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## Linking child temperament, physiology, and adult personality: Relations among retrospective behavioral inhibition, salivary cortisol, and shyness



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#### ABSTRACT

Shyness has been linked to several distinct behavioral antecedents and biological correlates across development, including early behavioral inhibition and neuroendocrine dysregulation. In the present study, we examined whether self-reported history of childhood behavioral inhibition, concurrent cortisol output, and sex affected shyness levels in adults. Results revealed that a history of childhood social behavioral inhibition predicted higher shyness among female adults with high levels of cortisol output. Among women with low cortisol levels, there was no relation between childhood social behavioral inhibition and shyness levels. These associations were not consistent when examining a history of nonsocial behavioral inhibition, or among adult males. These findings highlight the importance of differentiating social versus nonsocial behavioral inhibition when examining relations between childhood temperament and adult shyness. Further, these findings raise the possibility that neuroendocrine dysregulation may have a unique role in predicting and maintaining social behaviors such as shyness depending on sex and individual differences in temperament.

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#### 1. Introduction

Behavioral inhibition is an early emerging temperament that is characterized by fearfulness and wariness in response to novelty, including unfamiliar environments, objects, and/or people (Garcia-Coll, Kagan, & Reznick, 1984). There is a well-established link between this temperament and the emergence of distinct behavioral, psychological, and physiological correlates across development (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001). Most noteworthy, behaviorally inhibited children experience more socioemotional maladjustment, including social withdrawal and shyness in later childhood, as well as an increased risk for anxiety related disorders, particularly social anxiety, in adolescence and adulthood (e.g., Chronis-Tuscano et al., 2009; Hayward, Killen, Kraemer, & Taylor, 1998; Hirshfield-Becker et al., 2007; Kagan, Reznick, & Snidman, 1987; Pérez-Edgar & Fox, 2005).

Although a behaviorally inhibited temperament is a strong predictor for the development of social tendencies such as shyness, not all fearful children experience and maintain these social inhibitions across time (Fox et al., 2001). Inconsistent relations may indicate the presence of moderators linking childhood temperamental inhibition and later shyness. Therefore, it is important to examine multiple aspects of an

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individual that may interact with childhood temperament to predict shyness.

First, behavioral inhibition is a construct that is comprised of both social and nonsocial components, both of which are unique from each other (Dyson, Klein, Olino, Dougherty, & Durbin, 2011; Kochanska, 1991; Neal, Edelmann, & Glachan, 2002; Schofield, Coles, & Gibb, 2009). However, much research examining the association between behavioral inhibition and later social behavioral tendencies (e.g., shyness, social anxiety, sociability) has failed to differentiate the specificity of social versus nonsocial childhood behavioral inhibition in these relations. There is evidence to suggest that social fearfulness and inhibition in early life may have particularly strong links with the development of shyness and social anxiety given its strong social basis (Brooker, Kiel, & Buss, 2016; Chronis-Tuscano et al., 2009; Hayward et al., 1998; Kochanska & Radke-Yarrow, 1992; Mick & Telch, 1998; Schofield et al., 2009). This highlights the importance of considering different types of childhood behavioral inhibition (i.e., social versus nonsocial) when examining the association with later shyness.

Second, beyond temperamental vulnerabilities, there are several physiological systems, such as the hypothalamic pituitary adrenal (HPA) axis, that have been implicated in fear responses, including those with a social basis (e.g., shyness). The end product of the HPA axis is cortisol, which has been noted as playing an important role in the maintenance of fear responses (Schulkin, Morgan, & Rosen, 2005). For example, behaviorally inhibited children have been shown to

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display high cortisol levels under both stressed (e.g. Kagan et al., 1987) and non-stressed (e.g. Schmidt et al., 1997) conditions, which may be reflective of a hyperactive HPA axis. Increased HPA axis activity may be an underlying biobehavioral process predisposing individuals to avoid and withdraw from normative social contexts in everyday life, whereas lower activation of the HPA axis may reflect the tendency to approach social encounters. However, the majority of previous research has examined cortisol as an outcome or correlate of a fearful and shy temperament (e.g., Beaton et al., 2006; Kagan et al., 1987; Schmidt, Santesso, Schulkin, & Segalowitz, 2007; Schmidt et al., 1999), and surprisingly less literature has examined whether individual differences in HPA activity may act a moderating factor on the relation between temperament and personality development across time. As a result, the role of cortisol in moderating human anxiety and related phenomena such as shyness, particularly beyond childhood, is not clearly understood. By treating cortisol as a moderator, we may be able to better understand its role in shyness as a potential mechanism underlying links between early temperament and later shyness, as well as under what conditions temperamental biases are most likely to affect levels of shyness. Importantly, examination of the interaction of childhood temperament and current biological influences may provide a more complete picture of the developmental processes underlying adult shyness.

Finally, there is evidence to suggest that the relation between temperament and socioemotional functioning in later life may be different in males and females. As a direct association, behavioral inhibition in toddlers and social anxiety in adolescence has been shown to be stronger in females than males (Schwartz, Snidman, & Kagan, 1999), and shyness has been more strongly linked to social anxiety in females than males in both childhood and adolescence (Hayward et al., 2008; Tsui, Lahat, & Schmidt, 2017). Although limited, some research has examined how individual differences in physiological regulation may influence these associations in males and females in samples of children. For example, high cortisol in preschool-aged children predicted withdrawal behaviors in those who had a negative reactive temperament in infancy, but was stronger in boys than girls (Pérez-Edgar, Schmidt, Henderson, Schulkin, & Fox, 2008). Beyond the influence of cortisol output, other biological markers associated with shyness have differentially affected the association between temperament and social behavior across sexes, with right frontal EEG asymmetry having a stronger influence in preschool aged boys than girls (Henderson, Fox, & Rubin, 2001), and respiratory sinus arrhythmia (RSA) having a stronger influence in preschoolaged girls than boys (Morales, Beekman, Blandon, Stifter, & Buss, 2015). Importantly, this demonstrates that individual differences in the physiological systems involved in social behavior such as shyness may exert different effects in male and female children and the extent to which sex differences emerge is not consistent. As well, these sex differences have also not been extensively examined into later development (e.g., adulthood).

#### 1.1. Research aims and hypotheses

We addressed at least four gaps in the current literature by examining behavioral and biological factors hypothesized to be implicated in adult shyness: We examined 1) the specificity of social versus nonsocial self-reported childhood behavioral inhibition, 2) if hyperactivity of the adult HPA axis interacted with temperamental vulnerabilities, 3) if sex differences existed in these relations, and 4) the association of these factors on shyness in a later developmental period that has been previously unaddressed. In doing so, this study helped delineate the specificity of social and nonsocial components of early fear, as well as the interactive effects of this temperament with individual differences in biological functioning and sex. Notwithstanding the cross-sectional nature of the study, this investigation is an important first step in understanding multiple factors of an individual's past and current development that may influence shyness in adulthood.

We hypothesized that cortisol output would moderate the association between retrospective childhood *social* behavioral inhibition and adult shyness. Specifically, we predicted that high cortisol levels combined with a history of high social behavioral inhibition would be linked to the highest levels of adult shyness. Given that previous literature has found inconsistent relations in examining how physiological processes may interact with temperament to predict social behavior in males and females, no specific predictions were made with respect to the effect of sex.

#### 2. Method

#### 2.1. Participants

A convenience sample of healthy adults was recruited from Centralwest Ontario as part of a larger study examining behavioral and physiological correlates of socioemotional functioning in adulthood. A total of 81 adults (44 females), primarily Caucasian (81%), participated in this study ( $M_{\rm age}=30.21, {\rm S.D.}=11.23$ ). Participants were recruited from McMaster University, as well as through advertisements in the community and online.

#### 2.2. Procedures

After a complete description of the study was provided, written informed consent was obtained from the participants. Upon acclimation to the laboratory, participants provided their first saliva sample. Participants then completed a series of self-report questionnaires pertaining to personality dimensions and mental health, and then provided their second saliva sample. The participants then completed computer tasks that comprised a face processing task and ERP measures. These measures were collected as part of the larger study and are presented elsewhere (Jetha, Zheng, Schmidt, & Segalowitz, 2012). Finally, participants provided their third saliva sample prior to leaving the laboratory. All procedures were completed at McMaster University and approved by the University's Research Ethics Board.

#### 2.3. Measures

#### 2.3.1. Retrospective childhood behavioral inhibition

History of childhood behavioral inhibition was assessed using the Retrospective Self Report of Inhibition (RSRI; Reznick, Hegeman, Kaufman, Woods, & Jacobs, 1992). The RSRI is a 30-item self-report questionnaire assessing a broad range of childhood behaviors associated with behavioral inhibition. The items ask respondents to think about how they felt when they were in early elementary school and to rate items using a five-point Likert-scale. From the total scale, fearfulness related to social (12 items;  $\alpha = 0.82$ ) and non-social contexts (12 items;  $\alpha = 0.77$ ) can be derived to create subscales related to these two dimensions of behavioral inhibition. An example item from the social behavioral inhibition subscale includes "Did you enjoy meeting new children your age?" and an example of an item from the nonsocial behavioral inhibition subscale includes "Were you scared of the dark?". The RSRI has demonstrated good postdictive validity when self-reports are corroborated with objective accounts of inhibition in childhood, indicating that it is a useful and valid instrument to assess behavioral inhibition in childhood, particularly when prospective data are not available (Reznick et al., 1992). The RSRI (including the social and non-social subscales) has been employed extensively beyond the original validation study (Reznick et al., 1992; e.g., Coles, Schofield, & Pietrefesa, 2006; Hayward et al., 1998; Mick & Telch, 1998; Neal et al., 2002; Schmidt & Fox, 1995; Van Ameringen, Mancini, & Oakman, 1998; Schofield et al., 2009). In support of the separability of the two components of BI, the social and non-social subscales demonstrate low correlation in previous studies (Neal et al., 2002; Schofield et al., 2009).

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