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Shared genetic influences among childhood shyness, social competences, and cortical responses to emotions



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ABSTRACT

Visual event-related potentials (ERPs) evoked by facial expressions are useful to map socioemotional responses among shy children and to predict transition into social phobia. We investigated the sources of covariation among childhood shyness, social competences, and ERPs to other children's happy, neutral, and angry expressions. Electrophysiological and twin analyses examined the phenotypic and etiological association among an index of childhood shyness, an index of social competences, and ERP responses to facial expressions in 200 twins (mean age = 9.23 years). Multivariate twin analyses showed that the covariation among shyness, social competences, and a composite of a frontal late negative component occurring around 200–400 ms in response to happy, neutral, and angry expressions could be entirely explained by shared genetic factors. A coherent causal structure links childhood shyness, social competences, and the cortical responses to

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facial emotions. A common genetic substrate can explain the inter-relatedness of individual differences for childhood shyness, social competences, and some associated electrophysiological responses to socioemotional signals.

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Introduction

A major purpose of translational developmental neuroscience is to understand the transactional processes at the basis of socioemotional functioning in both healthy and disordered populations (Wiggins & Monk, 2013). However, establishing clear and reciprocally coherent connections among the etiological, neurofunctional, and behavioral levels remains a daunting task for developmental psychopathologists (Battaglia, 2012; Battaglia et al., 2009; Rutter & Pickles, 2016). The encoding and identification of facial expressions of emotions (EoEs) is a popular tool to investigate socioemotional processes in healthy and clinical populations. The ability to identify and use EoEs as elements of social communication matures early in life, informs social exchanges in humans and primates, and is influenced by both genetic factors (Wilmer et al., 2010) and environmental factors (Moulson, Westerlund, Fox, Zeanah, & Nelson, 2009). The processing of emotional stimuli is a complex function involving multiple cortical and subcortical regions (Vuilleumier & Pourtois, 2007), including areas in the ventral temporal lobe such as the fusiform gyrus and amygdala (Holmes, Lit, Murphy, Gold, & Crawley, 2003; Ishai, Ungerleider, Martin, Schouten, & Haxby, 1999; Monk, 2008; Stein & Stein, 2008).

Reactivity to EoEs has been employed to study socioemotional responses in children with different degrees of social anxiety and their possible transition into clinical conditions such as social anxiety disorder (SAD) (Battaglia et al., 2012; Jarcho et al., 2013; Lau et al., 2012; Schwartz, Wright, Shin, Kagan, & Rauch, 2003). Whereas the constructs of childhood social anxiety, withdrawal, and shyness collectively map individual gradients of uneasiness in social contexts, SAD represents a proper clinical condition with a peak of onset during adolescence (Cartwright-Hatton, McNicol, & Doubleday, 2006; Kessler, Chiu, Demler, Merikangas, & Walters, 2005). These constructs have been employed by different research traditions of developmental psychopathology and developmental science (Gazelle & Rubin, 2010; Rubin, Coplan, & Bowker, 2009). Gazelle and Rubin (2010) compared a model focusing on the concept of behavioral inhibition (BI) and an approach based on shy temperament in relation to the affective behavioral profiles of anxious withdrawn children and their parents and of clinical SAD and concluded that multilevel interaction models may help to solve apparent inconsistencies and promote the integration of different constructs and approaches (Gazelle & Rubin, 2010). Among these diverse but related constructs, the temperamental approach to shyness has three attractive features. First, shyness can be defined in a relatively straightforward, reliable, operational manner as a set of behaviors that indicate discomfort in social interactions and avoidance of novel and uncertain situations (e.g., Kagan, Reznick, & Snidman, 1988). Second, it has received validation from biological investigations (e.g., Schwartz et al., 2003). Third, longitudinal studies show that it bears a developmental relationship with SAD (Biederman et al., 2001; Hirshfeld-Becker et al., 2007). For example, a retrospective analysis of the U.S. National Comorbidity Survey controlling for neuroticism, self-criticism, and perceptions of low maternal care showed that childhood shyness was specifically associated with lifetime SAD (Cox, MacPherson, & Enns, 2005). Longitudinal general population data further show that shyness at 10 or 11 years of age predicts SAD during young adulthood (Mason et al., 2004). Thus, although childhood shyness is not a pathological condition per se, and many children outgrow their shyness into good adaptation (Schwartz et al., 2003), there is an association between childhood shyness and later development of SAD.

From a cognitive perspective, SAD is characterized by information processing biases and higher sensitivity to social cues, including EoEs (Clark & McManus, 2002). Research (e.g., see Tillfors et al., 2001, for a review) on perceptually induced anxiety has shown altered patterns of cortical activation

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