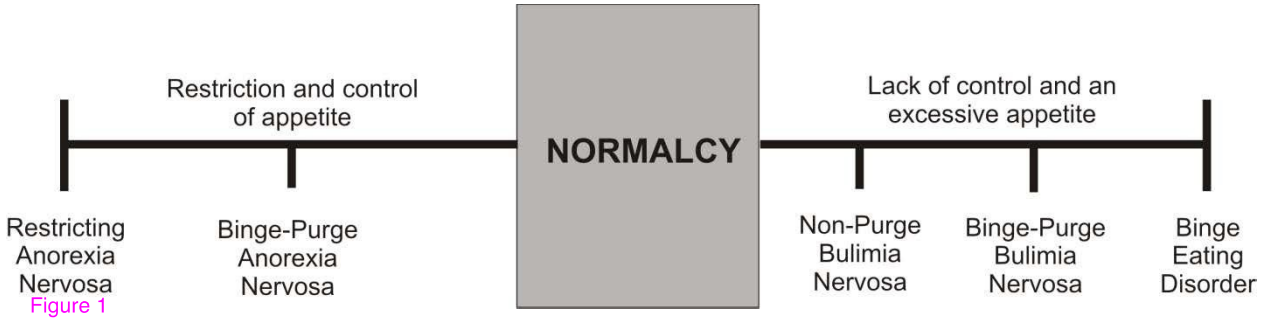


Figure 1

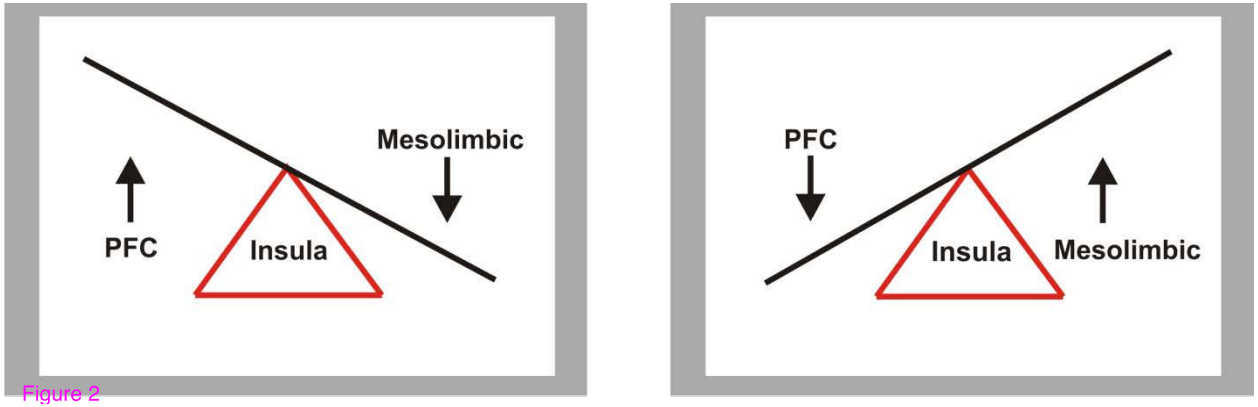


Figure 2

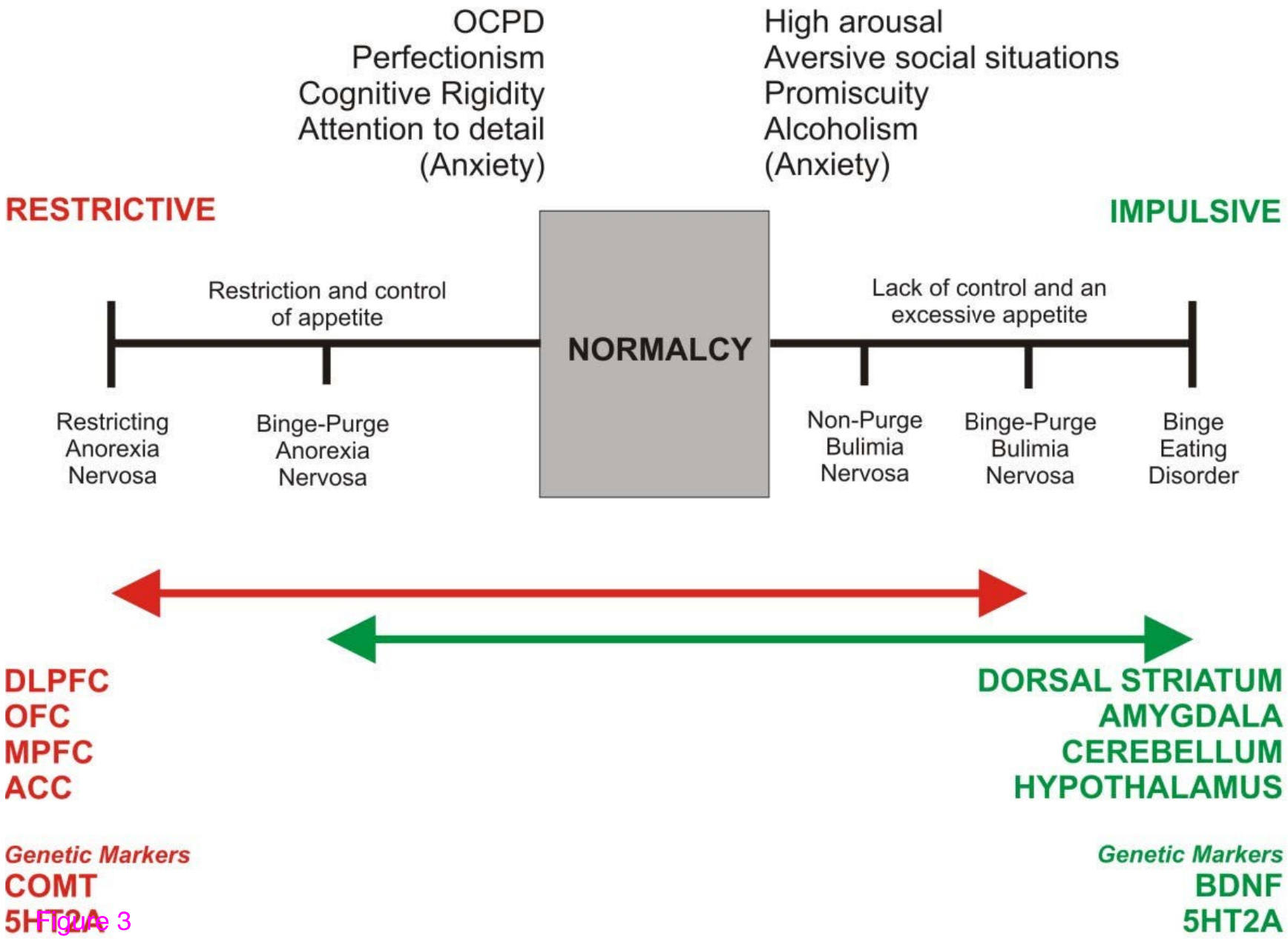
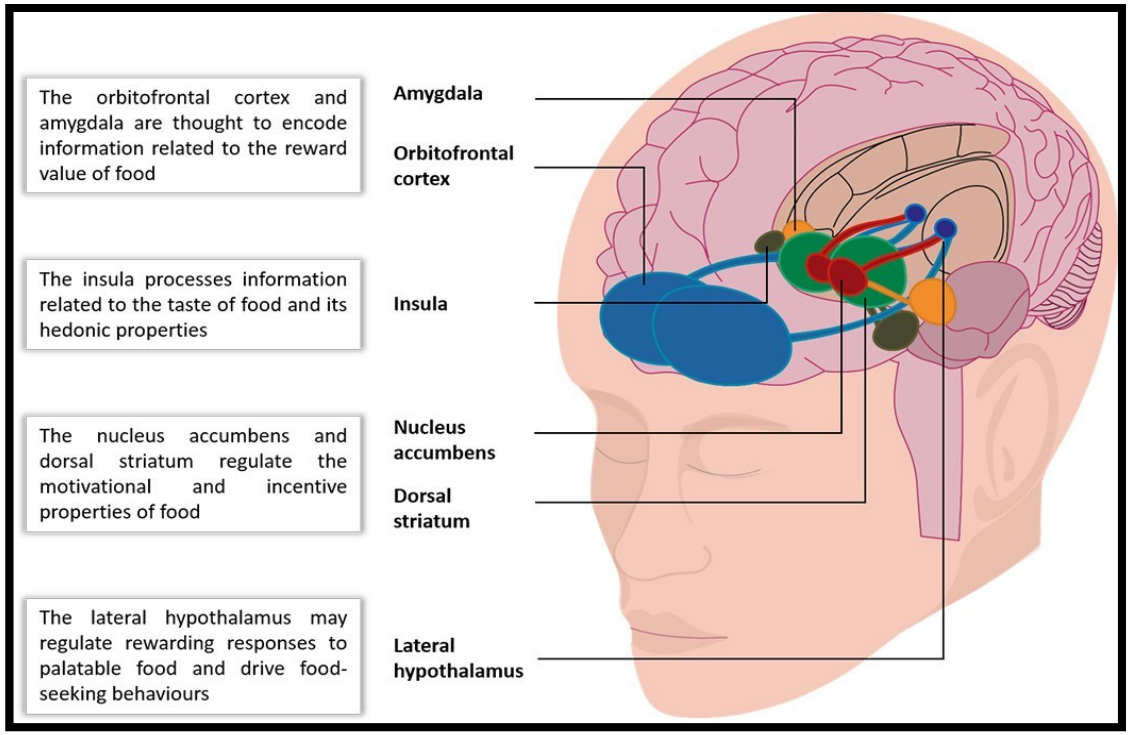

OCPD
Perfectionism
Cognitive Rigidity
Attention to detail
(Anxiety)
High arousal
Aversive social situations
Promiscuity
Alcoholism
(Anxiety)
RESTRICTIVE
IMPULSIVE
Restriction and control of appetite
Lack of control and an excessive appetite
NORMALCY
Restricting Anorexia Nervosa
Binge-Purge Anorexia Nervosa
Non-Purge Bulimia Nervosa
Binge-Purge Bulimia Nervosa
Binge Eating Disorder
DLPFC
OFC
MPFC
ACC
DORSAL STRIATUM
AMYGDALA
CEREBELLUM
HYPOTHALAMUS
Genetic Markers
COMT
5HT2A
Genetic Markers
BDNF
5HT2A

Figure 3



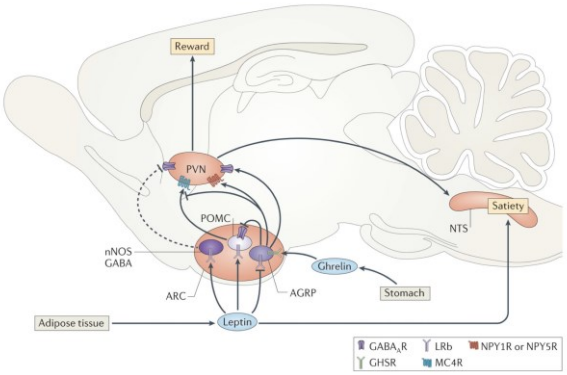


Figure 4. Neurocircuits involved in the homeostatic regulation of feeding

Neurons in the hypothalamic arcuate nucleus (ARC) and nucleus of the solitary tract (NTS) sense and respond to peripheral energy signals to promote energy homeostasis. Neuropeptides such as neuropeptide Y (NPY) and neurotransmitters such as GABA, among others, are released onto downstream neurons including those in the paraventricular nucleus (PVN). In the PVN, oxytocin and other neurons tonically inhibit feeding and, during energy deficit, are inhibited by orexigenic input from the ARC, thereby stimulating feeding. The same agouti-related protein (AGRP) neurons (which co-express GABA and NPY) that are involved in short-term feeding also contribute to long-term energy balance through the release of AGRP, an inverse agonist of melanocortin receptor 4 (MC4R) and, through GABA release, inhibit neighbouring pro-opiomelanocortin (POMC) neurons. POMC neurons are stimulated by input from leptin, and the release of α-melanocyte stimulating hormone (α-MSH) activates MC4R, thereby inhibiting food intake. In addition, recent evidence also implicates leptin-responsive GABAergic neurons that express neuronal nitric oxide synthase (nNOS) in the regulation of energy homeostasis. These neurons are found in the ARC and dorsomedial nucleus (not shown) and are hypothesized (dashed line) to inhibit downstream neurocircuits that drive feeding. Collectively, this input is relayed to the PVN and lateral hypothalamic area (not shown) and integrated to modulate the rewarding properties of food and the response to satiety signals. $GABA_AR$, type A GABA receptor; GHSR, growth hormone secretagogue receptor (ghrelin receptor); LRb, leptin receptor; NPY1R, NPY receptor type 1.

Selected Findings in Reward-Related Brain Areas in Clinical Populations

Disorder	*Dopamine*	*Opioids*	*Serotonin*	*Acetylcholine*
Binge eating	↑ DA in striatum ↓DA reuptake	Mu-antagonist ↓ food palatability	↓ transporter binding	
Bulimia nervosa	↓ HVA ↓DA transporter availability ↓DA receptor binding ↓DA release	↓ β-endorphin levels ↓ mu-opioid receptor binding		
Anorexia nervosa	↓ metabolite CSF levels ↑ D2/D3 receptor binding	gene polymorphisms of the delta-1 receptor	↓basal concentration of metabolites in CSF 5-HT1A and 5-HT2A receptors and 5-HT transporter dysregulated in cortical and limbic regions	

Nat Rev Neurosci. Author manuscript; available in PMC 2015 June 01.

THE (DIS)EMBODIED SELF IN ANOREXIA NERVOSA

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Abstract—This paper deconstructs the debate that has been raging for over a decade between proponents of the feminist cultural model of eating disorders and supporters of the traditional medical model of illness and treatment, bringing the level of analysis one step deeper—to the question of the constructions of "the self" employed in these discourses and the implications of these constructions for the successful understanding and treatment of anorexia nervosa. The paper argues that while feminist theorizing has largely dislodged the current representations of anorexia nervosa from the clamps of myopic medical discourses devoid of detailed cultural analysis, it has in fact produced similar theoretical dichotomies and blind spots that preclude the successful theorizing of an embodied self and its particular articulation in anorexia nervosa. It is proposed here that Foucault's [(1986) *The Care of the Self. The History of Sexuality*, Vol. 3. Vintage, New York] notion of "technologies of the self" can provide us with a useful tool for bridging the split between the "inside" and "outside" produced and reified in both the medical model and the feminist cultural formulation of anorexia; a framework is suggested for the implementation of this interpretative position, based on a reconceptualization of the particular ritualistic behaviors associated in anorexia as articulating the core issues of the illness—a reconfiguration and repositioning of the "inside" and the "outside" as a means of tailoring the self along a particular line of "attitude". The essay is based on eight months of fieldwork counseling in an eating disorders treatment center.

Key words—anorexia nervosa, addiction and recovery, culture and psychiatry, philosophies of the self

Susie Orbach (1986) writes,

has produced similar theoretical dichotomies and


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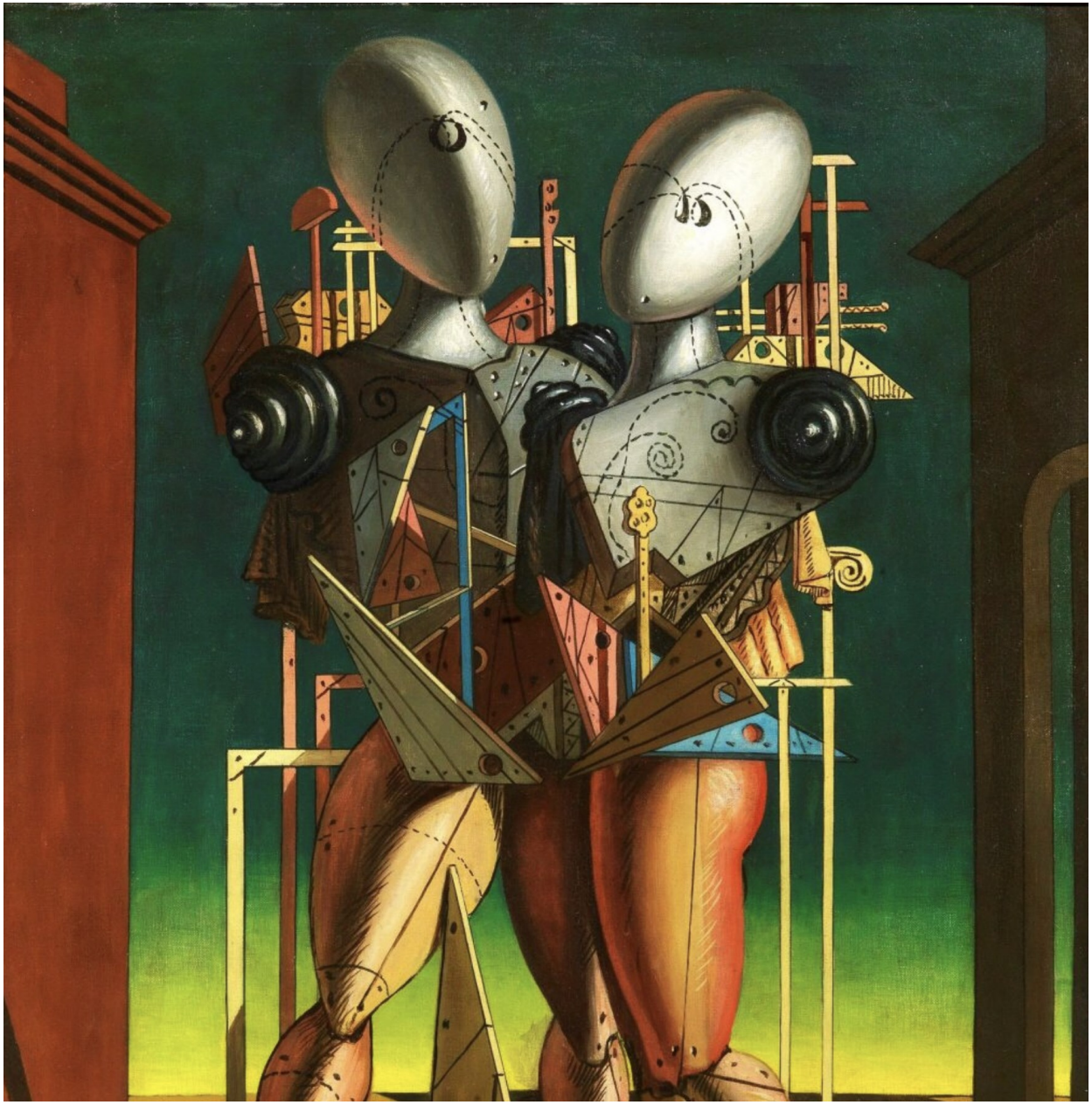
HOW TO MAGIA
LE STREGHE SON

<<così gli individui presi come corpi posso non essere di per sé nient'altro che sintomi relativamente ad altri corpi>>

<<se ciò non avviene il corpo è sintomo di un altro sintomo>>

<<il sintomo isterico è il sintomo che interessa al sintomo dell'altro in quanto tale, ossia che non richiede il corpo a corpo>>

Lacan, Joyce e il sintomo



aspetti clinici rilevanti

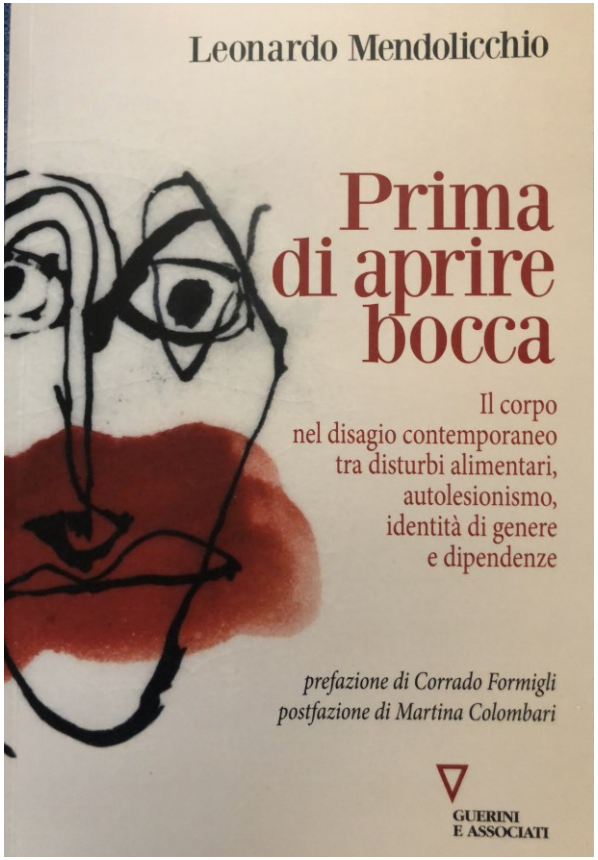
- idealizzazione immagine corporea
- prevalenza aspetti proiettivi
- erotizzazione fame
- erotizzazione morte
- egosintonia
- atemporalità del corpo
- Asessualità/enfasi sessulità del corpo

Lessico psicoanalitico:

Identificazione

Io ideale

Pulsione



Logica dei D.C.A.

$$\frac{i(a)}{(-)}$$

→ Fase dello specchio

$$\frac{\frac{i(a)}{a}}{(-\varphi)}$$

→ Logica di funzionamento DA

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Il Faut Bien Mager

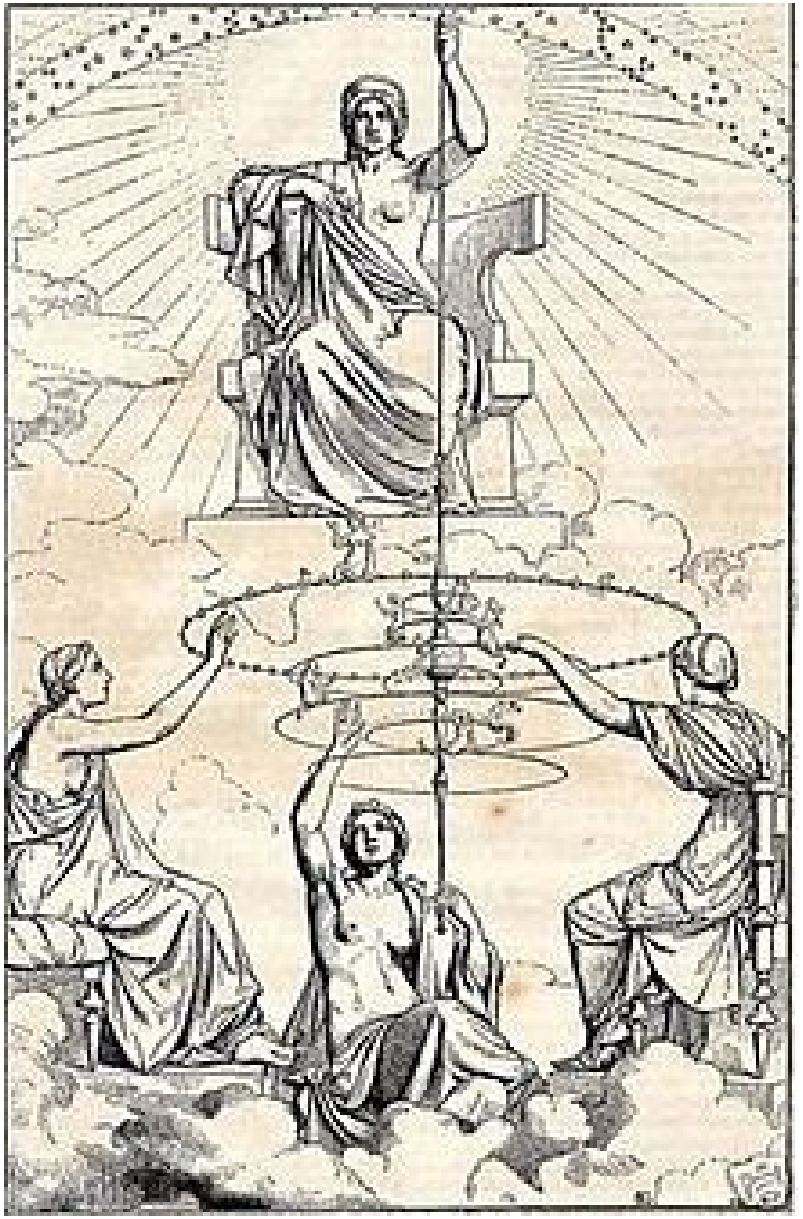
Il mangiare come metonimia dell'introiezione

Il nutrimento come eterogenesi della soggettività

J. Derrida

Progetto “Ginestra”:Nucleo trattamento pazienti gravi

- nutrizione artificiale
- Holding intensivo
- intensificazione approccio psicopedagogico
- servizi assistenziali con rapporto 1:1



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