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Letter to the Editor

Social dysfunction in preclinical, at risk stages of psychosis: A developmental view☆

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Difficulties in the social domain are allegedly an early clinical feature in the neurodevelopmental progression to psychosis, as clearly shown by the recent study of D'Angelo et al. (2018). Crucially, such study revealed that a parent-measure of social functioning (i.e. the Social Responsiveness Scale-II), which was originally developed within the context of autism spectrum disorder (ASD), is able to detect an early social impairment in children and adolescents at risk of or with psychosis. Such early social impairment presents a severity commensurable to the one reported in ASD in approximately one-third (30.6%) of the Ultra High-Risk (UHR) sample and in almost half (52.4%) of the psychotic sample. Clearly, these findings have implications both for the identification of social impairment itself and for the differential diagnosis between ASD and schizophrenia spectrum disorder (SSD).

Indeed, the finding of an early impairment of social functioning agrees with meta-analytical evidences on the association of adult psychosis with childhood social isolation, social withdrawal and adolescent social maladjustment (Matheson et al., 2013; Tarbox and Pogue-Geile, 2008); for example, teacher-rated social functioning appears to be predictive for a subsequent psychosis (Tsuji et al., 2013). Vulnerability to psychosis is phenotypically expressed in the social domain relatively early and in progressively more specific ways, along the increasing performative demands intervening during the transition from childhood to adolescence. Such social dysfunction might not be simply due to psychological traits of shyness/behavioral inhibition, but rather expresses more pervasive intersubjective difficulties in grasping the implicit social rules of interaction with peers. Furthermore, over and above its immediate bio-psychosocial consequences, an early withdrawal from social situations and interactions, by reducing the exposure to social stimuli during a critical developmental period, may trigger a deafferentation-like reorganization in brain networks underlying social cognition; this phenomenon may fuel a vicious cycle of social withdrawal-progressive deafferentation from social stimuli as well as a facilitate the development of psychotic symptoms (Hoffman, 2007; Michael and Park, 2014). Other additional mechanisms, such as the reduced functional connectivity between dMPFC subsystems of the Default Mode Network associated with familial risk for schizophrenia, may impact on interpersonal functioning (Dodell-Feder et al., 2014) and contribute to the overall social dysfunction in schizophrenia spectrum disorder.

In adolescence, when the affective salience of peer-networks reaches its peak, this developmental dynamic in the social domain may manifest as a propensity to introvertive withdrawal and isolation sometimes accompanied by increased perceived negative attitude of others and heightened interpersonal sensitivity (Solakangas et al., 2012). Individuals incurring in a UHR state might experience distortions of intersubjective resonance, a sense of loss of spontaneity (i.e. naturalness) in the way of relating to and communicating with others, social hypo-hedonia, and increasing propensity to de-socializing and solipsistic modes of experiences (Raballo and Krueger, 2011). Therefore, in the premorbid and prodromal stages of psychosis (often coinciding with childhood and the transition to adolescence), core features of intersubjective vulnerability to schizophrenia start to surface. According to phenomenology, such vulnerability resides in the distortion of a basic dimension of intersubjective attunement, i.e. the normally tacit feeling one inhabits a shared lifeworld with others, the meaning of which is co-created and maintained by everyday forms of "participatory sensemaking", that include coordinating activities, sharing emotions, negotiating intentions, and bridging distinct perspectives into alignment and mutual understanding (Stanghellini and Ballerini, 2002).

From a developmental viewpoint, the progressive distortion of this basic dimension of intersubjective attunement may be triggered or amplified by a deafferentation-like process, ultimately reverberating in a compromised social functioning. Such socio-functional decline which is often already perceivable by parents and peers in prodromal and early clinical stages, in a subgroup of UHR individuals reaches a magnitude akin to the one reported in ASD. Therefore, the findings by D'Angelo et al. (2018), first, strengthen the importance of social/intersubjective functioning as primary symptomatic area in preclinical and clinical stages of SSD, especially in those individuals with an early phenotypic expression of risk, in childhood and adolescence; and, second, highlight the challenge of the differential diagnosis between SSD and high-functioning ASD, both of which might have and early symptomatic onset in infancy, with similar severity of social dysfunction. The challenge is represented not only by the symptomatic overlap in the social domain, but also in other symptomatic areas, as shown by the partial overlap between autistic traits and schizotypy (e.g. Ford et al., 2018) and the frequent presence of thought disturbances and psychotic symptoms in ASD (Cochran et al., 2013) (Fig. 1). In this perspective, Sprong et al. (2008), compared the clinical profiles of UHR adolescents and adolescents with Multiple Complex Developmental Disorder (a group defined by research criteria aimed to characterize children and adolescents with DSM-IV diagnosis of ASD-not otherwise specified, and presenting severe, early childhood-onset deficits in affect regulation, anxieties, disturbed social relationships, and thought disorder). Although the groups clearly differed in developmental trajectories and autism traits, they did not differ with regard to schizotypal traits and basic symptoms, as well as disorganized and general prodromal symptoms, with group differences limited to positive and negative prodromal symptoms. Furthermore, the majority of children with Multiple Complex Developmental Disorder met UHR criteria, suggesting

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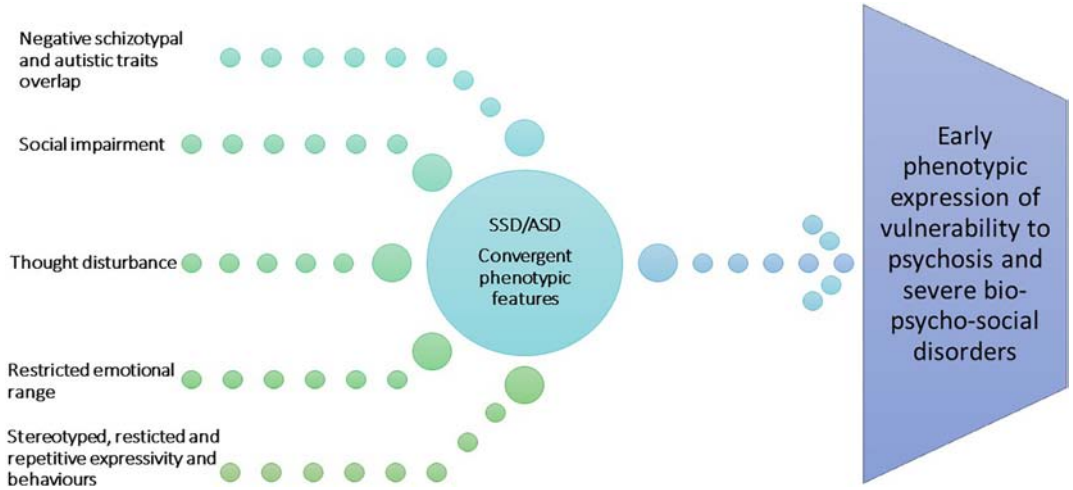

Fig. 1. Areas of potential phenotypic overlap between autistic spectrum disorders and schizophrenia spectrum disorders during developmental age.

that the phenotypical expressions of psychotic risk in children and adolescents may be the long-term result of different neurodevelopmental pathways.

In conclusions, social dysfunction represents and early phenotypic expression of the vulnerability to psychosis and clinicians should be aware that this dysfunction in developmental years partially overlaps with ASD.

Conflict of interest
None.

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