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Differential Diagnosis on the Autism Spectrum: Theorizing an “Ordinary Autism”

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Abstract

Objectives. – Data on the prevalence of Autism Spectrum Disorder (ASD) reveal several clinical evolutions inducing new psychiatric definitions and diagnostic practices. Thus, autism has shifted from being a rare syndrome with severe clinical forms to a new paradigm: the paradigm of “ordinary” or “invisible” autism, in terms of the frequency and the intensity of the disorders. These changes incorporate new populations into our conception of autism, with new phenotypes that pose theoretical and clinical challenges to clinicians. In response, we propose the hypothesis—based on psychoanalytic theories of psychic structures—of an “ordinary autism” as a definition of a non-prototypical autistic psychic functioning that falls outside the DSM diagnostic framework. This idea seems to provide us new theoretical references that nourish our practices as well as fundamental research.

Method. – First, we will review the nosographic mutations of the DSM-5 and their implications for non-prototypical psychic modes of functioning of autistic people that may not be contained within the autism spectrum's blurry boundaries—especially for the adult population without intellectual delay and in the case of complicated differential diagnosis for clinical and societal reasons. Next, we will discuss the definition of “ordinary” or “invisible” autism in a psychoanalytic structural model, as a possible epistemological orientation for identifying and designing practice with the clinical heterogeneity of autism outside the boundaries of psychiatric ASD.

Results. – The autistic population targeted by the DSM-5 criteria is different from that previously defined by DSM-IV. This leads to two consequences: on the one hand, autistic modes of functioning are not limited to individuals who have been diagnosed with Autism Spectrum Disorders as defined by the DSM-5; thus individuals with autism do not have access to the diagnosis of ASD or are given other diagnoses. The alternative diagnoses proposed by the DSM-5 that attempt to correct this diagnostic exclusion—such as Social (Pragmatic) Communication Disorder—are unsatisfactory. Therefore, there is an entire segment of the autistic population that

has subclinical, non-prototypic autistic manifestations or more subtle phenomena discernible in the broader autistic phenotype or sub-threshold autism spectrum that does not have access to the ASD diagnosis and raises differential diagnostic issues. On the other hand, it appears that the autism spectrum brings together extremely different entities and false positives such as schizophrenia and schizophrenic spectrum personality disorders under one diagnostic rubric. Then, the differential problem appears central: both at the theoretical level and in diagnostic practices. The recognition of these limits should encourage us to promote research and clinical applications on this subject. One solution that we envisage is to be found in an extension of Maleval's structural psychoanalytical model: we propose the notion of "ordinary autism"—an echo of ordinary psychosis—to define attenuated or compensated non-prototypical autistic phenotypes, increasingly frequent and with fewer "extraordinary" phenomenological expressions than the classic cases of autism which now call into question the relationship between the normal and the pathological.

Discussion. – "Ordinary autism" seems to offer clinicians the opportunity to formalize the new contemporary and extensive clinical reality of autism. This term situates itself within a theoretical model whose current and future developments might help us respond to clinical and diagnostic issues, but also to therapeutic and societal ones. We propose to continue on the path of the operationalization of these theoretical models in order to identify autistic structural constants that could be found throughout the "ordinary" clinic of autism and could serve as differentiating tools for diagnosis as well as a support in developing and refining therapeutic practices.

Conclusion. – We conclude that there is an urgent need to conceive of "ordinary autism" to provide us with reference points to respond to new clinical issues, but also to reintroduce respect for the autistic person in his or her subjectivity to the center of our therapeutic practices.

Key-words: Autistic spectrum disorder; Differential diagnosis; Social communication disorder; Pathological demand avoidance syndrome; Psychopathological structure.

1 Introduction

Anyone following the French and American media could easily come to the conclusion that we are experiencing an “epidemic” or a “sanitary catastrophe” regarding autism, which has been declared an “urgent” public health problem [1,2], following the publication of new epidemiological data from the Centers for Disease Control and Prevention [3] suggesting that one in 59 children, or one in 40 [4], is autistic. These statistics were generated by studies of eight-year-olds carried out in 2000, 2008, and 2012 that found that one in 150 children, then one in 88, then one in 68 scored positive on an autism spectrum disorders (ASD) screening test – compared to one in 2,778 eight-to-12-year-olds in a study from 1980. Naturally, these numbers should be handled with care, and should be used for informational purposes only given their limits – primarily methodological – and the variations between studies performed in different countries [5].

Without wasting our time on alarmist and farfetched hypotheses such as the exposition to vaccines or the consumption of organic products, such studies have demonstrated that policies aimed at raising awareness among the general public [6, 7], as well as screening and better training in detection and diagnosis for professionals [4, 8] lead to increases in the number of diagnoses of autism, particularly in cases of “sliding diagnoses” with better differential evaluations in favor of autism [6, 9, 10]. Certain environmental and cultural factors are emphasized or excluded in an attempt to explain these numbers [6], but without completely silencing the debate on the hypothesis of a veritable increase in these disorders [7].

However, the factor that is the most unanimously put forward to explain the exponential evolution of patients diagnosed with an ASD is that of the progressive expansion of the diagnostic

criteria [8, 10, 11] in nosographical works such as the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, published by the American Psychiatric Association [12]) and the ICD-11 (International Classification of Diseases, published by the World Health Organization [13]). These updated criteria offer a new canvas on which to conceive of an expanded clinical world of autism, based on a dimensional approach that is particularly represented by the inclusion of the large category of autism spectrum disorders in an even vaster category of neurodevelopmental disorders – the reasoning behind this inclusion remaining, for the moment, obscure.

The first data suggest that, on one end of the spectrum (that of those disorders whose degree of severity is the most intense), diagnostic practices have become more refined with the abovementioned nosographical evolution, and that there might have been substitutions or accretions of other diagnoses, such as learning disabilities or language disorders [9, 14] – and perhaps retrospectively, for example, in adults who received a later, corrected diagnosis of ASD.

However, it's on the other end of the spectrum that the new inclusions are the most numerous: the growing prevalence of ASD has certainly been influenced by a progressive evolution of the diagnostic threshold, in particular at the moment of the transition from the DSM-III and DSM-IV-TR to the recent DSM-5, which coincided with the spectrum-based conception of autism. This allowed for the diagnostic recognition of people who present less severe autistic phenotypes [15-17], as well as of women, teenagers, and adults – populations who remain under-identified [18].

It seems that the statistical data and the prevalence of the ASD diagnosis reflect a complex reality that bears witness to the theoretical and social developments in the way that we view autism – a disorder that has acquired a different status, which commits us to a paradigm shift: from one of scarcity and rareness with isolated and clearly identified clinical categories, to one that underscores the ordinariness of autism, both in terms of its frequency and of the intensity of the different disorders. Indeed, autism is now seen as greater than the rare *princeps* cases cited by Kanner, who

refused to expand his diagnosis to “the entire population, while [his] observations only concerned eleven cases.” Autism is no longer the province of Asperger’s “little professors” with their exceptional skills, nor of “autistic savants” whose voices were the most audible at the end of the 20th century. Thus autism – having become ASD – is becoming an ordinary diagnosis, no longer an obscure and marginalized syndrome whose most severe, fascinating, and exceptional cases were objects of fantasmatical investment and provided the basis for the first theoretical and nosographical texts [17].

These evolutions mean that clinicians are now faced with new theoretical and clinical challenges in their encounters with a less “extraordinary” autistic population made up of multiple, less visible autisms – or even invisible autisms, if we extend the analogy with the invisible, infra-rouge, or ultra-violet parts of the electromagnetic spectrum – that are no longer identifiable by stereotyped behavior or hypermnesic capacities, but by a collection of more subtle traits that the classical psychiatric models [17] struggle to quantify and to qualify.

Thus, just as the notion of “ordinary psychosis” [19] represented a liberation from a certain nosographical and theoretical psychoanalytic tradition, a heuristic that enriched both clinical practice and research, and that encapsulated something of the modernity and the evolution of psychopathology at the moment of this syntagm’s creation, this essay is the occasion for us to test out the analogous possibility of formalizing the hypothesis of an “ordinary” or “invisible” autism as the balanced or resting state of the fourth, “autistic” psychic structure, which Maleval began theorizing in 1998 [20].

It is necessary for clinicians to equip themselves with new tools in order to apprehend this “new” autism, whose contemporariness and whose frequency should pique our curiosity; in order to support this new comprehension, we will first of all present the nosographical mutations in the DSM-5, associated with the spectral conception of autism and the various problems that arise from

it, both regarding the evaluation of non-prototypical autistic modes of functioning and regarding cases of differential diagnoses. We will then turn our attention to the idea of an “ordinary” or “invisible” autism, also known as an adapted autistic structure, proposed in an innovative Lacanian psychoanalytic structural perspective. This will serve as our theoretical compass, following the example of “ordinary psychosis,” allowing us to think about autism, its evolutions, and the differential problematics that arise at the borders of the autism spectrum when other nosographical entities blur the clinical picture. We conclude by underscoring the urgent theoretico-clinical need to formalize and to orient ourselves within the vast, heterogenous dimensionality of autism, which, for us, calls for a necessary reintroduction of the notion of subjectivity in clinical work with autism, as a corollary to our diagnostic and therapeutic practices.

2. Autism’s Diagnostic Evolutions in the DSM-5

The DSM-5 has developed in the direction of a dimensional nosography (with a single psychopathological axis) while attempting to abandon the categorial model that was present in the first four editions of the manual [21], in support of the scientific data [22] on the classifications of schizophrenia [23] and, especially, of autism [24]. The DSM-5 favors a (quasi)dimensional approach: each symptom can be coded on a scale of severity, on a continuum of intensity that should allow clinicians to refine their diagnoses in the interface of therapeutic and educational recommendations. This new nosographical formalization forces clinicians to move beyond the usual, ordered categories based on the grand syndromes, with their unequivocal signs and symptoms, and has greater descriptive power, while allowing for the recognition of heterogenous forms that gravitate around the old categories – for example, partial syndromes (or sub-syndromes) –, thereby adding nuances to their potential interventions.

The DSM-5 thus codified significant changes in its classificatory system of autism, with the emergence of autism spectrum disorders (ASD), for which the diagnostic criteria are simplified: a move away from the autistic triad to a nomothetic conception of the “social/communication” criterion, combined with a single criterion on “restrictive and repetitive behaviors, activities, or interests,” in order to incorporate different categories or forms of autism that were previously recognized as distinct or exclusive into a single continuum (even if this continuum is tempered by specifications and indications on severity). This modification raised the hackles of associations of people with Asperger’s syndrome and of writers who have argued in favor of this syndrome’s specificity [25, 26]. The new classification is, however, meant to reflect recent advances in research [27, 28], in particular with the updates reflecting common genetic and neuropsychological factors that the much-decried DSM-IV didn’t take into account [28, 29].

Thus, the adoption of an autistic “spectrum” allows us to insist upon the shared cardinal points of the different autistic groups or clusters, which should facilitate clinical practice by allowing clinicians to establish more precise, trustworthy, and valuable diagnoses, while respecting real phenotypical heterogeneity, and thereby supporting the effort to identify biological and neuropsychological determinants that could solidify and refine the ASD construction [30]. This conceptual ideal seems more and more out of reach, in light of the complexity that is revealed by experimental research [17]. For the time being, the scientific postulates that underpin the changes to the DSM are reduced to hypotheses based on a relative scientificity that rests on a matrix of meta-analyses and statistical data presented as absolute, but which reflect only a part of published research [31, 32]. This leads to an avoidance of some uncertainty and a tendency to over-estimate the theoretical results and stake out firm positions. In this way, the DSM-5 promises a common language built on a hegemonic – but fallacious – scientific position, and promotes a psychiatric *epistēmē* liberated from early psychopathological and psychodynamic models, while moving towards the articulation of a semiological interpretive framework. Autism, in the DSM’s conception, is crystallized around notions of inherited neurological disorders that are therefore

distinguishable and treatable by the heteronomic use of *ad hoc* tools (diagnostic criteria, standardized scales, symptoms, duration, intensity). These paradigm shifts, whose multiple determinants fit together in a complex way, infiltrate general representations of autism and provoke profound transformations that lead to deep-reaching modifications for psychiatry and for clinical work, thereby forcing us to confront new diagnostic and clinical problematics.

3. Diagnostic Problems Raised by Nosographical Evolutions

Because of adjustments made to the DSM-5, autism is now diagnosed with more sensitivity (that is, a heightened probability that the diagnostic procedure correctly identifies an autistic person) and with more specificity (that is, the heightened probability that the diagnostic procedure correctly excludes a non-autistic person). A note in the DSM-5 specifies that “individuals with a well-established DSM-IV diagnosis of autistic disorder, Asperger’s disorder, or pervasive developmental disorder not otherwise specified should be given the diagnosis of autism spectrum disorder” [12]. However, several authors have commented on the repercussions of these modifications, especially for people who don’t meet, or no longer meet, the criteria of ASD and may therefore find themselves in other categories – or for people who meet the criteria for ASD but who present an atypical evolution, for example, towards the schizophrenic spectrum. In the following, we will summarize some of these problems.

3.1 Autism Without ASD

The DSM-5 features a reworking of its diagnostic criteria provoked by two major changes: the condensation of the criteria “deficits in nonverbal communicative behaviors used for social interaction” (with the suppression of the notion of a language delay) and the addition of sensorial anomalies to the B criterion (which were not mentioned in the DSM-IV). McPartland and his team [33] predicted that the first modification would exclude a significant number of individuals who had been diagnosed with autism using the DSM-IV-TR criteria, while the addition of the notions of

hypo- or hyper-sensitivity to sensorial stimuli would inevitably include other individuals [34]. In reality, even if some authors [35] feared a significant diagnostic inflation following the lowering of the threshold for meeting the criteria of ASD (the false-positive problem), it seems, on the contrary, that it is more difficult to attain the diagnostic threshold for ASD using the DSM-5's criteria, of which there are only 11 possible combinations, compared with the 2,027 possible combinations that were included in the DSM-IV [33].

These updates have led to a new diagnostic filtering. Thus, the autistic population targeted by the DSM-5 criteria will be different from the population that was defined using the criteria of the DSM-IV and IV-R. This population may indeed become larger, which would have important implications in terms of public health: the exclusion of a part of the population that no longer meets the diagnostic criteria and the inclusion of new subjects who can request treatment, thereby causing medico-social and financial conflicts of interest. Some authors [36, 37] claim that these changes have not led to significant nosographical displacements and that the individuals who were diagnosed by the DSM-IV were rediagnosed as presenting an ASD (or, in some cases, a pragmatic social communication disorder), using the DSM-5. Frazier, who carried out studies validating the DSM-5 criteria, concedes that some autistic subjects were left out of the new ASD entity: "We are not missing a ton of them, but we are missing some, and it's a reasonable amount. It raises the question of whether or not we could do better" [38]. These fears, shared by McPartland and his colleagues [33], have been confirmed by four consequential meta-analyses [39-43], which conclude that there has been a significant decrease in the number of ASD diagnoses in both children and adults, because, in general, people who received a diagnosis of Pervasive Developmental Disorder using the DSM-IV or DSM-IV-TR were less likely to receive a DSM-5 diagnosis of ASD, with a reduction of around 40% of the population. One possible explanation is that the DSM-5 criteria increase the nosographical model's specificity, when compared to that of the DSM-IV-TR, hence the exclusion of 94.9% of individuals who should not have received an ASD diagnosis, while the previous manual's weaker sensitivity [33, 44, 45] creates other exclusions that are less unjustified.

As a general rule, several studies have demonstrated that the individuals who met the DSM-5 criteria had more serious incapacities than those who met the DSM-IV criteria but who did not meet the DSM-5 criteria for ASD [33, 46]. In taking a closer look, we can see that the largest reductions concern populations of high-functioning autistic individuals who have an I.Q. of 70 or more and/or early diagnoses of Asperger's syndrome; moreover, we can observe high percentages [39-43, 47] of individuals who are not seen as compatible with the ASD diagnosis and who had previously received a diagnosis of PDD-NOS (pervasive developmental disorder-not otherwise specified, or atypical autism), diagnoses that used to be given to people who didn't completely meet the criteria for autistic disorders, but who demonstrated comparable characteristics and problems. This older category disappeared from the DSM-5 in its dimensional makeover, whereas it had been liberally employed in the past in diagnosing less severe autistic modes of functioning. These diagnostic evictions seem to concern both young children [48-50] and adults [51], with the latter population appearing to be more concerned, especially regarding women and cases without intellectual disabilities [45, 52].

3.1.1 Autism Spectrum and Social (Pragmatic) Communication Disorder

One solution was imagined in the DSM-5, with the creation of Social (Pragmatic) Communication Disorder (SCD) or pragmatic language impairment. SCD is not included among the autism spectrum disorders, with the American Psychiatric Association [12] stating that a SCD diagnosis can only be given if a diagnosis of ASD is excluded. However, SCD is useful in that it gives a "diagnostic cover" [39, 40, 51, 53] for people who present symptoms in the domain of social communication, but who have never demonstrated repetitive or restricted behaviors or interests.

It is possible that some individuals who had previously received a diagnosis of Asperger's syndrome or of a PDD-NOS and who, today, don't fit the criteria for ASD could be diagnosed with SCD. Robison calls SCD an "autism lite" [54], that is, that a person presents certain characteristics associated with autism, but not enough to be diagnosed using the new definition [55]. Other authors

[55, 56] agree with the APA, arguing that SCD could be an appropriate alternative diagnosis for people who no longer fully fulfill the new criteria in the DSM-5 for ASD.

However, if early conclusions suggested that the drop in the number of ASD diagnoses was balanced by more people being given SCD diagnoses [37, 57], other research draws attention to the significant lacunae in this category, which only includes a minority (less than a third) of individuals who met the DSM-IV-TR's diagnostic criteria for PDD-NOS but who don't meet the DSM-5 criteria for ASD [39, 40, 51]. Social communication disorders are not a pure substitute for PDD-NOS, as some researchers hoped; an article by Mandy, *et al.* [55] showed that of 219 children who met the DSM-IV criteria for PDD-NOS, 156 of them were diagnosed with ASD and 26 with SCD using the DSM-5, while the remaining 37 children didn't receive any of these diagnoses. Flax *et al.* [58] draw a similar conclusion. If the SCD entity fails to include individuals who have been excluded from ASD by the DSM-5, this could have multiple consequences in the future. SCD also raises the question of the heterogeneity of the profiles that it attempts to group together; new discoveries show that the diagnosis creates a limbo category between autism and non-autism [59], while a retrospective study [60] of the development of patients shows that the SCD label brings together ASD individuals and individuals with specific language disorders. SCD tends to create ambiguity, whereas it was supposed to minimize it, begging the question of the interest of integrating a hybrid and distinct category of autism spectrum disorders into a model that is attempting to break down categories in order to promote a dimensional approach. The diagnostic validity of SCD [55, 60] is also called into question, in part because the similarities and differences between SCD and ASD is not yet clear – and also because the question of whether or not social communication disorders belong on the autistic spectrum has not yet been resolved. (Officially, the APA has not defined SCD as disorders that belong on the autistic spectrum, but Mandy remains open on the question of whether SCDs are distinct from autism [61]). The characteristics of what treatment for SCD should look like, as well as the evaluation of the services offered by the few

institutions who care for SCD patients are open questions that should be rigorously examined [35, 39, 40, 42, 62], but the scientific literature on the topic remains limited [59].

Today, clinicians only rarely use the SCD diagnosis, and when they do, those who are thus diagnosed often have the impression of knowing nothing about their disorder, due to the lack of information on the topic [59]. Moreover, the lack of a link between the DSM-5's SCD and its direct equivalent in the ICD ("Developmental language disorder with impairment of mainly pragmatic language") is another problem [63]. The relationship between these two entities is murky at best and, at worst, not firmly established, which constitutes a real challenge for researchers and clinicians.

3.1.2 Autism Spectrum and Pathological Demand Avoidance

On the borders of the autism spectrum, one finds yet another diagnostic entity whose soundness and whose links with autism remain unclear: pathological demand avoidance, or PDA, individualized in 1980 by Newsom *et al* [64] and brought to light recently in France in an article by Philippe and Contejean [65]. PDA is characterized by an avoidance of demands, a superficial sociability, versatility, and a pronounced interest in imaginary worlds, which alter social integration.

Newsom and her colleagues [64] write about their use of this diagnosis in their encounters with atypical autistic children presenting particular behavioral patterns, whose relationships with autism were questioned by other professionals or by the children's parents (for example: their language development was atypical for autism and less disorganized, pragmatically speaking, than in Asperger's syndrome). This syndrome requires us to think about its place and its nosographical meaning; initially, Newsom and her colleagues [64] attempted to have it recognized as a something distinct from PDD (particularly from PDD-NOS) and from Asperger's syndrome. Thus, PDA could be a kind of double whammy: that is, a comorbidity that is not necessarily caused by ASD, but with which it could be associated [66]. Some authors tend to see these behavioral characteristics as a phenotype that belongs on the autism spectrum [67, 68], sometimes fleetingly present in childhood

[69], and infrequently used to qualify complex behaviors in children diagnosed as being on the autism spectrum or just beneath the threshold, but whose test scores for autistic traits are comparable [67]. Philippe and Contejean [65] interrogate the syndrome's overlap with Kanner's description of aloneness.

Green and his collaborators [70] underscore the paucity of writing on the subject and conclude that there is no proof of the construction, nor of the discriminatory or predictive validity of the constellation of symptoms of pathological demand avoidance as a diagnosis or as a distinct syndrome, inside or outside of the autism spectrum. Diagnosing PDA is not an easy task, since neither the ICD nor the DSM use it. Obviously, this interrogates the treatment that would result from this diagnosis. Ignorance surrounding this diagnostic entity can lead to a modification of best practices and to misunderstandings between healthcare professionals and patients' families [70].

3.1.3 Optimal Evolution and the Expanded Autistic Phenotype

At this point, we find ourselves in a dichotomous and paradoxical situation: as we pointed out in our introduction, an improved clinical recognition of the range of autistic phenotypes, including the most periphery forms, and the attempt to circumscribe this understanding in a new, data-based model, has ultimately led only to the diagnostic exclusion of a part of the autistic population. The alternative diagnoses that attempt to rectify this exclusion are largely unsatisfying, as evoked above, and only underscore the difficulty of clinically accounting for the semiological heterogeneity that exists on the new autism spectrum, notably on its borders. These alternative diagnoses only demonstrate the insufficiency of the behavioral markers and of the criteria that, ultimately, pass by people whose autistic functioning is more discreet or atypical [34]. While some observers celebrate what they consider to be an overdue correction of the over-pathologizing of normal eccentricity and social awkwardness [35], it would seem, however, that the research [71] suggests that autistic functioning and the particular difficulties that this induces are not limited to individuals who have been diagnosed with an ASD (or even with an alternative diagnosis), according to the DSM-5.

Who, then, are these undiagnosed subjects, who have not been identified with the ASD label nor with any other diagnostic alternative?

In this gray area outside of the ASD threshold [72], we encounter subjects who present autistic traits in the form of an ensemble of residual traits, the traces of an earlier autistic development that might have been gradually overcome (Fein's "optimal outcome" or OO [73]), or, for other authors, a "post-autism" [74], defined by Meltzer as "the post-autistic personality [...] meaning both outside the autistic states proper and the sequellae of Early Infantile Autism" [75], or "exits from autism" or an "after autism" [75], or – and the term is controversial – "recovery" from autism [77]. Thus, while autism spectrum disorders are relatively stable and lifelong, studies have shown that a subgroup of subjects present significant improvements and no longer meet the diagnostic criteria for ASD [78, 79]. These subjects earn higher scores on standardized tests that explore changes in cognition, in language, or in adaptive behaviors and social competencies [73, 78, 80], but have residual symptoms that can have an impact on the quality of their relationships and on their emotional stability, thus heightening the risk of psychiatric comorbidities [79, 80].

Recent studies shine a light on endophenotypical characteristics that are analogous to those found in autism, both in terms of cognitive style, personality traits, alterations or particularities in subjects' interactions or in their social communication, as well as special interests [81-84]. These characteristics are present in the general population, but with lesser degrees of severity, and are linked to genetic risks [85], which raises questions about societal evolutions that influence our relationships with others [86, 87]. Clinicians and researchers use a variety of terms when evoking these clinical forms: "residual autism" in the DSM-III, "subtler forms," "autism shadow syndrome," "residual Asperger's syndrome," "solitary," "autistic tendencies," or "phantom autism" [88]. Specifically, the current literature suggests the concept of "broader autism phenotype" or BAP, which refers to subclinical characteristics associated with ASD, that is, the expression of behavioral and cognitive similarities to ASD, but at a lower threshold than the one the DSM maintains [89]. Already mentioned in the 1940s by Asperger and Kanner, BAP was originally used to describe traits

of certain members of autistic persons' families. Observations from that time suggested that the expression of autistic traits overflows the clinical borders of autism syndrome to include sub-threshold, light phenotypes that one often finds in the parents of autistic children, which supports the hypothesis of a strong genetic component to the etiology of autism [82]. These traits correspond with the diagnostic criteria of ASD, and are considered to represent a weaker phenotypical expression linked to a genetic vulnerability to autistic traits [90, 91].

Today, BAP can be conceptualized as an ensemble, rather than a distinct category of traits organized clinically. These traits are seen as qualitative or quantitative variations of continuous behavioral traits, which are observed not only in relatives of ASD subjects who are genetically vulnerable or in psychiatric populations, but also in the general public [92, 93]. Current research describes a continuum, stretching from individuals who barely present any autistic traits to individuals who have been diagnosed with severe forms.

Finally, beyond the gray area of the BAP, this pole of the spectrum allows us to understand the psychological aspect of autistic traits, which can – but do not always – hinder a subject's overall functioning and quality of life. These traits also feed into the continuum of normalcy and include the positive aspects of autistic characteristics, such as originality, creativity, and divergent thinking.

3.1.4 Diagnosing Autism in Adults: Between “Camouflage” and Comorbidities

In France, the “Autism France” organization reveals that 90% of autistic adults have not been diagnosed and do not receive adequate care. This accounts for a significant amount of so-called inappropriate hospitalizations and represents a real problem in adult psychiatry [94, 95]. Certain authors evoke a “lost generation” [96, 97] composed of a large proportion of individuals, particularly those with fluid language and a normal I.Q., who were not identified during childhood for environmental reasons, such as a lack of means or of training in detection and diagnosis for healthcare professionals in the area where they grew up, as well as individual reasons, such as the muted nature of the disorder in childhood [97]. These individuals find themselves, having reached

adulthood, without a diagnosis, for which they have a legitimate need [95, 96]. This issue is even more pressing today, as adults are increasingly likely to request assessments (for example, an increase in requests of 50% at the Southern Lorraine Autism Resources Center), which means that more and more professionals are encountering autistic adults with attenuated behavioral manifestations.

Establishing an initial diagnosis of ASD in an adult can be difficult: for practical reasons (for example, the difficulty of collecting elements of a person's development history); because of limited tools [98]; because of incoherence in the diagnostic repertoire [99]; because of the individual's acquisition of compensatory strategies, referred to as "camouflage" [100, 101], which infiltrate their clinical presentation; or because of the lack of specialized institutions and of professionals trained in detecting autism in adults. Healthcare professionals may also find it difficult to obtain information about the patient's early development and may be led astray by a previous psychiatric diagnosis, or by the presentation of acquired skills that are considered to be incompatible with autism [96]. Clinically, these adult subjects frequently present concomitant disorders or comorbidities that make it more difficult to establish a diagnosis. Moreover, symptoms of ASD, even if they are present from childhood, may not clearly express themselves as long as the demands of society do not overwhelm the patients' capacities [97, 102] and may even be attenuated with age, since two to 15% of individuals appear to attain a level of adjustment and of functioning similar to that of the general population by the time they reach adulthood [103].

The precise and timely identification of autism spectrum disorders in the adult population – even those that are not particularly pronounced – constitutes an important clinical challenge [97, 99], even more so with the new nosographical mutations, since the criteria proposed by the DSM-5 might considerably modify the composition of the autism spectrum – to the detriment, indeed, of attenuated forms of autism.

3.1.5 The Problematic Borders of the Autism Spectrum

On a practical level, the question of a threshold remains problematic, since only an ASD diagnosis gives a person access to economic and social benefits. Thus, when the DSM-5 was published, there was great concern that publicly funded special educational services could be taken away from those children who were no longer “admitted” into the new diagnosis; and, in the United States, a grandfather clause was added that allowed people who had been diagnosed with an autism disorder using the DSM-IV criteria (Asperger’s syndrome or PDD-NOS) to continue receiving support. This clause, however, is not legally binding, but autism-rights organizations have been fighting to make sure that schools respect it [35]. The question, however, remains open for those people currently undergoing a diagnostic procedure. The outcome of the evaluation is relative, in that these individuals can land on one side or the other of the diagnostic threshold because of the way in which their autistic functioning manifests itself, and also because of positive or negative influences of their environment, their age, and their gender. Furthermore, the diagnostic tools recommended by health authorities, and based on algorithms that use a binary ASD/non-ASD logic, are not always well-adapted to the particularities of certain groups [98]. The recognition, or refusal, of an ASD diagnosis in this context is difficult, and if a lesser degree of social or communication dysfunction may not always signify an ASD, and if the use of this label may not be necessary or relevant, even when there is a slight deficit, we cannot overlook the fact that many individuals, whose autistic functioning can lead to daily suffering, seek help (for example, [104]). It might be necessary to dissociate the severity of the symptomatological presentation from individual repercussions in terms of psychological pain, especially since the current literature recognizes that camouflage strategies weigh heavily on those who deploy them (for example, [100, 101]). Thus, the acknowledgment of their disorder is often an important step in these individuals’ personal journeys, particularly for those who are diagnosed in adulthood. In many cases, this late diagnosis brings about feelings of relief and a new perspective on personal experiences, for the diagnosed person and for their entourage. This is why the announcement of the diagnosis is the object of keen anticipation, even sacralization: individuals have been waiting for it for a long time. Coman [72] reminds those

professionals who establish a diagnosis of their responsibility. They must take into account and unpack all of these variables in order to produce sound clinical diagnoses that are in the interest of the patient.

The DSM-5 has taken an immense step forward in proposing a quasi-dimensional nosography for autism spectrum disorders. However, if something has been gained in an evidence-based formalization of the large clinical variety of autism, something has also been lost. In effect, this attempt has been circumscribed by questionable diagnostic criteria that establish a cut-off score, and that sometimes fail to recognize subclinical, non-prototypical autistic manifestations and subtler phenomena that can be discerned in the broader autism phenotype or the sub-threshold autism spectrum. These attenuated clinical forms are, however, increasingly the object of scientific investigation, and clinicians are becoming more aware of them, largely because of their preponderance in contemporary clinical practice and because of the numerous comorbidities that are associated with them. Not only are these forms difficult to apprehend using a psychiatric model and psychiatric tools, an additional difficulty is that, in this clinical borderland, other related clinical phenotypes add themselves to the differential diagnostic issue.

3.2 Non-Autistic People on the Autism Spectrum

According to several studies, the modifications to the DSM-5 do not always prevent falsely positive diagnoses, in light of factors related to successive nosographical evolutions, but also, as we will see, in light of the social aspects.

3.2.1 Confusing Nosographical and Clinical Factors

While the new matrix of the DSM-5 for ASD is built around a criteria-based algorithm that excludes the older entities PDD and Asperger's, the authors of the manual proudly announce that the DSM-5 is wonderfully specific and excludes 94.9% of individuals who should not be diagnosed with an

ASD [35]. However, these results are obtained by including other classifications, from previous diagnostic manuals, in PDD. In this, one could indeed expect a greater specificity linked to the new criteria that eliminate an insignificant number of clinical forms that would have required a differential diagnosis in order to be counted among the old nosographical categories. This new specificity, however, has only excluded one part, and perhaps the easiest, of the non-autistic clinical forms that are close to what we, today, conceive of as making up the autism spectrum. In effect, what we mean by “autism” has evolved, and it is certain that formerly distinct clinical forms that have only recently been added to the conceptual framework are found among the 95% of “justifiable” ASD. Therefore, following Verhoeff [105], we should let go of this image of a “fundamental continuity with the original description of autism made by Leo Kanner,” that is, autism as a stable, positive, and essential entity that successive discoveries have simply updated and refined. Verhoeff reminds us not to forget that autism is the product of discontinuities and irregularities, and that it only acquired a coherent history *a posteriori*.

In fact, autism’s borders have shifted after each successive diagnostic regrouping, and early childhood autism, initially isolated, was joined by other clinical forms. The concept of autism has constantly been enlarged, up until the appearance of its spectral form in the DSM-5. The first step in this nosographical work was the creation of a framework that incorporates all the forms of broad disorders, followed by the pervasive developmental disorders in the era of the DSM-III and the DSM-IV-R, condemned at the time, notably because of the breakup of the forms of childhood-onset psychosis [106-108], which were absorbed by PDD and then diluted in ASD. Autism, which up until that point was considered to be a rare syndrome more closely related to different forms of early-onset psychosis, became, with the advent of the notion of the autism spectrum, a central and generic entity, the prototypical form of a serious disturbance of early childhood development [109], and, in the eyes of psychoanalysis at least, a step backward in the comprehension of the diversity and evolutions of childhood disorders [108].

Hochmann [32] thus relates the worries expressed by Allen Frances (the president of the commission that created the DSM-IV), for whom the increased prevalence was essentially due, on the one hand, to the enlargement of the pervasive developmental disorders to include the quasi-totality of encephalopathic intellectual disabilities, and, on the other hand, the non-discriminatory character of the criteria for Asperger's syndrome, and, finally, the enlargement of the poorly defined category of PDD-NOS, which today includes almost all of our psychotic dysharmonies and borderline pathologies, not to mention subjects who present obsessive tendencies or who are socially awkward – symptoms that occur on the border with “normalcy.” To this, we would add, all the forms of autism whose origin is syndromic (the result of a genetic mutation, *i.e.*, the deletion or duplication of one part of a chromosome). In fact, there is a great deal of genetic and etiological diversity (subgroups such as syndromic and non-syndromic autism), influenced by environmental aspects, contained within the ASD label. It would be fair to say that, at times, the clinical denomination refers more to a behavioral syndrome than it does to a specific psychological ailment [110].

Hence the hypothesis that ASD, this powerful “nosographical captor,” doesn't “bring together, under one rubric, the extremely different entities” [108] that, formerly distinguished from what was then considered to be autism, found a place under its umbrella. Everyone can find a place on the autism spectrum, even if their psychic organization or structure isn't necessarily regulated on an autistic mode. We can find an illustration of this in the numerous stories of subjects who were initially diagnosed as ASD and who later presented schizophrenic symptoms or psychiatric forms that are related to the schizophrenia spectrum, and who occasionally suffer psychotic decompensation [108, 111, 112]. In fact, even if the criterion of the moment of the appearance of the disorder usually suffices to give a good diagnostic indication of its developmental evolutions, a psychiatric reading of schizophrenic symptomatology that can appear very early in life can be obscured by ASD's unilateral lens, which groups together all early-onset and pervasive disorders,

and which, therefore, can hamper the potential appreciation of their differential evolutive specificities [113]. This also calls into question studies that tackle the complex problem of the links and the co-occurrences between psychiatric/schizophrenic disorders and autism [114-116]. In fact, these two psychiatric populations share a common history and an ensemble of symptomatic clinical manifestations and of cognitive and phenomenological particularities. Even new data from the fields of neurobiology and genetics contribute to the confusion, by demonstrating the similarities [117, 118]. Moreover, the DSM-5 makes use of a distinction between autism spectrum disorders – with the presence of fundamental social and communicational deficits – and the spectrums of schizophrenia, whose essential characteristics are hallucinations, delusions, and thought disorders, and whose caricatural qualities would seem to extinguish any hope of clearly distinguishing between “impure” and prototypical clinical forms of these spectra.

In this, false positives can arise from the fact that certain characteristics of ASD overlap with those of other disorders, for example, with schizophrenia and personality disorders on the schizophrenic spectrum [115, 119, 120] – this is, by the way, a research question that is progressively gaining scientific attention [112, 114, 121] – but also with social anxiety disorders, obsessive-compulsive disorders, intellectual disabilities that are not part of the autism spectrum, attention deficit hyperactivity disorders unrelated to autism, and neurological and/or somatic disorders [122]. These false positives can appear in childhood, or at any point of the symptoms’ evolution through adolescence and adulthood [123-125]. Vlasaliu’s [126] and Cath’s [124] teams, in order to carry out a differential diagnosis, compare ASD to a list of other nosographical entities, such as behavioral disorders, social phobia, selective mutism and other anxiety or depressive disorders, obsessive-compulsive disorder, or different personality disorders, in order to tease out areas of overlap. This tableau is made more complex by the very frequent comorbidities associated with autism at any age [127, 128], and this even more so for the ASD population as it’s defined according to the new criteria in the DSM-5 [129]. It is rare to find an autism spectrum disorder in its

“pure” clinical form, without any other associated disorders. Undiagnosed concomitant disorders contribute to the fact that many individuals do not receive appropriate services, benefits, or treatment, in light of the similarities between ASD and other disorders [94, 130, 131]. The diagnostic tools habitually used to establish a diagnosis of ASD can show their limitations in these situations where a differential diagnosis is complicated [98], thus contributing to false positives making their way through the porous borders of ASD.

3.2.2 Confusing Social Factors

This clinical situation reaches a point of hyper-complexity when one takes into account the social and economic context surrounding the changes in public policies regarding programs of treatment for autism [2]. There are concrete, non-medical stakes in France, with a sizable budget allotted for the recent “National Strategy for Autism” (2018-2022), but even more so in the United States, where a diagnosis of ASD can mean access to resources, notably financial, for subjects and their families. Therefore, if an incorrect diagnosis of ASD (a false positive) can provoke familial stress or a certain stigmatization, or lead to useless investigations and treatments, or mean a supplementary expense for the healthcare system, some families, justifiably, seek out this diagnosis and some professionals prefer it, since it allows subjects to obtain additional resources and to gain access to services that might remain out of reach in the absence of this diagnosis [17, 115, 132]. This is a point that was brought up recently during an international colloquium in Montreal [133]. Even if there are no statistics on these practices of diagnostic inclusion, evolutions of the prevalence of ASD can be affected by them. Fombonne relates the results of a study in which trained researchers carried out independent evaluations of over 200 children who had received a diagnosis of autism; the researchers identified at least 30% of these children who were not autistic [132]. Some authors argue that an autism diagnosis has become lucrative “merchandise” in light of the real financial interests that are involved [31, 134-136], which can turn a child into a product of a healthcare system that profits from the suffering of children and their parents. This also generates inequalities,

since funneling money to autism could constitute a form of discrimination against other children who do not present autism but who may suffer from problems in their development, learning, emotivity, and behavior that handicap them just as much. In this sense, Matson [137] points out that correctly identifying false positives does not necessarily make life easier for individuals who were thus incorrectly diagnosed.

However, this leads us to further question the extension of the diagnosis of autism to any individual whose strange or disruptive personality doesn't easily fit into another other diagnostic category; this tendency only underscores the urgency of considering the issue of over-inclusion, with the related risk of a trivialization of ASD. Kanner himself feared that the notion he formalized would become little more than a hodgepodge, devoid of meaning [135]. Tanguay [86] writes that the question of knowing where to place the limit between "social strangeness" and light autism is controversial, and one that will require lengthy discussions between expert clinicians, parents, and autistic people.

Also at stake is the question of social representations of ASD, particularly of Asperger's syndrome, which has become particularly attractive in the popular imagination, thanks to different positive representations in media and in the cultural landscape [138], which leads more and more people to self-diagnose as Asperger's [88] and to form online support communities. Alongside the testimonies and autobiographies of famous autistic people, one finds numerous testimonies online, as well as the publication of various detection tools in order to support the recognition of different social subgroups, fostering mutual identification and reinforcing the "Barnum effect" [139]. Mottron [140] invites caution regarding self-diagnoses and predicts an increasing number of adults who see themselves in the description of Asperger's syndrome, at least the version of the syndrome that is portrayed on social media – and the possibility at these people present a "simulated" disorder that expresses, in certain cases, a form of "histrionic avidity," the expression of people's identification with positive media representations. This recognition can be officially actualized by a medical

diagnosis, or a “scientifically validated diagnostic baptism” [141]. Online, one can find lists of psychiatrists who are more likely to validate an ASD diagnosis and who are, therefore, seen by the Asperger’s communities as being particularly skilled connoisseurs of autism, since they satisfy the communities’ requests.

One might wonder if the combined factors of an expanded definition of ASD; the accompanying scientific, social, and political context; and favorable diagnostic and clinical practices might lead to ASD functioning as an “ecological niche” or a “cultural biotope,” to use the philosopher Ian Hacking’s terms [142]. Hacking has elucidated five vectors of an “ecological niche” that, although formulated in relationship with “transitory mental illness,” allow us to question the place occupied by contemporary “autism” in evolution. For indeed, autism spectrum disorders validate the five vectors that contribute to the attractiveness and the propagation of a given clinical entity, instead of others, namely:

- 1) ASD can be identified using the precise taxonomy of a rigorous and technicist diagnostic army, and can be targeted by best practice recommendations; autism spectrum disorders also fit into a larger framework of neurodevelopmental disorders.
- 2) ASD has large-scale name recognition and an increased social recognition, thanks to different projects and campaigns carried out on a national level, beginning, in France, with the first “A New Impetus for Autism” plan in 2005, and up through autism being dubbed, in 2012, a “grand national cause.” In addition, we can mention the multiplication of scientific articles on the subject and its popularization in the mainstream press, for proof of which we need look no further than the exponential growth of the number of hits generated for the word “autism” in online search engines [107].
- 3) ASD condenses a “double cultural polarity” [142]: on the one hand, a fascination with autistic geniuses and, on the other hand, the rejection of autism, largely perceived of as a defect (the adjective “autistic” is often used as an insult [143]).

4) ASD reflects the irresponsibility of the individual, with the overvaluation of genetic causality or of other exterior “objective” agents; this point of view is strongly defended by the current actors.

5) The growth of associations and advocacy groups that have formed around ASD has led to an increased understanding of diversity; this is particularly noticeable with the use of preferred terms such as “(neuro-)atypical” or “neurodiversity,” or with the recognition of new biosocial identities that interrogate the borders between the normal and the pathological [144-146]. This is perhaps not unrelated to a certain malaise inherent to our “society of normalization” [147] that leads to so much subjective alienation. Also of note is the strong feeling of belonging to a community that is expressed by people who are diagnosed with an autism spectrum disorder [146], which contributes to this “feedback loop” [142].

This diagnostic category, with its particularly gratifying [145] “aura” [120], is more appealing for psychiatric and non-psychiatric communities, who would gladly exchange a stigmatizing diagnostic “label” for that of Asperger’s [139]. This “autistic” identity acts as a kind of “social creation,” or a “garment” [136, 138] made from the media’s popularization of Kanner’s and Asperger’s clinical descriptions, and put on by individuals who, in so doing, find a way out of their solitude and are able to draw attention to their existential struggles in a different register than those of illness or madness.

4. A Solution? An Adapted Autistic Structure

The differential issue appears to be central to a theoretical discussion of autism spectrum disorders, as well as to the diagnostic practices; and acknowledging our limits in terms of nosographical and diagnostic tools should encourage further research on the subject. The borders of the autism spectrum remain blurred, especially when they touch on the most severe forms: beyond “Kannerian” autism resides an unstable pole, where the differential diagnosis with childhood-onset schizophrenia can be difficult. And, at the other end of the spectrum, beyond Asperger’s syndrome

lies an invisible pole made up of autistic individuals who have achieved independence, and who are either diagnosed late in life, or never [74].

Between “too large” and “too tight,” there is, for the moment, no nosographical “good fit.” This leads to a situation of simultaneous diagnostic abuse and an under-recognition of autistic subjects, which, given the current state of our knowledge, means that we must continue to look for ways to orient ourselves in the expanded world of autism. It seems that, even if studies on the biology of ASD are more common, the results are still fragmentary, and the discovery of an unequivocal causality remains out of reach, as do unique biomedical diagnostic procedures inherited from a model that would allow for an exhaustive elucidation of this issue [76], hence the necessity remain open to a fecund and self-enriching dialogue between theoretico-clinical approaches and treatment. Maleval [148] thus suggests a way to work through this diagnostic problem: more clinical practice. Following this idea, we can cite the dimensional model of the Adult Autism Subthreshold Spectrum (AdAs), proposed by Dell’Osso and her colleagues [120, 149-152] who, in collaborative research carried out between Italy and the United States, propose a psychopathological and neurobiological model, building on the dimensional approach that was introduced with the DSM-5, and integrating more subtle phenomena such as those described in the broad autistic phenotype or the subthreshold autism spectrum. These authors are particularly attentive to the syndromic model of ASD; while they pay attention to the classic diagnostic criteria, they also take subthreshold signs and symptoms, as well as light/atypical endophenotypical manifestations into account. They pay attention to behaviors and personality traits that are associated with the diagnostic category of ASD in the adult population. They aim to provide a more precise representation of clinical syndromes, including the recognition of “prodromal,” subthreshold symptoms that can lead to an early diagnosis, and to provide preventive strategies for autistic adults who do not quantitatively or qualitatively present the clinical criteria for ASD. It is becoming widely understood that these adults will only be diagnosed later in life – if ever; and they remain especially vulnerable, because of their autistic particularities, to stress, leading to the frequent

development of psychiatric comorbidities ([128], for example). This model can be improved upon and refined, especially for clinical work with children, but it provides a structure for useful theoretical tools that can orient clinical work. There is more promising phenomenological research on, for example, childhood autism in a child psychiatry context ([153], for example); a phenomenological approach can also be used for differential ends with adults ([154], for example).

In order to advance our understanding of what autism is, and to refine treatment practices and our diagnostic approaches, we turn to Maleval. He suggests renewing with clinical practice in an attempt to elucidate a functional logic that exists above and beyond observable behaviors, following a psychodynamic logic that is open to, and capable of articulating with, multidisciplinary discoveries [148, 151-157]. We will attempt to put the theoretico-clinical concept of “ordinary” or “invisible” autism to the test. Maleval’s psychoanalytic structural model can provide a way of thinking about autism beyond psychiatric nosography, thereby modifying our understanding of autistic functioning in its contemporary evolutions.

4.1 A Psychoanalytic Structural Perspective on Autism

We locate the bases for thinking about autism within this clinical framework in the Lacanian-oriented structural model sketched out by the Leforts in the late 1990s [158], taken up by Laurent [159], and redeployed by Maleval, starting in 1998 [[20]).

Maleval [74, 155-157, 160] constructs a resolutely structural model using Lacan’s teaching as well as a critical review of clinical research on autism; his model also takes into account recent writings by autistic people [161]. His principal aim is to grasp an essential quality of autism that exists despite its clinical heterogeneity – in other words, to grasp the structural psychic specificities that are found in Kanner’s “version” of autism, in Asperger’s, and all along the autism spectrum. From this wealth of observations, he attempts to apprehend a general logic that is foundational for the subjective logics of autistic subjects [162]. His project is based on the clear affirmation that

“autism is no longer a psychosis” [160] – which constitutes something of a theoretical break within the psychoanalytic discipline on the question of psychosis, which Maleval [157, 160] encourages us to bypass: “the structural permanence of autism in psychoanalysis demands that we differentiate it from the field of the psychoses and even from the pre-psychoses.” Indeed, it would be possible to imagine certain overlaps with psychosis [107], in that autism could be considered a radical modality of psychotic foreclosure (a particular foreclosure, prior to the foreclosure of the signifier that is the Name-of-the-Father: a radical foreclosure of the progenitor for the Leforts; an elision for Laznik; or the foreclosure of a stupefying signifier for Didier-Weill). However, a study of autism that considers more than just the question of foreclosure reveals a psychic economy that differs significantly from psychotic modes of functioning, with the keystone idea that “autism is an original subjective mode of functioning, characterized by the return of *jouissance* on the edges” [159] (whereas, in schizophrenia, *jouissance* returns inside the body). Maleval [156] adds that “autism evolves towards autism” and not towards psychosis – as can be the case with schizophrenia, paranoia, and melancholy.

From here, autism could be considered the fourth subjective structure, differing from the psychotic structure, and characterized by a specific subjective functioning rooted in the differences provoked by a difficult first encounter with *jouissance*. This structure is based on two principal characteristics: a gripping-onto of objects of the drives, which is especially prevalent with the object that is the voice, which is at the origin of problems of enunciation; and a return of *jouissance* on the edges, on the border erected by the autistic subject using the object (physical objects, persons, animals, or hypertrophied thoughts) – between her/his secure and unchanging world and the world of others, experienced as incoherent and anxiety-producing [162].

This notion of an autistic structure is a prism that provides us with multiple possible readings of the formal heterogeneity of autism, which is organized differently, depending on a subject’s genetic predisposition and on the environment into which the subject is born and that orients her/his

development; in addition, there is always a particular management of the voice and of the regulation of borders, in order to control the subject's relationship to the world. Hence the heterogeneity in autistic ways of functioning: each autistic person has her/his own subjective organization, articulated with the world in terms of the possibilities, creations, and impossibilities [26, 163] that are expressed in behaviors that are more or less noticeable.

This model would appear to be a kind of watermark through which to "read" the psychiatric clinical picture of an autism spectrum, as outlined in the DSM. Autism spectrum disorders in the DSM-5 are laid out on a continuum based on how severely individuals are handicapped socially, and based on the degree of presence of restrictive, repetitive behaviors; these disorders constitute heterogeneous phenotypes that, one could argue, are linked to subjective psychic organizations that are regulated, in a variety of ways, on the autistic structure and that engage different utilizations of the autistic "edge." Here, Maleval [74, 163] points out that if a clinical point of view on autism integrates ASD, it is no longer limited to research on early childhood-onset autism; it embraces forms of autism without compensation and those that are close to the cases Kanner described, as well as forms in which the autistic subject's double allows her/him to compensate – not without difficulty – for a lack of primordial identification and difficulties in enunciation. In fact, we can imagine different configurations of the autistic "edge" (a border that isolates, a dynamic edge...) whose evolution and whose complexification is of great importance for autistic people's development. These different autistic ways of configuring the edge are also a resource that allows us to conceptualize a kind of core of the autism spectrum. These processes sketch out a continuum that extends from Kanner's autism to Asperger's syndrome, while reaching above and beyond, towards an invisible pole [26, 163].

Maleval thus suggests a way of working with the avatars of the autistic structure, notably by going beyond Asperger's syndrome, outside of the boundaries of psychiatry's autism spectrum, and by making use of the analysis of different subjective workarounds that autistic subjects put in place in

order to condition their ways of being in the world, regulating their relationship to the Other thanks to a particular way of dealing with *jouissance* by making use of edge-objects.

4.2 “Ordinary” or “Invisible” Autism

Our idea of an “ordinary” or “invisible” autism fits into this structural model, and allows us to consider all the various autistic ways of functioning that make up an “invisible pole” or “the limits of the autism spectrum,” [74] “Asperger’s and beyond,” [88] or even a “subthreshold autism spectrum” [120, 149-152] that escapes psychiatry’s nosological criteria. Maleval and Grollier [163] give the name of “post-Asperger’s” to these subjective ways of functioning that develop on the basis of an autistic structure that progressively adapted itself to structural constraints by a complexification of an edge, followed by its erasure. Maleval and Grollier describe situations in which autistic subjects can gradually undo their attachment to the edge, a mediator with the outside world that played the role of an initiator, a “pseudopod,” but which the subject no longer requires in her/his dealings with the outside world. In this sense, Maleval [157, 160] suggests that “autism fades away when an element of the edge [...] that was initially used to protect the subject from the other and for self-gratification becomes a true social skill, made up of signs that the subject appropriates.” With Maleval, it is possible to understand autism without pathologizing it, nor considering it to be a deficiency – thus following psychoanalysis’s understanding of other forms of psychic functioning. An autistic structure is not incompatible with high-level social and intellectual success for those people whose ways of functioning allow them to perform inventive work, and the edge – an idiosyncratic and original tool – allows subject to keep their anxiety in check. This subjective inventiveness around the edge is what will allow the autistic person to adapt to social life, in as much as their subjective functioning can find solutions that are coherent with their autistic structure, which might mean that few prototypical autistic traits are visible, or only unspecified symptoms. This sketches out the contours of a transnosographical, balanced autism, that is not

completely included in the ASD entity, whose manifestations can be confused with other differential diagnoses, or might remain undetected by the psychiatric nosographical framework entirely.

Here we can return to the idea that an autistic person cannot escape her/his autism, that it is her/his way of being in the world – instead, the subject can make subjective arrangements with this specific mode of functioning. We also find references that come close to what we’re attempting to pinpoint in these autistic forms in the terms “exits from autism,” “post-autistic personalities,” “optimal outcomes,” [73] or when Kato [87] evokes the possibility of “resilience” in people presenting Asperger’s syndrome through use of adaptive mechanisms that allow them to find a form of relative balance leading to a “successful Asperger’s syndrome.” This brings up the question of what triggers the onset of autism, since some subjects are diagnosed with ASD, then lose this diagnosis over the course of their development, perhaps after having achieved a structural equilibrium that some others might never find. This is a question that is not often treated in the literature [107], with Maleval [155] stating that “autism is not triggered” – unlike psychosis, for example.

At this point, we can now attempt to discuss the definition of an “ordinary” or “invisible” autism that is, however, the site of fundamental terminological stakes. We will consider the use of two different adjectives to describe this clinical reality, without finding either one fully satisfactory.

“Invisible” autism is a concept that seems to have been already used by Mottron and his colleagues [164] to designate autistic phenotypes, particularly among women, whose symptomatology is not represented by tests modeled on the DSM’s criteria and thresholds. Baker [88], like Maleval and Grollier [163], evokes these autistic modes of functioning at the “invisible” end of the autism spectrum, for people whose cognitive difficulties and emotional problems might be dissimulated and are only expressed through superficial signs – that is, largely invisible in a social setting, and hidden behind camouflage. In these cases, the behavioral manifestations that the psychiatric diagnostic framework seeks out are not explicit; there is no manifest autistic disorder.

Structural traces exist, subtly; if not “invisible,” they are certainly “less visible” or “barely visible.” These traces, behind a certain adaptive equilibrium, allow glimpses of several markers of autism that express themselves in something discreetly atypical in the subject’s interactions with the other or in her/his intellectual development [76], or in the continued presence of the positive aspects of autistic traits such as originality, creativity, and divergent thinking – and this at a marked distance, both qualitatively and quantitatively, from the typical deficient symptomatology.

The proposition of an “ordinary” autism is based on a comparison with ordinary psychosis, conceptualized by Miller [19] in an attempt to name a contemporary clinical reality: the arrival, in mental healthcare facilities, of numerous psychotic patients who are less visible, “more modest,” [19] or common, and who do not present major deliriums or hallucinations, but instead an ensemble of subtler traits that provide a contrast with “extraordinary” psychotics. The ordinariness of psychosis should be understood in terms of its frequency and the intensity of its symptoms, but also includes – and this appears between the lines – as a reference to a certain social norm. We are interested in the adjective “ordinary” because of its polysemic qualities, which also apply to autism as long as there are populations whose symptoms are subdued and who can therefore fit in and adapt to the social in a more or less “invisible” way.

However, this comparison is not completely symmetrical. When one speaks of ordinariness as a synonym for frequency, this is in no way comparable with the frequency of psychosis in the general population. This is true even if the psychiatric conception of autism is in the process of being extended (sometimes wrongly) and is increasingly confronted with particular autistic subjects (such as adults and/or women). One can assume that there are numerous psychotic subjects who have achieved and maintained a form of compensation within their structure, a sometimes permanent state of relative stability, *i.e.* without clinical consequences (clinical psychosis). Yet after a psychotic breakdown, certain subjects can find a new balance. The onset of the pathology is only one possible, and non-automatic, outcome of the psychotic structure: “the

subject's rebellion can act as a substitute to clinical psychosis, the latter returning to its initial state (without clinical manifestations) while also attaining a new state" [166]. If this is the case, we should assume that psychotic structures occur at a higher rate than 1% – the percentage of the Western population that is considered to be on the autism spectrum.

Yet, with autism, the major difference is that, as we have seen, "autism is not triggered:" [155] autism is already "there." Of course, insidious forms of schizophrenia do exist, as do forms of autism that appear later, when, for example, babies seem to behave "normally" during the first year of life, autistic behaviors manifesting themselves in the second or third year. The cases of Owen Suskind or of Donna Williams are examples of this. However, this later appearance of a disorder is restricted to early childhood; in this, it is clearly different from psychosis, which can be triggered at any age. It's also possible to imagine an evolution from an initial autistic blockage and a sort of subjective exit from autistic withdrawal that takes root in an "edge." The conditions of this evolution will be unique for each subject and will preserve the traces and the constraints of the early autistic mode of functioning. Thus, structural equilibrium, ordinarily expressed by the structure, is located in mechanisms that are significantly different than those used by the psychotic structure: in autism, a complexification of the "edge," and in psychosis, a suppletion or a sinthome that allows the subject to remain in balance in a Borromean ordinary psychosis.

For lack of a better term, we prefer to use the notion of "ordinary" autism, as the term seems already to be widely used and more intuitive in our field. Our preference is also motivated by the continuity with ordinary psychosis, or, for example, with "ordinary neurosis" [167] or "ordinary perversion" that "integrates this structure into the program of normality" [168]. This term draws attention to the continuum stretching between the normal and the pathological, which is our primary concern.

Therefore, and without locking ourselves into a rigid definition, this syntagm appears to offer clinicians the opportunity to put words to a new, contemporary clinical reality of autism: a

conception that takes into account a range of autistic subjectivities and that arises from a theoretical model whose current and future developments seem promising in their capacity to help us respond to a variety of stakes: clinical and diagnostic, as well as therapeutic and societal.

Why, one might ask, is there a need for a conceptual matrix that includes a consideration of a “non-disordered” autistic subjectivity (autism without ASD) for individuals whose semiology does not indicate the necessity for a psychiatric diagnosis, and thus indications for treatment and accompaniment? Indeed, we have pointed out that ordinary autistic subjects have been able to find a pacified balance in the interface between their being-in-the-world and the demands of social life; practically speaking, these subjects rarely seek help from clinicians [120]. However, they make up a clinical population that remains particularly vulnerable and that has a reduced capacity of adaptation to life’s stressors and, in consequence, a marked sensitivity to anxiety, to mood, to traumas, and to stress-related disorders [150]. When these subjects do occasionally seek help, it’s often at moments in their life when their self-organization frays ([108], for example), during times of social difficulty after interpersonal events that may lead to illegal acts and the attendant medico-legal consequences [120] or situations of abuse. These autistic subjects could then make up a significant, but difficult-to-quantify, number of clinical cases encountered in psychiatric facilities, seeking help with comorbid mental disorders or with suicidal ideation, acting-out, or self-harm [101, 120, 128, 150, 169], appearing in states of shutdown or meltdown, and forcing clinicians to deal with a blurred nosographical tableau. The encounter with these subjects is liable to lead to a number of false starts, both diagnostic [94, 95, 130] – we have already discussed the problematic nature of the differential diagnosis – and also therapeutic.

Regarding diagnosis, making these theoretical models operational is a path that should be pursued. As Miller advised [170] regarding ordinary psychosis, this happens through the attentive observation of “a variety of little clues” that express themselves in the cracks of autistic individuals’ subjective construction (a subjective construction that has a distinctively hand-made, DIY aspect).

We believe it's important to tease out distinctive clinical markers that could replace the DSM's criteria, which present a caricatural model of autism. The DSM model fails to recognize the extended range of autism, in terms of the subtlety of its symptomatic intensity, and the range of possibilities for subjective organization. A better model could help with the tricky differential diagnosis with ordinary psychosis [163] that is overlooked by psychiatric nosography. In an earlier article [104], we sketched out a possibility for operationalizing this project by using the Rorschach test to highlight the differential elements in clinical situations where the use of classic diagnostic criteria doesn't help elucidate the diagnostic situation. More recently, we have more strongly reiterated our thesis, based on a literature review, of the value of using the Rorschach test in evaluating ASD [165]. In effect, this test is particularly effective at revealing the semiological autistic characteristics that are sought for a psychiatric diagnosis. It also allows clinicians to study, using Maleval's theoretical postulates, intrapsychic cognitive and affective processes (underlying elements of behavioral manifestations), and the disturbances of these processes, in a dynamic perspective, in order to tease out qualitative and quantitative clues that might influence a differential diagnosis, in particular with schizophrenia [165]. As we've already pointed out, the stakes of differential diagnosis are real; a diagnosis of autism or of schizophrenia, for example, implies very different treatment plans.

Thus recognizing an ordinary autistic subject, regulated on the basis of an autistic structure, implies taking a position in the representation that we have of the autistic subject, and orients the accompaniment and the therapeutic treatments that can be proposed. The latter will be less focused on highly visible and debilitating autistic symptoms (these are not likely to be present), but, instead, will involve the work of supporting the subject while they come to grips with their difference, supporting them through the subjective processes they put in place to deal with their dynamic dissatisfaction, and accompanying their insertion in the social world [74, 161]. Thus, the therapeutic posture is one that remains in contact with the subject, with her/his knowledge and the skills that

s/he has developed to create a personal being-in-the-world. The clinician's role is to accompany the set-up of the patient's personal "therapeutic machinery" that is constructed in the interface of the demands of the subject's structure (her/his anxieties and preoccupations) with her/his interests and potential, in sum, within the subject's own psychic universe in its interaction with objects and a singular environment. Our action is conditioned by a non-intrusive, confident positioning in relation to the subject's desire to change. By respecting her/his objects and special interests, we can work towards tempering the subject's "edge" by complexifying it; this can ultimately transform a special interest into a veritable social competency, allowing for a subjective exit from autistic withdrawal and for greater autonomy [163, 171, 172]. This approach leads to therapeutic recommendations that are similar to those of psychoanalysis, as well as different epistemologies [173, 174]. Further studies on the formalization of the method and results of such approaches would be valuable for promoting the interest of this approach in the current socio-political climate. Particularly since this therapeutic orientation breaks with the quest for a superficial social adaptation by the acquisition of so-called "positive" behaviors according to techniques that are applied uniformly, regardless of the individual. Also, we should point out that adaptive strategies that are spontaneously learned, or for which the autistic subject is coached, are not always beneficial and should not necessarily be encouraged, given their serious cost in terms of fatigue, exhaustion, and stress, or even alterations of the autistic person's perception of her/himself. These elements contribute to the degradation of the subject's well-being and can lead to a certain subjective alienation that is often at the origin of the development of comorbid symptoms [100, 101].

Finally, if our work is focused on a pacified mediation between subjective modes of functioning and the social world, if we have insisted upon the specificities of different, and numerous, subjective organizations in order to understand individuals' needs and potential, we should also reflect on making social organizations and habitats more inclusive, which could benefit not only atypically functioning individuals, but the whole social fabric.

5. Conclusion

Of all the psychiatric entities of the 20th century, autism is the one that has made the transition into the 21st century with the greatest success in our field, while becoming a reference point in modern social discourse: Grollier [175] suggests that if the passage from the 19th to the 20th century occurred through an insistent interrogation of the notion of hysteria, the passage from the 20th to the 21st century is taking place through a questioning of autism. Leo Kanner's original description from 1943 has undergone significant revisions, linked to the evolution of ideas, of societies, of practices, of research, and of data – all of these having led to the multiplicity of definitions proposed that have, in turn, led to significant changes to the definition, the diagnosis, and the etiology of autism, and to a range of political and social proposals [31, 115, 135, 136, 141]. The DSM's recent spectrum-based model was a sign of progress in the understanding of ASD, with respect to earlier formalizations (links between formerly differentiated categories and a better appreciation of clinical heterogeneity). But the spectral model remains limited to a description of a behavioral semiology that only underscores clinical amalgamations [110], in particular with regards to the spectrum and the differential issue that arises. These successive and rapid changes to nosographical paradigms of autism spectrum disorders keep coming up against the same problem: how to include everything that we understand today as being part of autism, while excluding similar clinical forms that are not part of the spectrum?

One possible solution can be found in Maleval's structural psychoanalytic model, which inspired our use of the term "ordinary autism." Ordinary autism allows us to think about modified or "balanced" autistic phenotypes that are not prototypical, and that call into question fuzzy nosographical frameworks and interrogate contemporary clinical practices. These phenotypes – newly "ordinary" by virtue of their newfound frequency and because of phenomenological expressions that are less "extraordinary" than those of the prototypical cases that form the basis of the theoretical model of autism – interrogate the links between the normal and the pathological. A parallel could be drawn between this model and dominant psychiatric nosographies; we might also

adopt a common language that would add value to all models and lend them the depth of psychoanalysis's theorizations. This structural model of autism was born out of the theoretical renaissance, in a context of epistemic break [172, 173], marked by a new theoretical position staked out within the very discipline of psychoanalysis [161] and a break with earlier positions, in particular regarding the distinction between autism and psychosis.

Thus, to expand upon the analogy, just as ordinary psychosis imposed itself as a veritable theoretical necessity in response to a contemporaneous clinical reality, in a similar context, ordinary autism seems to us to respond to the same urgent need for markers, and this, in several ways.

1) Firstly, considering the range of autistic subjects without ASD is an urgent necessity in the public health context, since, as we have illustrated, a non-negligible proportion of autistic persons can not, or can no longer, receive a diagnosis, despite a clearly identified subjective suffering and a need for appropriate treatment. There is an ever-growing demand for diagnoses, and yet the professionals who are apt to respond to this demand, the means that are dedicated, and the tools provided to them are no longer sufficient, or are simply limited, especially for adult populations. The dimensional conceptualization of autism created clinical problems that must be addressed.

2) Finally, there is an urgent need to rehabilitate an understanding of the subjectivity of the autistic person, especially when current scientific discourse has a certain tendency to “negate the subject” in psychology – an inheritance of a factual medical science founded on proof and that affirms an “erasure of the sick person behind the sickness” [32]. This conceptual position has a particular resonance with an autistic state of being: walled-off in retreat, immutability, and the negation of the subject of the unconscious. Thus, moving away from “thinking about” autism to “talking about it” [76], thereby adopting a position that implies the recognition of an autistic subject whose psychic organization is complex and rich, compels us to rethink care: moving away from adaptive programs that seek to reeducate modes of functioning that are deemed deficient, oriented towards ideas of conformity that match the school system's or the family's ideas of “normalcy,” and

instead proposing an accompaniment and support of what the subject is able to do for her/himself, for example, in her/his “choice” of a particular object that responds to her/his difficulties, and this in order to accept and establish bonds with others [176]. This type of therapeutic objective is based on a conception of therapy with autistic subjects that has certain points in common with the ideas of Canadian cognitivists like Dawson and Mottron [173, 174]. In this way, ordinary autism, situated in a renewal of psychoanalysis’s structural considerations of autism, promotes the idea that the autistic person is a subject whom we can encounter with a subtle, global, and coherent understanding, beyond symptomatology, of her/his relationship with the world, a personal construction based on the demands of her/his unique structure and the way in which s/he grapples with this. The role of the therapist is then to accompany the subject in her/his “inventive discoveries” [171]. This perspective is essential for reintroducing respect for the autistic person’s subjectivity, too often overlooked by fossilized practices that are intolerant of a subjective position [32, 134, 172, 177] and whose sole focus is the reeducation of a deficient subject.

Psychoanalysis, too, must be able to reinvent itself, perhaps with regards to its own corpus [161, 173]. Psychoanalysis must push itself to transcend its classical models and to move towards an understanding of these new ways of being a “subject” – for example, with the idea of a new autistic subjective mode [178] -- while being in dialogue with and integrating the knowledge from and the progress made in other disciplines [179]. Given this, psychoanalysis will be able to offer a way of thinking about the interstices of psychiatric nosography and its modelizations, and this 19th-century discipline will thus be able to take part in current debates, contribute to research, and advance in our understanding of the enigma of autism – for autism remains enigmatic, both from a diagnostic and from a therapeutic point of view. Autism is not an inert “research object” but instead encompasses subjectivities with whom we should be actively engaging.

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Références

- [1] Fombonne E. L'autisme, une épidémie? *Inserm actualités*. 2006;(199):4–35.
- [2] Chamak B, Cohen D. (2007). Transformations des représentations de l'autisme et de sa prise en charge. *Perspectives psy*. 2007;46(3):218–27.
- [3] Baio J, Wiggins L, Christensen DL, Maenner MJ, Daniels J, Warren Z, et al. Prevalence of autism spectrum disorder among children aged 8 years—Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2014. *MMWR Surveill Summ*. 2018;67(6):1–23.
- [4] Zablotsky B, Black LI, Maenner MJ, et al. Prevalence and Trends of Developmental Disabilities among Children in the United States: 2009–2017. *Pediatrics*. 2019;144(4):e20190811.
- [5] Fombonne E. Editorial: The rising prevalence of autism. *Journal Child Psychol Psyc*. 2018;59(7):717–20.
- [6] Matson JL., Kozlowski AM. The increasing prevalence of autism spectrum disorders. *Res Autism Spect Dis*. 2011;5(1):418–25.
- [7] Fombonne E, Elsabbagh M, Clarke ME. Épidémiologie de l'autisme. *Encyclopédie sur le développement des jeunes enfants*. 2012;1–5.
- [8] Hansen SN, Schendel DE, Parner ET. Explaining the increase in the prevalence of autism spectrum disorders: The proportion attributable to changes in reporting practices. *JAMA Pediatr*. 2015;169(1):56–62.
- [9] King M, Bearman P. Diagnostic change and the increased prevalence of autism. *Int J Epidemiol*. 2009;38(5):1224–34.
- [10] Evrard P. L'autisme, une priorité de santé publique. *Études*. 2012;417(12):619–30.
- [11] Gernsbacher MA, Dawson M, Goldsmith H. Three reasons not to believe in an autism epidemic. *Curr Dir Psychol Sci*. 2005;14(2):55–8.
- [12] American Psychiatric Association (APA), DSM-5. *Diagnostic and Statistical Manual of Mental Disorders*. Washington; London : American Psychiatric Publishing; 2013.

- [13] Organisation mondiale de la santé. Classification statistique internationale des maladies et des problèmes de santé connexes-11^e version (CIM-11). Genève: Organisation mondiale de la santé; 2018.
- [14] Shattuck PT. Diagnostic substitution and changing autism prevalence. *Pediatrics*. 2006;117(4): 1438–9.
- [15] Lundström S, Reichenberg A, Anckarsäter H, Lichtenstein P, Gillberg C. Autism phenotype versus registered diagnosis in Swedish children: prevalence trends over 10 years in general population samples. *BMJ Open*. 2015;350:h1961.
- [16] Whitehouse AJO, Cooper MN, Bebbington K, Alvares G, Lin A, Wray J, et al. Evidence of a reduction over time in the behavioral severity of autistic disorder diagnoses. *Autism Res*. 2017;10(1):179–87.
- [17] Rødgaard EM, Jensen K, Vergnes JN, Soulières I, Mottron L. Temporal Changes in Effect Sizes of Studies Comparing Individuals With and Without Autism: A Meta-analysis. *JAMA Psychiat*. 2019;1956.
- [18] Jensen CM, Steinhausen HC, Lauritsen MB. Time trends over 16 years in incidence-rates of autism spectrum disorders across the lifespan based on nationwide Danish register data. *J Autism Dev Disord*. 2014;44(8):1808–18.
- [19] Miller JA. *La psychose ordinaire, La Convention d'Antibes*. Paris: Agalma Le Seuil; 1999.
- [20] Maleval JC. De l'autisme de Kanner au syndrome d'Asperger. *Evol Psychiatr*. 1998;63(3):293–309.
- [21] Demazeux S. L'échec du DSM-5, ou la victoire du principe de conservatisme. *Inf Psychiatr*. 2013;89(4):295–302.
- [22] De Beaurepaire, R. (2014). La schizophrénie et les autres psychoses dans le DSM-5. *Lett Psychiatr*. 2014;10(2):57–63.

- [23] Barch DM, Bustillo J, Gaebel W, Gur R, Heckers S, Malaspina D, et al. Logic and justification for dimensional assessment of symptoms and related clinical phenomena in psychosis: relevance to DSM-5. *Schizophr Res.* 2013;150(1):15–20.
- [24] Lord C, Bishop SL. Recent advances in autism research as reflected in DSM-5 criteria for autism spectrum disorder. *Annu Rev Clin Psychol.* 2015;11(1):53–70.
- [25] Sahnoun L, Rosier A. Syndrome d'Asperger : les enjeux d'une disparition. *PSN-Psychiatr Sci Hum.* 2012;10(1):25–33.
- [26] Grollier M, Leblanc M, Michel S. Autisme sévère, syndrome d'Asperger, écarts et similitudes. *Evol Psychiatr.* 2016;81(2), 446–459.
- [27] Swedo SE, Baird G, Cook Jr EH, Happe FG, Harris JC, Kaufmann WE, et al. Commentary from the DSM-5 workgroup on neurodevelopmental disorders. *J Am Acad Child Psy.* 2012;51(4):347–9.
- [28] Happé F. Criteria, categories, and continua: autism and related disorders in DSM-5. *J Am Acad Child Psy.* 2011;50(6):540–2.
- [29] Tsai LY, Ghaziuddin M. DSM-5 ASD Moves Forward into the Past. *J Autism Dev Disord.* 2014;44(2):321–30.
- [30] Lai MC, Lombardo MV, Chakrabarti B, Baron-Cohen S. Subgrouping the Autism “Spectrum”: Reflections on DSM-5. *Plos Biol.* 2013;11(4):e1001544.
- [31] Perrin M, Druel-Salmane G. L'autisme, au pays des sciences. *Clin Mediterr.* 2009;79(1):237–51.
- [32] Hochmann J. L'autisme entre Evidence Based Medicine et médecine narrative. In: Amy MD, editors. *Autismes et psychanalyses.* Toulouse: ERES; 2014. p. 24–47.
- [33] McPartland JC, Reichow B, Volkmar FR. Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *J Am Acad Child Psy.* 2012;51(4):368–83.
- [34] Weyland M. De la définition au diagnostic, du diagnostic à la prise en charge : DSM-5 dans le domaine de l'autisme et impacts d'une nouvelle version. *Les Cahiers de l'ASELF.* 2014;11(1):1–8.

- [35] Wakefield JC. Diagnostic Issues and Controversies in DSM-5: Return of the False Positives Problem. *Annu Rev Clin Psychol.* 2016;12:105–32.
- [36] Huerta M, Bishop SL, Duncan A, Hus V, Lord C. Application of DSM-5 criteria for autism spectrum disorder to three samples of children with DSM-IV diagnoses of pervasive developmental disorders. *Am J Psychiat.* 2012;169(10):1056–64.
- [37] Kim YS, Fombonne E, Koh YJ, Kim SJ, Cheon KA, Leventhal BL. A comparison of DSM-IV pervasive developmental disorder and DSM-5 autism spectrum disorder prevalence in an epidemiologic sample. *J Am Acad Child Psy.* 2014;53(5):500–8.
- [38] Zeldovich L. Why the definition of autism needs to be refined. [serial online] 2018 May. [cited 2020 Jan 27];[12 p]. Available from: URL: <https://www.spectrumnews.org/features/deep-dive/definition-autism-needs-refined/>.
- [39] Kulage KM, Smaldone AM, Cohn EG. How Will DSM-5 Affect Autism Diagnosis? A Systematic Literature Review and Meta-analysis. *J Autism Dev Disord.* 2014;44(8):1918–32.
- [40] Kulage KM, Goldberg J, Usseglio J, RomeroD, Bain JM, Smaldone AM. How has DSM-5 Affected Autism Diagnosis? A 5-Year Follow-Up Systematic Literature Review and Meta-analysis. *J Autism Dev Disord.* 2019;1–26.
- [41] Sturme y P, Dalfern S. The Effects of DSM5 Autism Diagnostic Criteria on Number of Individuals Diagnosed with Autism Spectrum Disorders: A Systematic Review. *Rev J Autism Dev Disord.* 2014;1(4):249–52.
- [42] Smith IC, Reichow B, Volkmar FR. The Effects of DSM-5 Criteria on Number of Individuals Diagnosed with Autism Spectrum Disorder: A Systematic Review. *J Autism Dev Disord.* 2015;45(8):2541–52.
- [43] Bennett M, Goodall E. A Meta-analysis of DSM-5 Autism Diagnoses in Relation to DSM-IV and DSM-IV-TR. *Rev J Autism Dev Disord.* 2016;3(2):119–24. [44] Taheri A, Perry A. Exploring the proposed DSM-5 criteria in a clinical sample. *J Autism Dev Disord.* 2012;42(9):1810–7.

- [45] Frazier TW, Youngstrom EA, Speer L, Embacher R, Law P, Constantino J, et al. Validation of Proposed DSM-5 Criteria for Autism Spectrum Disorder. *J Am Acad Child Psy.* 2012;51(1):28–40.
- [46] Matson JL, Belva BC, Horovitz M, Kozlowski AM, Bamburg JW. Comparing symptoms of autism spectrum disorders in a developmentally disabled adult population using the current DSM-IV-TR diagnostic criteria and the proposed DSM-5 diagnostic criteria. *J Dev Pays Disabil.* 2012;24(4):403–14.
- [47] Sung M, Goh TJ, Tan BLJ, Chan JS, Liew HSA. Comparison of DSM-IV-TR and DSM-5 Criteria in Diagnosing Autism Spectrum Disorders in Singapore. *J Autism Dev Disord.* 2018;48(10):3273–81.
- [48] Matson JL, Hattier MA, Williams LW. How does relaxing the algorithm for autism affect DSM-V prevalence rates? *J Autism Dev Disord.* 2012;42(8):1549–1556.
- [49] Mayes SD, Black A, Tierney CD. DSM-5 under-identifies PDDNOS: Diagnostic agreement between the DSM-5, DSM-IV, and Checklist for Autism Spectrum Disorder. *Res Autism Spect Dis.* 2013;7(2):298–306.
- [50] Mayes SD, Calhoun SL, Murray MJ, Pearl A, Black A, Tierney CD. Final DSM-5 under-identifies mild Autism Spectrum Disorder: Agreement between the DSM-5, CARS, CASD, and clinical diagnoses. *Res Autism Spect Dis.* 2014;8(2):68–73.
- [51] Wilson CE, Gillan N, Spain D, Robertson D, Roberts G, Murphy CM. Comparison of ICD-10R, DSM-IV-TR and DSM-5 in an Adult Autism Spectrum Disorder Diagnostic Clinic. *J Autism Dev Disord.* 2013;43(11):2515–25.
- [52] Lai MC, Lombardo MV, Auyeung B, Chakrabarti B, Baron-Cohen S. Sex/gender differences and autism: setting the scene for future research. *J Am Acad Child Psy.* 2015;54(1):11–24.
- [53] Murphy D, Glaser K, Hayward H, Eklund H, Cadman T, Findon J, et al. Improving outcomes through better diagnosis: the effects of changes in DSM-V on clinical diagnosis. Southampton (UK): NIHR Journals Library; 2018.

- [54] Robison ER. Social Communication Disorder—is it "Autism Lite?". *Psychology Today* [serial online] 2013 Jan. [cited 2019 Oct 18];[3 p]. Available from: URL: <https://www.psychologytoday.com/us/blog/my-life-aspergers/201301/social-communication-disorder-is-it-autism-lite>.
- [55] Mandy W, Wang A, Lee I, Skuse D. Evaluating social (pragmatic) communication disorder. *J Child Psychol Psyc*. 2017;58(10):1166–75.
- [56] Greaves-Lord K, Eussen ML, Verhulst FC, Minderaa RB, Mandy W, Hudziak JJ et al. Empirically based phenotypic profiles of children with pervasive developmental disorders: interpretation in the light of the DSM-5. *J Autism Dev Disord*. 2013;43(8):1784–97.
- [57] Regier DA, Narrow WE, Clarke DE, Kraemer HC, Kuramoto SJ, Kuhl EA et al. DSM-5 field trials in the United States and Canada, Part II: test-retest reliability of selected categorical diagnoses. *Am J Psychiat*. 2013;170(1):59–70.
- [58] Flax J, Gwin C, Wilson S, Fradkin Y, Buyske S, Brzustowicz L. Social (Pragmatic) Communication Disorder: Another name for the Broad Autism Phenotype? *Autism*. 2019;23(8):1982–92.
- [59] Tager-Flusberg H. Why no one needs a diagnosis of ‘social communication disorder’. *Spectrum*. [serial online] 2017 Apr. [cited 2019 Oct 20];[4 p]. Available from: URL: <https://www.spectrumnews.org/opinion/viewpoint/no-one-needs-diagnosis-social-communication-disorder/>.
- [60] Tager-Flusberg H. Evidence weak for social communication disorder. *Spectrum*. [serial online] 2013 May. [cited 2019 Oct 20];[5 p]. Available from: URL: <https://www.spectrumnews.org/opinion/viewpoint/evidence-weak-for-social-communication-disorder/>.
- [61] Furfaro H. New diagnosis covers ‘borderlands’ of autism spectrum. *Spectrum*. [serial online] 2017 Aug. [cited 2019 Oct 20];[4 p]. Available from: URL: <https://www.spectrumnews.org/news/new-diagnosis-covers-borderlands-autism-spectrum/>.

- [62] Swineford LB, Thurm A, Baird G, Wetherby AM, Swedo S. Social (pragmatic) communication disorder: A research review of this new DSM-5 diagnostic category. *J Neurodev Disord*. 2014;6(1):41–49.
- [63] Scheepers M, Kerr M. *Seminars in the Psychiatry of Intellectual Disability*. Cambridge: Cambridge University Press; 2019.
- [64] Newson E, Le Marechal K, David C. Pathological demand avoidance syndrome: a necessary distinction within the pervasive developmental disorders. *Arch Dis Child*. 2003;88(7):595–600.
- [65] Philippe A, Contejean Y. Le syndrome d'évitement pathologique des demandes : psychopathie autistique ? Syndrome d'Asperger ? Autisme atypique ? Ou trouble envahissant du développement (TED) spécifique ? *Neuropsychiat Enf*. 2018;66(2):103–8.
- [66] Wing L, Gould J, Gillberg C. Autism spectrum disorders in the DSM-V: better or worse than the DSM-IV? *Res Dev Disabil*. 2011;32(2):768–73.
- [67] O'Nions E, Viding E, Greven CU, Ronald A, Happé F. Pathological demand avoidance: exploring the behavioural profile. *Autism*. 2014;18(5):538–44.
- [68] O'Nions E, Gould J, Christie P, Gillberg C, Viding E, Happé F. Identifying features of 'pathological demand avoidance' using the Diagnostic Interview for Social and Communication Disorders (DISCO). *Eur Child Adoles Psy*. 2016;25(4):407–19.
- [69] Gillberg C, Gillberg IC, Thompson L, Biskupsto R, Billstedt E. Extreme ("pathological") demand avoidance in autism: a general population study in the Faroe Islands. *Eur Child Adoles Psy*. 2015;24(8):979–84.
- [70] Green J, Absoud M, Grahame V, Malik O, Simonoff E, Le Couteur A et al. Pathological Demand Avoidance: symptoms but not a syndrome. *Lancet*. 2018;2(6):455–64.
- [71] Gökçen E, Petrides KV, Hudry K, Frederickson N, Smillie LD. (2013). Sub-threshold autism traits: the role of trait emotional intelligence and cognitive flexibility. *Br J Psychol*. 2013;105(2):187–99.

- [72] Coman DC. Autism Spectrum Disorders. In: Wilson H, Braaten E, editors. *The Massachusetts General Hospital Guide to Learning Disabilities. Current Clinical Psychiatry*. Totowa, NJ: Humana Press; 2019. p. 149–69.
- [73] Fein D, Barton M, Eigsti IM, Kelley E, Naigles L, Schultz RT et al. Optimal outcome in individuals with a history of autism. *J Child Psychol Psyc*. 2013;54(2):195–205.
- [74] Maleval JC. *L'autiste et sa voix*. Paris: Seuil; 2009.
- [75] Meltzer D. *Explorations dans le monde de l'autisme (1975)*. Paris: Payot; 1980.
- [76] Passone S, Suarez-Labat H. *Après l'autisme. Comment sortir de l'état autistique*. Paris: In Press; 2013.
- [77] Lovaas OI. Behavioral treatment and normal educational and intellectual functioning in young autistic children. *J Consult Clin Psych*. 1987;55(1):3–9.
- [78] Helt M, Kelley E, Kinsbourne M, Pandey J, Boorstein H, Herbert M et al. Can children with autism recover? If so, how? *Neuropsychol Rev*. 2008;18(4):339–66.
- [79] Olsson MB, Westerlund J, Lundström S, Giacobini M, Fernell E, Gillberg C. “recovery” from the diagnosis of autism—and then? *Neuropsych Dis Treat*. 2015;11:999–1005.
- [80] Suh J, Orinstein A, Barton M, Chen CM, Eigsti IM, Ramirez-Esparza N, Fein D. Ratings of broader autism phenotype and personality traits in optimal outcomes from autism spectrum disorder. *J Autism Dev Disord*. 2016;46(11):3505–18.
- [81] Ruzich E, Allison C, Smith P, Watson P, Auyeung B, Ring H et al. Measuring autistic traits in the general population: a systematic review of the Autism-Spectrum Quotient (AQ) in a nonclinical population sample of 6,900 typical adult males and females. *Mal Autism*. 2015;6(1):2–14.
- [82] Sucksmith E, Roth I, Hoekstra RA. Autistic traits below the clinical threshold: re-examining the broader autism phenotype in the 21st century. *Neuropsychol Rev*. 2011;21(4):360–89.
- [83] Goussé V, Galéra C, Bouvard M, Michel G. Agrégation de déficits sociaux et de troubles psychopathologiques chez les parents de personnes avec autisme: vers une implication du tempérament? *Encephale*. 2011;37(2):119–26.

- [84] Palmer CJ, Paton B, Enticott PG, Hohwy J. 'Subtypes' in the presentation of autistic traits in the general adult population. *J Autism Dev Disord.* 2015;45(5):1291–301.
- [85] Robinson EB, St Pourcain B, Anttila V, Kosmicki JA, Bulik-Sullivan B, Grove J et al. Genetic risk for autism spectrum disorders and neuropsychiatric variation in the general population. *Nat Genet.* 2016;48(5):552.
- [86] Tanguay PE. Autism in DSM-5. *Am J Psychiat.* 2011;168(11):1142–44.
- [87] Kato S. Psychopathologie du syndrome d'Asperger et «aspérisation» de la société contemporaine. *Neuropsychiat Enf.* 2011;59(5):274–8.
- [88] Baker AR. The invisible end of the spectrum: Shadows, residues, 'BAP', and the female Aspergers experience. Paper presented to Inaugural World Autism Congress "Unity through Diversity"; 2002; Melbourne, Australia. Retrieved from <http://www.autismcongress.com>. 2002
- [89] Baron-Cohen S, Wheelwright S, Skinner R, Martin J, Clubley E. The autism-spectrum quotient (AQ): Evidence from asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *J Autism Dev Disord.* 2001;31(1):5–17.
- [90] Dawson G, Webb S, Schellenberg GD, Dager S, Friedman S, Aylward E et al. Defining the broader phenotype of autism: genetic, brain, and behavioral perspectives. *Dev Psychopathol.* 2002;14(3):581–611.
- [91] Hurley RS, Losh M, Parlier M, Reznick JS, Piven J. The broad autism phenotype questionnaire. *J Autism Dev Disord.* 2007;37(9):1679–90.
- [92] Landry O, Chouinard PA. Why We Should Study the Broader Autism Phenotype in Typically Developing Populations. *J Cogn Dev.* 2016;17(4):584–95.
- [93] De Groot K, Van Strien JW. Evidence for a broad autism phenotype. *Adv Neurodev Disord.* 2017;1(3):129–140.
- [94] Van Schalkwyk, G. I., Peluso, F., Qayyum, Z., McPartland, J. C., & Volkmar, F. R. Varieties of Misdiagnosis in ASD: An Illustrative Case Series. *J Autism Dev Disord.* 2015;45(4):911–8.

- [95] Alexander L, Farrelly N. A case of mistaken diagnoses: diagnostic and management challenges in a case of adult autism spectrum disorder. *Irish J Psychol Med.* 2019;1–4.
- [96] Lai MC, Baron-Cohen S. Identifying the lost generation of adults with autism spectrum conditions. *Lancet Psychiat.* 2015;2(11):1013–27.
- [97] Epstein T. *Autism in Adulthood.* Cham: Springer; 2019.
- [98] Frigaux A, Evrard R, Lighezzolo-Alnot J. L’ADI-R et l’ADOS face au diagnostic différentiel des troubles du spectre autistique : intérêts, limites et ouvertures. *Encephale.* 2019;45(5):441–8.
- [99] Murphy CM, Wilson CE, Robertson DM, Ecker C, Daly EM, Hammond N. et al. Autism spectrum disorder in adults: diagnosis, management, and health services development. *Neuropsych Dis Treat.* 2016;12:1669–86.
- [100] Hull L, Petrides KV, Allison C, Smith P, Baron-Cohen S, Lai MC et al. “Putting on My Best Normal”: Social Camouflaging in Adults with Autism Spectrum Conditions. *J Autism Dev Disord.* 2017;47(8):2519–34.
- [101] Livingston LA, Shah P, Happé F. Compensatory strategies below the behavioural surface in autism: a qualitative study. *Lancet Psychiat.* 2019;6(9):766–77.
- [102] Fusar-Poli L, Brondino N, Rocchetti M, Panisi C, Provenzani U, Damiani S et al. Diagnosing ASD in Adults Without ID: Accuracy of the ADOS-2 and the ADI-R. *J Autism Dev Disord.* 2017;47(11):3370–9.
- [103] Taylor JL, Seltzer MM. Changes in the autism behavioral phenotype during the transition to adulthood. *J Autism Dev Disord.* 2010;40(12):1431–46.
- [104] Frigaux A, Evrard R, Demogeot N. Au carrefour des spectres: problèmes de diagnostic différentiel entre autisme et schizotypie, autour du cas d’un jeune adulte. *Evol Psychiatr.* 2018;83(1):161–181.
- [105] Verhoeff B. Autism in flux: a history of the concept from Leo Kanner to DSM-5. *Hist Psychiatr.* 2013;24(4):442–58.

- [106] Bursztejn C. Autisme, TED, TSA: de quoi, de qui parle-t-on?. In: Berges-Bounes M, Forget JM, editors. Les psychoses chez l'enfant et l'adolescent. Toulouse: ERES; 2016. p. 61–78.
- [107] Brémaud N. Autisme vs psychose : une question toujours d'actualité? *Evol Psychiatr.* 2017;82(3):664–86.
- [108] Lazaratou H, Mouselimi A, Tsipa N, Golse B. L'évolution à long terme d'un cas de psychose infantile. Plaidoyer pour la réhabilitation du terme de «psychose». *Psychiatr Infant.* 2019;62(1):19–35.
- [109] Bursztejn C, Raynaud, JP, Misés R. Autisme, psychose précoce, troubles envahissants du développement. *Ann Med-Psychol.* 2011;169(4):256–9.
- [110] Demily C, Tordjman S. Vers une conceptualisation des troubles du spectre autistique comme un ensemble de maladies rares. *Perspectives Psy.* 2017;56(4):320–8.
- [111] Houzel D. 72. Nouvelles approches psychopathologiques de l'autisme infantile. In: Lebovici S, Diatkine R, Soulé M, editors. *Nouveau traité de psychiatrie de l'enfant et de l'adolescent: 4 volumes.* Paris: Presses Universitaires de France; 2004. p. 1225–54.
- [112] Rapoport J, Chavez A, Greenstein D, Addington A, Gogtay N. Autism Spectrum Disorders and Childhood-Onset Schizophrenia: Clinical and Biological Contributions to a Relation Revisited. *J Am Acad Child Psy.* 2009;48(1):10–8.
- [113] Touati B, Mercier A, Tuil L. Autisme : évaluation des diagnostics et des traitements dans un secteur de pédopsychiatrie. *Psychiatr Infant.* 2016;59(1):225–290.
- [114] Raja M, Azzoni A. Autistic spectrum disorders and schizophrenia in the adult psychiatric setting: diagnosis and comorbidity. *Psychiatr Danub.* 2010;22(4):514–21.
- [115] Bertelli MO, Piva Merli M, Bradley E, Keller R, Varruciu N, Del Furia C, et al. The diagnostic boundary between autism spectrum disorder, intellectual developmental disorder and schizophrenia spectrum disorders. *Adv Ment Health Intellect Disabil.* 2015;9(5):243–64.

- [116] Lugo Marín J, Alviani Rodríguez-Franco M, Mahtani Chugani V, Magán Maganto M, Díez Villoria E, Canal Bedia R. Prevalence of Schizophrenia Spectrum Disorders in Average-IQ Adults with Autism Spectrum Disorders: A Meta-analysis. *J Autism Dev Disord*. 2018;48(1):239-250.
- [117] Barlati S, Deste G, Ariu C, Vita A. Autism Spectrum Disorder and Schizophrenia: Do They Overlap? *Int J Emerg Ment Health*. 2016;18(1):760–3.
- [118] Gandal MJ, Haney JR, Parikshak NN, Leppa V, Ramaswami G, Hartl C. et al. Shared molecular neuropathology across major psychiatric disorders parallels polygenic overlap. *Science*. 2018;359(6376):693–7.
- [119] Lugnegård T, Hallerbäck MU, Gillberg C. Personality disorders and autism spectrum disorders: what are the connections? *Compr Psychiat*. 2012;53(4):333–40.
- [120] Dell’Osso L, Luche RD, Gesi C, Moroni I, Carmassi C, Maj M. From Asperger’s to DSM-5 Autism Spectrum Disorder and Beyond: A Subthreshold Autism Spectrum Model. *Clin Pract Epidemiol Ment Health*. 2016;12(1):120–31.
- [121] Ford TC, Apputhurai P, Meyer D, Crewther DP. Cluster analysis reveals subclinical subgroups with shared autistic and schizotypal traits. *Psychiat Res*. 2018;265:111–7.
- [122] McDougle CJ. (2016). *Autism Spectrum Disorder*. Oxford : Oxford University Press; 2016.
- [123] Seltzer MM, Shattuck P, Abbeduto L, Greenberg JS. Trajectory of development in adolescents and adults with autism. *Ment Retard Dev D R*. 2004;10(4):234–47.
- [124] Cath DC, Ran N, Smit JH, van Balkom AJ, Comijs HC. Symptom overlap between autism spectrum disorder, generalized social anxiety disorder and obsessive-compulsive disorder in adults: a preliminary case-controlled study. *Psychopathology*. 2008;41(2):101–10.
- [125] Matson JL, Williams LW. Differential diagnosis and comorbidity: distinguishing autism from other mental health issues. *Neuropsychiatry-Lond*. 2013;3(2):233–43.
- [126] Vilasaliu L, Jensen K, Hoss S, Landenberger M, Menze M, Schütz M, et al. Diagnostic instruments for autism spectrum disorder (ASD). *Cochrane Db Syst Rev*. 2016;1:CD012036.

- [127] Levy SE, Giarelli E, Lee LC, Schieve LA, Kirby RS, Cunniff C, et al. Autism spectrum disorder and co-occurring developmental, psychiatric, and medical conditions among children in multiple populations of the United States. *J Dev Behav Pediatr*. 2010;31:267–75.
- [128] Lugo-Marín J, Magán-Maganto M, Rivero-Santana A, Cuellar-Pompa L, Alviani M, Jenaro-Rio, et al. Prevalence of psychiatric disorders in adults with autism spectrum disorder: A systematic review and meta-analysis. *Res Autism Spect Dis*. 2019;59(1):22–33.
- [129] Romero M, Aguilar JM, Del-Rey-Mejías Á, Mayoral F, Rapado M, Pecina M, et al. Psychiatric comorbidities in autism spectrum disorder: A comparative study between DSM-IV-TR and DSM-5 diagnosis. *Int J Clin Hlth Psych*. 2016;16(3):266–75.
- [130] Dossetor D. 'All that glitters is not gold': misdiagnosis of psychosis in pervasive developmental disorders – A case series. *Clin Child Psychol P*. 2007;12(4):537–48.
- [131] Barlati S, Deste G, Gregorelli M, Vita A. Autistic traits in a sample of adult patients with schizophrenia: prevalence and correlates. *Psychol Med*. 2018;49(1):140–8.
- [132] Writh J. Inconsistent prevalence estimates highlight studies' flaws. *Spectrum*. [serial online] 2018 Aug. [cited 2019 Oct 22];[5 p]. Available from: URL: <https://www.spectrumnews.org/opinion/q-and-a/inconsistent-prevalence-estimates-highlight-studies-flaws/>.
- [133] Grégoire P, Mottron L. L'autisme en pédopsychiatrie : épidémie ou dérive ? *Sur le spectre*. 2019;7:9.
- [134] Alerini P. L'autisme : symptôme de l'antipsychanalyse ? *Essaim*. 2011;27(2):7–31.
- [135] Hochmann J. La bataille de l'autisme *Réflexions sur un phénomène social contemporain*. *PSN-Psychiatr Sci Hum*. 2009;7(3-4):99–111.
- [136] Hochmann J. Le communautarisme dans la bataille de l'autisme. *PSN-Psychiatr Sci Hum*. 2016;14(3):7–16.
- [137] Matson JL. *Handbook of assessment and diagnosis of autism spectrum disorder*. Cham: Springer; 2016.

- [138] Chamak B. L'autisme à l'écran. [serial online] 2015 Aug. [cited 2019 Oct 22];[8 p]. Available from: URL: Hal-SHS-01182764. <https://halshs.archives-ouvertes.fr/halshs-01182764/document>.
- [139] De Bucy G. Syndrome d'Asperger : un diagnostique désirable ? À propos de quelques demandes diagnostiques formulées par des adultes au CRA d'Amiens [Diplôme d'État spécialité psychiatrie]. Amiens: Université de Picardie Jules Verne; 2018.
- [140] Mottron L. L'autisme, une autre intelligence: diagnostic, cognition et support des personnes autistes sans déficience intellectuelle. Bruxelles: Mardaga; 2004.
- [141] Peuch-Lestrade J. Autisme et psychanalyse : pourquoi la guerre ? *Topique*. 2007;99(2):17–28.
- [142] Hacking I. Les fous voyageurs. Paris: Les empêcheurs de penser en rond (trad. fr. Françoise Bouillot); 2002.
- [143] Murray S. Representing autism: Culture, narrative, fascination. Oxford: Oxford University Press; 2008.
- [144] Chamak B, Bonniau B. Neurodiversité : une autre façon de penser. In: Chamak B, Moutaud B, editors. *Neurosciences et Société : enjeux des savoirs et pratiques sur le cerveau*. Paris: Armand Colin; 2014. p. 211–30.
- [145] Chamak B. Le concept de neurodiversité ou l'éloge de la différence. In: Déchamp-Le Roux C, Rafael F, editors. *Regards croisés sur l'idée de guérison et de rétablissement en santé mentale*. Montrouge: John Libbey Eurotext; 2015.
- [146] Chamak B. Autisme et militantisme: de la maladie à la différence. *Quaderni*. 2009;(68):61–70.
- [147] Foucault M. Il faut défendre la société. Cours au Collège de France, 1976-1997. cours du 17 mars 1976, p.225.
- [148] Maleval JC. Extension du spectre de l'autisme. Collectif de praticiens auprès d'autistes. *Ecouter les autistes*. [serial online] 2014 Oct. [cited 2019 Oct 22];[21 p]. Available from: URL: <http://www.autistes-et-cliniciens.org/Extension-du-spectre-de-l-autisme-60>.

- [149] Dell’Osso L, Dalle Luche R, Maj M. Adult autism spectrum as a transnosographic dimension. *CNS Spectr.* 2016;21(02):131–3.
- [150] Dell’Osso L, Gesi C, Massimetti E, Cremone IM, Barbuti M, Maccariello G, et al. Adult Autism Subthreshold Spectrum (AdAS Spectrum): Validation of a questionnaire investigating subthreshold autism spectrum. *Compr Psychiat.* 2017;73:61–83.
- [151] Dell’Osso L, Corsi M, Gesi C, Bertelloni CA, Massimetti G, Peroni D, al. Adult Autism Subthreshold Spectrum (AdAS Spectrum) in parents of pediatric patients with epilepsy: Correlations with post-traumatic stress symptoms. *Compr Psychiat.* 2018;83:25–30.
- [152] Dell’Osso L, Muti D, Carpita B, Cremone IM, Bui E, Gesi C, et al. The Adult Autism Subthreshold Spectrum (AdAS) model: a neurodevelopmental approach to mental disorders. *Journal of psychopathology.* 2018;24(3):118–24.
- [153] Coutelle R, Montaut T, Sibertin-Blanc D. Propositions pour une perspective phénoménologique dans l’autisme infantile. *PSN-Psychiatr Sci Hum.* 2011;9(4)198–207.
- [154] Kästner A, Begemann M, Michel TM, Everts S, Stepniak B, Bach C, et al. Autism beyond diagnostic categories: characterization of autistic phenotypes in schizophrenia. *BMC psychiatry.* 2015;15(1):115–26.
- [155] Maleval J. Pourquoi l’hypothèse d’une structure autistique ? (I). *La Cause Du Désir.* 2014;87(2):167–73.
- [156] Maleval J. Pourquoi l’hypothèse d’une structure autistique ? (II). *La Cause Du Désir.* 2014;88(3):153–64.
- [157] Maleval J. Pourquoi l’hypothèse d’une structure autistique ? (III). *La Cause Du Désir.* 2015;89(1):164–70.
- [158] Lefort R. *La distinction de l’autisme.* Paris: Seuil; 2003.
- [159] Laurent E. *L’autisme et la psychanalyse.* Toulouse: Presses Universitaires du Mirail; 1992.

- [160] Maleval JC. Pourquoi l'autisme n'est-il plus une psychose ? Collectif de praticiens auprès d'autistes. *Ecouter les autistes*. [serial online] 2013 Oct. [cited 2019 Oct 22];[21 p]. Available from: URL: <http://www.autistes-et-cliniciens.org/Pourquoi-l-autisme-n-est-il-plus>.
- [161] Bentata H. L'autiste et sa voix. *Cahiers de PreAut*. 2010;7(1):159–71.
- [162] Maleval JC. La rétention des objets pulsionnels au principe de l'autisme. In: Causse JD, Rey-Flaud H, editors. *Les paradoxes de l'autisme*. Toulouse: Erès; 2011. p. 28–38.
- [163] Maleval JC, Grollier M. Extension du spectre de l'autisme. *Evol Psychiatr*. 2015;80(4):764–81.
- [164] Beauchamp-Châtel A, Duret P, Mottron L. L'autisme « invisible »: le cas des femmes. Communication au : « Le point sur l'autisme invisible »; 31 mars 2017; Rouen.
- [165] Frigaux A, Evrard R, Lighezzolo-Alnot J. L'intérêt du test de Rorschach dans l'évaluation diagnostique des troubles du spectre autistique. *Evol Psychiatr*. 2020;85(1):
- [166] Arce-Ross G. Déclenchement des psychoses: les facteurs blancs, une valeur de nuisance vide. *Inf Psychiatr*. 2003;79(5):403–14.
- [167] Evrard R. Pour introduire la névrose extraordinaire. *Recherches en psychanalyse*. 2013;15(1):71–9.
- [168] Lebrun JP. *La perversion ordinaire*. Paris: Flammarion; 2015.
- [169] Vannucchi G, Masi G, Toni C, Dell'Osso L, Marazziti D, Perugi G. Clinical features, developmental course, and psychiatric comorbidity of adult autism spectrum disorders. *CNS spectr*. 2014;19(2):157–64.
- [170] Miller JA. Effet retour sur la psychose ordinaire. *Quarto*. 2009;94/95:40–51.
- [171] Maleval JC, Grollier M. La thérapie par affinités (ou le résistant retour d'une approche psychodynamique pour le traitement des autistes). *Evol Psychiatr*. 2017;82(3):646–63.
- [172] Gómez Cabra ML. *La clinique de la psychose et l'autisme chez l'enfant et la pratique à plusieurs* [thèse de psychologie]. Paris: université de Paris 8; 2015.
- [173] Lucchelli JP. *Autisme: quelle place pour la psychanalyse?* Paris: Michèle; 2018.

- [174] Maleval J.C. Nourrir l'intelligence de l'autiste ou mobiliser sa jouissance ? *La Cause Du Désir*. 2018;98:59–66.
- [175] Grollier M. L'autisme au xxie siècle? *Clin Mediterr*. 2007;76(2):271–86.
- [176] Bonnat J. Actualité de la clinique psychanalytique. Présentation. *B Psychol*. 2014;529:(1):3–4.
- [177] Golse B. Ce sur quoi nous ne pouvons pas céder. *Rev Fr Psychanal*. 2013;77(1):126–31.
- [178] Kupfer MC. Peut-il y avoir de nouveaux modes... de subjectivation où l'inconscient n'aurait pas de place ? *Analyse Freudienne Presse*. 2019;26(1):15–24.
- [179] Crespin G. Plaidoirie pour une approche intégrative des tsa. *Cahiers de PréAut*. 2017;14(1):9–24.