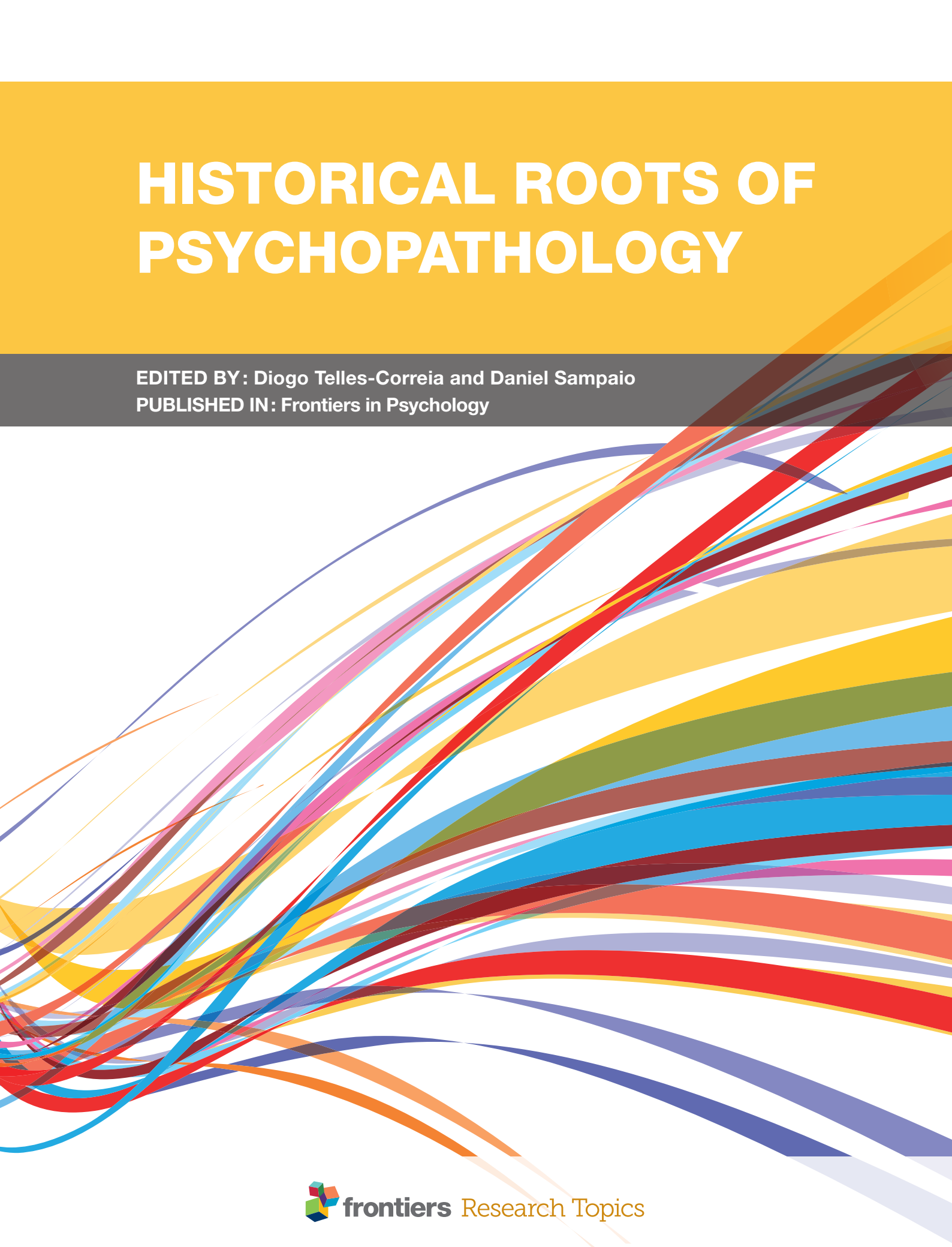
HISTORICAL ROOTS OF PSYCHOPATHOLOGY

EDITED BY: Diogo Telles-Correia and Daniel Sampaio
PUBLISHED IN: Frontiers in Psychology



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ISSN 1664-8714
ISBN 978-2-88919-933-4
DOI 10.3389/978-2-88919-933-4

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HISTORICAL ROOTS OF PSYCHOPATHOLOGY

Topic Editors:
Diogo Telles-Correia, University of Lisbon, Lisbon, Portugal
Daniel Sampaio, University of Lisbon, Portugal

New advances of the neuroscience supported by a refined, reliable and valid phenotyping (e.g., at the level of symptoms and not at the level of disorders), are bringing some promising results. The mapping of clinical phenomenology on specific brain dysfunction is now becoming plausible and the resulting functional psychopathology may in the future significantly replace the present nosology (Jablensky, 2010).

Nevertheless, as Andreasen (2007) points out: "Applying technology without companionship of wise clinicians with specific expertise in psychopathology will be a lonely, sterile and perhaps fruitless enterprise."

Some of the chapters of this Ebook deal with aspects which are essential to the historical understanding of mental symptoms and disorders.

Citation: Telles-Correia, D., Sampaio, D., eds. (2016). Historical Roots of Psychopathology. Lausanne: Frontiers Media. doi: 10.3389/978-2-88919-933-4

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frontiers in Psychology

EDITORIAL
published: 14 June 2016
doi: 10.3389/fpsyg.2016.00905

Editorial: Historical Roots of Psychopathology

*Diogo Telles-Correia * and Daniel Sampaio*

Faculty of Medicine, University of Lisbon, Lisbon, Portugal

Keywords: psychopathology, history of psychopathology, philosophy of psychiatry, symptoms, mental disorders

The Editorial on the Research Topic

Historical Roots of Psychopathology

Mental illness and mental symptoms depend on a construction that results from the decisions of certain social agents, which, in a specific social and historical context, according to an epistemological framework (how symptoms and disorders are constructed and detected) and an ontological framework (how they are defined, what they consist of), identify the behaviors which make up a symptom or a disorder (Berrios, 2011). Only after these theoretical hypotheses of mental symptom and disorder have been outlined are the data which empirically validate them searched and found. Therefore, the objects of psychiatry (mental symptom and disorder) being the result of a social conjecture and a philosophical perspective rooted in a specific time, they should also be studied with elements from social and human sciences (history, sociology, philosophy) (Telles-Correia, 2015).

Besides these elements, clinical experience is essential to find out new forms of presentation, as well as to name new clinical behaviors and manifestations. After all, this was the fundamental method of the great psychopathologists and nosologists of the late Nineteenth century and early Twentieth century, such as Falret, Kalbaum, Kraeplin, etc. (Jaspers, 1963; Goas, 1966).

These theoretical hypotheses, born out of clinical experience together with the historical, sociological and philosophical analysis of previously established theories are connected to reality through empirical validation. The latter aims to show that the said symptoms/diagnoses do really exist (Zachar, 2012).

According to Zachar (2012), there are two major paradigms of empirical validation. The first paradigm is the medical model. According to this model, validation includes the study of the natural history of disorders (which show to be consistent in terms of natural history with the proposed diagnosis), studies of family aggregation (which show a greater influence of heredity in the proposed diagnosis) and the search for neurobiological causality. This model was the basis for the development of classification systems such as the DSM.

At the same time a psychological model was developed. In this psychological/psychometric model current validation processes follow a different paradigm. According to this model, psychological/psychiatric variables are latent variables (which cannot be measured/observed directly but need instead to be assessed through other component variables). The validation of the instruments which measure this latent variable includes complex statistical methods (in the search for construct validity and criterion validity).

These two kinds of validation are not exclusive and nowadays there is a tendency for them to coexist and combine.

In recent years, the search for empirical validation has reached proportions never seen before, generally in line with the structures of diagnosis universally accepted. The so called content validation was thus devaluated. In this approach, the theoretical hypotheses are reassessed based

Edited by:
Antoine Bechara,
University of Southern California, USA

Reviewed by:
Paulo Jannuzzi Cunha,
University of São Paulo, Brazil

****Correspondence:***
Diogo Telles-Correia
tellesdiogo@gmail.com

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *24 March 2016*
Accepted: *01 June 2016*
Published: *14 June 2016*

Citation:
Telles-Correia D and Sampaio D (2016) Editorial: Historical Roots of Psychopathology. Front. Psychol. 7:905. doi: 10.3389/fpsyg.2016.00905

on an important revision of the history and evolution of the established concepts (which comprises the sociological movements and the ontological and epistemological philosophical frameworks of the different authors) and also on the new data from updated clinical practice. With the devaluation of content validation, the current guidelines are considered to be beyond criticism and irrefutable. Most of the times the technicians involved in the validation of psychometric instruments aimed at evaluating a specific concept which has been established decades ago (sometimes more than a 100 years ago) do not know the origin of these concepts nor the history of their development thoroughly (nor if they are socially, historically and philosophically appropriate nowadays). The same applies to the researchers who try to find the anatomical/chemical correlates of psychiatric manifestations but often do not grasp the conceptual basis of the object studied nor the validity of the methods used to detect the said object, nor have they in their team a member able to explain this to them (Telles-Correia, 2015).

New advances of the neuroscience supported by a refined, reliable and valid phenotyping (e.g., at the level of symptoms and not at the level of disorders), are bringing some promising results. The mapping of clinical phenomenology on specific brain dysfunction is now becoming plausible and the resulting functional psychopathology may in the future significantly replace the present nosology (Jablensky, 2010).

Nevertheless, as Andreasen (2007) points out: "Applying technology without companionship of wise clinicians with specific expertise in psychopathology will be a lonely, sterile and perhaps fruitless enterprise."

Some of the chapters of this Ebook deal with aspects which are essential to the historical understanding of mental symptoms and disorders.

The first text of this topic will briefly review the fascinating history of Asperger syndrome: why it was born, its tumultuous existence, and its downfall.

The second text presents an historical overview of the understanding of Obsessive Compulsive Disorder, highlighting the advances in neuroscience and how they influenced current perspectives on the nosology of this disorder.

The third text reviews many historical sources about the understanding and treatment of mental disorders in the early modern England and connects these with current trends in mental health care.

The fourth text will show a critical review about psychopathology classification systems on Sexual orientation and gender identity and argues for the broader respect and value of the diversity of human sexuality and of gender expressions.

The fifth text will review the history of histrionic personality disorder, one of the most ambiguous diagnostic categories in psychiatry reflecting attitudes about health, religion and gender across time.

The sixth text aims to review the evolution of the term "hallucination" up to present time, highlighting the difficulty in both defining and limiting this concept ever since its first appearance.

The seventh text presents some elements of the Freudian thinking on psychosis. Can the psychotic individual be invaded by a pulsating unconscious which demands a symbolic mediation?

The eighth text reflects recent changes in the Brazilian public policies for mental health since Diagnostic and Statistic Manual of Mental Disorders was introduced which might disregard the subject and its personal history.

The ninth text aims at reviewing the contributions by the different authors to the construction of the term "melancholia," throughout history, where it has been associated not only to affective disorders but also to abnormal beliefs.

AUTHOR CONTRIBUTIONS

DT: literature review and elaboration of the final paper. DS: literature review.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in Psychology

MINI REVIEW
published: 25 January 2016
doi: 10.3389/fpsyg.2015.02024

A Concise History of Asperger Syndrome: The Short Reign of a Troublesome Diagnosis

J. B.Barahona-Corrêa[1,2,3,4]* *and Carlos N. Filipe*[5]

[1] Department of Psychiatry and Mental Health, Nova Medical School/Faculdade de Ciências Médicas - Universidade Nova de Lisboa, Lisbon, Portugal, [2] Neuropsychiatry Unit, Champalimaud Clinical Centre, Fundação Champalimaud, Lisbon, Portugal, [3] Centro de Apoio ao Desenvolvimento Infantil – CADIN, Cascais, Portugal, [4] Department of Psychiatry and Mental Health, Centro Hospitalar de Lisboa Ocidental, Lisbon, Portugal, [5] Department of Physiology, Nova Medical School/Faculdade de Ciências Médicas - Universidade Nova de Lisboa, Lisbon, Portugal

First described in 1944 by Hans Asperger (1944), it was not before 1994 that Asperger Syndrome (AS) was included in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders, only to disappear in the Manual's fifth edition in 2013. During its brief existence as a diagnostic entity, AS aroused immense interest and controversy. Similar to patients with autism, AS patients show deficits in social interaction, inappropriate communication skills, and interest restriction, but also display a rich variety of subtle clinical characteristics that for many distinguish AS from autism. However, difficulties operationalising diagnostic criteria and differentiating AS from autism ultimately led to its merging into the unifying category of Autistic Spectrum Disorders. Here we briefly review the short history of this fascinating condition.

Keywords: Asperger Syndrome, autism spectrum disorders, DSM-5, psychopathology, nosology

OPEN ACCESS

Edited by:
Diogo Telles-Correia, University of Lisbon, Portugal

Reviewed by:
Andrea Raballo, University of Oslo, Norway
João Gama Marques, University of Lisbon, Portugal

***Correspondence:**
J. B. Barahona-Corrêa
bernardo.correa@cadin.net

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *30 July 2015*
Accepted: *18 December 2015*
Published: *25 January 2016*

Citation:
Barahona-Corrêa JB and Filipe CN (2016) A Concise History of Asperger Syndrome: The Short Reign of a Troublesome Diagnosis. Front. Psychol. 6:2024. doi: 10.3389/fpsyg.2015.02024

INTRODUCTION: REFRIGERATOR MOTHERS AND FINE-BONED ARISTOCRATS

The entry of autism and Asperger syndrome (AS) into the history of psychopathology was marked by extraordinary coincidences. Both disorders were first described by Kanner (1943) and Asperger (1944), respectively. Both were Austrian-born physicians and, though unaware of each other's writings, both used the term "autistic" to describe a unique group of children who shared features of impaired social interaction and restricted, repetitive behaviors and interests. Both Kanner (1943) and Asperger (1944) borrowed the term "autistic" from Eugen Bleuler, who used it in his "Dementia Praecox or the Group of Schizophrenias" to describe extreme social withdrawal and self-centeredness in patients with schizophrenia. Moreover, both authors emphasized that the syndrome they were describing differed from infantile (e.g., De Sancti's dementia praecocissima) and juvenile schizophrenia, namely by manifesting from birth and improving (in terms of social interaction) with growth, in contrast to the usual course of schizophrenia (Higier, 1923). Significantly, although Kanner (1943) initially considered language abnormalities (varying from sheer absence of language to atypical, socially ineffective use of well-developed language) to be a defining feature of his "Autistic Disturbances of Affective Contact," he later hypothesized that they could be secondary to the two nuclear features of the disorder: "extreme self-isolation" and "obsessive insistence on sameness" (Irwin et al., 2011). Furthermore, Kanner (1943) also noted that many of his patients possessed "good cognitive potential." Linguistic and cognitive ability would later sit in the eye of the storm unleashed by the appearance of AS as a discrete diagnostic

entity and the relentless polemic that accompanied it. While Kanner's syndrome eventually made its way to the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), Asperger's work, published in German, remained virtually unknown to the international scientific community for almost half a century. Indeed, the first English translation of Asperger's article "*Die Autistischen Psychopathen im Kindesalter*" first appeared in 1991 in Uta Frith's textbook "autism and AS." AS had already been described in 1981 by Lorna Wing, who first proposed the term to refer to a special subgroup of children who, according to Asperger's original description, were characterized by: social isolation and lack of reciprocity in social interactions; normal or precocious language acquisition, with above-average linguistic skills but subtle abnormalities of verbal and non-verbal communication (e.g., atypical syntax, pedantic vocabulary and absent or stereotyped prosody); a narrow focus of interests, often restricted to unpragmatic and highly original themes; overachievement in specific cognitive domains; and motor clumsiness (Wing, 1981). Unlike Kanner (1943), Asperger (1944) did not attempt to define diagnostic criteria for the disorder he was describing. Moreover, Asperger greatly emphasized subtle positive features in his patients: they often had extremely original thought, they tended to cultivate abstract and intellectualized interests, often had, in Asperger's own words, "a rare maturity of taste in art," and even a peculiar, fascinating physical appearance, with "finely boned features," of "almost aristocratic appearance" (Asperger, 1944). Asperger's captivating descriptions of his subjects certainly played a decisive role in the history of the syndrome that bore his name, especially as they contrasted sharply with Kanner's later recriminatory writings on "refrigerator-mothers" and the origin of autism (Irwin et al., 2011). Indeed, although Lorna Wing in her initial account of Asperger's work clearly stated her belief that AS and Kanner's autism were both part of an autistic spectrum, the idea of AS as an autonomous disorder, distinct from autism, quickly got hold of the opinion of many authors in the field, and certainly of the general public's curiosity for autism and related disorders (Wing, 1994). It is important to note here that Asperger himself referred to Kanner's paper, concluding that his subjects were clearly different from those described by Kanner (1943). The idea quickly made its way that Kanner's autism and AS were different disorders, distinguished mainly by the fact that AS children had good cognitive and linguistic skills and a normal development in the first 2–3 years of life (Klin, 2003). Moreover, for many authors the impairment in social interaction differed qualitatively between AS and Kanner's autism: while in the latter children seemed completely uninterested in others, AS children tried to relate with others but approached them in a dysfunctional and inconvenient way (Gillberg, 2002; Klin, 2003). Asperger himself contributed to this view that autism and AS subjects might be distinguished on the basis of cognitive ability and language development by emphasizing his patients' high intelligence and their acquisition of grammatical speech before they could walk (Wing, 1994). The number of publications on AS grew exponentially in the years following Wing's (1981) paper, and in 1994 AS was finally included in DSM – IV (**Figure 1**).

DEFINING ASPERGER SYNDROME: A TOWER OF BABEL

DSM-IV broadened the diagnostic boundaries of Autism, conceived for the first time as a spectrum of disorders that included Autistic Disorder, AS and Pervasive Developmental Disorder Not Otherwise Specified (American Psychiatric Association [APA], 1994). Also new was the inclusion of Childhood Disintegrative Disorder (Heller Syndrome) and Rett Syndrome, both characterized by developmental regression with severe autistic features (Matson and Mahan, 2009). A diagnosis of Autism required only six symptoms (in contrast with the minimum of eight required in DSM-IIIR), including at least two social interaction deficits, two communication deficits, and one symptom of interest restriction/repetitive behavior. Functional impairment had to be obvious before age three. The newly created category of AS required at least two symptoms of social interaction deficits and one symptom of behavioral and interest restriction, normal cognitive, and linguistic development before age 3, and age-adequate adaptive functioning in areas other than social interaction. Onset before age three was not mandatory. Importantly, the subject should not meet diagnostic criteria for Autistic Disorder – in which case the latter diagnosis should be given precedence, implying a differential diagnosis between AS and autism without cognitive delay, also called high-functioning autism (HFA; Klin et al., 2005). Meanwhile, other sets of diagnostic criteria for AS had appeared (**Figure 2**). In 1988 Carina and Christopher Gillberg (2002) proposed six criteria based on Asperger's original case-reports: socially impairing egocentricity, narrow interest patterns, compulsive routine adherence, peculiarities of speech and language, deficits in non-verbal communication, and motor clumsiness (Gillberg, 2002). Diagnosis required all six. There was no clause precluding a diagnosis of autism, and no mention of a minimum age limit or periods of normal development. 1 year later, Szatmari et al. (1989) proposed four mandatory criteria, comprising 22 symptoms: social isolation, impaired social functioning, deficits in non-verbal communication, and peculiarities of speech and language. As in DSM-IV, Autism was given diagnostic precedence over AS. Finally, WHO's 1993 International Classification of Diseases and Disorders (ICD-10) also suggested diagnostic criteria for AS, essentially similar to DSM-IV's (World Health Organization [WHO], 1992). Although not exactly contradictory, these several diagnostic schemes nevertheless produced a bewildering semiologic cacophony. Gillberg's criteria are too restrictive, and the only mentioning clumsiness as a mandatory symptom. Szatmari's criteria do not include interest restriction – a major criterion in the remaining diagnostic systems. DSM-IV and ICD-10 do not require abnormal non-verbal communication, mandatory in Gillberg's and Szatmari's sets. However, the most problematic clause, present in ICD-10, DSM-IV and Szatmari's criteria, was the exclusion of a diagnosis of AS if criteria for autism were met.

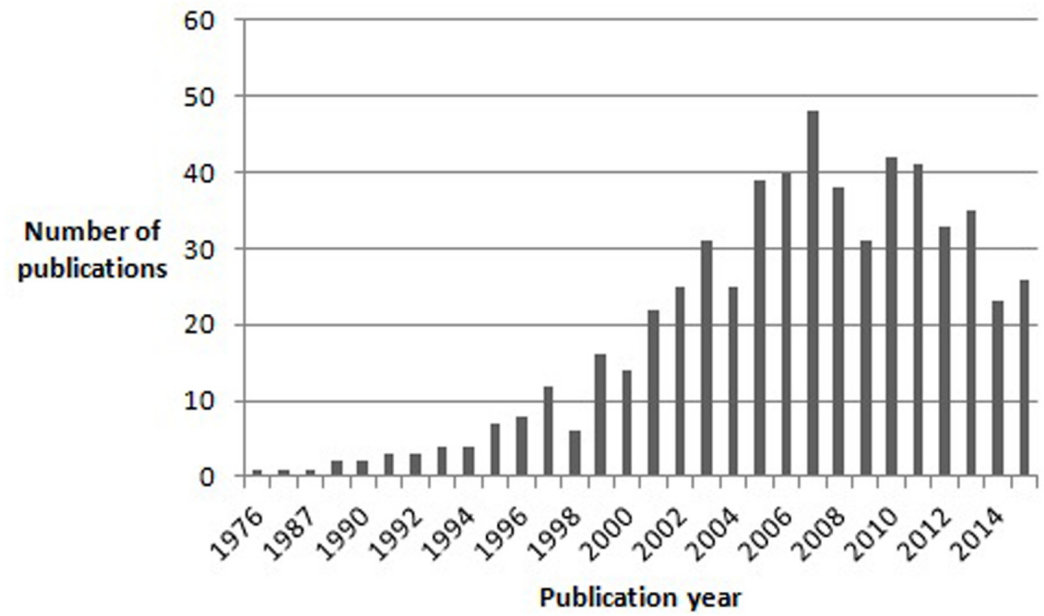

FIGURE 1 | Results of a pubmed search for articles containing the word "Asperger" in the title, published between 1976 and November 2015.

THE END IN THE BEGINNING

The consecration of AS as a distinct diagnosis was surrounded by controversy from the outset. Contradictions in the syndrome's definition soon became evident that would ultimately doom AS to extinction in DSM-5. The main problem was the precedence given to a diagnosis of autism. It soon became clear that most patients with significant impairments in social interaction and restriction of interests and activities also fulfill criteria for autistic disorder, thus precluding a diagnosis of AS. The requirement of normal cognitive and linguistic development failed to rescue a diagnosis of AS for the simple reason that cognitive and linguistic delay are not mandatory for diagnosing autistic disorder (Mayes et al., 2001; Happé, 2011). As Miller and Ozonoff (1997) demonstrated, even Asperger's own initial cases would fail to qualify for a DSM-IV diagnosis of AS. Moreover, it is often difficult to establish retrospectively if a patient had normal language before the age of three, and full-scale IQ is seldom a useful measure in AS, given the typically heterogeneous IQ profile (Gillberg, 2002; Spek et al., 2008). Consequences of this conundrum soon became visible in research. Researchers used AS and HFA as interchangeable diagnoses, modified DSM or ICD criteria, or used original, investigator-specific criteria, compromising comparability across studies (Klin et al., 2005). Gradually, two positions regarding AS emerged in the field: (1) diagnosing AS using DSM-IV criteria is impossible because AS does not exist and is indistinguishable from HFA; (2) DSM-IV's definition of AS is over-restrictive and additionally fails to discriminate AS from HFA (Szatmari, 2000; Mayes et al., 2001; Klin et al., 2005). Klin further argued that DSM-IV focuses excessively on superficial similarities between AS and HFA, ignoring AS's unique features: presence of social motivation with awkward, one-sided social approaches, normal or precocious language with pragmatic deficits and one-sided verbosity, pretend play of unusual content, and circumscribed interests with inordinate gathering of information (Klin et al., 2005; Baron-Cohen and Klin, 2006). Importantly (albeit inconsequently), Klin proposed a reversal of the precedence rule: in the presence of criteria for both HFA and AS, As should be diagnosed.

IS AS DIFFERENT FROM HFA?

Eventually, the controversy gradually converged onto knowing if AS and HFA can be distinguished qualitatively (suggesting different etiological and neurobiological mechanisms for each syndrome), or if they merely differ quantitatively and should therefore be regarded as variants of a single disorder (Macintosh and Dissanayake, 2004). Most studies used cross-sectional comparisons between subjects with either diagnosis to answer this question. Clinical differences between AS and HFA proved subtle at best. AS subjects have earlier language development, more appropriate intonation and pitch, and more pedantic speech and idiosyncratic vocabulary, while HFA subjects show more echolalia, pronoun reversal, and neologisms (Eisenmajer et al., 1998; Gilchrist et al., 2001; Macintosh and Dissanayake, 2004). AS children also display more imitative social play, attention and help-seeking, and reciprocal social interactions than HFA children (Prior et al., 1998; Macintosh and Dissanayake, 2006). Yet, these superior linguistic and social skills of AS children do not translate into superior ability to make friends or engage in reciprocal conversation. By adolescence, differences are no longer obvious, although AS subjects still show more sophisticated vocabulary and greater desire for friendship

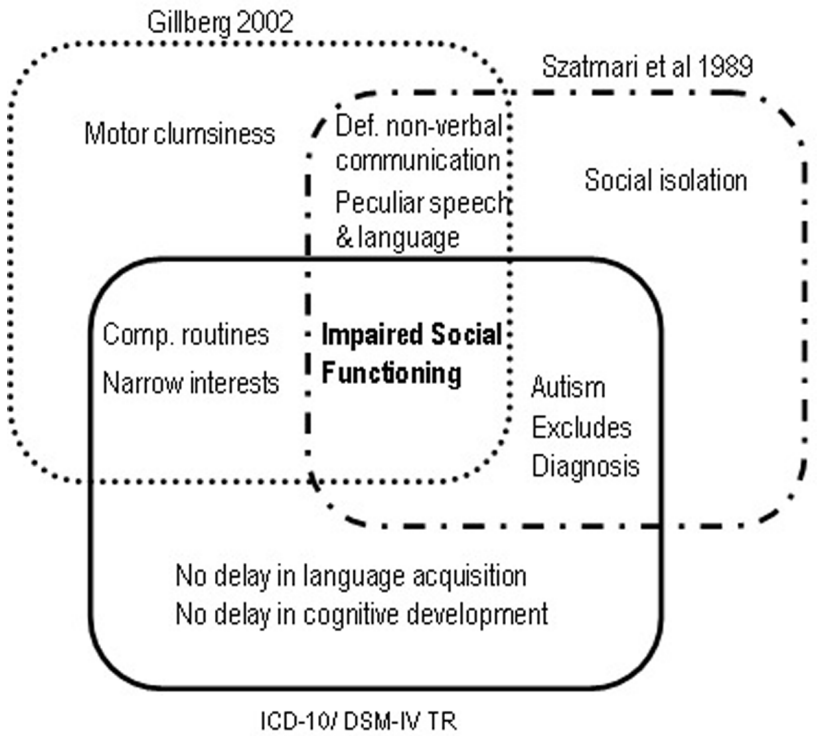

FIGURE 2 | Mandatory diagnostic criteria for Asperger Syndrome according to Szatmari et al. (1989), Gillberg (2002), ICD-10 and DSM-IV TR.

(Eisenmajer et al., 1998; Szatmari, 2000; Gilchrist et al., 2001; Wing et al., 2011). Cognitively, and as a group, AS subjects typically show a combination of superior verbal performance and visual-spatial, perceptual, and motor deficits (non-verbal learning disability profile), while the opposite profile characterizes HFA (Gillberg, 2002; Chiang et al., 2014). However, individual variability is huge, and it is difficult to control for the biasing effects of a differential diagnosis based on differences in language development (Klin et al., 2005). Studies that looked at theory of mind performance found mainly quantitative differences, with AS subjects scoring intermediately between HFA and healthy controls (Prior et al., 1998; Macintosh and Dissanayake, 2004).

Although many authors consider clumsiness as typical of AS, studies on motor control and gait have only found subtle differences in comparisons with HFA (Macintosh and Dissanayake, 2004; Rinehart et al., 2006). Again, differences decrease with age (Iwanaga et al., 2000). Finally, AS subjects show more intense preoccupations and circumscribed interests, while individuals with HFA have poorer imaginative play and more stereotyped behaviors, such as body rocking (Macintosh and Dissanayake, 2004; South et al., 2005). In terms of global functioning, AS subjects fare significantly better academically, but not in terms of employment or independent living (Tantam, 1994; Howlin, 2003).

An obvious approach to the question of whether or not AS and HFA are distinct entities is to look for biological differences between them. Despite the accumulated evidence on neurophysiological abnormalities in autism spectrum disorders (ASD) as a group, few data are available on possible differences between AS and HFA, apart from subtle differences in EEG connectivity patterns and left-hemisphere intra-cortical inhibition (abnormally decreased in HFA but not in AS; Duffy et al., 2013; Enticott et al., 2013; Luckhardt et al., 2014). Genetic studies have likewise produced little support for a discrimination between AS and HFA, although this must be tempered by growing evidence of a common genetic susceptibility shared by neurodevelopmental disorders in general, rather than a specific genetic etiology for each disorder (Lichtenstein et al., 2010).

Structural MRI studies comparing AS and HFA have produced contradictory results, with two recent meta-analyses and a recent systematic review reaching three different conclusions (Via et al., 2011; Yu et al., 2011; Pina-Camacho et al., 2013). The most consistent positive findings come from studies that differentiated AS from HFA based on language acquisition history: compared to AS and typical controls, HFA subjects have lower gray matter and white matter volumes, increased gyrification, and abnormal cortical folding in inferior frontal areas (including the pars opercularis; Nordahl et al., 2007; McAlonan et al., 2008, 2009; Jou et al., 2010); increased gray matter in supramarginal, superior temporal and inferior parietal gyri bilaterally (McAlonan et al., 2008; Jou et al., 2010; Toal et al., 2010), and decreased volume of the cerebellar vermis and posterolateral lobule (Scott et al., 2009; Hodge et al., 2010). However, these qualitative neuroanatomical differences are contradicted by studies that only found quantitative differences, with AS intermediately positioned between HFA

and typical subjects (Lotspeich et al., 2004; Haznedar et al., 2006).

On the whole, data on the distinctiveness between AS and HFA remain difficult to interpret. Many differences are quantitative rather than qualitative, and are distinctly more pronounced at younger ages, although the same might be said of many differences that discriminate HFA from typically developing subjects (Luckhardt et al., 2014). Moreover, research has been systematically plagued by difficulties ensuring independence between selection criteria and outcome measures (Macintosh and Dissanayake, 2004). Diagnostic and assessment methodologies vary wildly across studies, with the direction of findings influenced by the inclusiveness of diagnostic criteria for AS, notably by whether language acquisition delay was used as a criterion for diagnosing HFA (Via et al., 2011). Importantly, most studies are cross-sectional, missing potential differences between developmental trajectories in AS and HFA (Pina-Camacho et al., 2013). Indeed there is increasing evidence that AS and HFA correspond to distinct developmental trajectories (McAlonan et al., 2008; Lotspeich et al., 2004), with HFA marked by a delay or failure in the transition from right to left hemisphere dominance, occurring at around age 3 in typically developing children (Szatmari, 2000; Rinehart et al., 2002). Ultimately, the insuperable fragility of AS as a diagnostic entity resides in the lack of a biological marker, and in a phenotype that many see as insufficiently different from that of other related disorders.

A FORETOLD RESURRECTION?

Notwithstanding some clinical and biological differences between AS and HFA, the DSM-5 Neurodevelopmental Disorders Workgroup finally decided that there is insufficient evidence to support a meaningful distinction between them (Happé, 2011). DSM-5 therefore merged AS into a unitary category of autism spectrum disorders, characterized by a mandatory dyad of impaired social interaction and communication, and restricted, repetitive behaviors and interests (in contrast with the previously prevailing symptomatic triad; American Psychiatric Association [APA], 2013). All three symptoms of social interaction and communication deficits are required for a diagnosis. For the behavior and interest restriction criterion a polythetic definition was retained, albeit increasing the minimum of necessary symptoms from one to two, from a total of four (McPartland et al., 2012). Finally, a universal onset clause requires that symptoms be present from early childhood. Again, this profoundly modified definition of autism was met with criticism from the minute the first draft became known. Many patients and families, as well as adepts of the neurodiversity movement, were shocked that such an identity-defining diagnosis as AS ceased to exist overnight. Several specialists in the field felt that the decision was precipitate, that it ignored evidence supporting AS as a valid clinical and biological entity, and that DSM-5 fails to acknowledge the unique clinical features of those formerly diagnosed with AS (Wing et al., 2011; Spillers et al., 2014). Moreover, there are concerns that the new definition of ASD is too restrictive and will exclude many patients with AS from access to specialized treatment (Frazier et al., 2012; McPartland et al., 2012; Mayes et al., 2013). In fact, field trials showed that DSM-5 ASD has improved specificity at the cost of excluding more cognitively able individuals, including up to 75% of those previously diagnosed with AS (Frazier et al., 2012; Huerta et al., 2012; McPartland et al., 2012; Mayes et al., 2013). Concerns were further fueled by the inclusion of a new diagnosis of Social Communication Disorder in DSM-5, as this was felt by many to imply that higher functioning AS subjects would now migrate from the autistic spectrum to this new residual, consolation-prize category (Huerta et al., 2012). Others feel that the term AS should have continued to be mentioned in the manual as an admissible label for a particular group of patients within ASD, offering a clinical description of the syndrome but no diagnostic criteria (Wing et al., 2011). This would allow AS patients who regard the terms autism as unacceptably stigmatizing to keep their former diagnostic label. Indeed there is evidence that patients, families, education professionals, and health professionals connote AS with positive features and associate Autism with strange behavior, learning disability and family dysfunction (Kite et al., 2013; Spillers et al., 2014). Still others remain unreconciled with DSM-5 and hopeful of the syndrome's rebirth in future revisions of the manual (Tsai, 2013). Regardless of whether or not there will be a future for AS as a valid and meaningful clinical construct, its short existence had the undeniable merit of boosting the public's fascination with autism (Happé, 2011).

FUNDING

This work was supported by an unrestricted grant from Novartis Pharma.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

The reviewer João Gama Marques and handling Editor Diogo Telles-Correia declared their shared affiliation, and the handling Editor states that the process nevertheless met the standards of a fair and objective review.

frontiers in Psychology

REVIEW
published: 23 November 2015
doi: 10.3389/fpsyg.2015.01798

From Thought to Action: How the Interplay Between Neuroscience and Phenomenology Changed Our Understanding of Obsessive-Compulsive Disorder

J. Bernardo Barahona-Corrêa [1,2,3,4], Marta Camacho [3], Pedro Castro-Rodrigues [3,5], Rui Costa [6] and Albino J. Oliveira-Maia [2,3,6]*

[1] Department of Psychiatry and Mental Health, Faculdade de Ciências Médicas, Nova Medical School, Lisbon, Portugal, [2] Department of Psychiatry and Mental Health, Centro Hospitalar de Lisboa Ocidental, Lisbon, Portugal, [3] Champalimaud Clinical Centre, Champalimaud Centre for the Unknown, Lisbon, Portugal, [4] Centro de Apoio ao Desenvolvimento Infantil, Cascais, Portugal [5] Centro Hospitalar Psiquiátrico de Lisboa, Lisbon, Portugal, [6] Champalimaud Research, Champalimaud Centre for the Unknown, Lisbon, Portugal

OPEN ACCESS

Edited by:
Diogo Telles-Correia, University of Lisbon, Portugal

Reviewed by:
Sérgio Saraiva, Centro Hospitalar Psiquiátrico de Lisboa and University of Lisbon, Portugal
Lucas F. Manarte, University of Lisbon, Portugal

***Correspondence:**
J. Bernardo Barahona-Corrêa
bernardo.correa@fundacaochampalimaud.pt

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *04 August 2015*
Accepted: *07 November 2015*
Published: *23 November 2015*

Citation:
Barahona-Corrêa JB, Camacho M, Castro-Rodrigues P, Costa R and Oliveira-Maia AJ (2015) From Thought to Action: How the Interplay Between Neuroscience and Phenomenology Changed Our Understanding of Obsessive-Compulsive Disorder. Front. Psychol. 6:1798. doi: 10.3389/fpsyg.2015.01798

The understanding of obsessive-compulsive disorder (OCD) has evolved with the knowledge of behavior, the brain, and their relationship. Modern views of OCD as a neuropsychiatric disorder originated from early lesion studies, with more recent models incorporating detailed neuropsychological findings, such as perseveration in set-shifting tasks, and findings of altered brain structure and function, namely of orbitofrontal corticostriatal circuits and their limbic connections. Interestingly, as neurobiological models of OCD evolved from cortical and cognitive to sub-cortical and behavioral, the focus of OCD phenomenology also moved from thought control and contents to new concepts rooted in animal models of action control. Most recently, the proposed analogy between habitual action control and compulsive behavior has led to the hypothesis that individuals suffering from OCD may be predisposed to rely excessively on habitual rather than on goal-directed behavioral strategies. Alternatively, compulsions have been proposed to result either from hyper-valuation of certain actions and/or their outcomes, or from excessive uncertainty in the monitoring of action performance, both leading to perseveration in prepotent actions such as washing or checking. In short, the last decades have witnessed a formidable renovation in the pathophysiology, phenomenology, and even semantics, of OCD. Nevertheless, such progress is challenged by several caveats, not least psychopathological oversimplification and overgeneralization of animal to human extrapolations. Here we present an historical overview of the understanding of OCD, highlighting converging studies and trends in neuroscience, psychiatry and neuropsychology, and how they influenced current perspectives on the nosology and phenomenology of this disorder.

Keywords: habitual behavior, goal-directed behavior, orbitofrontal cortex, uncertainty, action value

INTRODUCTION

Obsessive-compulsive Disorder (OCD) is known to Western medicine at least since the Middle Ages (Berrios, 1996). The first historical accounts of OCD have been traced back to the 16th century, when people who suffered from repetitive blasphemous thoughts were believed to be possessed by demonic spirits (Muchembled, 2003). Phenomenologically, obsessions or obsessive thoughts have been considered to constitute the core of OCD. The term obsession derives from the Latin word *obsidere*, meaning to be possessed, occupied or preoccupied by something (Denys, 2011). It describes the occurrence of formal elements of thought (ideas, images, fears, doubts, ruminations) in a recurrent and persistent manner. These thoughts impose themselves on the individual, who experiences them as intrusive and anxiogenic, and incompatible with him or herself and/or his or her view of the world. In most cases, obsessive thoughts are accompanied by repetitive stereotyped behaviors, i.e., compulsions, from the Latin term *compellere*, meaning to be forced to something (Denys, 2011). Compulsions frequently assume a ritual-like nature and may either consist of motor acts (e.g., washing rituals) or purely mental acts (e.g., counting or praying; Burchard, 1980). Similarly to obsessions, compulsions are not in themselves pleasurable or gratifying to the patient. In most cases they are performed in order to reduce the anxiety evoked by obsessions, and are recognized by the subject as disproportionate or unrealistically related to the harm they are intended to avoid (Hollander et al., 2008).

Currently it is estimated that, in the USA, 2.3% of the adult population suffer from this condition (American Psychiatric Association, 2000). Moreover, there is evidence suggesting under-diagnosis (Fullana et al., 2009) and that isolated obsessive-compulsive symptoms (OCS) are extremely frequent in the general population (Grabe et al., 2001). OCD tends to present a chronic course and is frequently comorbid with mood disorders, eating behavior disorders and substance abuse (Torres et al., 2006). The World Health Organization (Ayuso-mateos, 2000), in an analysis of the indirect costs of the disorder (e.g., inability to work, impact on the family, early retirement) placed OCD in the 11th position of diseases with the greatest impact of non-fatal disease burden, at a level similar to schizophrenia in terms of years lost to disability.

From the early times of modern psychiatry, OCD has exerted a particular fascination on clinicians and researchers alike. Over more than a century of clinical and neurobiological research, OCD moved from being considered a typical neurosis (Black, 1974) to being the prototypical neuropsychiatric disorder, or at least, the most accomplished example of a mental disorder with clear underlying biological correlates. In the process, our understanding, and even our wording, of the disorder's manifestations changed radically (Berrios, 1989). Here we briefly review the current state of the art with regard to the neurobiology of OCD, and how neurobiological models have opened new perspectives on the phenomenology of this disorder.

CLASSIFICATION OF OCD

The first early modern descriptions of OCD already acknowledged the composite nature of this nosological entity. Indeed, in his *Études cliniques sur les maladies mentales et nerveuses*, Jules Falret was the first to comment on a clinical distinction between *folie du doute* (doubting madness) and *délire du toucher* (touching madness; Falret, 1890). More recently, factor analytical studies of OCS have consistently confirmed this notion and defined 4 to 5 clusters of OCS: 1. symmetry and "just right" obsessions with counting, ordering or repeating compulsions; 2. contamination obsessions with washing and cleaning compulsions; 3. hoarding compulsions; 4. aggressive obsessions with superstitious and checking compulsions; 5. sexual and religious obsessions (in four-factor models categories 4 and 5 are grouped together; Mataix-cols et al., 2005; Bloch et al., 2008; Landeros-Weisenberger et al., 2010). In a prospective study, it was shown that, even though specific obsessions may vary in individual patients over time, they generally tend to remain within the same broad symptomatic category (e.g., obsessions about body fluids evolve to excessive concern with contagious illnesses; Mataix-cols et al., 2005). Importantly, both structural and functional imaging studies support the hypothesis that particular neurobiological substrates underlie these different phenomenological dimensions of OCD (Menzies et al., 2008). Moreover, there is consistent evidence that distinct symptom dimensions may respond differently to specific treatments (Mataix-cols et al., 2005). In any case, such attempts to divide OCD into distinct entities have had a limited impact on the classification of the disorder.

Since its original inclusion in the first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-I; American Psychiatric Association, 1952), that classified OCD as a neurotic disorder, OCD has remained a single discrete nosological entity, included in subsequent editions of the manual within the broader category of the anxiety disorders. It was only in the very recent 5th edition of DSM that the nosological heterogeneity of OCD has been acknowledged for the first time, with hoarding being considered as a distinct, albeit related, disorder. Moreover, the two disorders were removed from the Anxiety Disorders category and placed in a novel category—Obsessive Compulsive and Related Disorders—comprising OCD and hoarding, as well as body dysmorphic disorder, trichotillomania and compulsive skin-picking. The decision to create this new nosological category was based on the increasing evidence that these disorders are related in terms of phenomenology, comorbidity, neural substrates, and treatment response (American Psychiatric Association, 2013). The new DSM category additionally reflects the notion that OCD constitutes one of the extremes of an obsessive-impulsive spectrum of disorders, that hold in common a failure of inhibitory behavioral control (Hollander et al., 2008). Psychopathology at the obsessive end of this spectrum mainly revolves around harm avoidance, with reward-seeking predominating at the opposite end of the spectrum and materializing in disorders such as pathological gambling or certain sexual paraphilia's. Although this view of an obsessive-impulsive spectrum has been challenged by accumulating evidence from both human and animal-based research showing that obsessiveness and impulsiveness are actually orthogonally related dimensions, the construct of an obsessive-impulsive group of syndromes, with psychopathology combining varying intensities of reward-seeking (impulsiveness)

and harm-avoidance (obsessiveness), continues to be supported by many authors (Fineberg et al., 2010).

NEUROBIOLOGY OF OCD

Neuroanatomy of OCD

The concept of OCD as a neuropsychiatric disorder of behavior emerged initially in the first quarter of the 20th century from observations that OCD frequently developed as a complication of encephalitis lethargica, and that subtle neurologic signs, similar to those present in recovered encephalitis patients, occur frequently in primary OCD (Schilder, 1938). Constantin von Economo was the first to describe, in 1931, the association between OCD and post-encephalitic Parkinsonism, and was also the first to suggest a causal link between basal ganglia lesions and secondary OCD (Schilder, 1938; Swedo and Snider, 2004).

Globally, the findings of structural and functional neuroimaging studies in OCD seem to converge on the orbitofrontal corticostriatal circuit and its limbic connections, mainly the cingulate gyrus and the temporal amygdala. The most consistent and replicated evidence from structural studies is of a reduction in gray matter and white matter volume of the orbitofrontal cortex (OFC), medial frontal gyrus, and anterior cingulate cortex (ACC), and increased volume of the ventral putamen (Pena-Garijo et al., 2010; De Wit et al., 2014). The most replicated findings of functional imaging studies tend to concentrate on these very same structures: most reports describe an increase in metabolic activity or in blood-oxygen-level dependent (BOLD) signal intensity in the orbitofrontal corticostriatal circuit, and a positive correlation between OCS severity and activity in that circuit (Westenberg et al., 2007; Pena-Garijo et al., 2010; Freyer et al., 2011). Furthermore, such activity is sensitive both to symptom provocation and to successful pharmacological or psychotherapeutic treatment. One study, combining meta-analysis of voxel-based morphometry studies and of functional neuroimaging studies with symptom provocation, demonstrated that, in OCD patients, the lateral OFC is the only brain region where structural findings coincide with functional findings (Rotge et al., 2010). It is, however, impossible to ignore the consistency of findings concerning other significant brain regions, namely the parietal cortex, the dorsolateral prefrontal cortex (dlPFC), the ventrolateral prefrontal cortex, the cerebellum and the caudate (Westenberg et al., 2007; Guehl et al., 2008; Menzies et al., 2008; De Wit et al., 2014).

Neurochemistry of OCD

Serotonin

Most of the evidence supporting a role for serotonin in the pathophysiology of OCD is based on the established therapeutic effects of serotonin reuptake inhibitors and the inefficacy of antidepressants with no effect on serotonin transmission or metabolism (Baumgarten and Grozdanovic, 1998; Westenberg et al., 2007; Goddard et al., 2008). Positron emission tomography (PET) and single positron emission computed tomography (SPECT) studies have shown a decreased availability of postsynaptic 5-HT2A receptors in the dlPFC, OFC and temporo-parietal cortices of OCD patients, as well as a reduced density of serotonin reuptake transporters in the striatum (Hesse et al., 2005; Westenberg et al., 2007; Perani et al., 2008). Both of these changes correlate with OCS severity. In spite of its popularity, the evidence base supporting the serotonergic model of OCD is mostly indirect and conceptually fragile. For many authors the available evidence suggests a role for serotonin not so much in the etiology of OCD as in the mechanism of action of the drugs used in its treatment (Baumgarten and Grozdanovic, 1998; Westenberg et al., 2007). In fact, only approximately half of OCD patients show any significant improvement when treated with serotonin reuptake inhibitors, a lower response rate than the one obtained in depression using the same agents. Moreover, in OCD patients, tryptophan depletion has no discernible effect on symptoms (Baumgarten and Grozdanovic, 1998; Westenberg et al., 2007).

In animal studies, evidence for a role of serotonin in compulsive-like behaviors has also been presented. In rats developing compulsive-like behaviors after OFC lesions, an increase of serotonin transporter density was found in the striatum (Joel et al., 2005). Stereotyped behavior can also be obtained pharmacologically, in rodents, by administration of 5-HT-2A antagonists (Fineberg et al., 2010), and decreased 5-HT-2A receptor activation decisively impairs reversal learning by increasing the number of perseverative responses to previously reinforced stimuli (Boulougouris et al., 2008). Furthermore, systemic administration of 5-HT-2A antagonists, or local infusion into the nucleus accumbens or medial prefrontal cortex (mPFC), *reduces* impulsivity in animal models, which might partially explain the limited success of "serotonergic" treatments in OCD (Boulougouris et al., 2008). Interestingly, while 5-HT-2C knock-out mice were found to develop compulsive chewing of a non-nutritive substance in one study (Chou-Green et al., 2003), treatment with the non-specific 5-HT-2C agonist m-chlorophenylpiperazine acutely induces OCS in humans and compulsive-like behaviors in animals (Tsaltas et al., 2005; Fineberg et al., 2010). 5-HT-2C antagonists, on the other hand, decrease perseverative and total number of trials in reversal learning tasks, and exacerbate premature responses (i.e., impulsivity), when administered systemically or when infused directly into the nucleus accumbens (Winstanley et al., 2004; Boulougouris et al., 2008).

Dopamine

One of the main arguments for a role of dopamine in the pathophysiology of OCD is the beneficial effect of antipsychotic medication in many cases that are resistant to SSRIs, particularly when tics are present (Miguel et al., 2001; Perani et al., 2008). Furthermore, OCS are frequent in movement disorders associated to hyperdopaminergic dysfunction, such as Sydenhams's chorea or Huntington's disease. In Parkinson's disease, OCD and other behaviors of the impulsive-compulsive spectrum have been described, mostly, though not exclusively, in patients treated with high doses of L-dopa (Evans et al., 2004; Koo et al., 2010). Finally, there has been extensive demonstration of both the induction of *de novo* stereotyped compulsive-like behaviors and the aggravation of pre-existing OCS by drugs that stimulate dopamine receptors directly (e.g., bromocriptine, apomorphine) or indirectly (e.g., amphetamines, cocaine, methylphenidate; Denys et al., 2003;

Perani et al., 2008; Koo et al., 2010). Treatment with bupropion, a dopamine reuptake inhibitor, has also been shown to aggravate OCD symptoms in a small trial (Vulink et al., 2009).

Similarly to what has been described for serotonin, direct evidence of a role for dopamine in the neurobiology of OCD also derives from PET and SPECT studies, which have revealed an increased density of dopamine transporter (DAT; Landeros-Weisenberger et al., 2010) and a reduction of D2 receptor availability in the basal ganglia (Denys et al., 2003; Kim et al., 2003). In some studies these changes were reverted after treatment with a selective serotonin reuptake inhibitor (SSRI; Kim et al., 2003; Moresco et al., 2007). One study reported a lower availability of D1 receptors in the striatum of OCD patients when compared to healthy subjects (Olver et al., 2009). In line with these findings, DAT knockout mice, which present synaptic dopamine levels 70% or more above normal, have stereotyped grooming behaviors, especially when exposed to unfamiliar environments (Wang et al., 2012). Furthermore, an often-cited animal model for OCD consists of repeated subcutaneous injections of the D2-agonist quinpirole, which leads to stereotyped, compulsive-like behavior in rats. This phenotype reverts with clomipramine treatment and subthalamic nucleus (STN) inactivation through deep brain stimulation (DBS; Winter et al., 2008; Klavir et al., 2009).

A potential role for dopamine in OCD has been frequently interpreted in the context of evidence for the participation of this neurotransmitter in reinforcement learning (Pizzagalli et al., 2008; Wise, 2009), including negative reinforcement learning, i.e., the performance of a behavior to prevent anticipated adverse consequences (Pessiglione et al., 2006). In OCD patients, a hypothetical excess of dopaminergic input into the ventral striatum could lead to excessive aversion avoidance behaviors, as well as to failures in reversal learning (Wunderlich et al., 2011). This prediction was recently partially confirmed in a study showing that, in a shock avoidance task, OCD patients will sustain avoidant responses beyond the interruption of shock delivery (Gillan et al., 2014a). Additionally, mesocorticolimbic dopaminergic hyperactivity could enhance the reinforcement of anxiety-reducing stereotyped actions, the performance of which would thus become rewarding and confer to compulsions properties akin to those of other "behavioral addictions" (Holden, 2001; Aouizerate et al., 2004; Wise, 2009). However, there is also evidence that ventral striatum activation during reward anticipation is blunted in OCD, which may be inconsistent with this hypothesis (Figee et al., 2011).

While there is extensive evidence pointing toward increased synaptic concentration of dopamine in the striatum of OCD patients, there is also evidence suggesting the reverse (Sesia et al., 2013). One SPECT study found reduced striatal DAT density in drug-naïve OCD patients when compared to healthy controls (Hesse et al., 2005). In another study midbrain DAT density was increased after citalopram treatment (Pogarell et al., 2005). Moreover, the risk of developing OCD appears to be increased in chronic cocaine abusers, who have down-regulated dopamine receptors and dopamine function (Crum and Anthony, 1993). There are also isolated reports of OCD cases where treatment with amphetamine, methylphenidate or bromocriptine led to an improvement of OCS—an intriguing observation given the often described aggravation of symptoms in OCD patients exposed to these substances (Denys et al., 2003; Perani et al., 2008; Koo et al., 2010). Furthermore, most atypical antipsychotics, and even haloperidol, can, in some patients, cause significant OCS (Lykouras et al., 2003). In summary, there is a definite role for dopamine, and possibly dopamine-signaled reward processing, in the pathophysiology of OCD, but the more exact characteristics of this role are still unclear.

Glutamatergic and GABAergic Neurotransmission in Corticostriatal Circuits

Recent findings suggest the involvement of other neurotransmitter systems, beyond serotonin and dopamine (Kariuki-Nyuthe et al., 2014). The unequivocal evidence that corticostriatal pathways are hyperactive in OCD has led to research into changes of glutamatergic neurotransmission, specifically at corticostriatal synapses. There is some evidence that, in OCD patients, the concentration of glutamate in the cerebrospinal fluid is increased (Ting and Feng, 2008), and magnetic resonance spectroscopy studies have found evidence of increased glutamate levels in the OFC, ACC, and striatum (see Naaijen for a review; Naaijen et al., 2015). In some studies, striatal glutamate levels have been found to correlate with OCS severity (Starck et al., 2008), and to decrease after pharmacological treatment or cognitive-behavioral (Rosenberg et al., 2000; O'Neill et al., 2013). Furthermore, some genes of interest in OCD research, identified in animal models of the disorder, are related to glutamatergic receptors (such as the GRIN2B and SLC1A1 genes) or to corticostriatal glutamatergic synapses (SAPAP3 gene; Wan et al., 2014). Consequently, in the last years, several pharmacologic agents that modulate glutamatergic transmission have been tested in OCD, such as memantine, topiramate, and riluzol (that reduces the synaptic release of glutamate). While the results are, in general, promising, these studies are still rare, mostly unreplicated, and based on small samples (Ting and Feng, 2008).

Gamma-aminobutyric acid (GABA), the main inhibitory neurotransmitter in the central nervous system, has been much less studied in OCD. Magnetic resonance spectroscopy studies found decreased levels of GABA in prefrontal cortical areas, including the ACC (Simpson et al., 2012), with one study reporting that an acute increase in mPFC GABA levels coincides with OCS relief after successful ketamine treatment (Rodriguez et al., 2015). Moreover, studies using transcranial magnetic stimulation found shortened cortical silent periods and increased intracortical facilitation in the left motor cortex of OCD patients—both of which are considered measures of $GABA_B$ receptor mediated neuronal inhibition (Richter et al., 2012). Taken together, these observations have been interpreted as reflecting reduced activity or number of cortical GABAergic interneurons which could, both directly and indirectly (through ACC to OFC projections), lead to abnormal striatal activation (Simpson et al., 2012). In line with this hypothesis, an association has been found between polymorphisms in the $GABA_B$ receptor 1 gene and OCD (Zai et al., 2005). Finally, studies in primates have shown that injecting a $GABA_A$ receptor antagonist (bicuculline) in specific

areas of the ventral striatum leads to OCD-like behavior changes (Worbe et al., 2009).

Genetic Studies in OCD

Environmental factors are relevant for the occurrence of OCD (Krebs et al., 2015). Maternal consumption of alcohol or caffeine during pregnancy, *hyperemesis gravidarum* or dystocic labor, adverse life events, including childhood physical or sexual abuse, were all found to increase the risk of developing this disorder (Cath et al., 2008). Nevertheless, it is not rare to uncover, in patients with OCD, a family history of the disorder, especially in cases with disease onset during childhood or adolescence, with co-morbid tics or with a predominance of symmetry/organization symptoms (Nicolini et al., 2009; Samuels, 2009). Thus, a possible role for genetic factors in OCD has been explored since at least 1929 (Lange, 1929). While many of the earlier studies had important methodological limitations (Pauls, 2010), more recent twin studies have shown that 67.5% of homozygous twins are concordant for OCD, with 31% concordance in dizygous twins (Aouizerate et al., 2004). The estimated contribution of genetic factors for the risk of developing OCD varies between 45 and 65% for child-onset disease, and 27 to 47% for adult-onset OCD (Nicolini et al., 2009), with genetic association studies pointing mostly to genes related to the serotonergic and dopaminergic systems (Frisch et al., 2000). Importantly, theoretical models derived from genetic segregation analysis suggest the simultaneous existence of mendelian (monogenic) and polygenic genetic transmission (Nicolini et al., 2009), with the description, in linkage studies, of major genetic loci in chromosomes 1, 3, 6, 7, 9, 10, 11, 14, and 15 (Samuels, 2009).

Neural Circuit Models of OCD

From the currently available information, two models have been proposed regarding OCD pathophysiology: the "cortical" model, centered primarily on frontal cortex dysfunction, and the "subcortical" model, that attributes OCD to dysfunction of the basal ganglia and their cortical connections (Modell et al., 1989; Deckersbach et al., 2006). The proponents of both models tend to focus on the importance of neurodevelopmental factors, given the frequent onset during childhood or adolescence, the comorbidity with developmental disorders, namely autism spectrum disorders, and the presence of discreet neurological signs in patients suffering from OCD (Bradshaw, 2001).

The cortical model for the neurobiology of OCD essentially proposes that OCD results from an imbalance between hyperactive OFC and ACC and hypoactivity of the dlPFC. According to this model, hyperactivity of the OFC and ACC, which has been consistently demonstrated in OCD, elicits egodystonic behavioral commands and uncertainty error signals (Bradshaw, 2001; Westenberg et al., 2007). These error signals are resistant to feedback information from sensory and limbic areas leading to persistent ruminations of uncompleteness or doubt and behavioral perseveration with stereotyped responses such as verification and repetition, aiming to reduce uncertainty (Pena-Garijo et al., 2010). Activity in the nucleus accumbens and amygdala would be secondarily modulated through extensive reciprocal connections with the OFC and ACC, leading to the profound anxiety and apprehension that is characteristic of OCD (Westenberg et al., 2007). Recent data on resting-state corticostriatal connectivity in children with OCD are broadly consistent with this hypothesis: reduced connectivity between the dorsal striatum and ACC coexists with increased connectivity between both dorsal and ventral striatum and medial frontal cortical areas involved in emotional processing (Fitzgerald et al., 2011). The net result would be an inability to suppress pre-potent security concerns and error signals while simultaneously investing them with excessive emotional salience (Fitzgerald et al., 2011; Baioui et al., 2013). Also consistent with this ventral-dorsal imbalance cortical model, decreased connectivity between the caudate nucleus and the dlPFC has been shown in OCD, possibly contributing to the cognitive inflexibility that is described in OCD, and participating in the inability to suppress thoughts and behaviors that are maladapted to environmental circumstances (Westenberg et al., 2007; Harrison et al., 2009). The classical cortical model thus ascribes OCD to a primary prefrontal dysfunction, essentially consisting of an imbalance between decreased dlPFC activity and increased activity in the OFC and ACC (McGuire, 1995; Fuster, 1997; Aouizerate et al., 2004). A less cited variant of the cortical model of OCD considers that intrusive feelings of anxiety originate from primary hyperactivity of the temporal amygdala. Such hyperactivity would lead to a secondary supplementary inhibitory effort from the medial and orbital prefrontal cortex. This supplementary inhibitory effort would be compromised by mesolimbic dopaminergic hyperactivity, that has been shown to reduce cortical inhibitory control over the amygdala (Denys et al., 2003; Westenberg et al., 2007).

The subcortical model of OCD neurobiology, first suggested by Modell (Modell et al., 1989), is currently the most popular and most frequently cited of the two models. According to this author, OCD results from a failure of the ventral pallidum to inhibit the mediodorsal thalamic nucleus and its connections to the OFC. This pallidal dysfunction would, at least in part, result from an unbalance between the activity of the main ventral striatum midbrain afferents, namely excitatory dopaminergic pathways from the ventral tegmental area, and inhibitory serotonergic pathways from the raphe nuclei (Aouizerate et al., 2004; Westenberg et al., 2007). The functional consequence would be an "anomalous reverberation" of the orbitofrontal cortico-striato-thalamo-cortical circuit. From a neurochemical perspective, the subcortical models are consistent with the role traditionally ascribed to hypoactive ascending serotonergic pathways in the physiology of OCD and with current evidence supporting a role for dopamine in the neurobiology of OCD and other disorders of the obsessive-compulsive spectrum.

In the last two decades, the subcortical model has been reviewed and perfected to accommodate novel research findings. According to the update proposed by Baxter et al (Baxter, 1987, 1992; Valente et al., 2005), OCD would result from a disruption of the caudate's "filter" function. The consequence would be a self-sustained, reverberant release of automatic programs, with a need for supplementary efforts to maintain adequate responses to relevant stimuli (Baxter, 1987, 1992). At the microstructural level, dysfunction of the caudate would mainly involve the striosomal compartment, which is predominantly implicated in

the direct pathway of the basal ganglia. Striosomes are particularly numerous in the ventromedial striatum, where they receive afferents from the OFC and ACC (Eblen and Graybiel, 1995; Aouizerate et al., 2004). In the Baxter model, hyperactivity in limbic and orbitofrontal corticobasal circuits is compounded by hypoactivity of the dlPFC, which has been consistently described in functional neuroimaging studies of OCD (Aouizerate et al., 2004; Menzies et al., 2008; Nicolini et al., 2009). Projections from the dlPFC to neurons in the striatal matrix are proposed to influence the indirect pathway, thus participating in the interruption of automatic behaviors and the adaptive switch from one behavioral program to another (Baxter, 1987, 1992).

Cortico-subcortical Models

In 2005, Chamberlain updated Baxter's model, centering OCD pathophysiology on the orbitofrontal corticostriatal circuit and its role in the acquisition and maintenance of stereotyped and automatic cognitive and behavioral patterns (Chamberlain et al., 2005; Menzies et al., 2008; Fineberg et al., 2010). The dysfunction of this network was proposed to explain the inability to suppress intrusive thoughts and automatic behaviors, as well as the deficits of psychomotor and cognitive inhibition that are abundantly described in OCD (i.e., the impulsive dimension of OCD phenomenology). These deficits of cognitive inhibition would in turn impair cognitive flexibility, that requires inhibition of prior cognitive processes, and working memory, that requires active suppression of distracting elements, both of which sustain the compulsive dimension of the OCD's phenotype (Chamberlain et al., 2005). The greatest merit of this model is that it reassigns a decisive role to cortical dysfunction and top-down cortical control of frontostriatal circuits (Fineberg et al., 2010). It thus avoids the somewhat arbitrary dichotomy between cortical and subcortical models. Moreover, it is more in line with recent evidence that the direct and indirect pathway do not necessarily exert a competitive influence on action control or selection, but rather act in coordination for adaptive action control (Cui et al., 2013).

Central to all of the successive subcortical models of OCD lies the concept of subtle dysfunction of the ventral caudate and of its role in activating and maintaining behavioral programs that are adaptive to updated information, transmitted by associative cortical regions (Aouizerate et al., 2004; Westenberg et al., 2007). Support for a central role of the caudate in the physiology of OCD has been provided by the demonstration, *in vivo*, of an abnormally high rate of neuronal depolarization in the caudate of patients with severe OCD undergoing DBS (Guehl et al., 2008). Furthermore, modulation of orbitofrontal corticostriatal circuitry using optogenetics can induce or decrease obsessive-compulsive-like behaviors in mice (Burguière et al., 2013; Ahmari et al., 2014), and DBS of frontostriatal targets has been shown to ameliorate OCS in OCD patients (Figee et al., 2013). Indeed, trials of DBS for OCD have revealed an important role for the STN in this disorder. It has been shown that the cortico-STN pathway (i.e., the hyperdirect pathway) is required to interrupt ongoing automatic motor and behavioral programs, before they become overtly expressed (Wylie et al., 2010; Alegre et al., 2013; Anzak et al., 2013). Consistent with this, in a recent study of OCD patients submitted to DBS, OCD severity was related to neuronal activity in the associative-limbic subdivision of the STN, with shorter bursts and interburst intervals, as well as higher intraburst frequency, in OCD patients (Welter et al., 2011).

THE EVOLVING NEUROPSYCHOLOGY OF OCD

Despite the substantial body of literature published over the last 25 years on neuropsychological performance in OCD, studies have yielded inconsistent results, mainly attributable to methodology differences (Abramovitch et al., 2013). The cognitive deficits that are more consistently described in OCD patients are found in tasks of non-verbal memory, response inhibition, interference control, cognitive flexibility, and visuospatial working memory, with executive dysfunction being the hallmark of OCD neuropsychological profile (Kashyap et al., 2013; Abramovitch and Cooperman, 2015). Despite the evidence of impaired executive functioning in OCD, it is not consensual whether these deficits are trait-related (stable individual characteristics) or state-dependent (dependent on the presence of symptoms). In general, there seems to be more support for the former hypothesis, since there is no correlation between the intensity of cognitive changes and the severity of OCS. In a longitudinal study, where executive functions were tested in OCD patients both before and after symptomatic remission, executive function deficits were found to be stable over time and independent of symptom remission (Bannon et al., 2006). Furthermore, treatment of OCS does not lead to improvements in cognitive performance suggesting that, in OCD, cognitive deficits are not merely a side effect of the core psychiatric symptoms (Kuelz et al., 2004).

Taken together, most studies suggest that cognitive deficits in OCD reflect OFC and dlPFC dysfunction as well as, possibly, temporo-parietal dysfunction of the non-dominant hemisphere (Whiteside et al., 2004; Menzies et al., 2008). In functional brain-imaging studies comparing the pattern of cortical activation between OCD patients and healthy individuals, cognitive deficits are associated both with decreases and increases of cortical BOLD signal or glucose metabolism over the OFC, dlPFC, and temporo-parietal cortices (Balleine and O'Doherty, 2010; Banca et al., 2015). The reason for such incongruences is unclear, but it has been ascribed both to state-dependent differences in cortical activation (Page et al., 2009; Banca et al., 2015), and to discrepancies in the interpretation of results from different imaging techniques (Whiteside et al., 2004).

Several authors have also underlined the analogy between core obsessive-compulsive psychopathology and cognitive deficits in OCD patients. Specifically, the inability to repress compulsive behaviors is interpreted in the context of difficulties in performing response inhibition tasks, while the inability to interrupt obsessive thoughts is related to difficulties in performing cognitive flexibility tasks (Pena-Garijo et al., 2010). Others have further suggested that, more than a simple phenomenological analogy, a genuine correspondence exists between impulsivity and response inhibition deficits, and between compulsivity and deficits in cognitive and behavioral flexibility (Fineberg et al., 2010). Here,

impulsivity is defined as a predisposition toward rapid, unplanned reactions to internal or external stimuli, with little regard for the adverse consequences of these actions. Compulsivity on the other hand is defined as a tendency to perform unpleasant repetitive actions in a stereotyped manner, in order to prevent perceived or anticipated negative consequences of not performing these actions. These behavioral dimensions are proposed to be sustained by two parallel corticostriatal circuits: a compulsive circuit, where compulsive behaviors are driven by the dorsal striatum and inhibited by the OFC; and an impulsive circuit where behavior is driven by the ventral striatum/nucleus accumbens shell, and inhibited by the ACC and ventromedial prefrontal cortex (vmPFC; Balleine and O'Doherty, 2010; Fineberg et al., 2010). In summary, the two core neuropsychological deficits described in OCD not only parallel the two main behavioral dimensions of this disorder—impulsivity and compulsivity—but also converge strikingly with the host of data pointing to structural and functional changes in the limbic and associative corticobasal circuits, and to deregulation of the two main midbrain monoaminergic ascending pathways (Aouizerate et al., 2004; Fineberg et al., 2010).

OCD and Action Control

More recently, advances in our understanding of the neurobiology of action control, specifically the distinction between goal-directed and habitual action strategies, have opened new perspectives on the neurobiology of OCD. Goal-directed behaviors are those that are sensitive to revaluation of the rewarding outcome (e.g., feeding to satiation), and also to degradation or extinction of the contingency between performing the action and obtaining the outcome. Habitual behavior, on the other hand, persists even when the outcome is no longer rewarding or the action-outcome contingency is degraded or reversed. The phenomenological analogy between habitual action control and compulsive behavior has led to the hypothesis that OCD patients may be predisposed to rely excessively on habitual action control rather than on goal-directed behavioral strategies (Gillan et al., 2011; Robbins et al., 2012). Evidence that patients suffering from OCD present structural and functional changes in the striatum and mPFC, areas so frequently implicated in the control of transitions between action strategies (Tricomi et al., 2009; Balleine and O'Doherty, 2010), further strengthens this hypothesis. Recently, in a series of studies with OCD patients, using diverse decision making paradigms, Gillan and colleagues provided empirical support for the hypothesis that such patients are biased toward habitual action control patterns, in comparison with control subjects (Gillan et al., 2011, 2014a), and fail to rely on prospective comparisons of action-outcome alternatives to guide decision making (Gillan et al., 2014b). In an fMRI study with symptom-provocation in OCD patients, a pattern of activation/deactivation was evoked, suggesting an imbalance in circuits involved in the control of habitual and goal-directed action strategies (Banca et al., 2015). In fact, the authors describe deactivation of caudate-prefrontal circuits simultaneous to hyperactivation of putamen and STN regions, which was not found in control conditions, or in healthy subjects. Importantly, in additional connectivity analyses, the vmPFC and OFC were found to be a critical node in the circuits involved in symptom provocation (Banca et al., 2015).

However, the parallel between habitual action patterns and the elaborately ritualized, egodystonic behaviors that plague OCD patients may be an over-simplification. Importantly, the approaches used to date to study action control patterns in OCD generally fail to take into account the fundamental role of habits in the formation of chunked action-sequences, i.e., relatively invariant, rapidly deployed sequences of single action-units (Ostlund et al., 2009; Dezfouli and Balleine, 2013). Action sequences, like habits, are mainly dependent on the sensorimotor striatum, with each action unit being triggered by the antecedent action rather than by environmental stimuli. Additionally, in action sequences, assessments of individual action-units and of inter-action states are bypassed (Dezfouli and Balleine, 2013). This could be, to some degree, the opposite of what is frequently described by OCD patients, namely the failure to develop adaptive and automatic action-sequences, performing them with effort, in constant doubt as to their correct execution and hampered by continuous state re-evaluation (Reuther et al., 2013). In fact, in a rat model of pharmacologically-induced compulsivity, evidence was presented that the inability to change between two actions is related to the occurrence of an abnormal phasic dip in VTA dopamine neuron burst firing at the completion of a compulsive-like action sequence, leading to the recurrent activation of the same action, rather than the expected transition to a new action sequence (Joel and Doljansky, 2003). It is interesting, in this respect, that OCD and OCS are particularly frequent in movement disorders that also disrupt the normal chunking of action sequences, such as chorea, Parkinsonism or primary dystonia (Tremblay et al., 2010; Barahona-Corrêa et al., 2011).

The above-mentioned failure to develop adaptive and automatic action-sequences could be interpreted in the context of maladaptive goal-directed uncertainty monitoring, leading to repetitive behaviors, such as checking, in order to reduce uncertainty. This possibility is consistent with the evolving evidence on the role of the STN in the regulation of behavior, and more specifically in the neurobiology and psychopathology of OCD. As mentioned previously, the STN has been one of the targets for DBS in OCD. Linked to frontal cortical areas by the so-called hyperdirect pathway (Nambu et al., 2002), the STN could constitute a potential route of entry, into striatal circuits, of higher-level decision signals for regulation of behavior (Weintraub and Zaghloul, 2013). One of these areas is the OFC, where, in rats, neurons have been shown to encode uncertainty regarding stimulus identity (Kepecs et al., 2008). Since, in OCD patients engaged in repetitive checking bouts, STN neuron spike rates increase prior to the repetition of checking actions (Burbaud et al., 2013), such neural activity changes could reflect uncertainty signals, possibly originating in the OFC, and leading to repetitive goal-directed behaviors, in an attempt to reduce uncertainty.

One other unresolved question regarding the predominance of habitual action strategies in OCD is that such strategies result in highly adaptive action control patterns, allowing the subject to engage in alternate and cognitively demanding tasks, while

performing a given well-learned action. This actually seems to be impaired in OCD patients, who not only engage explicit memory-linked medial temporal structures when performing implicit learning tasks (Rauch et al., 2007), but also show impaired sequence learning when faced with parallel, simultaneous explicit and implicit processing demands (Deckersbach et al., 2002). An alternative proposal is that OCD may result from an inability to inhibit the motivation to perform a compulsion, rather than simply from a bias in action control toward habitual behavior (Hinds et al., 2012). Repetition of compulsive behaviors would thus result from a primary overvaluation of action outcomes, with over-activity of the goal-directed action control system that is, by definition, sensitive to outcome values. In an example conceived according to this model, environmental danger-signaling cues could activate the motivation to engage in stereotyped, safety behaviors such as washing or checking, but performance of these actions would fail to effectively terminate the motivational state that elicited them, thus prompting repetition of the behaviors (Woody and Szechtman, 2011; Hinds et al., 2012).

FROM NEUROBIOLOGY TO PHENOMENOLOGY

It is intuitive that the biological understanding of medical disorders should progress from semiology to etiology, rather than the opposite. For most medical disorders, understanding the physiology of signs and symptoms paved the way to the formulation of pathophysiological models and ultimately to the discovery of the disorder's primary causation. Yet, in the case of OCD, it is clear that the evolution in our understanding of the disorder's pathophysiology has had a profound impact on our conceptualization of the disorder's phenomenology. Indeed we may say that the gradual dislocation of the main focus of pathophysiological models of OCD from cortical to subcortical structures has been paralleled by a metamorphosis of the conceptualisation of OCD phenomenology, whereby the initial emphasis laid on the control of explicit thought processes, thought contents, and thought-evoked anxiety, has been replaced by concepts rooted in behavioral neuroscience and animal models of action control. Contemporary literature on OCD is dominated by new phenomenological concepts, and even a new semantics for OCD-related concepts, now seen as characterized by a loss of inhibitory control over impulsive stimuli-response associations, by excessive harm-avoidance, or by a failure to flexibly shift between alternative action-sequences in response to changing environmental demands. Similarly, new insights into the role of dopamine and reward circuits in OCD led researchers in the field to emphasize the phenomenological analogies between addiction disorders and OCD, regarded here as a form of behavioral addiction, a disorder combining impulse control dysfunction with abnormal reward processing mechanisms. These phenomenological metamorphoses found echo in the very nosology of OCD, which in DSM-V was emancipated from anxiety disorders and raised to a new statute as an autonomous spectrum of disorders of behavior control.

In very recent years, the phenomenological analogy between habitual action control and compulsive behavior brought yet another renewal of the semantics and the phenomenology of OCD. The idea that OCD patients may be predisposed to rely excessively on habitual rather than on goal-directed behavioral strategies now faces the field with a host of new concepts stemming from animal experimentation. To some degree, there seems to be a tendency to reduce the psychopathology of OCD to a deregulation of automatic behavior and pathological streams of non-functional stimuli-response associations, downplaying the role of cognition in the process. At the extreme of this evolution from a cognitivist toward a behavioral control centered conceptualisation of OCD, obsessions are eventually seen as secondary, retrospective cognitive constructions developed by the subject to assimilate these intrusive behavioral programs back into the self (Gillan et al., 2011). Compulsions, on the contrary, gradually emerge as the new protagonist in OCD phenomenology, either as merely perseverative phenomena, as stereotyped behaviors pathologically invested with reward value, or as abnormally resilient habitual action-sequences.

CONCLUSION

Science has come a long way in understanding OCD. It has been a unique process, marked by dialectic interplay between the progress in neurobiological models of OCD and an increasingly sophisticated phenomenological characterization of this complex syndrome. Refined structural and functional imaging of the brain and technological progress in animal experimental modeling of OCD have opened the way to groundbreaking insights into the role of cortical and subcortical structures in the disorder's pathophysiology, and revolutionized our perception and interpretation of obsessive-compulsive phenomena. Yet, in spite of much progress, we still lack an explanation for many of the disorder's manifestations, and not least its extraordinary clinical variability. Extrapolations from animal models, themselves extrapolations of human phenomenology, while carrying extraordinary promise to further understanding of the disorder, necessarily ignore many complex aspects of human psychopathology. Care is thus needed to carefully cross-validate human-animal-human extrapolations, as well as analogies between psychopathology and constructs from experimental psychology and behavioral neuroscience literature, while avoiding impoverishing an extremely rich legacy of psychopathological detail and subtlety. In common with other psychiatric disorders such as bipolar disorder or schizophrenia, neuroimaging studies in OCD are often contradictory and difficult to interpret. This is due not only to phenotypic variability across subjects, but also to the far from clear meaning of volumetric and BOLD-signal differences between clinical subjects and healthy controls. Genetic studies have likewise failed to provide a much anticipated breakthrough in understanding and treating OCD and other mental disorders, probably because genetic factors lie too far upstream in the complex flow of factors and events that ultimately lead to the expression of mental illness in a given individual. A renewed interest for psychopathological refinement can certainly contribute toward the advancement of knowledge on the neurobiology of mental disorders in general, beyond explanation of each syndrome's gross features and toward

tackling of more subtle manifestations and of subtypes of each disorder. Nevertheless, the future also calls for more detailed studies using, for example, genetic animal models, improved neuroimaging technology, *in vivo* neuronal recordings and non-invasive neuromodulation in humans, in order to more fully elucidate the mechanisms that may underlie the details of OCD psychopathology.

ACKNOWLEDGMENTS

Preparation of this manuscript was supported by an ERA-NET NEURON grant to RMC and a Junior Research and Career Development Award from the Harvard Medical Portugal Program and Fundação para a Ciência e Tecnologia (HMSP/ICJ/0020/2011) to AJO-M.

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Conflict of Interest Statement: The Reviewer Sérgio Saraiva declares that, despite being affiliated to the same institution as the author Pedro Castro-Rodrigues, the review process was handled objectively and no conflict of interest exists. The other authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in Psychology

MINI REVIEW
published: 19 November 2015
doi: 10.3389/fpsyg.2015.01740

Being Mad in Early Modern England

Aleksandar Dimitrijevic [1, 2]*

[1] Psychology, University of Belgrade, Belgrade, Serbia, [2] International Psychoanalytic University, Berlin, Germany

It has become almost a rule that the birth of scientific psychiatry and what we today term clinical psychology took place in the short period between the last decade of the XVIII century and the 1820s. Everything that happened before that period—every description, diagnosis, and therapy—has been considered "pre-scientific," outdated, in a way worthless. In this paper, however, I am providing the argument that, first, the roots of contemporary psychiatry reach at least to England of the early modern period, and that, second, it may still turn out that in the field of mental health care historical continuities are more numerous and persistent than discontinuities. Thus, I briefly review the most important surviving documents about the treatment of mental disorders in England of Elizabethan and Jacobian period, organizing the argument around the well-known markers: diagnostics and etiology, therapy, organization of the asylum, the public image of the mentally ill.

Keywords: history of mental disorders, early modern period, history of melancholy, Shakespeare, Robert Burton

OPEN ACCESS

Edited by:
Diogo Telles-Correia, University of Lisbon, Portugal

Reviewed by:
Tiago Reis Marques, King's College London, UK
Eva Gebhardt, Studio Panama, Italy

***Correspondence:**
Aleksandar Dimitrijevic
a.dimitrijevic@ipu-berlin.de

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *04 August 2015*
Accepted: *29 October 2015*
Published: *19 November 2015*

Citation:
Dimitrijevic A (2015) Being Mad in Early Modern England. Front. Psychol. 6:1740. doi: 10.3389/fpsyg.2015.01740

It has become almost a rule that the birth of scientific psychiatry and what we today term clinical psychology took place in the short period between the last decade of the eighteenth century and the 1820s. The historians either hail Phillipe Pinel's efforts to free Parisian patients/inmates from chains, or they quote from German medical textbooks that have laid fundaments for psychiatry of the twentieth century, in whose shadows clinicians still work today.

More importantly, every description, diagnosis, and therapy that happened before that period has been considered "pre-scientific," or outdated. As modern medicine is based on natural sciences, its history is not regarded as a source of relevant knowledge, but is relegated among humanistic disciplines. In this paper, however, I am offering an argument that, first, the roots of contemporary psychiatry reach at least to England of the early modern period, and that, second, it may still turn out that in the field of mental health care historical continuities are more numerous and persistent than discontinuities.

In order to achieve that, I shall briefly review documents about the treatment of mental disorders published and used in England especially, but not exclusively, during Elizabethan and Jacobean eras (i.e., 1558-1625)—organizing the argument around the well-known markers: diagnostics and etiology, therapy, organization of the asylum, the public image of the mentally ill… I will review the books that were revolutionary for that period (like *The Discoverie of Witchcraft*), significantly influenced the development of the field (like *A Briefe Discourse of a Disease Called the Suffocation of the Mother*) or still quoted very frequently today (like *The Anatomy of Melancholie*). I will also add illustrations from Shakespeare's plays, universally considered very accurate and influential (Simpson, 1959; Edgar, 1970; Kail, 1986; Adams, 1989; Iyengar, 2014).

One may well argue that the modern treatment of the mentally ill started when the practice of exorcism used during the Christian Middle Ages was finally rejected. In practically all the earliest documents that have been preserved, mental disorders are described as possession (Zilboorg, 1941; Clarke, 1975; Wallis, 2010). So, when a Margery Kempe dictated her life story in 1420—which made her probably the first autobiographer in the English tradition and among women generally, quite

an achievement for an illiterate countrywoman—she described her experience of what we would now term Postpartum (or postanatal) depression (PDD), but using in her explanations a rhetoric completely different than ours: devils, fiends, witches, deception, powers; and recovery through the grace, Gospel, Cross, visions of Jesus Christ (after Porter, 1991, pp. 44–46, 165–167).

The codified view followed *Malleus Maleficarum*, published in 1487 and propagating the claim that mental disorders were consequences of witchcraft and should be treated as inspired by devil and punished severely. Special attention was given to the "revelatory discovery" that the manifest source of evil were in fact women, who seduced, charmed, and possessed men, often castrating them and keeping flocks of penises as amusing independent beings (Mackay, 2009). Not long after *Malleus* was translated in English, it received royal support, as in 1597, James, still only the King of Scotland, published *Daemonologie* (James, 1597)[1]. And even in Burton's famous *The Anatomy of Melancholie*, to which I shall return several times, the influence of devils is listed among possible causes of mental disorders (Burton, 1621).

The early modern period literature about psychopathology is especially important because it introduced the idea of internal causation. It may have all started when Reginald Scott, a judge, described (Scot, 1586) many cases of witches he had tried before the court of law and claimed that these women were in fact insane. In the chapter "Not witchcraft but melancholie. Voluntarie confessions untruly made," Scott explained that they were themselves deceived at believing they possessed supernatural powers.

In the beginning, ecclesiastical explanations were replaced by the even older notion of bodily humors. The book *De Proprietaribus Rerum* by Bartholomeus Anglicus (1240), professor of theology in Paris during the thirteenth century, was first translated in English[2] in 1470 and published many times before the end of that century. It contained descriptions of "frenesia"—"delirium due to disease of the brain" and "perafrenesi"—"delirium occurring in the course of febrile or other systemic disease," as well as dementia and mania, but gives best illustration of the then new approach in his depiction of depression:

> Melancholy is a humour, boystous and thicke, and is bredde of troubled drastes of blode [. . .] Of this humor havying maistry in the body, these ben the sygnes and tokens. Fyrste the colour of the skynne chaungeth into blacke or bloo: Soure savour, sharpe—and erthy is felte in the mouth. By the qualite of the humor the patient is feynte—and fereful in hert without cause, and oft sorry [. . .] Some dread enmyte of some man: Some love and desire dethe (Chapter 11, "OfMelancholy").

Hysteria was similarly explained in the Aristotelian manner as the movement of uterus throughout the body and its harmful effect on other organs. The term "suffocation of the mother" was widely accepted after the book of the same title by Jorden (1603). Just one token of this is King Lear's painful exclamation: "O, how this mother swells up toward my heart! *Hysterica passio* down, thou climbing sorrow; Thy element's below" (2.2.225-227; Norton Edition).

Not long after this, however, truly psychological categories were for the first time used as explanations of mental disorders. Juan Luis Vives (1492-1540), who, although Spanish by origin, lived at the court of Henry VIII, was highly influential, even considered by some to be the father of modern psychiatry (Zilboorg, 1941). Vives wrote that "The soul therefore is itself the author, the moving force (behind our functions), drawing its energy not elsewhere but within the body" (after Stone, 1998, p. 27). Just a century later, another sophisticated explanation for the conflicting nature of the mind was offered: "Therefore we must conceive in a godly man, a double selfe, one which must be denied, the other which must denie; one that breeds all the disquiet, and another that stilleth what the other hath raised. . . " (Sibbes, 1615, Chapter 9, "Of the souls disquiets").

Classification of disorders was also getting more and more detailed. Robert Burton differentiated between four categories: (1) diseases emanating from the body; (2) diseases of the head (brain); (3) madness (mania); (4) melancholy. But matters very quickly grew more and more complicated. Thomas Willis, in *Cerebri Anatome,* introduced the terms neurology and psychology and wrote that disorders were caused by problems in nerve transmission, later discussing vital and involuntary systems in the brain. In the first English book on medical psychology, *De Anima Brutorum,* Willis, in 1672, described fourteen categories, including the purely neurological ones. His insights may strike us as uncannily similar to the standpoints of contemporary psychiatry, as he has described *dementia praecox* (or, in nowadays parlance—*shizophrenia*): "young persons who, lively and spirited, and at times brilliant in their childhood, passed into obtuseness and hebetude during adolescence" (Willis, 1965, p. 176); he also observed the coexistence of melancholia and mania in the same person, now known as Bipolar Affective Disorder. Willis also rejected the idea of the wandering womb as a cause of hysteria, while Sydenham (1695) wrote of hysterical convulsions that resembled epileptic seizures and, more than two centuries before Freud, was the first to discuss hysterical disorders in men, although he thought they were more prone to hypochondria. Sydenham also believed that in each case there were many sources of influence, including the family context. And even before that, Christopher Langton wrote in 1550 that sorrow can overthrow the heart and life can be utterly extinct from the patient, whom today we would label psychosomatic.

Four centuries ago, the mentally ill were equated with the lowest in the human nature, frequently even with animals, or beasts. I will draw only from the two most famous sources. Burton's *Anatomie of Melancholie* was published 10 times during the XVII century and nine more times later on, so it is considered the most widely read and quoted book in the history of psychiatry (Smith and Mulhauser, 1959). Burton introduced each type of

[1]It is noteworthy that when James was crowned as successor of Queen Elisabeth's throne, in 1603, he was greeted in Oxford by a theater company that performed a play about witches, Scottish king-to-be and madness of his murderous wife (see Greenblatt, 2005).

[2]Although, "Anglicus" meant "coming from England; Englishman," he, of course, wrote in Latin.

mental disorders in verse, and here is the depiction of mania (in the chapter "Argument on the Frontispiece"):

> But see the *Madman* rage down right
> With furious looks, a ghastly sight.
> Naked in chains bound doth he lie,
> A roars amain he knows not why!
> Observe him: for as in a glass,
> Thine angry portraiture it was.
> His picture keeps still in thy presence;
> 'Twixt him and thee, there's no difference.'

Even more famous example comes from the second act of *King Lear*, where Edgar comes up with a plan to hide his identity in the following way:

> Whiles I may scape
> I will preserve myself, and am bethought
> To take the basest and most poorest shape
> That ever penury in contempt of man
> Brought near to beast. My face I'll grime with filth,
> Blanket my loins, elf all my hairs in knots,
> And with presented nakedness outface
> The winds and persecutions of the sky.
> The country gives me proof and precedent
> Of Bedlam beggars who with roaring voices
> Strike in their numbed and mortified arms
> Pins, wooden pricks, nails, springs of rosemary,
> And with this horrible object from low farms,
> Poor pelting villages, sheep-cotes and mills
> Sometime with lunatic bans, sometime with prayers
> Enforce their charity. 'Poor Tuelygood, Poor Tom.'
> (2.2.162-177; Norton Edition)

The aforementioned Bedlam was founded in 1247, but became a hospital exclusively for the mentally disturbed in 1377. For centuries, it did not offer anything close to caring and humane approach to the afflicted. It more often served for the "patients" "to be held in close confinement and totally *incommunicado*" (after Arnold, 2009, p. 25) or as a constant source of entertainment for the rich, who visited it on Sundays, bought tickets, and amused themselves by observing behavior of the psychotic. Worse still, Bedlam included a long history of corruption: in 1574, a woman was admitted without any medical cause and after that "for six weeks before her committal, she had been tied down in bed by her husband and another woman until she was 'well nigh famished"' (Arnold, 2009, p. 42). In 1614, the first account of "malpractice on the mad" was reviewed before the College of Physicians (Arnold, 2009, p. 64), and in 1631 a surprising visit found out that Bedlam's thirty patients shared mere five pounds of cheese per day.

In the beginning of this period there was hardly anything we would consider therapeutic. The treatment recommended for witches was strangulation, beheading or burning at stake, but it was only slightly less cruel for the ill. Thomas More, who from 1516 to 1523 lived near Bedlam hospital, described the approach characteristic of his days:

> "[…] he had therfor ben put uppe in bedelem, and afterward by betynge and correccyon gathered hys rememberaunce to hym, and beganne to come agayne to hym selfe beynge theruppon set at liberty and walkynge about abrode, hys olde fansyes beganne to fall agayne in his hed" More (1533, p. 198, Ch. 36).

It sounds uniquely heartless when Thomas Willis describes a case he attended: "A countrywoman, aged about 45, for long melancholic, was seized by mania […] so much so that it was necessary to bind her with chains and ropes to keep her in bed. On the fifth day half a pint of blood was drawn from the basilica vain […] In the evening I visited her. She was now shouting wildly, now singing, now weeping. She breathed rapidly, drawing the breath in with a hiss, her lips being drawn inwards. I prescribed a liniment of Vigo's ointment with [word illegible] smeared on a rose cake to be applied to her forehead and temples and a poultice of gently cooked water-hemlock to be put on the region of spleen; and also repeated drafts of a cardiac julep. The next night she died" (after Porter, 1991, p. 287).

Gradually, the prescriptions were becoming milder, but no more effective or scientific. Some treatment procedures of the time included shaving the head and washing it with rose water and vinegar in order to help evaporate "grosse vapors which hurt the Memory" (after Hunter and Macalpine, 1963, p. 23). And the mentally ill were offered herbal cures, leeching and vomiting, while no less than one Pope recommended "a roasted mouse, eaten whole" (Hunter and Macalpine, 1963, p. 12). Bright (1586) wrote that depression was a physiological disorder caused by bad diet, so that the first step in recovery was to avoid "beets, cabbage, dates, olives, bread of fine unleavened flour, pork, beef, quail, peacocks, fresh-water fish, red wine, beer and ale." At almost the same year, a more comprehensive regime was prescribed: "Let the sicke use wyne that is white, thinne, and not very old, and let them eschewe wine that is thick and black, let there exercises be meane, let them ryde or walke by places pleasant and greene, or use sailing on water. Also a bath of sweet water with a moist dyet let the sicke use often as one of his remedies, sleep is wonderful good for them, as also moderate carnal copulation. Let them be mery as much as may be, and heare musicall instruments and singing" (Barrough, 1583, p. 36, Ch. 28, "Of Melancholie"). On his part, Robert Burton listed hundreds of herbal remedies and distracting activities (music being one of the most important among them), believing that these were effective in the cases of depression. More importantly, Burton thought that the melancholic should be encouraged to become open and confess their sorrows to an empathetic friend, thus foretelling contemporary psychotherapeutic approaches.

Similarities between contemporary psychopathology and that of the early modern England are, I believe, striking! Notions of possession and exorcism are overruled, but we are still debating the relationship between psychological and "external" factors in psychopathology. Mental disorders that we meet in our clinical practices were delineated and described about four centuries ago. Public image of the mentally ill is more affirmative then it used to be, but during the last five decades stigma has constantly been on the rise (Kecmanovic, 2010) and prevailing representation of persons with psychotic disorders is that they are dangerous and unpredictable (Link and Phelan, 1999). Asylums are still in use across Europe (Mental Health Europe, 2012) and with them

discrimination, loss of human rights, torture, corruption. Our treatment approaches are not bizarre as they used to be, but their effectiveness is far from being perfect. If, however, we would like to continue improving, it may be important that we remain aware of indebtedness, past continuities, and roots of contemporary psychopathology that reach at least four-and-a-half centuries back.

ACKNOWLEDGMENTS

The preparation of this paper was supported by Ministry of Education and Science of the Republic of Serbia, Grant No. 179018, and by "Trauma, Trust, and Memory" Project funded by "German Academic Exchange Service [DAAD]," Grant No. 57173352.

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Conflict of Interest Statement: The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in Psychology

MINI REVIEW
published: 01 October 2015
doi: 10.3389/fpsyg.2015.01511

Sexual orientation and gender identity: review of concepts, controversies and their relation to psychopathology classification systems

Carla Moleiro and Nuno Pinto*

Instituto Universitário de Lisboa ISCTE-IUL, CIS, Lisboa, Portugal

Numerous controversies and debates have taken place throughout the history of psychopathology (and its main classification systems) with regards to sexual orientation and gender identity. These are still reflected on present reformulations of gender dysphoria in both the Diagnostic and Statistical Manual and the International Classification of Diseases, and in more or less subtle micro-aggressions experienced by lesbian, gay, bisexual and *trans* patients in mental health care. The present paper critically reviews this history and current controversies. It reveals that this deeply complex field contributes (i) to the reflection on the very concept of mental illness; (ii) to the focus on subjective distress and person-centered experience of psychopathology; and (iii) to the recognition of stigma and discrimination as significant intervening variables. Finally, it argues that sexual orientation and gender identity have been viewed, in the history of the field of psychopathology, between two poles: gender transgression and gender variance/fluidity.

Keywords: sexual orientation, gender identity, transgender, discrimination, psychopathology, mental health care

OPEN ACCESS

Edited by:
Diogo Telles-correia, University of Lisbon, Portugal

Reviewed by:
Hélder Lourenço, Centro Hospitalar do Médio Ave, EPE, Portugal
João Gama Marques, Faculdade de Medicina da Universidade de Lisboa, Portugal

****Correspondence:***
Carla Moleiro, Instituto Universitário de Lisboa ISCTE-IUL, CIS, Avenida das Forças Armadas, 1649-026 Lisbon, Portugal
carla.moleiro@iscte.pt

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *29 July 2015*
Accepted: *18 September 2015*
Published: *01 October 2015*

Citation:
Moleiro C and Pinto N (2015) Sexual orientation and gender identity: review of concepts, controversies and their relation to psychopathology classification systems. Front. Psychol. 6:1511. doi: 10.3389/fpsyg.2015.01511

Numerous controversies and debates have taken place throughout the history of psychopathology and mental health care with regards to lesbian, gay, bisexual and transgender (LGBT) people. The present paper aims to review relevant concepts in this literature, its historical and current controversies, and their relation to the main psychopathology classification systems.

Concepts and Definitions

Concepts and definitions that refer to sexual orientation and gender identity are an evolving field. Many of the terms used in the past to describe LGBT people, namely in the mental health field, are now considered to be outdated and even offensive.

Sexual orientation refers to the sex of those to whom one is sexually and romantically attracted (American Psychological Association, 2012). Nowadays, the terms 'lesbian' and 'gay' are used to refer to people who experience attraction to members of the same sex, and the term 'bisexual' describe people who experience attraction to members of both sexes. It should be noted that, although these categories continue to be widely used, sexual orientation does not always appear in such definable categories and, instead, occurs on a continuum

(American Psychological Association, 2012), and people perceived or described by others as LGB may identify in various ways (D'Augelli, 1994).

The expression gender identity was coined in the middle 1960s, describing one's persistent inner sense of belonging to either the male and female gender category (Money, 1994). The concept of gender identity evolved over time to include those people who do not identify either as female or male: a "person's self concept of their gender (regardless of their biological sex) is called their gender identity" (Lev, 2004, p. 397). The American Psychological Association (2009a, p. 28) described it as: "the person's basic sense of being male, female, or of indeterminate sex." For decades, the term 'transsexual' was restricted for individuals who had undergone medical procedures, including genital reassignment surgeries. However, nowadays, 'transsexual' refers to anyone who has a gender identity that is incongruent with the sex assigned at birth and therefore is currently, or is working toward, living as a member of the sex other than the one they were assigned at birth, regardless of what medical procedures they may have undergone or may desire in the future (e.g., Serano, 2007; American Psychological Association, 2009a; Coleman et al., 2012). In this paper we use the prefix *trans* when referring to transsexual people.

Since the 1990's the word transgender has been used primarily as an umbrella term to describe those people who defy societal expectations and assumptions regarding gender (e.g., Lev, 2004; American Psychological Association, 2009a). It includes people who are transsexual and intersex, but also those who identify outside the female/male binary and those whose gender expression and behavior differs from social expectations. As in the case of sexual orientation, people perceived or described by others as transgender – including transsexual men and women – may identify in various ways (e.g., Pinto and Moleiro, 2015).

Discrimination and Impact on Mental Health

Lesbian, gay, bisexual and transgender people often suffer from various forms of discrimination, stigma and social exclusion – including physical and psychological abuse, bullying, persecution, or economic alienation (United Nations, 2011; Bostwick et al., 2014; European Union Agency for Fundamental Rights, 2014). Moreover, experiences of discrimination may occur in various areas, such as employment, education and health care, but also in the context of meaningful interpersonal relationships, including family (e.g., Milburn et al., 2006; Feinstein et al., 2014; António and Moleiro, 2015). Accordingly, several studies strongly suggest that experiences of discrimination and stigmatization place LGBT people at higher risk for mental distress (Cochran and Mays, 2000; Dean et al., 2000; Cochran et al., 2003; Meyer, 2003; Shilo, 2014).

For example, LGB populations may be at increased risk for suicide (Hershberger and D'Augelli, 1995; Mustanski and Liu, 2013), traumatic stress reactions (D'Augelli et al., 2002), major depression disorders (Cochran and Mays, 2000), generalized anxiety disorders (Bostwick et al., 2010), or substance abuse (King et al., 2008). In addition, transgender people have been identified as being at a greater risk for developing: anxiety disorders (Hepp et al., 2005; Mustanski et al., 2010); depression (Nuttbrock et al., 2010; Nemoto et al., 2011); social phobia and adjustment disorders (Gómez-Gil et al., 2009); substance abuse (Lawrence, 2008); or eating disorders (Vocks et al., 2009). At the same time, data on suicide ideation and attempts among this population are alarming: Maguen and Shipherd (2010) found the percentage of attempted suicides to be as high as 40% in transsexual men and 20% in transsexual women. Nuttbrock et al. (2010), using a sample of 500 transgender women, found that around 30% had already attempted suicide, around 35% had planned to do so, and close to half of the participants expressed suicide ideation. In particular, adolescence has been identified as a period of increased risk with regard to the mental health of transgender and transsexual people (Dean et al., 2000).

In sum, research clearly recognizes the role of stigma and discrimination as significant intervening variables in psychopathology among LGBT populations. Nevertheless, the relation between sexual orientation or gender identity and stress may be mediated by several variables, including social and family support, low internalized homophobia, expectations of acceptance vs. rejection, contact with other LGBT people, or religiosity (Meyer, 2003; Shilo and Savaya, 2012; António and Moleiro, 2015; Snapp et al., 2015). Thus, it seems important to focus on subjective distress and in a person-centered experience of psychopathology.

On the History of Homosexuality and Psychiatric Diagnoses

While nowadays we understand that higher rates of psychological distress among LGB people are related to their minority status and to discrimination, by the early 20th century, psychiatrists mostly regarded homosexuality as pathological *per se*; and in the mid-20th century psychiatrics, physicians, and psychologists were trying to "cure" and change homosexuality (Drescher, 2009). In 1952, the American Psychiatric Association published its first edition of the *Diagnostic and Statistical Manual* (DSM-I), in which homosexuality was considered a "sociopathic personality disturbance." In DSM-II, published in 1968, homosexuality was reclassified as a "sexual deviation." However, in December 1973, the American Psychiatric Association's Board of Trustees voted to remove homosexuality from the DSM.

The most significant catalyst to homosexuality's declassification as a mental illness was lesbian and gay activism, and its advocacy efforts within the American Psychiatric Association (Drescher, 2009). Nevertheless, during the discussion that led to the diagnostic change, APA's Nomenclature Committee also wrestled with the question of what constitutes a mental disorder. Concluding that "they [mental disorders] all regularly caused subjective distress or were associated with generalized impairment in social effectiveness of functioning" (Spitzer, 1981, p. 211), the Committee agreed that homosexuality by itself was not one.

However, the diagnostic change did not immediately end the formal pathologization of some presentations of homosexuality. After the removal of the "homosexuality" diagnosis, the DSM-II contained the diagnosis of "sexual orientation disturbance," which was replaced by "ego dystonic homosexuality" in the DSM-III, by 1980. These diagnoses served the purpose of legitimizing the practice of sexual "conversion" therapies among those individuals with same-sex attractions who were distressed and reported they wished to change their sexual orientation (Spitzer, 1981; Drescher, 2009). Nonetheless, "ego-dystonic homosexuality" was removed from the DSM-III-R in 1987 after several criticisms: as formulated by Drescher (2009, p. 435): "should people of color unhappy about their race be considered mentally ill?"

The removal from the DSM of psychiatric diagnoses related to sexual orientation led to changes in the broader cultural beliefs about homosexuality and culminated in the contemporary civil rights quest for equality (Drescher, 2012). In contrast, it was only in 1992 that the World Health Organization (World Health Organization, 1992) removed "homosexuality" from the International Classification of Diseases (ICD-10), which still contains a diagnosis similar to "ego-dystonic homosexuality." However, this is expected to change in the next revision, planned for publication in 2017 (Cochran et al., 2014).

Controversies on Gender Dysphoria and (Trans)Gender Diagnoses

Mental health diagnoses that are specific to transgender and transsexual people have been highly controversial. In this domain, the work of Harry Benjamin was fundamental for *trans* issues internationally, through the Harry Benjamin International Gender Dysphoria Association (presently, the World Professional Association for Transgender Health, WPATH). In the past few years, there has been a vehement discussion among interested professionals, *trans* and LGBT activists, and human rights groups concerning the reform or removal of (trans)gender diagnoses from the main health diagnostic tools. However, discourses on this topic have been inconclusive, filled with mixed messages and polarized opinions (Kamens, 2011). Overall, mental health diagnoses which are specific to transgender people have been criticized in large part because they enhance the stigma in a population which is already particularly stigmatized (Drescher, 2013). In fact, it has been suggested that the label "mental disorder" is the main factor underlying prejudice toward *trans* people (Winter et al., 2009).

The discussion reached a high point during the recent revision process of the DSM-5 (American Psychiatric Association, 2013), in which the diagnosis of "gender identity disorder" was revised into one of "gender dysphoria." Psychiatric diagnosis was thus limited to those who are, in a certain moment of their lives, distressed about living with a gender assignment they experience as incongruent with their gender identity (Drescher, 2013). The change of criteria and nomenclature "is less pathologizing as it no longer implies that one's identity is disordered" (DeCuypere et al., 2010, p. 119). In fact, gender dysphoria is not a synonym for transsexuality, nor should it be used to describe transgender people in general (Lev, 2004); rather, "[it] is a clinical term used to describe the symptoms of excessive pain, agitation, restless, and malaise that gender-variant people seeking therapy often express" (Lev, 2004, p. 910). Although the changes were welcomed (e.g., DeCuypere et al., 2010; Lev, 2013), there are still voices arguing for the "ultimate removal" (Lev, 2013, p. 295) of gender dysphoria from the DSM. Nevertheless, attention is presently turned to the ongoing revision of the ICD. Various proposals concerning the revision of (trans)gender diagnoses within ICD have been made, both originating from transgender and human rights groups (e.g., Global Action for Trans* Equality, 2011; TGEU, 2013) and the health profession community (e.g., Drescher et al., 2012; World Professional Association for Transgender Health, 2013). These include two main changes: the reform of the diagnosis of transsexualism into one of "gender incongruence"; and the change of the diagnosis into a separate chapter from the one on "mental and behavioral disorders."

Mental Health Care Reflecting Controversies

There is evidence that LGBT persons resort to psychotherapy at higher rates than the non-LGBT population (Bieschke et al., 2000; King et al., 2007); hence, they may be exposed to higher risk for harmful or ineffective therapies, not only as a vulnerable group, but also as frequent users.

Recently, there has been a greater concern in the mental health field oriented to the promotion of the well-being among non-heterosexual and transgender people, which has paralleled the diagnostic changes. This is established, for instance, by the amount of literature on gay and lesbian affirmative psychotherapy which has been developed in recent decades (e.g., Davis, 1997) and, also, by the fact that major international accrediting bodies in counseling and psychotherapy have identified the need for clinicians to be able to work effectively with minority clients, namely LGBT people. The APA's guidelines for psychotherapy with lesbian, gay, and bisexual client (American Psychological Association, 2000, 2012) are a main reference. These ethical guidelines highlight, among several issues, the need for clinicians to recognize that their own attitudes and knowledge about the experiences of sexual minorities are relevant to the therapeutic process with these clients and that, therefore, mental health care providers must look for appropriate literature, training, and supervision.

However, empirical research also reveals that some therapists still pursue less appropriate clinical practices with LGBT clients. In a review of empirical research on the provision of counseling and psychotherapy to LGB clients, Bieschke et al. (2006) encountered an unexpected recent explosion of literature focused on "conversion therapy." There are, in fact, some mental health professionals that still attempt to help lesbian, gay, and bisexual clients to *become* heterosexual (Bartlett et al., 2009), despite the fact that a recent systematic review of the peer-reviewed journal literature on sexual orientation change efforts concluded that "efforts to change sexual orientation are unlikely

to be successful and involve some risk of harm" (American Psychological Association, 2009b, p. 1).

Moreover, there is evidence of other forms of inappropriate (while less blatant) clinical practices with LGBT clients (e.g., Garnets et al., 1991; Jordan and Deluty, 1995; Liddle, 1996; Hayes and Erkis, 2000). Even those clinicians who intend to be affirmative and supportive of LGBT individuals can reveal subtle heterosexist bias in the work with these clients (Pachankis and Goldfried, 2004). Examples of such micro-aggressions (Sue, 2010) might be automatically assuming that a client is heterosexual, trying to explain the etiology of the client's homosexuality, or focusing on the sexual orientation of a LGB client despite the fact that this is not an issue at hand (e.g., Shelton and Delgado-Romero, 2011). Heterosexual bias in counseling and psychotherapy may manifest itself also in what Brown (2006, p. 350) calls "sexual orientation blindness," i.e., struggling for a supposed neutrality and dismissing the specificities related to the minority condition of non-heterosexual clients. This conceptualization of the human experience mostly in heterosexual terms, found in the therapeutic setting, does not seem to be independent of psychotherapist's basic training and the historical heterosexist in the teaching of medicine and psychology (Simoni, 1996; Alderson, 2004).

With regards to the intervention with *trans* people, for decades the mental health professionals' job was to sort out the "true" transsexuals from all other transgender people. The former would have access to physical transition, and the later would be denied any medical intervention other than psychotherapy. By doing this, whether deliberately or not, professionals – acting as gatekeepers – pursued to 'ensure that most people who did transition would not be "gender-ambiguous" in any way' (Serano, 2007, p. 120). Research shows that currently *trans* people still face serious challenges in accessing health care, including those related to inappropriate gatekeeping (Bockting et al., 2004; Bauer et al., 2009). Some mental health professionals still focus on the assessment of attributes related to identity and gender expressions, rather than on the distress with which *trans* people may struggle with (Lev, 2004; Serano, 2007). Hence, *trans* people may feel the need to express a personal narrative consistent with what they believe the clinicians' expectations to be, for accessing hormonal or surgical treatments (Pinto and Moleiro, 2015). Thus, despite the revisions of (trans)gender diagnoses within the DSM, more recent diagnoses seem to still be used as if they were identical with the diagnosis of transsexualism – in a search for the "true transsexual" (Cohen-Kettenis and Pfäfflin, 2010). It seems clear that social and cultural biases have significantly influenced – and still do – diagnostic criteria and the access to hormonal and surgical treatments for *trans* people.

Conclusion

Controversies and debates with regards to medical classification of sexual orientation and gender identity contribute to the reflection on the very concept of mental illness. The agreement that mental disorders cause subjective distress or are associated with impairment in social functioning was essential for the removal of "homosexuality" from the DSM in the 1970s (Spitzer, 1981). Moreover, (trans)gender diagnoses constitute a significant dividing line both within *trans* related activism (e.g., Vance et al., 2010) and the health professionals' communities (e.g., Ehrbar, 2010). The discussion has taken place between two apposite positions: (1) trans(gender) diagnoses should be removed from health classifying systems, because they promote the pathologization and stigmatization of gender diversity and enhance the medical control of *trans* people's identities and lives; and (2) trans(gender) diagnoses should be retained in order to ensure access to care, since health care systems rely on diagnoses to justify medical treatment – which many *trans* people need. In fact, *trans* people often describe experiences of severe distress and argue for the need for treatments and access to medical care (Pinto and Moleiro, 2015), but at the same time reject the label of mental illness for themselves (Global Action for Trans* Equality, 2011; TGEU, 2013). Thus, it may be important to understand how the debate around (trans)diagnoses may be driven also by a history of undue gatekeeping and by stigma involving mental illness.

The present paper argues that sexual orientation and gender identity have been viewed, in the history of the field of psychopathology, between two poles: gender transgression and gender variance/fluidity.

On the one hand, aligned with a position of "transgression" and/or "deviation from a norm," people who today are described as LGBT were labeled as mentally ill. Inevitably, classification systems reflect(ed) the existing social attitudes and prejudices, as well as the historical and cultural contexts in which they were developed (Drescher, 2012; Kirschner, 2013). In that, they often failed to differentiate between mental illness and socially non-conforming behavior or fluidity of gender expressions. This position and the historical roots of this discourse are still reflected in the practices of some clinicians, ranging from "conversion" therapies to micro-aggressions in the daily lives of LGBT people, including those experienced in the care by mental health professionals.

On the other hand, lined up with a position of gender variance and fluidity, changes in the diagnostic systems in the last few decades reflect a broader respect and value of the diversity of human sexuality and of gender expressions. This position recognizes that the discourse and practices coming from the (mental) health field may lead to changes in the broader cultural beliefs (Drescher, 2012). As such, it also recognizes the power of medical classifications, health discourses and clinical practices in translating the responsibility of fighting discrimination and promoting LGBT people's well-being.

In conclusion, it seems crucial to emphasize the role of specific training and supervision in the development of clinical competence in the work with sexual minorities. Several authors (e.g., Pachankis and Goldfried, 2004) have argued for the importance of continuous education and training of practitioners in individual and cultural diversity competences, across professional development. This is in line with APA's ethical guidelines (American Psychological Association, 2000, 2012), and it is even more relevant when we acknowledge the significant and recent changes in this field. Furthermore, it is founded

on the very notion that LGBT competence assumes clinicians ought to be aware of their own personal values, attitudes and beliefs regarding human sexuality and gender diversity in order to provide appropriate care. These ethical concerns, however, have not been translated into training programs in medicine and psychology in a systematic manner in most European countries, and to the mainstreaming of LGBT issues (Goldfried, 2001) in psychopathology.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in Psychology

MINI REVIEW
published: 25 September 2015
doi: 10.3389/fpsyg.2015.01463

Historical roots of histrionic personality disorder

Filipa Novais [1], Andreia Araújo [2] and Paula Godinho [1]*

[1] Neurosciences and Mental Health Department, Santa Maria Hospital, Lisbon, Portugal, [2] Child and Adolescent Psychiatry Department, Hospital Dona Estefânia, Lisbon, Portugal

Histrionic Personality Disorder is one of the most ambiguous diagnostic categories in psychiatry. Hysteria is a classical term that includes a wide variety of psychopathological states. Ancient Egyptians and Greeks blamed a displaced womb, for many women's afflictions. Several researchers from the 18th and 19th centuries studied this theme, namely, Charcot who defined hysteria as a "neurosis" with an organic basis and Sigmund Freud who redefined "neurosis" as a re-experience of past psychological trauma. Histrionic personality disorder (HPD) made its first official appearance in the Diagnostic and Statistical Manual of Mental Disorders II (DSM-II) and since the DSM-III, HPD is the only disorder that kept the term derived from the old concept of hysteria. The subject of hysteria has reflected positions about health, religion and relationships between the sexes in the last 4000 years, and the discussion is likely to continue.

Keywords: hysteria, histrionic, personality disorder, history, historical roots, neurology

OPEN ACCESS

Edited by:
Diogo Telles-correia, University of Lisbon, Portugal

Reviewed by:
Hélder Lourenço, Centro Hospitalar do Médio Ave, EPE, Portugal
Filipe P. H. Arantes-Gonçalves, Clinica de Saúde Mental do Porto, Portugal

****Correspondence:***
Filipa Novais, Neurosciences and Mental Health Department, Santa Maria Hospital, Avenida Professor Egas Moniz 1649-035 Lisboa, Lisbon, Portugal
filipa.novais@chln.min-saude.pt

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *31 July 2015*
Accepted: *14 September 2015*
Published: *25 September 2015*

Citation:
Novais F, Araújo A and Godinho P (2015) Historical roots of histrionic personality disorder. Front. Psychol. 6:1463. doi: 10.3389/fpsyg.2015.01463

Introduction

Histrionic personality disorder (HPD) is the only modern category in diagnostic classifications that conserved a derivative of the old concept *Hysteria* (Sulz, 2010). Several psychiatric disorders derived from the original term hysteria such as the conversion disorder, the somatization disorder, somatoform disorders, phobic anxiety, the term mass hysteria, and finally the HPD. Although different authors extensively studied this theme across time, the authors will focus on HPD.

The word *hysteria* derived from the Greek term "hystera," meaning the womb or uterus. It has been used since ancient times and appears in texts of the Egyptians, Greeks, and Romans. Since then, the meanings of *hysteria* have mirrored the preoccupations of the societies at each time.

In the old Rome, the word "Histrione" was already used to define the actors that represented coarse farces representing those who are false and theatrical (Zimmerman, 1999).

From Egyptians to Hippocrates

The oldest record is an Egyptian medical papyrus dating from around 1990 BC, the *Kahun Papyrus*, which is the first known medical gynecological text. Plato (429 ± 347 BC) described this phenomenon as "The animal within them is desirous of procreating children, and when remaining unfruitful. . .gets discontented and angry, and wandering in every direction through the body. . .drives them to extremity, causing all varieties of disease. . ." (Illis, 2002). Many women's afflictions, including choking, mutism, and paralysis were attributed to a condition called the "wandering womb" or "the wicked womb." It was Hippocrates (460 ± 377 BC) who first introduced the term "Hysteria" and described it as the consequence of a dry womb rising toward the throat searching for humidity, thereby impeding breathing. The neurotoxic effects of the "frustrated uterus"

would affect widows and virgins (Bogousslavsky, 2011). Galen (AD 129 ± 216 BC) instead blamed the blocked menstrual flow and sexual abstinence. One of his most striking views was that men could also suffer hysterical symptoms caused by retained sperm. His ideas contributed to initiate a debate, which had run for centuries, over whether men could or not have hysteria. Galen's views persisted among the medical practitioners of Christian post-Roman Britain (Edwards, 2009).

From Middle Ages to the 18th Century

During Middle Ages, as the attitude toward sickness changed from naturalistic to demonotheologic, with Augustine of Hippo (354–430 AC) and other theologians, hysteria came to be seen as a manifestation of demonic possession. Convulsions and the so-called "suffocations of the matrix" were considered as an expression of sexual pleasure and, therefore, sin. Devil could enter women's body to possess them and the hysterical become the witch, persecuted by the Catholic Church and many of those were killed by the inquisition (Roudinesco and Plon, 2000).

With the renewed interest on empiricism and science during Renaissance, old Greek concepts of hysteria were recuperated. Similar therapies to those prescribed in ancient Greek civilizations, such as genital stimulation by horse riding, dancing and, in particular, marriage and sexual intercourse were still prescribed for such condition (Edwards, 2009).

Several researchers including Charles Lepois, Thomas Willis, Thomas Sydenham, and Pierre Pomme had great interest in the study of hysteria (Teive et al., 2014).

Some of these authors defied the original theories that connected hysteria to the uterus and some defended that the disease was originated in the brain. One of the first was Thomas Willis (1621 ± 1675) who argued that hysterical disorders, the so-called "convulsive distempers," were caused by an excess of animal spirits carried by the nerves to different parts of the body, introducing a new etiology for the disease. He believed in a nervous origin instead of vapors opening the door to the desexualisation of the disease (Risse, 1988).

The famous clinician Thomas Sydenham, (1624–1689) was one of the most important contributors to the study of hysteria at his time. He published a treatise on hysteria called *Epistolary Dissertation on the Hysterical Affections* and stated that hysteria was the most common of all diseases afflicting both men and women and the more richer and civilized a patient was, the more likely he or she was to be afflicted. One of his most remarkable conclusions was that hysteria could take multiple forms in order to imitate several other diseases, frequently triggered by intense emotions such as anger, grief, terror or passions (Gilman et al., 1993).

William Cullen, (1769), a noted Scottish physician, published *Synopsis Nosologiae Methodicae*, a classification of diseases where hysteria figured on the group called *neuroses*. These diseases were considered to result from nervous system malfunction involving changes in sensibility and motion. Hysteria was included in the class of illnesses characterized by irregular muscular contractions, the so-called spasmodic diseases, but Cullen still admitted that in its origin were gynecological problems (Risse, 1988).

Philippe Pinel, (1745–1826) considered that diagnostic difficulties were associated with the numerous disorders and symptoms attached to it, so he defended the study of hysteria in its uncluttered or "pure state." He included hysteria in his "*Nosographie Philosophique*" (1813) placing it in the group called "Neuroses" (Whitaker et al., 2007).

During this period, hysteria was a serious subject in medical schools and textbooks. Some authors considered it to reflect psychological frustrations directly linked to the restricted role of women in society (Risse, 1988). Griesinger, (1817–1868) kept the view that hysteria was related to genital disorders and sexual frustration but also involving "morbid action of... the brain" (Gilman et al., 1993).

In 1859 Pierre Briquet, (1796–1881) published his "*Traité Clinique et Therapeutique de L'Hysterie*" presenting data from 430 hysterical patients collected in 10 years. He rejected the idea of the uterine origin of the disease and considered it as a "neurosis of the brain" in someone of the "hysterical type." Briquet had a remarkable contribution for development of the HPD; he considered this type of personality traits as the ground for the development of the histrionic disorders (Mai and Merskey, 1981). He introduced sociological and material concepts in the comprehension of hysteria, such as living and working conditions. The industrialization, with the development of the trains and the subsequent numerous traumatic accidents, brought up the discussion about the hysteria in men. Between 1880 e 1900, hysteria was epidemic: writers, doctors and historians agreed to refer to the industrial social crises, like strike, as a sign of the feminine convulsive nature and frequently applied the terms hysteria and "uterine furies" to designate them (Roudinesco and Plon, 2000).

At the end of the 19-century, Salpêtrière Hospital acquired a remarkable importance on the study of hysteria and hypnotism due to the famous French neurologist, Jean-Martin Charcot (1825 ± 1893), who created the study of Diseases of the Nervous System there, in 1862. He had many remarkable collaborators such as Albert Pitres, Paul Richer, Georges Gilles de la Tourette, Paul Sollier, Joseph Babinski, Sigmund Freud, and Pierre Janet creating the famous Salpêtrière' s School of Neurology. His interest on hysteria probably started after 1870, when Charcot's took charge of the Delasiauve service, a place where mainly epileptics and hysterics were admitted (Bogousslavsky et al., 2009). Using a photographic camera, after long and detailed observations and methodical comparisons of hysteria with other conditions, he considered two main forms of hysteria—with and without convulsions. The hysteroepilepsy or "*grandes crises d'hystérie*" were described as having four stages: 1. Epileptoid; 2. Contortions and acrobatic postures (Clownism); 3. Emotional gestures ("*attitudes passionnelles*"); and 4. Final delirium (Teive et al., 2014). Charcot considered hysteria as a "neurosis" with an organic basis and described permanent clinical features in patients who were also prone to paroxysmal fits, the "stigmata": sensory dysfunction, hyperexcitability and visual field narrowing (Bogousslavsky et al., 2009).

According to him, the presumed neurological impairment was dynamic in nature and produced by unconscious mental processes (Macmillan, 1997). Hysterical symptoms occurred in genetically

predisposed individuals and were manifested within familiar circumstances. Therefore, he stated that a fundamental condition of the treatment should be the isolation from family members and called this "the moral or mental side of treatment" (Illis, 2002).

One of Charcot's most remarkable students was Babinski, (1857–1932) who defined hysteria as a psychic state that would give the patient the ability of "auto-suggestion," so that the patient would be able "to be persuaded" and therefore was prone to "healing" by suggestion (Philippon and Poirier, 2009). Consequently he recommended the term "pithiatism" (from the Greek: created by suggestion and curable by persuasion). Despite the influences of his master, he presented his own theory about hysteria, as well as several approaches and specific criteria in order to differentiate organic from hysterical symptomatology (Mai, 2004; Allilaire, 2007; Clarac et al., 2008).

Later, Charcot introduced hypnosis as a therapeutic technique and also as an experimental tool to the study of hysterical phenomena and its underlying neurophysiology and psychogenic trauma-related mechanisms of the hysterical neuroses (Levin, 1978).

The principles of hypnosis have been previously established by Franz Anton Mesmer, (1734–1815) who created the so-called "Animal magnetism," a pervasive property of nature that could be used as an effective therapy for a wide variety of conditions and its therapeutically application—the "mesmerization" (Lanska and Lanska, 2007).

Charcot and his group have been criticized by the School of Nancy and his main investigator Hippolyte Bernheim, (1840–1919), a French physician and neurologist. While Charcot believed that hypnosis was based on physiologically well-determined phenomenon only applied, as a therapeutic and diagnostic technique, to hysterical patients, Bernheim proposed that it was based on changes in psychological functioning; different features of hypnosis would therefore reflect different degrees of suggestibility. He also argued that suggestibility was a normal human trait and not an abnormal phenomenon as Charcot defended (Macmillan, 1997).

Both Bernheim and Charcot had important influences on Sigmund Freud's, (1856–1939) latter theories. Freud, who later developed the psychoanalytic theory leading to the redefinition of hysteria and the creation of different syndromes that came from the original concept, went to the famous Salpêtrière in October 1885 in order to study with Charcot. He started translating some of Charcot's lectures and defending his views. 2 years after, he translated Bernheim's work and visited Nancy in the summer of 1889 (Macmillan, 1997).

Another important author that had influenced the work of Freud was Pierre Janet, (1859–1947), also a Charcot follower. Many of Freud's basic concepts were developed or elaborated by Janet, such as psychological automatism, consciousness, subconsciousness, narrowed field of consciousness, dissociation, suggestibility, fixed idea, and emotion (Hart and Horst, 1989). Janet considered that hysteria results from the idea the patient has about pathology, translating it into a physical disability. He studied five hysteria's symptoms: anesthesia, amnesia, abulia, motor control diseases, and modification of character (Tasca et al., 2012).

In 1895, Freud and Breuer, (1842–1925) published the "Studies on Hysteria," including the famous case study of Anna O and the formulation of three types of hysteria: defense, retention and hypnoid hysteria (Breuer and Freud, 1955). Freud defied the traditional idea that defended that hysteria was caused by the lack of conception and motherhood, proposing that hysteria was a disorder caused by a lack of libidinal evolution (setting the stage for the Oedipal conflict), so the consequence, and not the cause, would be the lack of conception as a result of the incapacity of the hysterical to live a mature relationship. Hysterical symptoms would therefore be the expression of the impossibility of fulfillment of the patient's sexual drive. Freud also added, to this paradigm, the concepts of "primary benefit" and "secondary advantage" associated with the use of these symptoms to satisfy patient's needs (Tasca et al., 2012). This new paradigm concerning the emotional origin of hysterical symptoms was often applied to shell shock and other "war neurosis" during the World War I and II (Crocq and Crocq, 2000). In fact, with the war and after that, during the 1940s and the 1950s, the interests in this matter grew rapidly.

Freud explored traumatic experiences occurring in the family in order to provide an explanation for hysteria. Unacceptable feelings connected to seduction were repressed and converted into somatic symptoms. Latter he found that many of these reports were false, so he concentrate on intrapsychic factors. Patients repressed not actual happenings but their own sexual fantasies (Slipp, 2014).

Histrionic Personality Disorder

Although the roots of modern histrionic personality can be traced back to Freud's description of "hysterical neuroses" (Sperry, 2003), personality was already a matter of attention before.

In the mid-19th century, Ernst von Feuchtersleben, (1765–1834) who wrote the Textbook of Medical Psychology (1845) made the first psychosocial description of what would become the histrionic personality. He described hysterical women as being sexually heightened, selfish and "overprivileged with satiety and boredom" (Millon, 2011).

Ernst Kretschmer, (1888–1964), a German psychiatrist known for the establishment of a typology based on the human constitution, suggested that hysterics show "a preference for what is loud and lively, a theatrical pathos, an inclination for brilliant roles. . .(and) a naïve, sulky egotism" (Bornstein et al., 2015). Another Kretschmer's important contribution was the demand for objective criteria in order to distinguish hysteria from simulation (Lerner, 2003).

The first providing a detailed psychoanalytic description of the hysterical personality style was Wilhelm Reich, (1897–1957), an Austrian psychoanalyst. He wrote "coquetry in gait, look or speech betrays, especially in women, the hysterical character type. . .We find fickleness of reactions. . .and. . .a strong suggestibility, which never appears alone but is coupled with a strong tendency to reactions of disappointment. . ."

A decade after his work, Otto Fenichel, (1897–1946), a psychoanalyst of the so-called "second generation," added another characteristic to this description: the pseudo-hypersexuality,

noting that these individuals "are inclined to sexualize all nonsexual relations. . ." (Bornstein et al., 2015).

Easser and Lesser, (1965) seek to integrate two different earlier approaches: the ego psychology school and Freud's libido theory. They proposed a classification of hysterics consisting on two extremes—the hysterical personality and the "hysteroid" (borderline) personality. Zetzel, (1968) also divided patients into "good" hysterics, who function well, and "bad" hysterics, who have weak egos and poor object relations. This latter group of patients has a profile and level of functioning similar to the one seen in borderline patients (Slipp, 2014).

Several theorists studied the particular traits of this type of personality including histrionic's impressionist cognitive style and inattention to detail. In his book, "Hysterical Personality Style and the Histrionic Personality Disorder," Horowitz (1991), focused on the connection between perception and behavior in histrionic personality; he argued that it was based on an underlying information processing bias. A disturbed mental representation of the self would constitute the link to the various features of this type of character. On the other hand, according to the biosocial-learning model, proposed by Theodore Millon and other authors, this personality type may arise from unconscious patterns of reinforcement provided by parents and others (Blaney et al., 2015). The cognitivists Beck et al. (2004) suggested that histrionic person believe that potential caregivers are not trustful and should be manipulated instead. According to these authors, their core believes include "I am inadequate and unable to handle the life on my own" and "It is necessary to be loved by everyone, all the time."

Since the first attempts to the establishment of diagnostic criteria in hysteria, there has been considerable controversy, considering the etiology, the definition and even the existence of such condition.

Discussion and Conclusion

The terms hysteria, hysterical personality, and HPD mark the development of unceasing attempts to identify a distinct pattern of psychopathology (Bakkevig and Karterud, 2010).

The first edition of the American Diagnostic and Statistical Manual (DSM-I), published in American Psychiatric Association (1952), had no category for hysterical personality although some of its features were included in the "emotionally unstable personality." The DSM-II (American Psychiatric Association, 1968) was strongly impacted by psychoanalysis: some personality disorders had to be differentiated from other neuroses with the same name (e.g., hysterical, obsessive-compulsive, and neurasthenic personalities and neuroses). Following the medical model created by Emil Kraepelin, in DSM-III (American Psychiatric Association, 1980), and the subsequent DSM-III-R (American Psychiatric Association, 1987) and DSM-IV (American Psychiatric Association, 1994), personality disorders were described as discrete types and grouped into three clusters. The term *hysterical* from DSM-II was replaced with "histrionic" in DSM-III following the proposition of Paul Chodoff who considered pejorative the description of the "hysterical female" as labile, egocentric, seductive, frigid and childish, as described in his article "The diagnoses of hysteria: An overview" (Chodoff, 1974). From DSM-III to DSM-IV-TR, (American Psychiatric Association, 2000) diagnostic criteria of HPD had several changes mainly due to the argument of "unspecificity." An important change occurred from DSM-III-R to DSM-IV:5 criteria were considered the threshold for obtaining the diagnosis, as compared to 4 criteria in DSM-III–R. This lead to a decline in the number of patients diagnosed with HPD (Blais and Baity, 2006).

The merits of compounding typological and dimensional models of personality were questioned during the preparation of DSM-5, reopening a century-old debate (Crocq, 2013). Data, although sparse, actually suggest that the rate of presentation of "hysteria" in neurological practice has remained stable over time (Stone et al., 2008).

Bakkevig and Karterud (2010), in a study carried out with a sample of patients attending psychiatric day hospital, concluded that the prevalence of HPD was very low (0.4%) and comorbidity was high, especially with borderline, narcissistic, and dependent personality disorders. They suggested that the HPD category should be deleted from the DSM system, excepting that clinical phenomena of exhibitionism and attention-seeking, which are the dominant personality features of HPD, should be preserved in an exhibitionistic subtype of narcissism. Nevertheless, HPD remains present in DSM-5 (American Psychiatric Association, 2013).

Edwards (2009) advocates that those who argue that hysteria has disappeared from clinical practice, "miss the point" and "that it has merely changed to reflect the preoccupations of our society. . ."

Concerns with stigma and lack of specificity of the term hysteria, and its derivative histrionic, led to its residual presence in modern classifications but the theme and its modern diagnosis that emerged from the original concept kept their topicality and importance in clinical practice.

For about 4000 years the construct of hysteria and its derivatives has reflected attitudes about health, religion and relationships between the sexes and the interest raised by this condition is likely to continue (Illis, 2002).

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in Psychology

REVIEW
published: 27 July 2015
doi: 10.3389/fpsyg.2015.00991

Hallucinations and related concepts—their conceptual background

Diogo Telles-Correia, Ana Lúcia Moreira and João S. Gonçalves*

Clinica Universitaria de Psiquiatria e Psicologia, Faculty of Medicine, University of Lisbon, Lisbon, Portugal

Prior to the seventeenth century, the experiences we now name hallucinations were valued within a cultural context, they could bring meaning to the subject or the world. From mid-seventeenth to eighteenth centuries, they acquire a medical quality in mental and organic illnesses. However, the term was only fully integrated in psychiatry by Esquirol in the eighteenth–nineteenth centuries. By then, a controversy begins on whether hallucinations have a perceptual or intellectual origin. Esquirol favors the intellectual origin, describing them as an involuntary exercise of memory and imagination. By the twentieth century, some authors maintain that hallucinations are a form of delusion (Ey), while others describe them as a change in perception (Jaspers, Fish). More integrated perspectives like those proposed by Alonso Fernandez and Luque, highlights the heterogeneity of hallucinations and the multiplicity of their types and causes. The terms pseudohallucination, illusion, and hallucinosis are grafted into the concept of hallucination. Since its introduction the term pseudohallucination has been used with different meanings. The major characteristics that we found associated with pseudohallucinations were "lack of objectivity" and "presence of insight" (differing from hallucinations). Illusions are unanimously taken as distortions of real objects. Hallucinosis, first described in the context of alcohol consumption, is generally considered egodystonic, in which insight is preserved. These and other controversial aspects regarding the evolution of the term hallucination and all its derivative concepts are discussed in this paper.

Keywords: hallucinations, pseudohallucinations, hallucinosis, illusions, psychopathology

OPEN ACCESS

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****Correspondence:***
Diogo Telles-Correia,
Clinica Universitaria de Psiquiatria e Psicologia, Faculty of Medicine, University of Lisbon, Avenida Egas Moniz, 1649-028 Lisboa, Portugal
tellesdiogo@gmail.com

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *20 April 2015*
Accepted: *29 June 2015*
Published: *27 July 2015*

Citation:
Telles-Correia D, Moreira AL and Gonçalves JS (2015) Hallucinations and related concepts—their conceptual background. Front. Psychol. 6:991. doi: 10.3389/fpsyg.2015.00991

Introduction

Hallucination is one of the most relevant symptoms in psychiatry. It is also one of the hardest to define and delimitate from other psychopathological concepts. This latter aspect in particular led to the emergence of other related concepts like "pseudohallucination," "illusion," and "hallucinosis." The etymology of the word hallucination is controversial. It may have had origin in the Latin word *allucinor, allucinaris*, used by Cicero, meaning the intent to mislead or equivocate (Corominas, 1973). It may also have originated from the Latin compound *ad lucem* (ad-*next to*; *lucem*-light; Barcia, 1903). Evidence for similar concepts may be found in Christian authors. Saint Augustine, for instance, tried to characterize visions in mystical experiences by distinguishing three meanings for the expression *videre* (latin word for "to see"): corporal (as in the visual experience of the external world through the senses), imaginative (as in the representation of images and objects that have a temporal but not spatial location), and intellectual (as in abstract concepts that lack a spatial and temporal location;

Sarbin and Juhasz, 1967). Saint Thomas Aquinas established a difference between normal and false perceptions and argued that a vision (*visio*) was a natural phenomenon instigated by God or the devil (Sarbin and Juhasz, 1967).

Only after the seventeenth/eighteenth centuries have hallucinations acquired a scientific/medical sense. The term was then used to designate organic conditions (afflictions of the cornea and diplopia) and mental disorders (strange noises, premonitions, and appearances; Luque and Villagrán, 2000). According to Berrios (1996), "variously named, these experiences were in earlier times culturally integrated and semantically pregnant, i.e. their content was believed to carry a message for the individual or the world" (p. 35). With the medicalization of the term, the semantic nature was lost and hallucinations came to be considered, first, as diseases or independent syndromes and, later on, as symptoms that characterize different diseases (Berrios, 1996).

In this article we aim to review the evolution of the term "hallucination" up to present time, as well as its related concepts, such as "pseudohallucination," "illusion," and "hallucinosis."

Eighteenth and Nineteenth Centuries

Esquirol achieved the major theoretical advance on hallucinations, introducing the term to psychiatry. According to Ey (1939), Esquirol brought together psychiatry and the patient with hallucinations. Before, hallucinations were only considered in one sensory modality (vision), but Esquirol (1845) combined in his designation experiences from various sensory modalities: "Hallucinations of sight. . . have been denominated visions. Who would dare to say visions of hearing, visions of taste, visions of smell? . . . A generic name is needed. I have proposed the word hallucination" (p. 110). Furthermore, Esquirol argued that hallucinations were a form of *delirium* ("*une certaine forme de délire*"), or a symptom of *delirium* (*delirium* was then considered a synonym for madness, a syndrome that affected several psychopathological areas: thinking, perception, humor, etc.). For Esquirol (1845), "a person is said to labor under a hallucination, when they have a thorough conviction of the perception of a sensation, when no external object. . . has impressed the senses" (p. 94). Esquirol (1845) also noted that "this symptom of *delirium* has been mistakenly identified by all authors with local lesions of the senses" (p. 94). According to him, hallucinations were different from illusions and only the latter truly correspond to sensory errors: "In hallucinations everything happens in the brain: visionaries dream awake. The activity of the brain is so energetic that the visionary or the hallucinated gives a body and reality to images and ideas that memory reproduces, without the intervention of the senses. In illusions, on the contrary, the sensibility of the nerve extremities is altered" (Esquirol, 1845, p. 111). According to Esquirol (1845), every type of hallucination was based on the same pathophysiology: "the images, ideas and notions which seem to belong to the function alteration of these free senses (hearing, taste, smell) . . . are produced by the same causes" (p. 110). By this time, the hallucinations were often referred to as "perceptions devoid of an object," a definition often erroneously associated with Esquirol. On the contrary, Esquirol always maintained that the hallucination was not a perception but a "form of *delirium* that makes patients believe they have a perception," meaning, the conviction of having a perception but not actually having a perception. In fact, Falret (1864) states that "the hallucination is a perception without object, as has been often repeated" (p. 264).

The work of Esquirol emerged at a stage when there was a controversy about hallucinations in French Psychiatry, a debate which revolved around two fundamental dichotomies: (1) If hallucinations arise simply by an "involuntary exercise of memory and imagination"—as advocated by Esquirol and others—or, rather, are the result of a sensory abnormality (central or peripheral); (2) If hallucinations were always pathological and, therefore, only occurring in the context of mental disease or whether they could occur without mental disease. This last question arises from the first, and goes back to the self-report of two patients suffering from these symptoms.

Nicolai published in 1799 an essay called "*Memory of the Apparition of Ghosts or Spectres Caused by Disease with Psychological Considerations*" (Ferriar, 1813; Brierre de Boismont, 1862). In this work, Nicolai described his own visual hallucinatory experiences, where insight had been preserved. This case was included by Brierre of Boismont in the category of "hallucinations compatible with reason" (Berrios, 1996). In his book "*Les Farfadets, ou Tous les démons ne sont pas de l'autre monde*" (published in 1821) Berbiguier reported experiences compatible with hallucinations and delusional ideas, where insight was not preserved. This case was considered paradigmatic of pathological hallucinations (Berrios, 1996). Baillarger, in 1844, wrote an essay (for a contest of the Royal Academy of Medicine), in which he classified hallucinations in its several modalities (claiming that the auditory hallucinations were "the most frequent," although visual hallucinations were "easier to study and understand") and tried to respond to the dispute in which the psychological or psycho-sensorial origin of hallucinations was questioned. In this context Baillarger (1886) proposed two types of hallucinations: psycho-sensorial (arising from a combination of the action of imagination and the sensory organs) and psychological (independent of the sense organs). Falret (1864), who found Baillarger's division unhelpful regarding clinical practice, defended Esquirol's strictly intellectual perspective of hallucinations: "the hallucination demands the intervention of the intellectual faculties, the memory and the imagination. . . it is a phenomenon with a psychic nature and not a sensory nature" (p. 23). Michéa (1851) in its "*Du Delire Des Sensations*," from 1846, also considers hallucinations as a transformation—usually involuntary—of memory and imagination into something that resembles a sense-perception. Griesinger (1867) defines hallucinations as "subjective images that are projected externally and acquire an apparent objectivity and reality" (p. 86). Tamburini (1881) questioned the accepted view (particularly defended by the French school) that proposed the origin of hallucinations to be predominantly psychological. According to Tamburini (1881) all hallucinations were a result of excitation of the centers of formation of images and other sensations in the brain. Several authors of this time, such as Chaslin (1912), accepted this position and developed it. This unitary vision of hallucinations promoted a divorce between

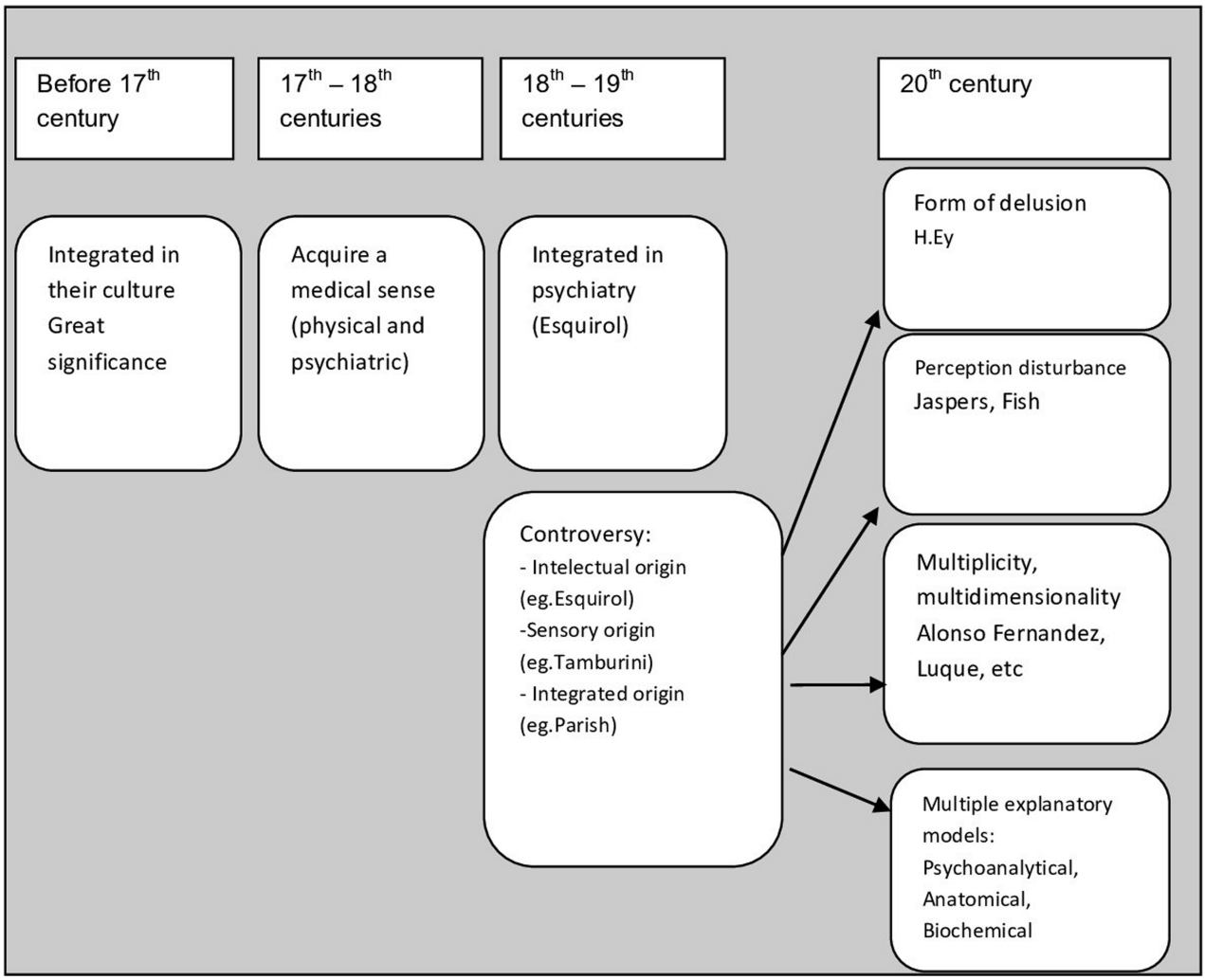

FIGURE 1 | Prior to the seventeenth century, the experiences we now name hallucinations were valued within a cultural context, they could bring meaning to the subject or the world. From mid-seventeenth and eighteenth centuries, they acquire a medical quality to designate mental and organic illnesses. However, the term is fully integrated in psychiatry by Esquirol in the eighteenth-nineteenth centuries. By then, a controversy begins on whether hallucinations have a perceptual or intellectual origin. Esquirol favors the intellectual origin (involuntary exercise of memory and imagination). Tamburini believes they are the result of the stimulation of the centers of imaging formation in the brain, but Parish advances with a more integrative approach. In the twentieth century, some authors maintain that hallucinations are a form of delusion (Ey), while others describe it as a change in perception (Jaspers, Fish). A more integrated perspective, like the one proposed by Alonso Fernandez and Luque, highlights the heterogeneity of hallucinations and the multiplicity of their types and causes, even within hallucinations with a psychiatric (and non-organic) cause. Multiple explanatory models were also developed during the twentieth century.

history and the social context of the patient and the content of the hallucinations, which was caused by random stimulation of the nerve centers. According to this view, even the presence or absence of insight was determined by the intensity of stimulation of those centers (Berrios, 1996) (**Figure 1**).

Edmund Parish von Senftenberg (1861–1916), a German researcher, published, in 1894, a seminal work in this area, "*Uber die Trugwahrnehmung*" ("*Hallucinations and Illusions*"; Parish, 1897). Here, the author suggests that the term "misleading perceptions" could encompass all concepts related to hallucination and illusion: "all hallucinations and illusions can be designated by misleading perceptions, whether observed in the sane or the insane, whether occurring in sleep or in the waking state, whether arising spontaneously or experimentally induced" (Parish, 1897, p. 17). Parish puts forward a new solution, conceptualized as the theory of "associative pathways" and that included the afferent and efferent pathways of the brain. According to him, in a normal perception there is information originated by the peripheral sensory organs and transmitted to the brain, affecting region A, which in turn communicates with other regions B, C, etc. From these regions efferent information is created, which is then, transmitted to the sensory organs a, b, c, etc. Thus, normal perception arises. Conversely, on occasion, after zones A, B, C, being excited, efferent information is locked in its usual channels, and has to find different ways to reach the sensory organs, thus creating a new perception: the hallucination.

In other cases, a given perception stimulates region A, but cannot stimulate B and C, resulting in an incomplete efferent information (for instance only a). In this case, illusion arises. On the other hand, Parish (1897) states that "the great controversy on whether hallucinations arise from sensory or ideational centres is meaningless since the sensory and imaginative centres are not separate and occupy close parts of the brain" (p. 134). In this theory, Parish overcomes extremist views and finds an integrative and original solution accepting the idea that central and peripheral sensory regions can be affected and that both the ideational and sensory regions of the brain are affected (**Figure 1**).

Twentieth Century

In turn, Jaspers defines hallucinations as perceptions of morbid origin (*sinnesvorgange*), which did not appear by transformation of real perceptions (as illusions do). Citing the work of Kandinsky, he says that, unlike pseudohallucinations, hallucinations have sensory consistency (objectivity, detail and corporality) and originate outside the subject's mind. Jaspers (1963) only intends to describe these psychopathological phenomena (as is characteristic of its descriptive psychopathology), without having the ambition to explain the pathophysiology (**Figure 1**).

Later, Ey defined hallucinations as a psychosensory disruption different from illusion and delusional interpretations (which for him was synonymous to the German term of delusional perception), consisting characteristically of a perception without an object (Ey et al., 1978). The three major conditions associated to hallucinations were: (1) sensory appearance of the experience; (2) conviction of its reality; (3) absence of a real object (Ey, 1973). On the other hand, he also insisted that hallucinations "are secondary to false beliefs or convictions" (Ey, 1973, p. 45), approaching the original definition by Esquirol, for whom the hallucination was a form of delusion that led patients to believe that they had a perception. This author, like other authors with a phenomenological orientation (Mayer-Gross, Mattussek, Zutt, etc), defends a unifying vision hallucination-delirium that meets in the psychotic patient with a deformation of the global reality (Castilla del Pino, 1984). He divides hallucinations into two major groups: (1) secondary to loss of structure of the field of consciousness (altered consciousness of the oneiroid type); (2) secondary to disruption of the self (present in schizophrenia; Ey, 1973). Cabalero Goas (1918–1977) also understood the hallucination as a disturbance in the distinction between the outer and inner world. To this author hallucinations have several features among which are highlighted: (1) a false perception, where falsity arises from not corresponding to any real object, and that occurs along with real perceptions; (2) a "new" phenomenon to the patient with a nature of perception that presents itself as being strange to his personality, coming from outside of the subject; (3) possession of an irresistible ability to convince the patient of its reality, to paraphrase Bumke (1946) who stated that "the power that these perceptions have over patients is higher than that of normal perceptions"; (4) aesthetic-spatial nature; (5) presentation of corporeality and its location outside the subject's mind; (6) disintegration of the real and replacement by a quasi-reality (this concept is imported from Merleau Ponty); (7) expression of an inner psychological experience in the form of a psychosensorial manifestation; (8) existence in conjunction with a disturbance of the mental activity characterized by a loss of insight, which causes this phenomenon to be accepted as a normal sensory perception (Goas, 1966). These features underline the fact that hallucinations are a very heterogeneous group of phenomena. They appear in key groups: (1) Schizophrenia, in which they arise by loss of consciousness of the ego activity, the patient feeling totally influenced, directed and governed by the external world, with a rupture or excision of the internal unit of the perceptual world in relation to the surrounding world. In this situation, the subjective and the objective are confused, thoughts that are determined by a delusion are verbalized and are heard as coming from the outside; (2) In another group, the acute confusional syndromes, where there is a dissolution of consciousness to an oneiroid level, with a characteristically associated obnubilation; (3) In another, acute psychosis ("*bouffée délirante*"), where there is also a characteristic dissolution of consciousness to the oneiroid level, with a predominance of visual hallucinations and a very marked distress (Goas, 1966).

Lopez Ibor (1906–1991) advances with a new pathophysiology for the hallucination. According to the author, in normal perception of real objects a transmission of a signal occurs that will be deposited in the CNS (central nervous system) in the form of an engram. In the hallucination there is an abnormal activation of these engrams by direct stimulus made by the center of ideas (with their affective, intuitive component), so that individuals will experience the phenomenon with all the characteristics of a normal sensory perception. In such cases, there is a clear fusion of the inner and external worlds, both objective and subjective (Lopez Ibor, 1964).

Slade and Bentall (1988) define hallucination as any experience similar to a perception that: (1) occurs in the absence of an appropriate stimulus; (2) has all the strength and impact of the corresponding real perception; (3) is not susceptible of being voluntarily directed or controlled by those who experience it. These authors suggested that hallucinations occur as a result of the inability of the subject to distinguish if an object is real or if it is a product of the imagination, i.e., hallucinations would be caused by a deficit in the metacognitive capacity of assessment/discrimination of reality (Slade and Bentall, 1988).

Based on a thorough review of the history of the concept, Berrios (1995) recently introduced a broader classification of hallucinations, defining them as "verbal reports of sensory experiences with or without insight, not vouchsafed by a relevant stimulus" (p. 229). By doing so, Berrios leaves little room to the development of related concepts such as "pseudohallucination" and "hallucinosis," whose theoretical validity is very debatable, according to this author.

The concept of hallucinations was also addressed within the phenomenological philosophy. The most representative example was given by Merleau-Ponty. He considers that the malfunctioning of both the power of summoning and perceptual faith originate hallucinations. The first is the capacity to bring appearances to existence. The second relates to promptings one extracts from the world. Individuals may perceive the world in an infinite number

of options. Until further experience there is room for illusion. In that sense, one needs faith in order to perceive the horizons as satisfiable, as real. When both perceptual capacities mentioned are altered hallucination arises (Merleau-Ponty, 2002; Romdenh-Romluc, 2007).

Beyond descriptive psychopathology and phenomenological models of hallucinations there were many explanatory models developed throughout the twentieth century, some of them are still in investigation. One of these models was the psychoanalytical model. Freud initially believed that hallucinations resulted from forgotten traumatic experiences from childhood, which returned and forced themselves into consciousness (Bloom, 2010). He revised his theory afterwards and argued that hallucinations were fantasies or wish fulfilments, recreating things which have been lost or destroyed earlier (Eigen, 2005). For Freud "wishing ends with hallucination" (Eigen, 2005, p. 41).

Jung argued for a need to focus on the psychology of hallucinations and their content. He reported that "hallucinations contain a germ of meaning" and that "a personality, a life history, a pattern of hopes and desires" lie behind such experiences (Jung, 1963, p. 127).

On the other hand, Lacan focused on the subjective effect of hallucinations on the person that experiences them. According to Lacan "hallucination is a perceptum that has a paradoxical effect on the percipiens. Rather than a perceptum without an object, it is a perceptum that disrupts the subject. Lacan first emphasises the effects of perplexity that hallucinations evoke, in terms of disrupted signification" (Vanheule, 2011, p. 102). Later on he elaborates on this viewpoint and argues the possibility of hallucinations having a stabilizing and pacifying effect (Lacan, 1993; Vanheule, 2011).

Other models are the cognitive-perceptual, the anatomical, and the biochemical ones. Regarding cognitive-perceptual theories developed since the twentieth century, hallucinations can be seen as erroneous perception or "sensory deceptions." Both bottom-up (or data-driven) and top-down (or conceptual) models have been distinguished. The first occurs with sensory perception impairments, for instance, when older or sensory deprived people present with auditory hallucinations, or when people with deficits in processing visual stimuli present with visual hallucinations. The second, that has been chosen by several authors, occurs in the brain of the perceiver and is not related to the external world. For Bentall (1990) hallucinations are the result of the failure of source monitoring, which is a metacognitive skill necessary to discriminate between both internal and external sources of information. For (Hoffman and McGlashan, 2006), who uses a psycholinguistic approach, a parasitic memory can disrupt language production and thus originate auditory hallucinations. For David (1994) inner speech may be at the basis of verbal hallucinations through a disruption of the "inner-voice-inner-ear system." Other authors consider a misattribution of internal events to an external source. In this sense, hallucinations could be related to "normal intrusive thoughts" externalized due to motivational factors (Morrison et al., 1995; Kumar et al., 2009).

Several functional and structural neuroimaging studies have been preformed to try to find the anatomical correlates of hallucinations [mainly in auditory verbal hallucinations (AVH)]. The most consistent findings are an association between AVH and: (1) structural abnormalities in the Superior Temporal Gyrus and Inferior Frontal Gyrus; (2) an association between the hyperconnectivity in the Arcuate Fasciculus, (3) functional activation in Superior Temporal Gyrus and Inferior Frontal Gyrus, insula, cingulate, cerebellum, and supramarginal gyrus (McCarthy-Jones, 2012). Nevertheless, most of these studies are done in patients with many additional symptoms other than hallucinations (usually in patients with schizophrenia) and therefore these changes might not be specific to hallucinations.

A biological model based on neurotransmitters has also been proposed. This model has been developed after the emergence of antipsychotics. In schizophrenia there is evidence that very high levels of dopamine in the limbic system play a major role in the emergence of hallucinations and delusions. Antipsychotic medications, which block central dopamine activity, alleviate hallucinations in psychosis (Kapur, 2003). These medications do not only improve hallucinations but also other psychotic symptoms such as delusions. Again this model might not be specific to hallucinations.

Pseudohallucinations

The term "pseudohallucination" (literally, false hallucination) has been used with different meanings throughout the history of its existence. The term appears to have been introduced in 1868 by Hagen, to define "illusions or sensory errors," such as hypnagogic hallucinations (that occur in normal subjects when falling asleep and are experienced passively, unintentionally, with a lack of clarity and objectivity and maintaining awareness of their falseness; Hagen, 1868). But before Hagen, other authors have described similar phenomena without designating them with this term. On the other hand, the development of the concept pseudohallucination occurs close to the term hallucination and has even been used to respond to many of the conceptual issues that arose in relation to the latter (normality vs. abnormality, if they come from inside or outside the subject's mind). Although the importance of distinguishing between complete perceptual experiences and pseudoperceptions had been stressed ever since the medieval christian authors (like Saint Augustine and Saint Thomas Aquinas), the first real contributions to the development of the term pseudohallucination emerged in France in the mid-nineteenth century.

By defining the concept of hallucination as a "conviction of perceiving a sense to which there is no external object," Esquirol gives way to the possibility of the arousal of pseudohallucinatory experiences in which the patient's conviction is significantly weaker (Esquirol, 1845).

Other French authors also contributed to the construction of the concept of pseudohallucination, introducing some constructs that have much in common to the later concept of pseudohallucination. Buchez proposed that hallucinations should be divided between involuntary and voluntary (the latter considered normal, occurring in artists; Berrios, 1996). Maury, on the other hand, stated that hallucinations observed in mystics, usually in the context of exhaustion and prolonged fasting,

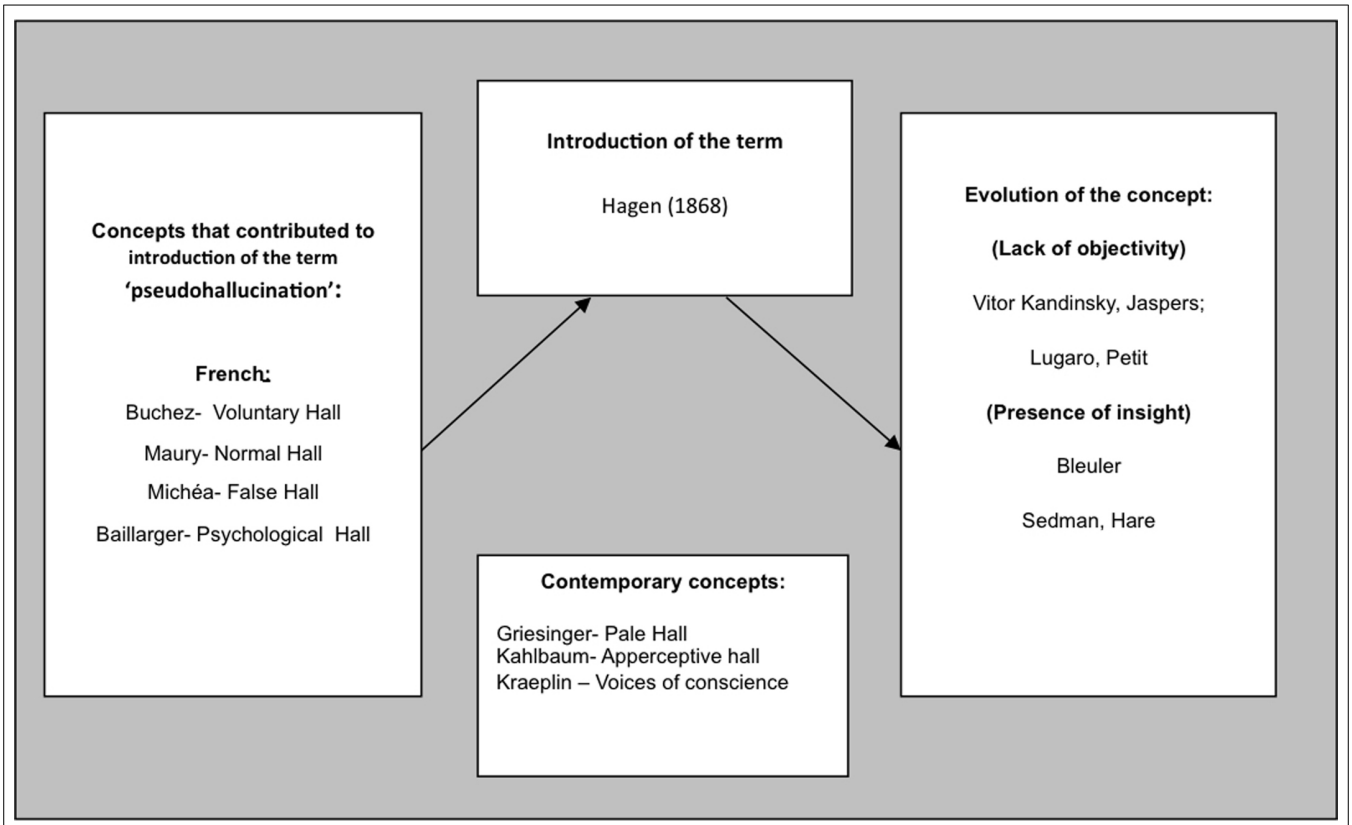

FIGURE 2 | HALL-Hallucination. Here are represented the French concepts that could have influenced the introduction of the term "pseudohallucination" by Hagen (1868); the contemporary German concepts that are very similar to pseudohallucinations but have different names; and the further evolution of the term: some authors emphasize the lack of objectivity as the main difference between hallucinations and pseudo hallucinations (Kandinsky, Jaspers, Lugaro, Petit) but others focus instead on the presence of insight (as Bleuler, Sedman, Hare).

correspond to the "normal hallucination type" and Boismont introduced the term "physiological hallucinations" to include the hallucinations of mystics and other visionaries whom he refuses to consider alienated (Berrios, 1996). Michéa, in 1840, referred to a type of phenomenon he called "false hallucinations," which he considers as intermediates between an idea and true hallucinations: "False hallucination is more than an idea, since its object reveals a vivid and defined shape, which is very close to the appearance of a physical element, but is less than a true hallucination, as it will never impose itself as a real perception, however vivid and defined it is" (Michéa, 1851, pp. 113–114) (**Figure 2**).

Baillarger distinguishes between psychological hallucinations (depending on imagination) and psycho-sensory hallucinations (depending on both imagination and senses). He said about psychological hallucinations: "their voices are intellectual—they occur within the soul," and that the "patients hear their thought by means of a sixth sense" (Baillarger, 1886, pp. 384–389). These statements are very close to the actual meaning of pseudohallucinations.

Nevertheless, the origin of the term pseudohallucination has been attributed to Hagen, as stated before. It was in his 1868 book "Zur Theorie der Hallucination," that he defined them as "illusions or sensory errors," as mentioned above, and that they could be confused with true hallucinations. In this work Hagen cites several authors such as Esquirol, Falret, Bailarger, Boismont, among others, that had described concepts close to pseudohallucinations before him. Therefore, it is not fair to attribute the origin of pseudohallucinations solely to Hagen (1868), and to forget the important contribution of other authors, namely from the French School (as apparently did Jaspers) (**Figure 2**).

Other Hagen contemporary authors from the German school also played a key role in developing the concept of pseudohallucination. Griesinger (1861) stated that "there is a difference between hallucination and the inner exaltation of the imagination (. . .) we can ask ourselves whether this difference is specific or just a matter of degree (. . .) I've seen an interesting transformation of obscure hallucinations, pale, internal (*blassen Mithallucinirens der inneren Sinne*), accompanying the perception of hallucinations with a real objective clarity" (p. 91).

Kahlbaum also published an important work on the pathophysiology and the clinic of hallucinations. According to him, there would be an organ of apperception that generated centrifugal hallucinations (or apperceptive hallucinations–closely related to the spontaneous activity of memory and little with true sensory content; Lange, 1900).

In addition to hearing clear hallucinations as voices from the outside world (which as he stated were predominant)

Kraeplin (1919) noted that patients with *dementia praecox*, also sensed "voices of conscience," "false voices," "voices that do not speak with words," where there is an "inner feeling in the soul." These descriptions aligned with those corresponding to pseudohallucinations.

The term "pseudohallucination" was then further explored by other authors such as the Russian psychiatrist Kandinsky (1885) In his work, he described his hallucinatory experiences defining pseudohallucinations as "subjective perceptions similar to hallucinations, with respect to its character and vividness, but that differ from those because these do not have objective reality" (Kandinsky, 1885, p. 134) (**Figure 2**).

It was based on the descriptions of Kandinsky that Jaspers developed his theory about pseudohallucinations, which is, still today, the most widespread in the scientific community. According to this author pseudohallucination is a false hallucination, and a phenomenon that could be, at first sight, mistaken for a true hallucination. In pseudohallucinations there is a lack of sensory consistency (objectivity, detail, and corporality) and they are not located outside the subject's mind. Other features of pseudohallucinations correspond to the fact that they do not usually persist for long over time and can be modified by will (initiated or interrupted; Jaspers, 1963).

Bleuler (1934) has defined pseudohallucinations in a slightly different way from Jaspers, describing them as perceptions with full sensory clarity and normal localization, but whose falsity is recognized, thus emphasizing the importance of insight.

Lugaro, a Florentine alienist, published, in 1903, a paper on pseudohallucinations in which he defines them in a very broad and current way: (1) pure representations, without the objectivity of hallucinations; (2) they present ego-dystonia (*carattere di estraneità alla personalità*); (3) they result from an irritation in the associative centers (and not in the sensory centers); (4) they may produce secondary delusions; (5) they can be seen in prolonged psychotic states as in chronic schizophrenia (Lugaro, 1903).

Petit (1913) writes *Essai sur une varieté de pseudo-hallucinations: les auto-représentations aperceptives*. According to him this kind of phenomena, whose conceptualization dated back to Baillarger, Kandinsky, among others, could acquire multiple presentations and had only in common that they looked very much like hallucinations. Among the various subtypes of pseudohallucinations, Petit (1913) described a group of automatic phenomena (i.e., which arise spontaneously and impose their presence on the subject), that are recognized as not being caused by any sensory source, that lack the attributes of external perceptions, and that are directly experienced in consciousness, while also being perceived as egodystonic (*créations exogenes, étrangéres par leur origine à son Moi conscient et créateur*).

It was only in the second half of the twentieth century that the Anglo-Saxon school also took its part in the conceptual development of pseudohallucinations. Sedman (1966) considered them a kind of hallucination perceived through the senses but recognized as false. Hare (1973), defined them as subjective sensory experiences of morbid origin that are not interpreted in a morbid way because they are recognized by the patient as real (Hare, 1973). Hare was thinking of classic authors, such as Bleuler, who called attention to the importance of insight, something that cannot be present in true hallucinations (**Figure 2**).

In DSM-IV, pseudohallucinations are mentioned in the section on Conversion Disorders and defined as a possible symptom of conversion disorder which occurs with preservation of insight in the absence of other psychotic symptoms, often involving more than one sensory modality, and frequently having a naive, childish or fantastic content and a psychological meaning (American Psychiatric Association, 2000). In DSM-5 this definition faded out, and there is no reference to the term (American Psychiatric Association, 2013).

Illusion

The term *illusion* also appears to have been originally coined by Cicero from the Latin *illusio*, *illusionis* (deception), *illudere* (to deceive).

Esquirol called illusions "sensorial errors" and defined them as perceptions derived from sensory stimulation that are distorted by certain ideas or passions. Ever since then, most authors find consensus in defining illusion as a perceptual distortion of a real stimulus.

Ey et al. (1978) defines illusions as "falsifications of the perception of real objects" and Bleuler (1934) as "pathologically altered real perceptions." Jaspers (1963) also incorporates Esquirol's original concept, defining illusion as experiences that correspond to transpositions (or distortions) of real perceptions where external sensory stimuli unite with certain transposing (or distorting) elements so that in the end we cannot differentiate one from the other (Jaspers, 1963). He discriminates three types of illusions: those that stemmed from inattention, those from altered affective states, and finally "pareidolias." Fish followed Jaspers' view and defined illusions as stimuli from a perceived object combined with a mental image that produces a false perception (Fish, 1967).

In DSM-IV, illusion is defined as misinterpretation or misperception of a real external stimulus, and DSM-5 keeps this definition (American Psychiatric Association, 2000, 2013).

Hallucinosis

This term was introduced by Wernicke (1906)—*Alkoholhalluzinose*, alcoholic hallucinosis—referring to the presence of vivid and threatening acoustic hallucinations in excessive alcohol consumers who maintained insight and showed no disturbance of consciousness associated (Telles-Correia et al., 2014). Later on, Ey et al. (1978) defined "eidolie hallucinosique" as an hallucinatory setting with no associated delusions in patients with insight and that were egodystonic regarding the non-real characteristics of the hallucinations (the hallucinatory experience is disintegrated from the patient's personality).

In the ICD-10, "organic hallucinosis" is defined as: "A disorder of persistent or recurrent hallucinations, usually visual or auditory, that occur in clear consciousness and may or may not be recognized by the subject as such. Delusional elaboration of the hallucinations may occur, but delusions do not dominate the clinical picture; insight may be preserved"

(World Health Organization, 1992). There is no reference to the term in both the DSM-IV and DSM-5 editions (contrary to previous editions, for instance, DSM-III).

Throughout the twentieth century, the term hallucinosis was progressively attributed to other types of psycho-organic syndromes and substance-abuse associated behavioral disturbances, other than alcohol consumption.

Conclusion

We conclude that there has been a major difficulty in both defining and limiting the concept of hallucination, ever since its first appearance; and this is likely because it belongs to a heterogeneous group of symptoms that might be found in a variety of psychiatric disorders and normal physiological states as well (as in hypnagogic hallucinations). On the other hand, there is no cause that necessarily produces a hallucination. This justifies Alonso Fernandez's (1968) statement that "the fact that psychic phenomena like hallucinations don't possess an intrinsic unity results in an impossibility of attributing a globally valid definition to them" (p. 501).

The types of classification of hallucinations also ranged widely throughout history, including etiological (psychic or psychosensory), nosological: (normal/pathological), and phenomenological (with or without insight, with or without sensory consistency) definitions.

If defining hallucination poses such a difficult task one can argue that defining its dependent concepts is an even bigger challenge. For instance, there is a great deal of confusion between the meanings of pseudohallucination and hallucinosis. Luque calls our attention to the fact that hallucinations, as well as delusions, should not be considered one-dimensional but, instead, as the expression of a series of dimensions or factors that constitute them (Luque and Villagrán, 2000). Chen and Berrios have isolated eleven dimensions of hallucinations in a clinically applicable scale: insight, vividness (perceptive detail), complexity, localization (spatial origin attribution), intensity, voluntary control, constancy, bizarreness, situation, attribution (toward a specific event) and connection with delusions (Chen and Berrios, 1996). Haddock conceived a scale to measure dimensions of hallucinations and delusions. The hallucination section was divided into four dimensions (emotional characteristics—negative content, impact; physical characteristics—duration, frequency; control; and cognitive interpretation; Haddock et al., 1999).

Beyond descriptive psychopathology and phenomenological models of hallucinations there were many explanatory models developed throughout the twentieth century, some of them are still in investigation. The anatomical and biochemical models are perhaps the most recent ones and that have not yet found a specific pattern for hallucinations.

There is still a lot to be clarified regarding hallucination as a psychopathological phenomenon and all its derivative concepts. The problem does not lie in its description or classification in terms of descriptive psychopathology.

The problem lies in clarifying in what the hallucination generator structure consists and if it exists as a unitarian or a multiple etiology. This is probably the major issue that supports the differences between distinct authors opinions regarding the concept of hallucination. The fact that the mentally ill patients consider it an image derived from the external world, reaching them by means of a perceptive organ, allows the hallucinatory phenomenon to be classified as a psychosensory disturbance, as it unanimously happens in current days.

Nevertheless, presently, there is no clear notion about pathophysiological mechanisms underlying the phenomenon. Therefore, the descriptive component remains essential, even though, sometimes, blinded by a fierce nosological ambition. In order to reach real biological correlates it is imperative that we go back to fundamental psychopathological symptoms and use them as a primary basis in all investigations.

Studying the history of psychopathology is a powerful way of calibration, by which language in Psychiatry can be improved and prepared for more rigorous quantification.

As Berrios (2011) postulated, the epistemology of psychopathology "has to include a combination of methods as history, philosophy and empirical investigation" (p. 39). The history of psychiatry and psychopathology brings to us some information about the social processes where concepts have evolved, philosophy clarifies if the language used is sufficiently powerful, and empirical investigation tests the validity of the new concepts toward reality (Berrios, 2011).

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in Psychology

MINI REVIEW
published: 15 July 2015
doi: 10.3389/fpsyg.2015.01001

Possible relation between psychosis and the unconscious: a review of "The Unconscious," by Freud

Jacqueline de Oliveira Moreira[1] and Carlos R. Drawin[2]*

[1] Extended General Practice in Health, Department of Psychology, Pontifical Catholic University of Minas Gerais, Belo Horizonte, MG, Brazil, [2] Philosophy, Federal University of Minas Gerais and Jesuit School of Philosophy and Theology, Belo Horizonte, MG, Brazil

OPEN ACCESS

Edited by:
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****Correspondence:***
Jacqueline de Oliveira Moreira, Extended General Practice in Health, Department of Psychology, Pontifical Catholic University of Minas Gerais, Rua Congonhas, 161, Belo Horizonte, MG, Brazil
jackdrawin@yahoo.com.br

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *20 April 2015*
Accepted: *02 July 2015*
Published: *15 July 2015*

Citation:
de Oliveira Moreira J and Drawin CR (2015) Possible relation between psychosis and the unconscious: a review of "The Unconscious," by Freud. Front. Psychol. 6:1001. doi: 10.3389/fpsyg.2015.01001

This review intends to present some elements of the Freudian thinking on psychosis, focusing on the relations between psychosis and the unconscious. The unconscious phenomena which episodically cross the neurotic individual are massively and continuously shown on psychosis. The psychotic individual appears to be constantly invaded by the *other*, like a strange person, which bursts inside of him/her and presents itself as a threat to the process of construction of this person's identity. But what is the relation between the unconscious and psychosis in the Freudian text? It could be hypothesized that the psychotic individual may be invaded by a pulsating unconscious which demands a symbolic mediation. This reveals the importance of associating verbal construction to medication in cases of psychosis.

Keywords: psicose, unconscious, Freud, Freudian Theory, metapsychology

It is known that Freud's professional background was in Neurology, under the orientation of the intensely anatomic 19th century German medicine (although Freud was Austrian, his most influential teachers were representative of German neurophysiologic tradition and supported the scientific presuppositions of the Berlin Physicalist Society). Therefore, Freud's medical practice did not take place in psychiatric institutions and his oeuvre is not extensively dedicated to the issue of psychosis. Nevertheless, it is undeniable that Freud's efforts to theorize psychic pathologies offer relevant contribution for a dynamic comprehension of psychosis, which had been systematically investigated by Emil Kraepelin. One should not fail to mention the fact that Freud made an appropriation of the clinical material collected by Bleuler and Jung in order to corroborate the hypothesis which suggests that, in cases of schizophrenia, words may be treated as things (D*inge*), approximating schizophrenic speech and neologisms to the "organ speech," which is a characteristic of hypochondria.

Freud's interest in psychosis can be noticed since the very beginning of his theoretical work, as, motivated by his investigations regarding neurosis' etiology, he compared characteristics of neurosis to classic psychotic pictures, such as melancholy (Freud and Draft, 1895a) and paranoia (Freud and Draft, 1895b). This comparative and etiological investigation method appeared also in seminal articles such as "The Neuro-psychosis of Defense" (Freud, 1894) and"Further Remarks on the Neuro-psychosis of Defense" (Freud, 1896). At that time, Freud intensely dedicated himself to unveil the psychic mechanisms of phobias, obsessions, and, more specially, of hysteria. The studies conducted during this period led to two major theoretical formulations: "The Interpretation of Dreams" (Freud, 1900) and "Three Essays on the Theory of Sexuality" (Freud, 1905). Nonetheless, clinical phenomena were obscure, and matted, demanding, thus, well-thought-out theoretical criteria for grounding psychopathological classifications. It is importante to mention that the

matter of transference is also taken into consideration in the lacanian thought. In *On a Question Preliminary to any Possible Treatmentof Psychosis* (Lacan, 1957–1958), Lacan argues that there is a possible treatment of psychosis. Further discussions on this, however, would extrapolate the proposal of this article, which is to review a Freudian text.

Firstly (Freud, 1985–1900), Freud accepts the distinction between neurosis and psychosis elaborated by German psychiatry, but his focus was directed toward the distinction between "current neuroses" (originally translated to English as "actual" neuroses), originated from sexual somatic dysfunctions, and "psychoneuroses" originated from psychic conflicts.It is important to mention, here, Freud's classic reading of an autobiographic report: the Schreber case (Freud, 1911b), which described a case of paranoia. Later, in a moment in which Freud faced difficulties to obtain a wider theoretical synthesis (1914–1920), he deepened the distinction between neurosis and psychosis through the discovery of the narcissistic constitution of psyche. This was the point in which he elaborated core texts, such as "On Narcissism: An Introduction" (Freud, 1914) and the "Papers on Metapsychology" (Freud, 1915–1917), that showed a deep connection between theory, clinical work and psychopathology. Thus, the 1915 classification maintains the distinction between "current (actual) neuroses" and "psychoneuroses," and also observes the differences between two types of psychoneurosis: "transference neuroses" (obsessional neurosis, hysteria, and phobia) and the "narcissistic neurosis." The firsts preserve the libidinal connection with objects, allowing the establishment of transference in the analytical process. In the case of the latter, libidinal connection is compromised, and the libido originated from objects returns to the Ego and is crystallized in a narcissistic position, limiting the individual's access to external reality.

In his final psychopathological classification (Freud, 1924a,b), Freud retakes the consecrated designation of "psychosis," which now included pictures of schizophrenia and paranoia, leaving the term "narcissistic neurosis" to refer only to pictures of melancholia. In the article "Neurosis and psychosis" (Freud, 1924a) and "The Loss of Reality in Neurosis and Psychosis" (Freud, 1924b), the thinker distinguishes these two concepts using the hypotheses proposed 1 year earlier, in "The Ego and the Id" (Freud, 1923): "Neurosis and Psychosis," for instance, proposes a topic differentiation based on "The Ego and the Id" and, thus, proposes a third picture, namely the "narcissistic neuroses." While transference neurosis presents a conflict between ego and id and the psychoses present a conflict between the ego and the external world, the narcissistic neuroses consist on a conflict between ego and superego.

This brief introduction to the long trajectory of Freudian investigation does not make justice to its subtleties and theoretical oscillations, but may be useful for the focalization of a moment of crucial transformation in his comprehension of psychosis. This moment is represented mainly by two exceptionally fecund metapsychological texts (written almost simultaneously, but published within a 2 years interval): "The Unconscious" (Freud, 1915a) and "Mourning and Melancholia" (Freud, 1917).

It is known that, during the First Topography (Freud, 1900–1920), Freud manifested great interest in the "Psychoneuroses of Defense." However, after acknowledging the existence of narcissism, he started using the term "Narcissistic Neurosis" to define the clinical experience of psychosis, based on metapsychology, and psychopathology. The concept of transference represents, in this perspective, the defining criteria for the two types of psychoneurosis, namely, transference neurosis and narcissistic neurosis. In the narcissistic neuroses with reflux of libido, there is a barrier for an effective process of transference, that is, the transference bond with the analyst does not occur, and the libido gets repressed. Nonetheless, this review article focuses, mainly, in presenting the Freudian thoughts on psychosis and its articulation with the concept of unconscious and the issue of the other, explaining, thus, the focus on "The Unconscious" (Freud, 1915a).

The phenomena of the unconscious episodically cross the neurotic individual and announce, through various symptoms, dreams, and faulty acts, the discontinuity in the egoic functioning. In the psychotic individual's case, these phenomena are even more common, appearing continuously and massively in their lives. These individuals appear to be constantly invaded by the *other* which bursts inside of him/her and often presents itself as a threat to the process of construction of this person's identity. In the aforementioned 1915 article, Freud develops an association between the schizophrenic's conscious speech and the oneiric processes through which this individual goes. He concluded that the psychic processes of psychosis are subject to the primary process, to a free flow of energy which announces a regression to the hallucinatory satisfaction of desire (*Wunsch*): in the case of schizophrenia, the individual regresses to auto-erotism; and, in the case of paranoia, he/she regresses to primary narcissism.

This fixation with the primary process, which is ruled by the principle of pleasure, produces a schism with reality that compromises a wide range of gains originated by the principle of reality. Skills such as attention, judgment, and rational thinking are lost when there is a schism with reality in the psychotic mind. According to Freud, this is a difference between neurotic and psychotic individuals: while the first do not repudiate reality (they only ignore it), the latter not only repudiate it, but try to replace it (Freud, 1924b).

In this perspective, the author notices that "all observers have been struck by the fact that in schizophrenia a great deal is expressed as being conscious which in the transference neuroses can only be shown to be present in the *Ucs*. [Unconscious] by psycho-analysis[1]" (Freud, 1915b).This statement led to the proposition of the hypothesis that, in narcissistic psychoneuroses (psychoses), one can witness the "unconscious open to the sky," since words explicitly and directly reveal unconscious content. However, Freud also shows that the character of strangeness of schizophrenia results from the predominance of word-representation (*Wortvorstellung*) over thing-representation (*Sachvorstellung*).The second proposition seems to contradict

[1]The author has used as source for consult a Brazilian edition in which this passage can be found on Freud (1915b, p. 225).

the hypothesis which simply defines psychosis and unconscious. Word-representation, articulated with thing-representation, belongs to the preconscious and is what makes communication with the outside world possible. In the unconscious, on the other hand, this articulation between word-representation and thing-representation does not exist: only thing-representation is observed. Therefore, it can be **hypothesized** that, if in psychosis there is a domain of word-representation over thing-representation, and the first is absent in the unconscious, it is not correct to affirm that psychosis is a direct expression of the unconscious, or the unconscious "open to the sky."

Nonetheless, a doubt soon came to Freud's mind: how can we affirm the presence of the unconscious in psychosis if there has been a rejection of castration (*Verwerfung*), and repression has not taken place? On the one hand, the unconscious system is ruled by the primary process. Since the psychotic individual's psychic apparatus is also ruled by the primary process, with a domain of the principle of pleasure over the principle of reality, it can be **hypothesized** that there is a predominance of the unconscious system in cases of psychosis. On the other hand, the theory of psychic apparatus, developed in Freud's First Topography, had established a structural relation between the unconscious and repression. Is it possible to conclude, thus, that this affirmation of the First Topography is incorrect and the immediate association between unconscious and repression should be abandoned? It can be noticed that, by facing the issue of psychosis, Freud was obligated to review and question metapsychology. He, then, introduced the concept of Id as a possible response for such crucial matters.

It appears, hence, that in psychosis repression keeps on operating as a primal repression (*Urverdrängung*), but a later incidence or proper repression and its effect of psychic splitting would also be seriously affected. In this perspective, the unconscious in psychosis can be understood in a dynamic and economic manner, but not in a topic way. There is no structural splitting in the two great systems, which means that the preconscious does not censor the unconscious contents and the borders are rather fluid. Nevertheless, a clear manifestation of this structural absence of barriers, i.e., limits that circumscribe different systems, only occurs after a first episode in which the individual is taken over by the *other*, by a stranger who inhabits his/her body. Therefore, it is very common for the psychotics not to recognize themselves and speak of themselves in the third person.

But how would this refusal of reality occur in the perspective of the First Topography? It is possible to suggest that the perception of external objects results from a combination of word-representation and thing-representation, an articulation which takes place in the preconscious system. In psychosis, the perception and interpretation of reality fail, since, instead of being libidinously invested in the preconscious system, the thing-representation occurs in the unconscious. This means that the preconscious system is invaded by the unconscious in a search for primitive forms of libidinous satisfaction, such as hallucination. It is more of an invasion performed *by* the unconscious than a topic regression *to* the unconscious, although it can be argued that there is a temporal regression to archaic forms of egoic organization, such as auto-erotism, and primary narcissism. Temporal regression and primitive forms of satisfaction help the individual bypass frustration and are impregnating, as they represent means of self-satisfaction and the capability of autistically finding satisfaction.

Freud's image of a bird's egg, with its provision of food inside the shell (Freud, 1911a), can well illustrate these psychotic mechanisms. In schizophrenia, object-cathexes are abandoned through the annulment of preconscious thing-representation: only an intensely invested word-representation remains. However, as the thing-representations inscribed in the unconscious, the primitive object-cathexes have proximity with word-representation, revealing a strange phenomenon in which words and things are equal. In this perspective, in psychosis, the words turn into things, i.e., the psychotic speech is characterized by a concreteness which interferes in the symbolic procedures of communicational language. In summary, this structural weakening of the psychic apparatus splitting, which prevents the distinction and articulation of unconscious thing-representation and preconscious word-representation, worsened by the emptying of preconscious thing-representation, distorts the perception of external reality and, more especially, hampers a socially shared interpretation of this reality. In psychosis, there is commonly an imaginary mold and a concrete understanding of culture's symbolic net.

It is valid to mention that, in several of Freud's texts throughout his career, the division of consciousness hypothesis is presented based on an intense affective experience which is incompatible with other representations and cannot be assimilated by the ego, constituting, thus, a situation of trauma. In the article "The Neuro-Psychosis of Defense" (Freud, 1894), the traumatic experience is seen as responsible for the triggering of defense hysteria (*Abwehrhysterie*). Later, after elaborating the idea of death drive, Freud shows in "Beyond the Pleasure Priciple" (Freud, 1920) that the principle of pleasure is in service of death drive, facing trauma as a structural condition of psychism. In "Splitting of the Ego in the Process of Defense" (Freud, 1940), Freud carries on with this theory and argues that psychic conflict produces a splitting of Ego which, in some cases, may lead to a severe loss of reality.

Returning now to the matter of psychosis in *The Unconscious* (Freud, 1915a), one can affirm that the primary process, the free flow of energy, becomes dominant in the way the psychic apparatus functions in psychosis. According to the first pulsional dualism, it can be stated that the psychotic individual is ruled by the principle of pleasure/displeasure, which does not mean that the psychotic experience is drowned in a quest for pleasure, as this would contradict clinical experience. Despite the occasional insinuation of this confusion in Freudian texts, the logic of his theorization is clear: pleasure/displeasure refers to a principle of psychic functioning rather than an affective experience, and, consequently, its predominance or intrusion may (and they frequently do) produce great psychic suffering. This apparent contradiction may evoke a certain perplexity, hence the need to explain the reiteration of suffering. Freud (1920) saw the necessity to define death drive as something that goes "beyond the pleasure principle," as an extreme and destructive limit to the functioning

of the psychic apparatus. Such hypothesis, anticipated in the 1895 "Project for a Scientific Psychology" (Freud, 1895) through the idea of *neuronal inertia* as total energy flow, had been counterbalanced by the idea of constancy, in which the balance of libidinous investments oppose to the tendency to point zero, i.e., the tendency to return to death (?), to the condition of inanimate matter. This initial idea was reconsidered, with a deeper elaboration, when ideas regarding psychosis confronted the theorization of the First Topography.

Therefore, it appears that the *other* (stranger) who frightens the psychotic individual and invades his/her body represents the imaginary image of death drive, and the intense suffering that it produces testifies not the return of what has been repressed, as happens in neurosis, but the threatening and disruptive presence of a pulsating unconscious. Chaotic excitements are mixed with external sources of stimulation, imprisoning the psychotic individual, who has no contact with the stabilizing counterpoint of social reality. This overpowering "pulsating unconscious" presents itself as a profoundly deformed type of alterity, an uncontrollable *other* that does not submit itself to the regulations imposed by the encounter with the other of mediations and symbolic interactions. In fact, the psychotic experience represents a lack of comprehension of the alterity dimension as the recognition of difference built in the process of intersubjective relations.

In the perspective of Freudian psychoanalysis, the psychotic episode happens when the individual is eclipsed; when, facing the frustration of the loss of an object, this individual directs the libido to him/herself, in an effort to desperately invest in his/her ego, which justifies the designation of narcissistic neurosis. In this desperate movement to support themselves, the psychotic and, more especially, the schizophrenic individual, to use the Freudian terminology, are concealed in the auto-erotic circuit of a shattered body. Delusional thinking would have, thus, especially in cases of minor paranoia and its systematic construction, an ordering function, since object-investments would make a re-encounter with the other possible, in a real dimension of alterity which would be represented in and by delusions.

Nonetheless, in the vortex of psychotic episodes emerges the *other*, with no social alterity, the devouring and impersonal presence of a "pulsating unconscious," a presence of this unrepresented drive which cannot serve as a mold for the disordered excitements and pulsating chaos. Mental automatism, common in psychosis, may be understood as the presence of an *other*, of an exteriority, in the core of a person's intimacy. The psychotic individual, affected by mental automatism, complains about invasions, abuse, usurpation, voices which scream, and other revealing phenomena of a stranger inhabiting the mind and unsettling the idea of unity and identity.

Therefore, when facing psychotic phenomena through the perspective of Freudian theorization, clinical strategy should consist on introducing symbolic mediation, sometimes represented by the analyst in the institutions, which could offer the psychotic individual an opportunity to reconstruct his/her dimension of alterity. Managing these situations in a context of transferential failure is not at all easy and demands of the analyst the ability to make good use of opportunities of stabilization provided by delusion. In spite of the major difficulties of this task, always taken into account by Freud, it is not unreasonable to conclude that coping with it is a true ethical imperative for the analyst.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in Psychology

OPINION
published: 13 April 2015
doi: 10.3389/fpsyg.2015.00401

DSMs and the Brazilian psychiatric reform

Fuad Kyrillos Neto[1]*, *Jacqueline de Oliveira Moreira*[2] and *Christian I. L. Dunker*[3]

[1] Research and Extension Center, Department of Psychology, Federal University of São João del-Rei, São João Del Rei, Brazil, [2] Extended General Practice in Health, Department of Psychology, Pontifical Catholic University of Minas Gerais, Belo Horizonte, Brazil, [3] Laboratory of Social Theory, Philosophy and Psychoanalysis, Department of Clinical Psychology, Institute of Psychology, University of São Paulo, São Paulo, Brazil

Keywords: psicopatologia, diagnosis, DMS, psychiatric reform, ways of living

The present text proposes a reflection on the point of recent history of changes in the Brazilian public policies for mental health in which DSM's (*Diagnostic and Statistic Manual of Mental Disorders*) practical psychopathology was introduced in services inspired by psychiatric reform.

The counterpoint of these considerations is psychoanalytic clinic, which focuses on "spontaneously achieved diagnosis"—which means that the person who is being submitted to analysis is able to name and to understand their own condition. Psychoanalytic clinic is based on free association, on transference and on discourse; these being the principles which make it possible for the subject to build knowledge on their own symptoms throughout the treatment. This enables us to think that the ability to name and understand their own suffering can be achieved by forms of life which have productive naming practices (self-diagnosis). We believe that using DSM and insisting in the movement of reinsertion (often forced) may produce the effect of silencing the individual and diminishing the possibilities of clinically listening to singular experience.

In this manuscript what interests the most are the impacts of using DSM's classification combined with a discourse which privileges citizenship, "unmedicalization" and social acts in the movement against madhouses.

This movement, which began in Brazil in the 1980s, can be related to major social and ideological changes that took place in the end of Brazilian Military Dictatorship. These changes reached many different sectors of society, including mental health services, which were drastically modified in regards to the way they are organized and how they understand and offer mental health care. In the beginning of the aforementioned decade, there were two main forms of mental health care: the public mental hospitals (where patients actually spent the rest of their lives) and a rapidly growing chain of private psychiatric clinics (Delgado, 2008). In that period, marked by the democratization of Brazilian political system and by the consequent public acceptance of political-ideological debate, some previously marginalized psychiatric and psychoanalytical perspectives found an opportunity to be broadcasted, and, thus, show alternatives for the treatment of psychological phenomena.

The Brazilian Movement of Mental Health Professionals proposed, in that context, a critical review of the hegemonic and centralizing role of psychiatric hospitals as well as of mental health services. According to this new proposition, financial resources and the forms of care and treatment should invariably preserve personal dignity and human and civil rights as well as provide ways of maintaining the patient in their own community, since isolation and social disconnection were considered the main problems of interning patients in mental institutions. Brazilian legislation should adapt in order to ensure that mental patients would have their human and civil rights respected, to reorganize community mental health services and to make sure these new configurations are taken into practice. Furthermore, according to the proposals of this movement, human resources in mental health and psychiatry should be trained in accordance to a model in which community service prevailed and the new principles for hospitalization were to be followed.

OPEN ACCESS

Edited by:
Diogo Telles-Correia, University of Lisbon, Portugal

Reviewed by:
Sérgio Saraiva, Centro Hospitalar Psiquiátrico de Lisboa, Portugal
Diogo Frasquilho Guerreiro, University of Lisbon, Portugal

***Correspondence:**
Fuad Kyrillos Neto, fuadneto@ufsj.edu.br

Specialty section:
This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology

Received: *24 December 2014*
Accepted: *21 March 2015*
Published: *13 April 2015*

Citation:
Kyrillos Neto F, Moreira JO and Dunker CIL (2015) DSMs and the Brazilian psychiatric reform. Front. Psychol. 6:401. doi: 10.3389/fpsyg.2015.00401

In order to make these directives possible, it is necessary to organize replacement services, which must be prepared to take on new tasks and to respond to the necessities of patients and their families. The Centers of Psychosocial Care (*Centros de Atenção Psicossociais*—CAPS) certainly constitute the most creative and advanced alternative for reaching these goals. There, a contract is established with the patient, regarding the type of treatment, the number of times the patient will go to the hospital every week and the way they will deal with the medication (either at home or at CAPS).

Today, the CAPS are ruled by Ordinance[1] no. 336/GM, published on February 19, 2002, and integrate the Unified Health System (*Sistema Único de Saúde*—SUS). This Ordinance recognized the Centers and amplified CAPS's functioning and complexity, defining that these services must provide continuous treatment to people who suffer with severe and persistent mental illnesses in a certain territory. They must also provide clinical care and psychosocial rehabilitation, substituting, thus, the hospital-centered model which prevailed in the past. CAPS must also make an effort to avoid hospitalization and promote citizenship and social inclusion of patients and their families.

The Centers which are open 24 h a day provide rear beds for both male and female patients, who have access to many diverse types of care offered by the service: day and night hospitality; daytime hospital; home visits; and access to psychiatric medication. In case of a more critical state, indicating the necessity of a more intense accompaniment, the patient will remain in the center (day and night hospitality) and will use one of the rear beds.

Nicácio (1994) points out five essential characteristics of therapeutic practices carried out by these services, namely: guaranteed rights to asylum in the center (which does not translate into isolation or exclusion); rapid responses to critical situations; insertion in their community; investment inversion (meaning to emphasize social production of patients, i.e., without worrying about clinical structure or psychopathologic outlines); and, finally, the process of social valorization, comprehended as the active institutional participation in the process of social interchange.

When analyzing these five characteristics, defined as essential for the ideological project which guide the functioning of the service, the absence of a clinical dimension is noticed. The "investment inversion" to which referred Nicácio (1994) proposes that psychiatry should not emphasize pathology; instead, it should contemplate the complex existence of these patients and their insertion in social context. Therefore, there has been an abdication of the clinical point of view in favor of social context.

In DSM's case, one can raise the hypothesis that this characterization proposal is inspired by pragmatism, renouncing, thus, the notion of mental illness in favor of the idea of a disorder: something that is not in accordance to the previously established order. Therefore, DSM is organized in order to find trustworthy, temporary and operational categories which allow the overcoming of terminological misunderstandings in the field of Psychopathology (Pereira, 2009).

In regards to the use of DSM in public policies concerning mental health, it can be stated that, motivated by the desire to organize an efficient form of investment in public health policies (including policies for mental health care), the managers must know which are the most frequent and prevailing clinical entities in a certain community as well as the real efficacy of the several available therapeutic possibilities. According to Pereira (1996), this is a perspective that thinks of medicine as a concrete form of intervention in the order of life, as well as in social institutions. Mental suffering, in this perspective, is seen as a matter of public health[2]. In contemporaneity, defining clinical practice through efficacy, based in rapid results, becomes an ethical ideal.

It is important to remember that, since the first version of DSM, in 1952, there can be noticed a movement of revitalization of several diagnostic classes which subdivide psychodynamic classes, such as neurosis and psychosis, into increasingly smaller symptomatic units. Thus, it becomes more and more common to see all sorts of people recognizing themselves in a group of clinical signs with some diagnostic value. It appears that this type of diagnosis finds itself in consonance with the globalization of capital, since there is a globalization of presentation and cataloging of the different ways to suffer.

Lately, the conflict between the possible diagnostic reasons and their implications in treatment and organization of services has been intensified. A response to this intensification, a third version of DSM has been developed, and it represents a turning-point in the relations between Psychiatry and Psychoanalysis, since it proposes a non-theoretical classificatory system, operational in great psychiatric syndromes, which can modify the concept of research and psychiatric practice (Mayes and Horwitz, 2005). This conducts us to a situation in which psychic phenomena are comprehended and defined through their topography, and the definition of a mental disorder happens based on the simultaneous manifestation of several symptoms. The subject and their life-story become dispensable for treatment choices.

Therefore, we argue that, even though they have given positive contributions to the thought regarding psychological treatments, DSM and the practices which insist in social re-insertion disconnected of any subjective consideration may lose sight of the individual and their story.

[1]BRASIL. Portaria n 336/GM, de 19 de fevereiro de 2002. Available online at: http://dtr2004.saude.gov.br/susdeaz/legislacao/arquivo/39_Portaria_336_de_19_02_2002.pdf [Accessed 02 February 2015].

[2]Foucault defines Liberalism as the frame of biopolitics. His analysis emphasizes the paradoxal role of society in regards to the government: society tends to limit itself, but the government also intervenes in society in order to produce, guarantee and multiply the liberty necessary for Economic Liberalism (Foucault, 2008).

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

frontiers in
PSYCHOLOGY

MINI REVIEW ARTICLE
published: 03 February 2015
doi: 10.3389/fpsyg.2015.00081

Melancholia before the twentieth century: fear and sorrow or partial insanity?

Diogo Telles-Correia* and João Gama Marques

University Clinic of Psychiatry and Psychology, Faculty of Medicine, University of Lisbon, Lisboa, Portugal

Edited by:
Anne Giersch, Institut National de la Santé et de la Recherche Médicale, France

Reviewed by:
Mark Elliott, National University of Ireland Galway, Ireland
Alexandre Todorov, Washington University School of Medicine, USA
Rebekka Lencer, University of Muenster, Germany

****Correspondence:***
Diogo Telles-Correia, University Clinic of Psychiatry and Psychology, Faculty of Medicine, University of Lisbon, Hospital Santa Maria, Avenida Egas Moniz, Lisboa 1649-028, Portugal
e-mail: tellesdiogo@gmail.com

Throughout the history of psychopathology, several meanings have been assigned to the term melancholia. The main ones were related to affective disorders (fear and sadness) and abnormal beliefs. At the time of Hippocrates melancholia was regarded mainly in its affective component. Since that time, and until the eighteenth century, authors and opinions have been divided, with both aspects (affective disorders and abnormal beliefs), being valued. Finally, in the eighteenth to nineteenth centuries, with Pinel at its peak, melancholia becomes exclusively a synonym of abnormal beliefs. At the turn of the nineteenth to the twentieth century, the affective component returns as the main aspect characterizing melancholia.

Keywords: melancholia, depression, psychopathology, history, eighteenth century, history, nineteenth century

INTRODUCTION

Throughout the history of psychopathology, many terms have been introduced. Some have disappeared, as "*Phrenitis*" and "*Carum*," while others have survived (Berrios, 1996a). Among the surviving terms, some have kept their original meaning, while others have seen their definition change. "Melancholia" is an example of the latter: since its appearance its meaning has differed widely from the current one.

Burton, who dedicated a book to the phenomenon in the seventeenth century, *The Anatomy of Melancholy*, states that: "The tower of Babel never yielded such confusion of tongues as the chaos of melancholy doth variety of symptoms" (Burton, 1883, p. 240).

Throughout history, the two most important meanings of melancholia had to do with affective disorders (mainly depressive mood), and abnormal beliefs. Since Hippocrates, the authors dealing with the subject have alternated between one kind and the other of the symptoms.

Existing work on the subject tends to prefer one side of the two: either insisting that the affective side of melancholia has been neglected before the twentieth century (Berrios, 1996a; Stanley, 1983), or else stating that this aspect has always been paramount (Radden, 2000).

This article aims at reviewing the contributions by the different authors to the construction of the current term melancholia, throughout history, holding the view that both affective and disorders and abnormal beliefs have been valued (and not one of them exclusively).

FROM HIPPOCRATES TO THE EIGHTEENTH CENTURY

Hippocrates (460–379 BC) attributed the melancholic state (as well as other disorders such as dysentery, skin rashes, etc.) to the excess of black bile, one of the four basic humors described by the author (blood, black bile, yellow bile, and *phlegm*) (Hippocrates, 1923). According to the author, these states were clinically characterized by several symptoms, above all fear and sadness.

Galen (129–216 AD) followed Hippocrates in his theory of temperaments, referring four types of temperament resulting from humoral excesses: melancholic (black bile) (in this case, melancholy was the greek name, *atrabilia* the latin name), optimistic (blood), choleric (yellow bile), and phlegmatic ("*phlegm*") (Galen, 1952).

For Galen, along with depressive symptoms ("all patients presented fear and despondency"), melancholic patients showed bizarre and fixed ideas (probably alike to what we call today "delusion") and a behavior which was consistent with such ideation: "There are patients who think to have become a sort of snail so that they must escape everyone in order to avoid having their shell crushed, while others fear that Atlas, who supports the world, may grow weary and vanish, among other imaginary ideas" (Galen, 1976). On the other hand, Galen states that these ideas are restricted to a theme, the remaining life of the patient being unaffected: "although he was able to discuss other issues reasonably and recognise the people who were present, he kept believing that some flutists were constantly playing next to his house. . ." (Galen, 1833).

Aretaeus of Cappadocia (First Century AD), regarding melancholic patients, states that: "Many fear to be given poison to drink; their senses redouble in penetration and acuteness rendering them suspicious and able to an extreme of discerning hostility everywhere" (Trélat, 1839, p. 73–82).

He characterized this illness through two fundamental dimensions: (1) as an emotional phenomenon, consisting of a state of anguish (*animi angor*); (2) as an intellectual phenomenon consisting of a delusional conception that absorbs and fixes the mind ("*in una cogitatione defixus*") (Trélat, 1839).

The notion of a partial delusion was to become dominant in the authors dealing with the subject after Aretaeus, and until the end of the eighteenth century.

Andreas Laurentius (1560–1609) was the author of an important revision of the meanings assigned to the concept of melancholia from Galen to the sixteenth century. He defines melancholia as the presence of "delirium with no fever, but with fear and sadness" (Laurentius, 1599). He adds that "all melancholic patients have a disturbed imagination," and dedicated a great part of his work specifying the type of delusions observed in these patients (fear that Atlas would drop the world, the idea of having been beheaded or swallowed by a serpent, etc.) (Laurentius, 1599). On the other hand, he stresses that "apart from these ideas their imagination remains undisturbed and they are able to speak marvellously of all other issues" (Laurentius, 1599). Laurentius believed that this pathology was caused by an excess of black bile (in the like of Hippocrates and Galen). "The coldness and darkness of this humour affect the mind, especially the imagination" (Laurentius, 1599).

Bright (1551–1615), in his *Treatise of Melancholy*, which is said to have inspired Burton in his later work, states in regard to melancholic patients that they "are for the most part sad and fearful, and such as rise of them: as distrust, doubt, diffidence, or despair" (Bright, 1586).

Burton (1577–1640) tries to gather in his work a summary of all the meanings of melancholia upheld to that date, comparing such a task to "capturing many-headed beast" (Burton, 1883, p. 51). Even so, Burton tries to consistently unify the description of the various authors.

He states that patients suffering from melancholia present multiple symptoms, which "may be infinite," the most frequent being fear and sorrow ("Fear and sorrow are the true characters and inseparable companions of most melancholy") (Burton, 1883, p. 109). However, among the several symptoms described are included ideas of persecution, poisoning and jealousy. He refers that these patients can be "most violent in all their imaginations, not affable in speech." But he stresses that these ideas are not generalized but contained within specific boundaries, as "they are of profound judgement in some things, although in others, non *recte judicant inquieti*" (Burton, 1883, p. 283).

William Cullen (1710–1790) uses the expression "partial insanity" to define the monothematic delusion in melancholia, contrasting with "universal insanity," which he equates to mania. Nonetheless, the author highlights the difficulty of drawing a line between the two situations: "the boundaries between universal and partial insanity cannot always be drawn with accuracy" (Cullen, 1793, p. 76).

For this reason, Thomas Willis (1621–1675), prefers to divide melancholia in two types: the universal type (in which the "delusion" is extensive to almost anything) and the particular type (in which the judgement of the individual is affected only in one or two areas) (Willis, 1683).

Sennert (1572–1637) defined melancholia as: "a concentration of the soul upon the same idea or a delusion acting on a false thought which is almost exclusive," "whose judgement is little changed or only in what regards one object." This author included in melancholic cases states of sadness or joy, as "in melancholy the delusion is sometimes joyful" (Trélat, 1839).

Sauvages (1706–1767) held a similar view, stating that "melancholia is characterised by exclusive delusion," while Lorry (1726–1783), refers to melancholia as "a partial delusion inflated with exciting passion" (De Matos, 2007, p. 25).

In the beginning of the nineteenth century, Benjamin Rush (1746–1813) described two varieties of partial insanity: tristimania and amenomenia. While the former replaced the term "hypochondria" (when the delusional ideas refer to the patient, their condition or matters and are painfully lived), the latter replaced the term "melancholia" (when the delusions refer to objects outside the patient and are lived with pleasure—or absence of pain). It should be stressed that at the time hypochondria was often regarded as a slight form of melancholia and not as it is seen today (Rush, 1827).

Pinel (1745–1826) abandons the humoral theory in favor of a more rigorous descriptive psychopathology, narrowing down mental disorders to four main groups: Melancholia, Mania, Idiocy, and Dementia (Pinel, 1806). In melancholia, the patients are "overwhelmed by an exclusive idea, endlessly recalled in their words, which seems to absorb all their faculties." It is distinguished from mania, "nervous excitation or extreme restlessness, sometimes to the point of fury, and a variable level of general delusion" (Pinel, 1806, p. 150).

For Pinel, melancholia takes on two opposite forms: (1) "a heightening of pride and the chimeric idea of possessing infinite richness and power without limits"; (2) "the most fearful despondency, a profound dejection or even despair, therefore considering two forms of melancholia: depressive and expansive" (Pinel, 1806, p. 136).

Hence, until the eighteenth century, most authors privileged abnormal beliefs to affective disorders in melancholia. Aretaeus' "*angor animi*" had disappeared leaving only the concept of the mind absorbed "*in una cogitatione*."

AFTER THE EIGHTEENTH CENTURY

One of the most important steps for the change taking place in the turn of the eighteenth to the nineteenth century was the work of Esquirol (1772–1840), who labeled "monomania" several situations of partial insanity, characterized by delusion which was limited to one object or to a restricted number of objects: "I propose the word monomania a term which express the essential character of that form of insanity, in which the delirium is partial, permanent, gay, or sad" (Esquirol, 1845, p. 200). "Regarding affectivity, they take on two forms: depressive (lypemania), and expansive (monomania itself)" (Esquirol, 1845, p. 202).

According to Esquirol himself, "lypemania" is very similar to Rush's "tristimania" and to Pinel's "depressive melancholia", while "monomania" is similar to Rush's "amenomania" and to Pinel's "expansive melancholia" (Esquirol, 1845).

Therefore, partial insanity is no longer comprehended in the term "melancholia" and is given a new designation, that is, "monomania." Esquirol states that: "writers have confounded monomania with melancholia because in both the delusion is fixed and partial." Within "monomania," a specific type may be distinguished due to its depressive affective content, "lypemania," which Esquirol uses as a synonym for "melancholia," ("lypemania or melancholia") throughout his work. In this way, melancholia regains its connotation of a depressive state. The further disappearance of monomania causes the term "lypemania" to fall in disuse, while "melancholia" remains in use.

Two of Esquirol's disciples, Falret (1794–1870) and Baillarger (1809–1890) coined the terms "circular insanity" ("folie circulaire") and "dual form insanity" ("folie à double forme"), based on the affective characteristics and motor activity in which manic excitement alternates with mental depression. These would come to inspire Kraepelin's nosology (Radden, 2000).

Tuke (1827–1895), in his compendium "A dictionary of psychological medicine, giving the definition, etymology and synonyms of the terms used in medical psychology, with the symptoms, treatment, and pathology of insanity," edited in 1892, points out affective symptoms as paramount in melancholia: "A disorder characterized by a feeling of misery which is in excess of what is justified by the circumstances in which the individual is placed" (Tuke, 1892, p. 787). He also refers the frequent presence of delusion: "as a rule, the disorder of feeling is accompanied, with more or less evidence, by a disorder of thought, and actual delusion accompanies the melancholia" (Tuke, 1892, p. 789). This delusion could take on any type. In Tuke's dictionary are included other reviewed concepts, such as "monomania" or "circular insanity," faithful to the designations of the original authors. Curiously, in Tuke's dictionary, the term "mental depression" is often quoted as an antonym of exaltation and a synonym of simple melancholia, without delusion, "characterised by the chief symptoms of simple melancholia; the patients look sad without having melancholic delusions" (Tuke, 1892, p.240). This term, with a physiological connotation, will be diffused until the twentieth century (Berrios, 1996b).

At the end of the nineteenth century, kraepelinian nosology separates affective disorders (Manic Depressive Illness, including melancholia) from Dementia Praecox. Kraepelin overvalues affective symptoms in the former and thought and cognitive changes in the latter. Therefore, at the turn of the nineteenth century, the idea of melancholia as a disorder mostly affecting the abnormal beliefs restricted to some objects is gradually abandoned in favor of a disease mostly characterized by affective symptoms, namely depressive ones.

In the beginning of the twentieth century the term melancholia was gradually displaced by the term depression, which had a physiologic connotation, showing up in the medical manuals as "mental depression" (Berrios, 1996b).

Nowadays the term melancholia is reserved to certain cases of severe depression, usually known as endogenous depression. This kind of depression is characterized by the presence of profound sadness, anhedonia, loss of emotional resonance, vegetative symptoms (insomnia, anorexia, circadian variability in mood), a seasonal pattern, motor retardation and presence of delusions and/or hallucinations. It is thought that the endogenous depression has mainly an organic cause (with several neurobiological alterations, including psychoimmunological), and a better response to medication (and in severe cases electroconvulsive therapy) than the reactive depression (also called neurotic or situational depression) (Telles-Correia, 2012).

It is in this perspective that the term melancholia appears in the DSM-IV and DSM-5, as "depression with melancholic features." In the ICD-10 the term melancholia is not present anymore.

DISCUSSION AND CONCLUSIONS

Since its appearance, the term "melancholia" has suffered an evolution in its meaning.

At the time of Hippocrates, "melancholia," resulting from an excess of black bile, was a state characterized by affective dimensions: fear and sadness.

Galen upheld Hippocrates' physiopathological theory of melancholia. However, apart from the affective characteristics, he

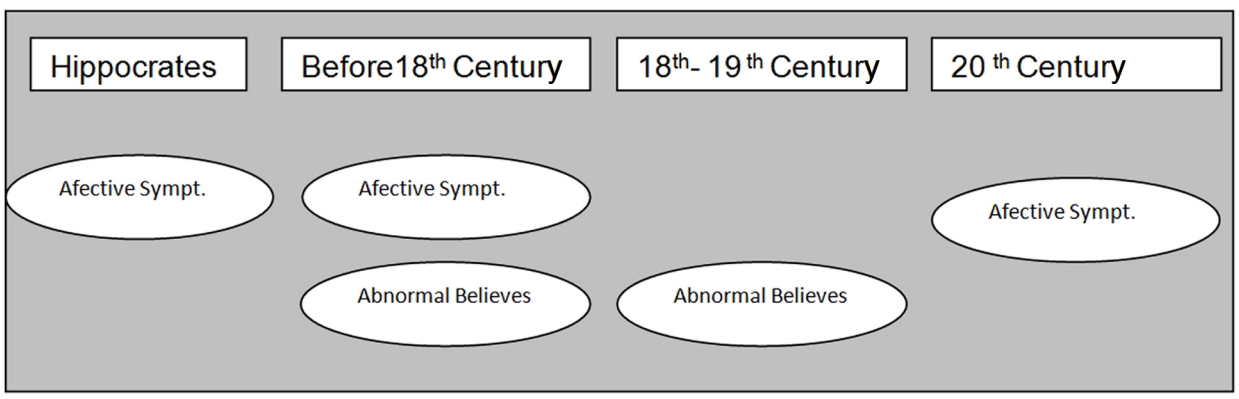

FIGURE 1 | Evolution of the meaning of the term melancholia throughout history.

also recognized fixed and bizarre ideas, restricted to a theme, with the other mental functions being preserved.

Aretaeus of Cappadocia, his contemporary, also upheld the idea that melancholia consisted of an emotional (*animi angor*) and intellectual (*in una cogitatione defixus*) phenomenon.

The idea of a partial delusion gradually became dominant in the characterization of melancholia in the authors who came after Galen until the end of the eighteenth century. This conception reaches its peak with Pinel, who distinguishes "mania" (accompanied by general delusion) from melancholia (accompanied by exclusive delusion; **Figure 1**).

At the turn of the nineteenth to twentieth century, the affective component is recovered as the main change characterizing melancholia. With Esquirol naming the cases of partial insanity "monomania" (the term "melancholia" being abandoned for this meaning), and later, the definition of the cases of manic excitement and cyclic mental depression by Falret, Baillarger, and Kraeplin, the affective component of melancholia would be revalued (**Figure 1**).

We thus conclude that throughout its evolution, "melancholia" has been associated to two fundamental meanings: affective disorders and abnormal beliefs. Some have minimized the importance of the affective component before the twentieth century. However, we agree with Radden, as from Hippocrates to the present day, many authors valued affective symptoms such as sadness, grief and fear in the definition of "melancholia" (Radden, 2000).

It is our opinion that studies reflecting on the evolution of concepts in psychopathology are important. Studying the history of psychopathology is a powerful way of calibration, by which language in Psychiatry can be improved and prepared for more rigorous quantification.

The epistemology of psychopathology "has to include a combination of methods as history, philosophy and empirical investigation" (Berrios, 2011, p. 39). The history of psychiatry and psychopathology brings to us some information about the social processes where concepts have evolved, philosophy clarifies if the language used is sufficiently powerful, and empirical investigation tests the validity of the new concepts toward reality (Berrios, 2011).

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received: 24 September 2014; accepted: 14 January 2015; published online: 03 February 2015.

*Citation: Telles-Correia D and Gama Marques J (2015) Melancholia before the twentieth century: fear and sorrow or partial insanity? Front. Psychol. **6**:81. doi: 10.3389/fpsyg.2015.00081*

This article was submitted to Psychopathology, a section of the journal Frontiers in Psychology.

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