

What gene–environment interactions can tell us about social competence in typical and atypical populations

Grace Iarocci^{*}, Jodi Yager, Theo Elfers

Department of Psychology, Simon Fraser University, Burnaby, BC, Canada

Accepted 3 January 2007

Available online 12 July 2007

Abstract

Social competence is a complex human behaviour that is likely to involve a system of genes that interacts with a myriad of environmental risk and protective factors. The search for its genetic and environmental origins and influences is equally complex and will require a multidimensional conceptualization and multiple methods and levels of analysis. Behavioural genetic research can begin to address the fundamental yet complex question of how children develop social competence by uncovering the various influences on social development and disentangling variance due to multiple genes, environments and experiences. In this paper, we review the current status of research on sociability, face recognition, emotion recognition, and theory of mind (TOM)—well defined and measured constructs that are likely to be useful indices for detecting genetic and environmental influences on social competence. We also propose specific milestones as indices of further progress in the field: the development of an operational definition of the construct of social competence, the identification of social endophenotypes—psychological processes that are validly and reliably measured components of social competence, and improving specificity and homogeneity with regard to social endophenotypes within a population of study by employing ‘extreme social phenotypes’. These efforts will lead to a better understanding of the specific contributions to the normal variation of social competence in the general population as well as to atypical social development.

© 2007 Elsevier Inc. All rights reserved.

Keywords: Social competence; Behavioural genetics; Endophenotypes; Extreme phenotypes

1. Introduction

The fundamental yet complex question of how children develop social competence requires a multidisciplinary approach that considers multiple variables, methods and levels of analysis. Social scientists are at an important juncture whereby they can capitalize on key advances in genetics, neuroscience, psychology and behavioral ecology in their quest to conceptualize and study complex and multidimensional abilities such as social competence. For example, advances in mapping the human genome promises unprecedented possibilities for discovering normal and aberrant genes and associated proteins involved in brain tissue development. Extraordinary innovations in measuring the structural and functional properties of the brain

have contributed to mapping connections between psychological processes and their neurophysiological correlates. Developments in constructivist thought (e.g., Bruner, 1988, 1996; Piaget, 1929) have led to refinements in conceptualizing the mind and amassed substantial empirical evidence that humans are active interpreters of their environment. Social constructivist and contextualist revelations (e.g., Vygotsky, 1934; Wertsch, 1999) that the essentially human aspects of mind (i.e., language and higher order thought) develop through human relationships as children interact with people, objects, and events in socio-cultural contexts have highlighted the dynamic and transactional nature of typical and atypical development (Chapman, 1991; Sameroff & Chandler, 1975). Human relationships are further shaped by prevailing beliefs, values, and social policies within specific social ecologies (e.g., neighborhoods, communities, schools) (Bronfenbrenner, 1979, 2000). Behavioural genetic research exemplifies the type

^{*} Corresponding author.

E-mail address: g iarocci@s fu. ca (G. Iarocci).

of study that can begin to uncover these various influences on development and disentangle variance due to multiple genes, environments and experiences.

In this paper, we propose that a behavioural genetic approach that considers the dynamic interplay between multiple genes and environments is needed to address the question of how children develop social competence. We begin by providing a conceptual framework for social competence that incorporates multiple processes that are hierarchically organized and contextually embedded. The polygenic model of inheritance is proposed to account for the multiple genetic and environmental factors that synergistically contribute to the development of social competence. Within this framework, polygenes associated with social competence would be influenced significantly by environmental factors and contribute to only a small proportion of the overall variance in this behaviour in the normal population. A developmental psychopathology perspective is needed to increase the discriminative power of the phenotype of social competence. One strategy involves identifying potential endophenotypes that refine the definition of the construct of social competence. We review genetic studies on sociability, face recognition, emotion recognition, and theory of mind (TOM)—endophenotypes that are well defined and measured and likely to be useful indices for detecting genetic and environmental influences on social competence. Another strategy focuses on defining the group of study more precisely. We discuss the benefits of employing ‘extreme social phenotypes’ such as the unusual sociability observed among individuals with William’s syndrome to flesh out the specific contribution of key processes such as sociability in the normal variation of social competence in the general population.

2. Defining the construct of social competence

Social competence is conceptualized as an emergent property of the dynamic interplay between characteristics of individuals and their environments (Guralnick, 1996; Rose-Krasnor, 1997; Sameroff, Seifer, & Bartko, 1997; Wyman, Sandler, Wolchik, & Nelson, 2000). In Fig. 1 we depict a hypothetical model of social competence that

focuses on specific cognitive and social abilities. Within this framework, social competence is thought to involve the active and skillful coordination of multiple processes and resources available to the child to meet social demands and achieve social goals in a particular type of social interaction (e.g., parent–child, peer relations) and within a specific context (e.g., home, school).

Basic sensory/perceptual, cognitive, and emotion processes (i.e., attention, memory, motivation) are fundamental to the development of higher-order social abilities. Sociability and the abilities to recognize faces, emotions and understand that others’ thoughts and feelings are different from one’s own are only a few of the higher-level social abilities involved in the development of social competence. Each of these abilities is a necessary building block of social competence. However, these abilities are not sufficient for socially competent behaviour to emerge. A child must be able to coordinate their social abilities along with available contextual resources to meet developmental goals in an adaptive way. Higher-order coordination of social abilities is a critical component of social competence because it permits the child to appropriately match their social goals with the demands of the social context (Bost, Vaughn, Washington, Cielinski, & Bradbard, 1998; Guralnick, 2005; Rose-Krasnor, 1997). Accordingly, social competence entails the development of appropriate strategic processes (i.e., tools) and resources to tackle the social demands of a particular task in given context. Social learning through mediation and scaffolding experiences will likely influence the development of the strategies or ‘tools’ that are particularly useful or meaningful within a particular socio-cultural context. Thus, the beliefs and practices of parents and other relevant agents of social mediation will play a significant role in shaping social competence.

Social competence is both a developmental phenomenon that can be measured over the course of a child’s development (i.e., ontogenesis) as well as a characteristic of a particular social encounter where the time scale is in the order of seconds/minutes (i.e., microgenesis). Thus, continuities and discontinuities in the development of social competence are expected as children are better able to coordinate abilities and take advantage of resources with increasing age but may be less competent at certain developmental stages or in specific social contexts. With any individual child, there is likely to be variation in social competence overtime and across contexts. However, within the general population, some children will show more consistently adaptive or maladaptive social behaviour in various social situations and over the course of their development.

3. Multiple genes and environments for social competence

Nature (genetics) and nurture (environments) effects on development are often described as additive and emanating from separate and independent influences despite considerable empirical evidence that the process is best characterized as dynamic, synergistic and interdependent (Plomin

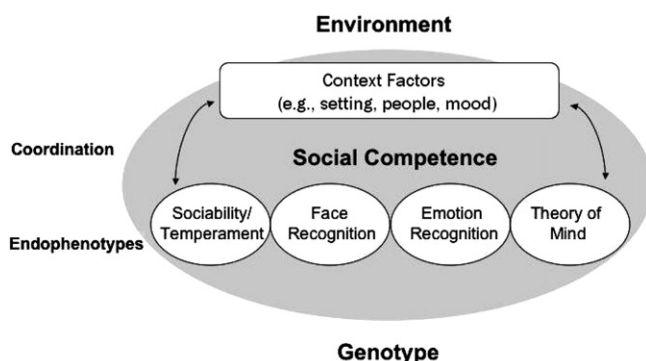


Fig. 1. A hypothetical model of social competence.

et al., 1994; Plomin & Rutter, 1998; Rutter, Simonoff, & Plomin, 1996). The transactional and dynamic nature of the construct of social competence further highlights the inadequacy of deterministic, dichotomized and static conceptualizations of gene–environment connections. (Rose, 2001). More nuanced developmental conceptualizations such as reaction range, affordance range, and reaction surface have been proffered to capture the transactional phenomena (Gottesman, 1963; Scarr-Salapatek, 1973). Within this framework, complex psychological abilities are considered both structured and malleable with different potential phenotypes as probable outcomes from the same genotype in response to varying environmental supports (Fischer, Rotenberg, Bullock, & Raya, 1993; Scarr-Salapatek, 1973). Accordingly, affordance range refers to the outcome of a specific environmental input over the range of all possible genotypes (Goldsmith & Gottesman, 1996). Hypothetically, the ranges of genotypes and possible environments could be plotted against each other in a virtual three-dimensional space with the resulting phenotypic outcome as z-axis. The surface that results in the plot may be used to visualize the interaction between the genotype and the environment. This surface is referred to as the reaction surface (Goldsmith, Gottesman, & Lemery, 1997) and is represented for the phenotype of social competence in Fig. 2. In this figure, genotypes vary with regard to their susceptibility to environmental conditions and environments vary with regard to how facilitative they are to genotypes. Thus, adaptive social competence is neither fully dependent on the environment or the individual but rather, the result of an optimal match between environments and the genes.

Developmental concepts such as reaction range, affordance range, and reaction surface encourage transactional thinking about gene–environment connections but can also motivate behavioural genetic designs that capture multivariate relations overtime. One of the many challenges of

translating multivariate and dynamic constructs into measurable units is that the theories are not readily captured by the methods. This idea is particularly evident in the empirical methods used to test behavioral genetic theories as they frequently artificially partition genetic and environmental effects (Wahlsten, 1994). However, using methods to decompose the sources of genetic and environmental variance of a measured behaviour and analyzing the variance statistically should not be interpreted to mean that these influences should be considered discrete and static conceptually. Further, Gottesman and Hanson (2005) point out that all interactions between a human's genes and the environment are occurring within the dimension of time. Thus, the interaction between a certain gene and a particular environment is likely to change dynamically over time, and demands methods that also take development into consideration.

4. Polygenes: Multiple gene systems

Complex behaviours such as social competence involve numerous genetic and environmental factors that work within a system. Genetic contributions may include a host of genes that co-act in an additive manner or interact with each other (Eley, 2001; Petrill, 2002; Skuse, 2001). In multiple gene-systems, genes do not act alone but rather each of the polygenes operates in conjunction with other relevant genes and environments to contribute to the phenotype (Plomin, Asbury, & Dunn, 2001). Thus, the genetic and environmental influences must be considered within a systemic framework that accounts for the co-action and/or interaction of the polygenes and multiple environmental factors (at various levels of analysis) that are hypothesized to be involved in the phenotypic expression of interest (Petrill, 2002).

Each of the polygenes is called a quantitative trait locus (QTL) and is comprised of a paternally and maternally derived allele (Plomin et al., 1994). In the general population, genes that are not closely located on the same chromosome recombine during the natural process of meiosis in the ovaries and testes when gametes are produced and chromosomes exchange parts (Plomin, 1999). The probability of recombination between two genes on the same chromosome is a function of the distance between them. Recombinations break up alleles for a marker (i.e., specific gene of interest) as well as the QTLs that happen to be close together on the same chromosome, unless the marker and the QTL are in close proximity on the same gene (Plomin & Craig, 2001). Thus, QTLs are genes involved in complex abilities that are normally distributed in the general population and may account for individual differences in behaviour. For example, polygenes are thought to contribute to the development of complex abilities such as IQ that are quantifiable and continuously distributed in the population (Plomin, 2001). At the extreme end of the distribution QTLs may be implicated in a cluster of behaviours that constitute a psychological disorder (Eley, 2001).

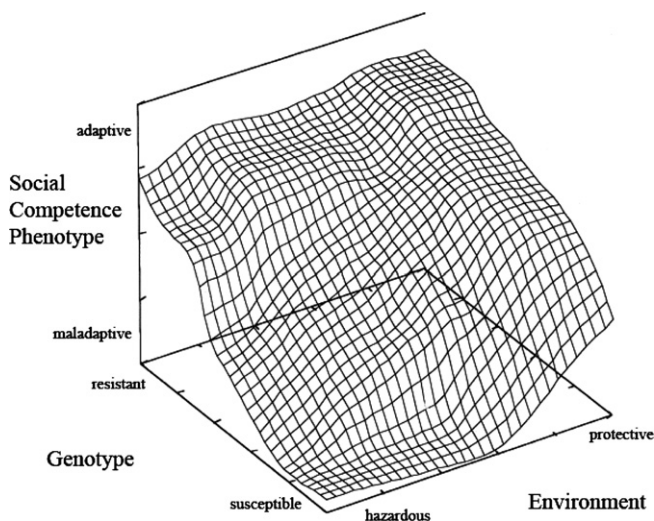


Fig. 2. A hypothetical depiction of reaction surface for social competence. Adapted from Goldsmith et al. (1997).

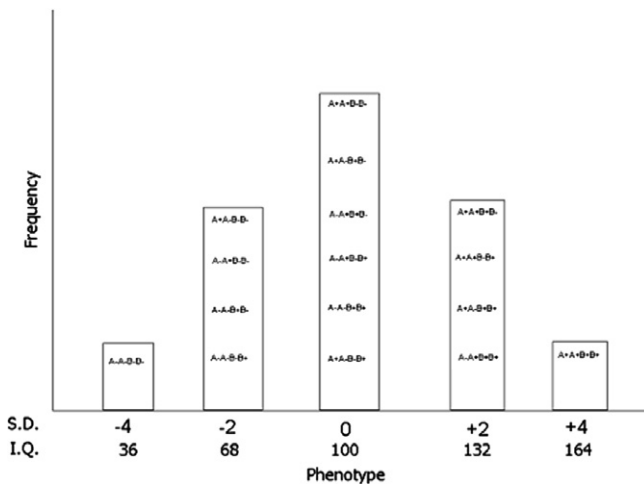


Fig. 3. Gottesman's five-gene model of the Normal distribution of IQ. The hypothetical case of two genes contributing to IQ, each including a maternal and a paternal allele that is either enhancing or nonenhancing with regard to IQ.

QTLs are continuously distributed in the population and contribute in an additive and probabilistic fashion to developmental risk (Plomin, 2001). For example, IQ, much like other quantitative traits such as height or weight, has a roughly Normal distribution. Gottesman (1963) illustrated the Normal distribution of IQ scores using a hypothetical and simplified five-gene model (see Fig. 3). He reasoned that if two genes influenced IQ and each gene has two alleles (one of maternal and the other of paternal origin), an offspring may inherit IQ enhancing alleles A+ B+ or IQ nonenhancing alleles A- B- from each parent. Thus, 16 permutations of alleles are possible and, if expressed completely, five categories of genotype for IQ would be produced. The distribution of IQ would be roughly Normal as one offspring inherits all IQ nonenhancing alleles, four offspring inherit mostly nonenhancing alleles, six offspring inherit an equal share of IQ enhancing and nonenhancing alleles, four offspring inherit mostly enhancing alleles and one offspring inherits all IQ enhancing alleles (Gottesman, 1963).

5. Environments: Multiple levels and experiences

According to the social bioecological model of development, the child is embedded within various socio-cultural systems that interact to either support or hinder his development (Bronfenbrenner, 1979, 1999, 2000). These dynamic systems are conceptualized as different spheres of influence and include those that have a distal (i.e., indirect) effect and those that have a proximal (i.e., direct) effect on the individual (Cicchetti & Toth, 1997). Indirect influences are thought to emanate from macrosystems or global-political contexts, mesosystems, which encompass the patterns, beliefs and values of the culture in which the child exists, and the exosystems which comprise the

various formal and informal social structures in the child's environment, including the neighbourhood, schools, and local government policies on education and health (Bronfenbrenner, 1979; Cicchetti & Toth, 1997). Direct influences include the child's interaction with significant persons or events in their lives (e.g., sensory and perceptual input, parenting customs, sibling and peer relations and teaching practices). Risk and protective factors may be present in each of the systems and may operate through distal and/or proximal effects that influence the course of the child's development in adaptive or maladaptive ways (Bronfenbrenner & Ceci, 1994).

For example, in infancy, social competence may be evident within the parent-child relationship as consistency in engaging with, and responding to the other establishes a secure and stable attachment that is integral to the infant's very survival. Later in development as the child is increasingly able to control their own behaviour and choose environments, social competence appears to transform into something more akin to a personal characteristic of the child (Bronfenbrenner, 1999; Sroufe & Jacobvitz, 1989). However, variability in the availability of social resources and in the quality of the parent-child relationship jointly influence a child's ability to generate and coordinate flexible, adaptive responses to demands and capitalize on social opportunities in the environment (Waters & Sroufe, 1983). Thus, contextual factors (at various levels of analysis) such as social opportunities, scaffolding practices, and cultural beliefs are also essential components of the behavioural expression of social competence (Fischer et al., 1993; Hauser-Kram, Warfield, & Shonkoff, 1999; Landry, Garner, & Pirie, 1994).

Traditionally, individual differences in behaviour are parcelled into three main sources of variance: inherited genes, shared family environment and nonshared environment (see Plomin, Fulker, & Corley, 1997). Shared environment refers to experiences that family members share (e.g., household income). Nonshared environment refers to events or experiences that family members do not share (e.g., school and peers). The variance on a measured ability due to shared family influences may be differentiated from variance due to nonshared influences that derive from extra-familial experiences. However, these distinctions are somewhat artificial. Goldsmith and colleagues (1997) suggest that environmental influences even when they are shared are likely to vary in the extent that they influence any specific member of a family. Moreover, defining 'shared' as pertaining to familial variables might not be appropriate in all cases (Goldsmith et al., 1997). For example, the authors note that some environmental influences that are shared among siblings are likely to occur outside the family (e.g., the same school resources).

A more differentiated conceptualization of 'environment' is especially pertinent to the study of social competence. Social activity in many situations is not a means to an end but an end in itself, and the products of this activity are co-constructed by two or more individuals with unique

thoughts, feelings and previous experiences. The implication is that human interactions (particularly when they entail social goals) are both expressions of underlying psychological processes and co-constructed experiences of subjective (self-other) reflections. Thus, the same objective event (or environmental conditions) may be interpreted quite differently even among genetically similar siblings (Plomin et al., 2001; Plomin & Daniels, 1987). Experience oriented approaches to the study of environmental influences are needed to focus on process variables, including the unique experiences of the individual in specific relationships (e.g., sibling, parent–child and peers) and social phenomenon (e.g., social comparison) that affect the individual experience of those relationships (McGuire, 2002). A within family design is one method that has been used to capture these subjective variables (Lemery & Goldsmith, 2002). The goal is to track children's differential experiences in the family and their role in their development (Turkheimer & Waldron, 2000). Within this approach, the researcher is able to capture both individual differences within the family (e.g., the siblings' differential experiences, unique experiences of the sibling relationship and different peer and teacher influences) as well as across families (e.g., comparing siblings across pairs of different degrees of genetic relatedness (McGuire, 2002).

An alternative to categorizing environmental influences would be to separate environment into vertical and horizontal influences, the former referring to factors handed down from one generation to the next and the latter referring to the influences of peers or same-aged siblings (Bodmer & Cavalli-Sforza, 1976; Goldsmith et al., 1997). Goldsmith and colleagues propose that a vertical transfer of environmental influences can be either directly phenotypically transmitted via parental role modeling or more indirectly transmitted via the socio-cultural environment. Broader environmental influences such as the cultural background are thought to have a similar effect on both parents and children. Although such a distinction might be generally useful (Goldsmith et al., 1997), we would caution against a linear conceptualization of environmental influences. Behavior cannot be directly transmitted to the next generation but is likely to be transformed via accommodation by the individual (Piaget, 1960).

6. Perspectives from psychopathology on how to narrow the search for multiple genes and environments

The construct of social competence is conceptually rich, however, it encompasses a host of genetic, environmental and experiential factors that are not likely to be easily disentangled and isolated for experimental study. Empirical challenges include a lack of consensus on the operational definition of social competence, no psychometrically sound measures of the construct and significant discontinuity across measurement contexts (see Topping, Bremner, & Holmes, 2000). The search for polygenes associated with the expression of social competence through behaviour

genetic designs wherein gene markers are linked to a complex behaviour or disorder would be futile since the phenotype of the hypothesized genetic link or association is not validly or reliably measured (Eley, 2001).

Clues about how to circumvent the problem of specificity and discriminative capacity of social competence are found in the literature on psychopathology. Psychological disorders are defined as a set of behavioural phenomena that co-occur and stem from a common underlying cause(s). The phenotypic outcome (usually defined in terms of a behavioural syndrome) is thought to represent the endpoint of pathological pathways of brain development that originates from the co-action of polygenic and environmental risk and protective factors (Heath, Eaves, & Martin, 1998). In certain cases, syndromes may reflect a set or subset of behaviours that fall at the extreme end of the Normal distribution in the general population (Plomin, 1999). Thus, atypical behaviour of this type would be due to quantitative (as opposed to qualitative) differences in genetic processes. Genetic processes that contribute to variation in abilities within the general population will in some (albeit few) cases also contribute to extreme abilities. Extreme IQ performance within the Normal population is the most compelling example of an extreme phenotype that nonetheless is part of the normal variation in IQ within the general population (Plomin, 1999).

Similarly, social competence, although not yet quantifiable, is thought to involve the coordination of a set of key abilities that are likely to vary within the general population. The assumption is that the genetic processes that contribute to social abilities that underlie normal social competence would also be implicated in exceptionally high or low social competence. In order to improve the odds of detecting the key genetic processes involved in social competence, two methodological refinements are needed. First, more precise, quantifiable and reliable measure(s) of the phenotype would improve discriminative capacity. Second, a homogeneous group of individuals (usually at the high or low end of the Normal distribution) with regard to genetic endowment for that particular phenotype would increase the power of detecting polygenes with small effect sizes. However, low social competence in certain extreme groups (e.g., people with autism) may have a very different etiology than low social competence found in the Normal population (Nijhout, 2003).

7. Increasing the discriminative power of the phenotype

7.1. Endophenotypes

In an effort to narrow the search for genetic susceptibility to mental disorder, Gottesman and Shields (1972a, 1972b, 1973a, 1973b) proposed the concept of endophenotypes. Endophenotypes were originally defined as “internal” biological indices of susceptibility and later broadened to “simpler clues to the genetics underpinning psychiatric diseases than the disease syndrome itself” (Got-

tesman & Gould, 2003). Endophenotypes are considered intermediate pathways in the developmental course that leads to a behavioural syndrome. Any fundamental process (e.g., neuroanatomical, neurochemical, neurophysiological, neuropsychological) that is theoretically and/or empirically linked to the phenotype of interest would be a candidate endophenotype. One notable example is the detection of an association between a directly measurable protein marker (apolipoprotein E) and cognitive decline due to Alzheimer's disease (Corder et al., 1993).

With regard to social ability, a few promising candidates for 'social endophenotypes' are sociability and social-cognitive processes such as one's ability to recognize faces and emotions and understand others' thoughts and intentions. Our review focuses on these abilities because they are hypothesized to be key building blocks of social competence. These processes are relatively well defined, have established measures, and have been studied empirically over time and across contexts. The goal would be to evaluate whether any or all of these constructs could be considered intermediate psychological processes that are more proximally related to the polygenes of interest than the higher-order distal phenotype of social competence (Glanon, 2003; Skuse, 2001). If these processes are potentially relevant 'social endophenotypes' they should show stronger and less complex genetic and environmental influences than the more complex construct of social competence, provided that the researcher uses appropriate behavioural genetic methods designed to detect these influences. The review of studies on candidate 'social endophenotypes' is prefaced by a description of the three main behavioural genetic methodologies (i.e., family/twin design, linkage and association) typically employed in genetic studies.

7.2. *Methods for detecting genetic and environmental influences*

Family/twin design and linkage and association methods attempt to increase the odds of detecting genetic and environmental influences on the variance in a measured phenotype (Eley, 2001). However, they each provide different types of information and not all are appropriate to study the genetic basis of complex behaviours in the Normal population.

7.2.1. *Family and twin studies*

Twin and adoption studies help to tease apart genetic and environmental influences by comparing monozygotic (MZ) twin pairs who share all of their genes with dizygotic (DZ) twin pairs who share only half their genes. In addition, these twin pairs are often compared with regard to whether they were reared by their genetically related caregivers (i.e., biological parents) or adoptive parents. Heritability estimates and concordance rates are then used to infer the role of heredity in the development of complex behaviours. For example, if heredity is involved in a particular behaviour then researchers would expect to see MZ

twins who share all of their genes to be more similar on a measured phenotype than DZ twins who share only some of their genes. Adopted children's resemblance to biological parents' phenotype is further support for heredity. Conversely, resemblance to adoptive parents' phenotype suggests a role for environmental influences. Heritability is the proportion of phenotypic variation in a population that is due to genetic variation. Heritability analyses estimate the relative importance of variation due to genetic and/or environmental factors.

A second measure often used to infer the role of heredity is the concordance rate. Researchers typically employ concordance rates to study the percentage of incidences in which both twins are diagnosed with a disorder or behaviour when it has been found to be present in one twin. Thus, high concordance rates indicate that the disorder or behaviour is highly heritable. For a thorough review of the role of heredity in behaviour genetics see Plomin and McClearn (1993).

Family and twin designs are useful to uncover whether certain phenotypes are heritable or run in families (Broman, Nichols, & Kennedy, 1975; Nichols, 1984; Plomin, 1994). However, these studies provide inferences on heritability rather than direct evidence of genetic etiology (Plomin & Colledge, 2001; Plomin et al., 1994). For example, the methodology used to determine concordance rates in twins would artificially dichotomize social ability (low versus high) that would be more appropriately conceptualized on a continuous scale (Plomin, 1999). Therefore, these techniques are not amenable to explore nonpathological inheritance of low or high social ability. The study of social competence requires a quantitative estimate of genetic etiology that could account for the lower and higher end of the normal variation in social ability.

7.2.2. *Linkage*

Methods such as linkage analysis are typically used to track disorders in genetically related individuals (i.e., families) and require a precise definition of the phenotype so that the characteristic of interest can be identified in the population studied. Linkage methods use the known locations of genes as markers for the hypothesized target gene. For example, if it is thought that the target gene is on a particular chromosome, a known gene on that chromosome (which is suspected to be related to the target gene) is selected as a marker. If the target gene is physically close to the marker gene, then the probability of a disorder and the marker genes being transmitted together from parent to offspring is high because they are less likely to be separated during meiosis. The likelihood that marker genes will be transmitted together based on the distance between them is computed and tracked in families. Two genes are considered 'linked' if they are transmitted together as expected. Linkage studies are not sufficiently powerful to detect QTLs of small effect size that are typically involved in normal variation of complex abilities such as social competence but may be useful in detecting genes associated

with disorders that affect social processes and run in families (e.g., autism).

7.2.3. Association

Association studies are better able to test whether the polymorphism (specific form of the gene of interest) is present in individuals who are more or less affected by the behaviour of interest (e.g., low or high face recognition ability). Association refers to covariation between allelic variation in a marker gene and phenotypic variation in the population. This pattern occurs when the molecular genetic marker, restriction fragment length polymorphism (RFLP), is located very close to the gene of interest, and therefore is almost never separated during meiotic crossing over of chromosomes. The advantages of association methods are that they are equally appropriate for a continuous dimension and a dichotomous disorder and allow the use of large samples of unrelated individuals, which can be used to provide sufficient power, to detect associations that account for a small amount of variance. However, success in identifying associations between a polymorphism and a complex behaviour is a function of the researcher's ability to precisely define the phenotype.

8. Current status of the genetic study on 'social endophenotypes'

8.1. Sociability/temperament

Family studies on sociability provide clues to uncovering genetic and environmental sources of influence on the development of social competence that may be shared by family members. For example, sociability is commonly studied as a core component of the larger construct of introversion-extraversion - a personality dimension encompassing social interest, positive emotionality, and reward seeking/sensitivity (Daniels & Plomin, 1985; Olino, Klein, Durbin, Hayden, & Buckley, 2005). Twin studies of sociability and extraversion have consistently yielded heritability estimates greater than 0.50 (Eid, Riemann, Angleitner, & Borkenau, 2003a, 2003b; Floderus-Myrhed, Pedersen, & Rasmuson, 1980; Loehlin, McCrae, Costa, & John, 1998; Scarr-Salapatek, 1973), suggesting that the preference for social activity may be, in part, inherited.

Similarly, low sociability or shyness expressed behaviourally as inhibition or fearful responses to strangers appears to be highly heritable. For example, in nonadoptive families, where both genetic and environmental influences are shared, parent ratings of infant shyness were related to mothers' self-reports of shyness, low sociability and introversion (Daniels & Plomin, 1985). Furthermore, genetic influences were highlighted by significant correlations between the shyness of adopted-away infants and the shyness/low sociability of their biological mothers. More detailed investigations of exposure to novel social situations, the nature of parent-child interactions, and the warmth/responsiveness of parents would be needed to

more precisely qualify the family influences on shyness in children (Plomin & Daniels, 1987).

Debilitating anxiety about social situations as observed in social anxiety disorder was also found to be moderately heritable (i.e., heritability estimates around 0.30) based on a review of the literature (Knowles, Mannuzza, & Fyer, 1995). Similarly, for cognitive components of the disorder such as irrational social fears (e.g., negative evaluation fears) heritability estimates ranged from 0.20 to 0.50 (Phillips, Fulker, & Rose, 1987; Rose & Ditto, 1983; Stein, Jang, & Livesley, 2002). However, these findings must be interpreted with caution as the studies relied exclusively upon self-report measures of fear and included small sample sizes.

In an effort to refine the endophenotype and improve the search for polygenes associated with sociability, social inhibition and introversion-extraversion, researchers are guided by hypotheses regarding the neurochemical systems (e.g., catecholamines, dopamine) believed to underlie the expression of these behaviours. Genes that have previously been identified as playing a role in neurotransmission are then tested for associations with sociability, social inhibition or introversion-extraversion. For example, based on previous associations between the serotonin transporter gene and anxiety (Cloninger, Adolfsson, & Svrakic, 1996) and the dopamine D4 receptor gene and novelty seeking in adults (Cloninger et al., 1996), Schmidt, Fox, Rubin, Hu, and Hamer (2002) hypothesized that in young children, the DRD4 gene would be associated with bold/exuberant behaviour and the serotonin transporter gene would be associated with shy/socially withdrawn behaviour. They found that children with long as opposed to short allelic repeats of the DRD4 receptor gene scored significantly higher on the Child Behaviour Checklist (Achenbach & Edelbrock, 1981) at age 4 years. However, no association was found between the serotonin transporter gene and shy behaviour in 4 year olds (Schmidt et al., 2002). The long allelic repeats of the DRD4 receptor gene was also associated with attention problems in the same group of children and suggests that it may be involved in sustaining attention to novel stimuli in typical and children at risk for attention disorders (Auerbach, Benjamin, Faroy, Geller, & Ebstein, 2001; Schmidt, Fox, Perez-Edgar, Hu, & Hamer, 2001). Thus, attentional processing and emotional reactivity to novel stimuli may underlie temperamental differences in infancy and predict social responsiveness later in development.

In one study where preschoolers were subjected to a socially demanding self-presentation task, researchers found that shy preschoolers, as compared to their non-shy peers, exhibited a significantly greater increase in heart rate, and a greater increase in right but not left EEG activation (Schmidt, Fox, Schulkin, & Gold, 1999). In addition, the children who were classified as low in social competence exhibited a greater change in salivary cortisol reactivity. These findings suggest that neurophysiological and endocrine reactivity to social stress may be promising

endophenotypes of shyness and low social competence (Schmidt et al., 1999; Schmidt et al., 2002). Future studies employing neurochemical, neurophysiological and endocrine markers of social inhibition or shyness may begin to uncover genetic underpinnings of nonpathological shyness as well as the relation to more extreme forms of social avoidance as in social phobia and social anxiety disorder (Stemberger, Turner, Beidel, & Calhoun, 1995).

8.2. Face recognition

A face is a unique stimulus that is thought to hold special status in human social development (Ristic, Friesen, Kelland, & Kingstone, 2002; Slater, 1989). As early as the first week of life, infants appear to show a preference for and orient toward face over non-face stimuli (Morton & Johnson, 1991). This initial interest blossoms into the development of highly skilled or “expert” processing of faces (Tanaka, 2001). The ability to recognize identity through faces constitutes an important foundational skill that bootstraps further social–cognitive processing. Thus, the ability to recognize faces is an endophenotype that is likely to be highly discriminative for behavioural genetic studies of complex social behaviours.

Face recognition is thought to be mediated by specialized neural systems (Nelson, 2001; Posamentier & Abdi, 2003), thus, neuroimaging indices may be particularly useful measures of face recognition. In one study, researchers examined the link between the serotonin receptor type 3 gene (HTR3A) and limbic/prefrontal activation in response to a face recognition task in which participants were asked to identify stimuli as a face or a house (Iidaka et al., 2005). Twenty-six individuals were genotyped for a specific polymorphism (C178T) in the regulatory region of the serotonin receptor gene (HTR3A) on chromosome 11. All participants underwent functional magnetic resonance imaging during the face recognition task. Participants with C/C alleles of this gene locus demonstrated greater activation in the amygdala and dorsolateral prefrontal cortices as well as faster face recognition responses than those with C/T alleles. Thus, results suggest that variations at this serotonin receptor gene locus are associated with differential face processing ability, which may be mediated by the gene’s effects on limbic/prefrontal brain activity.

There is extensive evidence to suggest that the development of face recognition ability depends largely upon experience and learning (Nelson, 2001). For instance, empirical demonstrations of the inversion effect (poor recognition of upside–down faces) and the “other-race” effect (improved recognition of faces of the same race) suggest that exposure to faces fine-tunes this social–cognitive ability (Sangrigoli & de Schonen, 2004). Furthermore, young children, who have less experience with upright faces, do not demonstrate the same magnitude of the inversion effect (Carey & Diamond, 1994). Nelson (2001) hypothesizes that the development of face recognition is an “experience-expectant

process” that operates in an analogous fashion to speech perception. More specifically, while genetic factors may result in the development of neural systems with the potential to become specialized for face processing, experience with faces is necessary for this specialization to occur. Research is needed to explore whether there is a sensitive developmental period for this exposure and what type of experience is sufficient.

8.3. Emotion recognition

In addition to recognizing people’s identity, many social tasks entail the ability to recognize and interpret the internal emotional states of others based on facial expressions (Adolphs, 2003). Investigations of the genetic underpinnings of this ability have highlighted the role of the serotonin transporter promoter gene. More specifically, the serotonin transporter promoter gene has a short (S) and long (L) allele that are differentially associated with greater amygdala activity in response to angry or fearful faces in healthy adults (Hariri et al., 2002). Battaglia et al. (2005) further examined the influence of the serotonin transporter promoter gene on children’s shyness/behavioural inhibition and cortical responses to happy, neutral, and angry facial expressions. In their study, children with one or two copies of the S allele (i.e., SS or S carriers) demonstrated higher levels of shyness/behavioural inhibition as well as different electrophysiological responses to angry and neutral faces (i.e., smaller ERP N400 amplitudes). Decreased N400 ERP amplitudes suggest reduced cortical activity and possibly heightened subcortical activity (e.g., increased amygdala response) in the group of SS and S carriers. Based on these results, the authors concluded that a pattern of reduced cortical activation in response to specific social cues is inherited and may reinforce a child’s disposition towards shyness/behavioural inhibition.

Studies on the role of early experiences on emotion recognition development are beginning to emerge. The focus of these studies is on early parenting influences on emotion processing. The emotional environment provided by mothers (indicated by maternal emotional disposition) appears to be related to the infants’ responses to facial expressions (de Haan, Belsky, Reid, Volein, & Johnson, 2004). Specifically, infants with highly positive mothers, as assessed with The Positive and Negative Affect Schedule, looked longer at and demonstrated a larger ERP negative central (Nc) component, indicating the allocation of greater attentional resources, to fearful versus happy faces. This pattern of findings was thought to reflect, in part, the infants’ familiarity with happy expressions due to exposure provided by mothers high in positive affect.

Further support for the notion that experience shapes emotion recognition ability comes from studies of infants of clinically depressed mothers. While typical infants tend to look longer at sad faces in comparison to happy ones, presumably because they are novel, infants of clinically depressed mothers fail to demonstrate this pattern (Field,

Pickens, Fox, Gonzalez, & Nawrocki, 1998; Striano, Brennan, & Vanman, 2002). In addition, the experience of maltreatment from a parent or caregiver may affect the child's ability to process emotions. For example, maltreated children have difficulty identifying facial expressions of affect due to impoverished expressive environments that may hinder the development of emotion recognition (Camras, Grow, & Ribordy, 1983; During & McMahon, 1991).

The type of maltreatment experienced may influence the nature of the processing deficit. In one study, Pollak and Sinha (2002) demonstrated that subgroups of maltreated children display differential patterns of emotion recognition ability. Physically abused children, exposed to high levels of threat and hostility in their family environments, were found to correctly identify facial expressions of anger on the basis of less perceptual information as compared to typically developing (TD) children. On the other hand, neglected children were less accurate at emotion recognition than TD or physically abused children. Taken together, these findings highlight the role that salience and/or relative frequency of exposure to particular emotional expressions play in the development of emotion recognition.

In addition to the emotional climate of the home, other aspects of the family environment may facilitate the development of emotion recognition. In a review of early developmental influences on emotion understanding, Harris (1994) identified several family factors of particular interest. These included the ease of family communication about emotion, the frequency of feeling-state talk within the home, the frequency or intensity of sibling interaction, and the engagement in joint pretend play (either with a caregiver or sibling) in the home. Although investigations of these variables are primarily correlational in nature, and thus, do not inform on the specific role in emotion recognition, they do, nonetheless, suggest promising avenues for future behavioural genetic study.

8.4. *Theory of mind*

Researchers have begun to investigate heritability of experimentally defined constructs that are hypothesized to underlie socially competent behaviour such as theory of mind (TOM). TOM constitutes a high-level social-cognitive component that involves inferring the mental states (beliefs, desires, feelings, and intentions) of others. The ability to interpret other people's intentions is essential for effective social understanding and prediction as well as the ability to empathize with others (Perner, 1991; Symons, 2004). TOM is an umbrella term that encompasses more specific abilities (e.g., first-order false belief comprehension, second-order false belief comprehension, faux pas detection), and thus, genetic studies on specific theory of mind components may be more fruitful (Adolphs, 2003). Using a twin design to study first-order false belief comprehension (the understanding that people act in accordance with their beliefs about reality even when

those beliefs are false), Hughes and colleagues (1999, 2000) obtained a heritability estimate of 0.67 for a 119 3-year-old twin pairs, suggesting significant genetic influences. However, in a subsequent study of 1116 5-year-old twin pairs who were assessed on a battery of experimental TOM tasks, shared and nonshared environment accounted for the majority of the variance in their performance, with common genetic influences accounting for only 15% of the variance (Hughes et al., 2005). The authors concluded that the discrepant findings may be due to developmental differences (3- versus 5-year-olds) or statistical power. Alternatively, the discrepancies may stem from methodological issues including differences in the measurement of TOM.

Ronald, Happé, Hughes, and Plomin (2005) proposed that the construct of TOM could be further differentiated into prosocial, or "nice" TOM, aimed at facilitating cooperation with others from antisocial, or "nasty" TOM, that enables the deception of others (Ronald et al., 2005). They hypothesized that TOM development may not be uniform across different social contexts and investigated the genetic underpinnings of "nice" and "nasty" TOM in a sample of over 5000 MZ and DZ twin pairs. Parents (primarily mothers) completed questionnaires assessing their children's TOM-related prosocial and antisocial behaviours at ages 2, 3, and 4 years. Both "nice" TOM and "nasty" TOM demonstrated moderate heritability for both males and females at all ages assessed (heritability estimates ranged from .25 to .57). Significant nonshared environmental influences were also suggested at all ages and accounted for approximately 24 and 46 percent of the variance. Shared environmental influences only appeared significant at ages 2 and 3 years, accounting for 17 to 43 percent of the variance. However, these findings are limited due to the use of only one (subjective) measure of TOM.

Hughes and colleagues (1999, 2000) employed additional model-fitting analyses to examine the environmental factors contributing to TOM development. Their best fit model of the data included only genetic and nonshared environmental factors (estimated from the residual difference between MZ twin pairs). Shared environmental factors were dropped from the full model. In sum, the authors proposed that genetic and non-shared environmental influences operate in a dynamic fashion—that is, superior social understanding may enable a child to engage in more sophisticated social interactions, which in turn, may facilitate further TOM development.

Individual differences in TOM have also been linked to family social background variables (Cutting & Dunn, 1999); the presence of older siblings (Ruffman, Perner, Naito, Parkin, & Clements, 1998); the quality of sibling relationships (Hughes & Ensor, 2005); and levels of family talk about feelings (Dunn, Brown, & Slomkowski, 1991). In addition, variations in parent caregiving practices were associated with TOM development. Harsh parenting practices (i.e., physical abuse) were associated with delays in the acquisition of TOM (Cicchetti, Rogosch, & Maughan,

2003; Pears & Fisher, 2005) whereas disciplinary responses requiring the child to reflect on the victim's feelings were associated with enhanced false belief understanding (Ruffman et al., 1998).

Hughes and Cutting (1999) examined parenting styles and found evidence of sex differences in the association between parenting and TOM—parental warmth appeared strongly related to emerging TOM for girls, whereas, parental disciplinary strategies featured more prominently for the TOM development of boys. Levels of parental emotional expression and control can vary for children within the same family (Deater-Deckard, 1996), suggesting that differential parenting styles may constitute a significant nonshared environmental factor impacting TOM. These studies highlight the importance of social interaction, and particularly child-caregiver and sibling interactions, in the development of TOM. However, because the studies are correlational, it is possible that findings are spurious, resulting from associations with a third variable, such as genetic factors. Thus, environmental factors need to be incorporated into behavioral genetic designs in order to fully understand their relative contributions to TOM development.

9. Increasing the discriminative power of the group of study

9.1. Extreme phenotypes

A significant limitation to the behavioural genetic study of social competence and its relevant social endophenotypes is the large variability in social ability within the population. Greater specificity and homogeneity with regard to social endophenotypes within a population of study would greatly advance the search for genes related to social competence. DF extremes analysis is one technique that has proven useful to the genetic study of cognitive ability and may be amenable to the study of social ability. This procedure is designed to measure differential regression to the mean and thereby yields “group” statistics that address the etiology of the average difference on a quantitative trait between the selected group (e.g., low or high IQ) and the rest of the population (DeFries & Fulker, 1985, 1988). DF extremes analysis is based on the premise that if a particular phenotype, such as low cognitive ability, is linked genetically with a measured quantitative trait, such as IQ, the mean quantitative trait score of twin partners within the group of persons with mental retardation will be more similar for identical twins than for fraternal twins (i.e., there will be less regression to the mean). Accordingly, if group heritability for low-IQ individuals differs from the heritability for the general population then, it can be inferred that the etiology of low cognitive ability is qualitatively different. Alternatively, if group heritability for low-IQ individuals is similar to the heritability for the general population, low cognitive ability can be conceptualized as part of the low end of a quantitative distribution (Saudino, Plomin, & Pedersen, 1994).

With regard to the study of social competence, it would be premature to use the DF extremes approach as a standardized measure of this complex ability does not currently exist and the phenotype is not well defined in the general population. However, we do have well defined genetically disordered groups that are known to display deficits or excesses in social-cognitive processes. A homogenous group with regard to known genetic deletions/mutations and a well-defined behavioural phenotype may provide clues about candidate genes involved in aspects of social competence that are abnormal in the group. For example, Skuse and colleagues (1997) employed a genetically homogeneous group to uncover genetic influences on social cognitive ability in a sample of women with Turner's syndrome. Turner's syndrome is a chromosomal abnormality caused by nondisjunction of the sex chromosomes that is associated with the presence of only one complete X chromosome and no Y chromosome and characterized by a female phenotype. Although cognitive functioning is usually spared, impairments in social competence and social-cognition are common in this group (Molko et al., 2004; Ross, Zinn, & McCauley, 2000).

Skuse and colleagues (1997) used a parent questionnaire to compare the overall social-cognitive skill (e.g., emotion perception, social understanding, perception of social cues) of two groups of females with Turner's syndrome aged 6–25 years old (55 with a maternally derived single X chromosome and 25 with a paternally derived X chromosome). Results indicated that the females with a maternally derived X chromosome demonstrated significantly greater social-cognitive dysfunction, as reported on a parent-report questionnaire, as compared to those with a paternally derived X chromosome. This pattern of findings suggests an imprinted genetic locus for social-cognition expressed from the paternally derived X chromosome. The implication is that there may be a selective silencing of the corresponding genes on the maternally derived X chromosome.

Although notable in their attempt to examine the genetic underpinnings of social-cognition using the extreme group approach, Skuse, James, and Bishop's (1997) results should be interpreted with caution. In particular, the investigators assessed social-cognition using an omnibus measure—a 12-item, parent report social-cognition questionnaire. Questionnaire measures are susceptible to the diverse motivations of responders (e.g., demand characteristics, social desirability biases, faking, and response sets) and/or memory problems (e.g., forgetting or recall biases) that may limit their validity. Also, there is extremely limited psychometric data available for the use of this particular questionnaire (Scourfield, Neilson, & Lewis, 1999). Questionnaire measures may not be well suited to assess the cognitive processes that underlie social behaviour. Direct and specific measures of component social-cognitive skills (e.g., face and emotion processing, theory of mind) are needed to advance this line of research.

Fragile X syndrome is another example of a sex-linked condition that may be informative to the behavioural genetic study of social competence. Fragile X syndrome is the most common inherited cause of mental impairment and the most common known cause of autism. Fragile X syndrome is a genetic disorder caused by a mutation of the FMR1 gene on the X chromosome (Hagerman, 2006). Typically, the FMR1 gene contains between 6 and 53 repeats of the CGG codon. In people with the disorder, the FMR1 allele has over 230 repeat sequences of this codon. In one study, researchers investigated the consequence of high repeat alleles on cognitive ability (Allen et al., 2005). They used the Wechsler Adult Intelligence Scales-III (WAIS-III) to assess cognitive ability in 66 males and 217 females with a wide range of repeat sizes. Among females only, they found that FMR1 repeat size and transcript level significantly explained approximately 4% of the variance in the Verbal IQ score, suggesting that this polymorphism is one of many factors that influence variation in cognitive performance.

There is evidence to suggest that social cognitive performance may also be affected by the mutation of the FMR1 gene. Recent research on the social phenotype of Fragile X has uncovered that individuals with this syndrome exhibit low sociability and deficits in social cognition, particularly theory of mind (Cornish et al., 2005a, 2005b; Dykens, 2001). More precise definitions of the social–cognitive profiles of these individuals are needed for future behavioural genetic study of the FMR1 gene and social competence.

The combined benefit of employing a homogeneous group with an extreme phenotype and well defined and measured social endophenotypes that improve the discriminative ability of the phenotype is evident in recent genetic studies of sociability in William's syndrome (WS). WS is a genetic disorder caused by a consistent deletion of approximately 20 contiguous genes on chromosome 7, including the gene for elastin (Korenberg et al., 2000). Individuals with WS exhibit characteristic physical features (e.g., facial and physical abnormalities, cardiovascular difficulties) and a range of cognitive deficits, including mild to moderate mental retardation and impaired spatial processing abilities (Bellugi, Lichtenberger, Jones, Lai, & St. George, 2000). Most notably, however, these individuals demonstrate a unique social phenotype characterized by hypersociability (i.e., overly friendly and socially disinhibited behaviour) as compared to chronological age-matched TD individuals (Doyle, Bellugi, Korenberg, & Graham, 2004; Gosch & Pankau, 1997), individuals with DS (Doyle et al., 2004) and those with non-specific MR (Gosch & Pankau, 1994). This observed pattern of hypersociability appears early in development (Jones et al., 2000) and has high sensitivity and specificity, suggesting that the hemizygous deletion of one or more of the affected genes is responsible for altering typical social developmental processes (Doyle et al., 2004; Mervis, Robinson, & Bertrand, 2000).

As a first step in the exploration of the genetic bases of hypersociability, Jones and colleagues (2000) describe an

ongoing program of research aimed at specifying and quantifying the social–behavioural features of WS. Multiple salient indices of hypersociability have been identified including heightened social approach behaviours (e.g., increased interest in approaching and engaging strangers, reduced fear of strangers, and overfriendliness) and increased linguistic expressiveness (e.g., frequent use of sound effects, emotional expression, and exaggerated vocal prosody). The authors have developed and employ a range of measures to assess these indicators of hypersociability in the WS population (e.g., the Salk Institute Sociability Questionnaire; Chiles, Bellugi, & Cassady, 1998).

The stage is now set to combine a more sophisticated understanding of the WS social phenotype with behavioural genetic methodologies in order to uncover the genetic origins of sociability. Future genotype–phenotype investigations in WS will likely focus on examining the impact of genotypic variability on phenotypic outcome (Mervis et al., 2000). In particular, this approach will involve studying the small percentage of individuals with WS (approximately 2%) who do not have the typical deletion breakpoints characteristic of the disorder, but instead, have smaller deletions in the WS region (Mervis et al., 2000). These individuals often display evidence of some, but not all, WS phenotypic characteristics. Comparing those with the typical versus atypical WS deletions may provide important clues regarding the contribution of specific genes to the behavioural expression of WS features such as hypersociability (Bellugi, Lichtenberger, Mills, Galaburda, & Korenberg, 1999; Doyle et al., 2004).

This behavioural genetic approach has already been applied to the study of visuospatial deficits in WS (Frangiskakis et al., 1996) and preliminary findings support its utility in the study of the social phenotype. For example, Doyle et al. (2004) described a young girl with WS who demonstrated an unusually small deletion - a subset of genes at the telomeric region that are typically absent in WS were present in this child. The girl's social behaviour differed markedly from others with WS, as she did not demonstrate hypersociability and instead displayed significant shyness around strangers. Thus, the authors suggested that the absence of this particular subset of genes may contribute to hypersociability in the disorder. Although the study of individuals with smaller deletions has the potential to facilitate the search for genes responsible for sociability, investigators caution against associating the hemizygous deletion of specific genes with particular impairments until findings are adequately replicated (Donnai et al., 2000).

10. Nurturing nature: Implications of the gene–environment connection in social competence

Social competence is a complex human behaviour that is likely to involve a system of genes that interacts with a myriad of environmental risk and protective factors. The search for its genetic and environmental origins and influences is equally complex and will require a

multidimensional conceptualization and multiple methods and levels of analysis. The development of an operational definition of the construct of social competence and its hypothesized processes or ‘building blocks’ and regulatory strategies or ‘tools’ will be a crucial milestone. Precise specification of key processes involved in social competence will contribute to the identification of social endophenotypes - psychological processes that are validly and reliably measured components of social competence. Social endophenotypes may be employed in behavioural genetic designs and prove to be sensitive intermediate measures of social competence and have more power to detect genes associated with social ability in studies of typical, at risk, and atypical populations.

To date, the behaviour genetic study of sociability, face and emotion recognition and TOM is limited by methodological challenges. There is great variability in the measures used across studies and a tendency to assess only one dimension of the construct. Moreover, other potentially relevant social endophenotypes such as social attention (e.g., eye gaze tracking) and perception of non-verbal cues other than facial expression (e.g., voice prosody/, biological motion) have not yet been explored. Behavioural measures of endophenotypes may be further explored using brain imaging techniques which could uncover where, when and how neural processes are engaged during a behavioural task. Neurophysiological as well as other biological indices may be especially useful to detect social endophenotypes that would not be evident from examining behavioural patterns alone.

The utility of social endophenotypes depends ultimately on the power of the behavioural genetic design. Sociability has been examined extensively with regard to how heritable it is—an important first step to determine whether there is genetic involvement. However, the heritability factor for a particular social ability is specific to a given sample and time period, and thus, can change with the age of the individual when there is an increasing influence of a QTL. Furthermore, heritability estimates are only considered meaningful when certain assumptions are met—namely, limited gene—environment covariation/interaction and little interaction among the genes involved (Rose, 2001). This is usually not the case and may result in heritability estimates that overestimate the value of the genetic contribution because they include the gene—environment covariation and interactions within the genetic effects. However, as Gottesman (2004) pointed out, “heritability was never intended to be an end, but a means to an end, an end tempered by considerations of developmental processes, gene by environment interactions, and ecological validity” (p. 222). Behavioural genetic studies of social endophenotypes, such as sociability and theory of mind, must now move beyond heritability investigations and take advantage of the more sophisticated genetic designs that are available. In addition, longitudinal studies would improve the power to detect genetic and environmental influences.

Another useful methodological strategy consists of examining the variability within a particular genotype to see how it affects the resulting phenotype. This type of research is currently being conducted with genetically atypical populations such as persons with Fragile X with pre-mutation as compared to those with the full mutation and in a subgroup of persons with WS who have smaller deletions. This research strategy is promising but limited by the problem of variability. Specifically, the phenotypic outcome of different subpopulations (e.g., at risk or atypical) can be caused by differing factors, including their genetic endowment, the environment, or complex gene—environment interactions (Dykens, 1995; Goldsmith et al., 1997; Nijhout, 2003).

Another significant benchmark in the behavioural genetic study of social competence will be the identification of risk and protective factors associated with the development of social competence. Ironically, it is the identification of relevant ‘social’ genes that will most likely lead to the identification of environments that either nurture or impede the development of social competence. Behavioural genetic paradigms remove “genetic noise”, thus, facilitating the search for environmental risk and protective factors in developmental outcomes (Eisenberg, 2004). Ultimately, research integrating the knowledge of genetic and specific environmental factors in the development of social competence is needed. For instance, twin and/or adoption studies should identify and measure putative environmental variables in their genetic investigations (as opposed to simply estimating shared or nonshared environmental influence broadly through statistical means) (see Moffitt, 2005, for a review of how such paradigms have been applied in the study of antisocial behaviour).

In sum, human social development (in all its forms) is the most compelling example of the reciprocal relation between genes and environments. It highlights the notion that biology, particularly when in the service of social goals, must be nurtured by experience. Moreover, it suggests that it is the dynamic relation or connection between genes and environments that is primary. The implication is that simply identifying vulnerable and resilient genes or facilitative and restrictive environments is insufficient. What is needed is an understanding of the optimal match between genotypes and environments. This is possible with behaviour genetic research that addresses how genetic and environmental effects interact and how these contribute to change and continuity in typical and atypical development.

Acknowledgments

Grace Iarocci and Theo Elfers’ work on this paper was supported by a research grant from the Social Sciences and Humanities Research Council of Canada to Grace Iarocci. Jodi Yager’s contribution was supported by a fellowship from the Autism Research Training Program (jointly funded by CIHR, NAAR and FRSQ).

References

- Achenbach, T. M., & Edelbrock, C. S. (1981). Behavioral problems and competencies reported by parents of normal and disturbed children aged four through sixteen. *Monographs of the Society for Research in Child Development*, 46(1), 82.
- Adolphs, R. (2003). Cognitive neuroscience of human social behaviour. *Nature Reviews Neuroscience*, 4(3), 165–178.
- Allen, E., Sherman, S., Abramowitz, A., Leslie, M., Novak, G., Rusin, M., et al. (2005). Examination of the effect of the polymorphic CGG repeat in the FMR1 gene on cognitive performance. *Behavioral Genetics*, 35(4), 435–445.
- Auerbach, J. G., Benjamin, J., Faroy, M., Geller, V., & Ebstein, R. (2001). DRD4 related to infant attention and information processing: A developmental link to ADHD? *Psychiatric Genetics*, 11(1), 31–35.
- Battaglia, M., Ogliari, A., Zanoni, A., Citterio, A., Pozzoli, U., Giorda, R., et al. (2005). Influence of the serotonin transporter promoter gene and shyness on children's cerebral responses to facial expressions. *Archives of General Psychiatry*, 62(1), 85–94.
- Bellugi, U., Lichtenberger, L., Jones, W., Lai, Z., & St. George, M. (2000). I. The neurocognitive profile of Williams syndrome: A complex pattern of strengths and weaknesses. *Journal of Cognitive Neuroscience*, 12(Suppl 1), 7–29.
- Bellugi, U., Lichtenberger, L., Mills, D., Galaburda, A., & Korenberg, J. R. (1999). Bridging cognition, the brain and molecular genetics: Evidence from Williams syndrome. *Trends in Neurosciences*, 22(5), 197–207.
- Bodmer, W. F., & Cavalli-Sforza, L. L. (1976). *Genetics, evolution, and man*. San Francisco, CA: W.H. Freeman.
- Bost, K. K., Vaughn, B. E., Washington, W. N., Cielinski, K. L., & Bradbard, M. R. (1998). Social competence, social support, and attachment: Demarcation of construct domains, measurement, and paths of influence for preschool children attending head start. *Child Development*, 69(1), 192–218.
- Broman, S. H., Nichols, P. L., & Kennedy, W. A. (1975). *Preschool IQ: Prenatal and early developmental correlates*. Oxford, England: Lawrence Erlbaum.
- Bronfenbrenner, U. (1979). *The ecology of human development—experiments by nature and design*. Cambridge, MA: Harvard University Press.
- Bronfenbrenner, U. (1999). Environments in developmental perspective: Theoretical and operational models. In S. L. Friedman & T. D. Wachs (Eds.), *Measuring environment across the life span: Emerging methods and concepts* (pp. 3–28). Washington, DC: American Psychological Association.
- Bronfenbrenner, U. (2000). Ecological systems theory. In A. E. Kazdin (Ed.), *Encyclopedia of psychology* (vol. 3, pp. 129–133). Washington, DC: American Psychological Association.
- Bronfenbrenner, U., & Ceci, S. J. (1994). Nature–nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review*, 101(4), 568–586.
- Bruner, J. S. (1988). The course of cognitive growth. In K. Richardson & S. Sheldon (Eds.), *Cognitive development to adolescence: A reader* (pp. 33–60). Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.
- Bruner, J. (1996). A narrative model of self construction. *Psyke & Logos*, 17(1), 154–170.
- Camras, L. A., Grow, J. G., & Ribordy, S. C. (1983). Recognition of emotional expression by abused children. *Journal of Clinical Child Psychology*, 12(3), 325.
- Carey, S., & Diamond, R. (1994). Are faces perceived as configurations more by adults than by children? Object and Face Recognition. *Special Issue of Visual Cognition*, 1(2/3), 253–274.
- Chapman, M. (1991). The epistemic triangle: Operative and communicative components of cognitive competence. In M. Chandler & M. Chapman (Eds.), *Criteria for competence: Controversies in the conceptualization and assessment of children's abilities* (pp. 209–228). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Chiles, M., Bellugi, U., & Cassady, C. (1998). *The sociability questionnaire*. Unpublished test protocol. La Jolla, CA: Laboratory for Cognitive Neuroscience.
- Cicchetti, D., Rogosch, R., & Maughan, A. (2003). False belief understanding in maltreated children. *Development and psychopathology special issue: Experiments of nature: Contributions to developmental theory*, 15(4), 1067–1091.
- Cicchetti, D., & Toth, S.L. (1997). Transactional ecological systems in developmental psychopathology. In S.S. Luthar, J.A. Burack, D. Cicchetti, & J.R. Weisz, (Eds.), *Developmental psychopathology: Perspectives on adjustment, risk, and disorder* (pp. 317–349).
- Cloninger, C. R., Adolfsson, R., & Svrakic, N. M. (1996). Mapping genes for human personality. *Nature Genetics*, 12(1), 3.
- Corder, E. H., Saunders, A. M., Strittmatter, W. J., Schmechel, D. E., Gaskell, P. C., Small, G. W., et al. (1993). Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families. *Science*, 261(5123(Print)), 921–923.
- Cornish, K., Burack, J. A., Rahman, A., Munir, F., Russo, N., & Grant, C. (2005a). Theory of mind deficits in children with fragile X syndrome. *Journal of Intellectual Disability Research*, 49(5), 372–378.
- Cornish, K., Kogan, C., Turk, J., Manly, T., James, N., Mills, A., et al. (2005b). The emerging fragile X premutation phenotype: Evidence from the domain of social cognition. *Brain and Cognition*, 57(1), 53–60.
- Cutting, A. L., & Dunn, J. (1999). Theory of mind, emotion understanding, language, and family background: Individual differences and interrelations. *Child Development*, 70(4), 853–865.
- Daniels, D., & Plomin, R. (1985). Origins of individual differences in infant shyness. *Developmental Psychology*, 21(1), 118–121.
- Deater-Deckard, K. (1996). Within family variability in parental negativity and control. *Journal of Applied Developmental Psychology*, 17(3), 407–422.
- de Haan, M., Belsky, J., Reid, V., Volein, A., & Johnson, M. H. (2004). Maternal personality and infants' neural and visual responsivity to facial expressions of emotion. *Journal of Child Psychology and Psychiatry*, 45(7), 1209–1218.
- DeFries, J. C., & Fulker, D. W. (1985). Multiple regression analysis of twin data. *Behavior Genetics*, 15(5), 467–473.
- DeFries, J. C., & Fulker, D. W. (1988). Multiple regression analysis of twin data: Etiology of deviant scores versus individual differences. *Acta Geneticae Medicae et Gemellologiae: Twin Research*, 37(3-4), 205–216.
- Doyle, T. F., Bellugi, U., Korenberg, J. R., & Graham, J. (2004). "Everybody in the world is my friend" hypersociability in young children with Williams syndrome. *American Journal of Medical Genetics Association*, 124(3), 207–263.
- Dunn, J., Brown, J., & Slomkowski, C. (1991). Young children's understanding of other people's feelings and beliefs: Individual differences and their antecedents. *Child Development*, 62(6), 1352–1366.
- During, S. M., & McMahon, R. J. (1991). Recognition of emotional facial expressions by abusive mothers and their children. *Journal of Clinical Child Psychology*, 20(2), 132–139.
- Dykens, E. M. (1995). Measuring behavioral phenotypes: Provocations from the 'new genetics'. *American Journal on Mental Retardation*, 99(5), 522–532.
- Dykens, E. M. (2001). Personality and psychopathology: New insights from genetic syndromes. In H. N. Switzky (Ed.), *Personality and motivational differences in persons with mental retardation* (pp. 283–317). Mahwah, NJ: Lawrence Erlbaum Associates, Publishers.
- Eid, M., Riemann, R., & Angleitner, A. (2003b). Sociability and positive emotionality: Genetic and environmental contributions to the covariation between different facets of extraversion. *Journal of Personality*, 71(3), 319–346.
- Eid, M., Riemann, R., Angleitner, A., & Borkenau, P. (2003a). Sociability and positive emotionality: Genetic and environmental contributions to the covariation between different facets of extraversion. *Journal of Personality*, 71(3), 319–346.
- Eisenberg, L. (2004). Social psychiatry and the human genome: Contextualising heritability. *British Journal of Psychiatry*, 184(2), 101–103.

- Eley, T. C. (2001). Contributions of behavioral genetics research: Quantifying genetic, shared environmental and nonshared environmental influences. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety* (pp. 45–59). New York, NY: Oxford University Press.
- Field, T., Pickens, J., Fox, N. A., Gonzalez, J., & Nawrocki, T. (1998). Facial expression and EEG responses to happy and sad faces/voices by 3-month-old infants of depressed mothers. *British Journal of Developmental Psychology*, *16*(4), 485–494.
- Fischer, K. W., Rotenberg, E. J., Bullock, D. H., & Raya, P. (1993). The dynamics of competence: How context contributes directly to skill. In R. H. Wozniak & K. W. Fischer (Eds.), *Development in context: Acting and thinking in specific environments* (pp. 93–117). Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.
- Floderus-Myrhed, B., Pedersen, N., & Rasmuson, I. (1980). Assessment of heritability for personality, based on a short-form of the Eysenck Personality Inventory: A study of 12,898 twin pairs. *Behavior Genetics*, *10*(2), 153–162.
- Frangiskakis, J. M., Ewart, A. K., Morris, C. A., Mervis, C. B., Bertrand, J., Robinson, B. F., et al. (1996). LIM-kinase hemizygosity implicated in impaired visuospatial constructive cognition. *Cell*, *86*(1), 59–69.
- Glannon, W. (2003). Key concepts endophenotypes. *Philosophy, Psychiatry, & Psychology*, *10*(3), 277–284.
- Goldsmith, H. H., & Gottesman, I. I. (1996). Heritable variability and variable heritability in developmental psychopathology. In M. F. Lenzenweger & J. J. Haugaard (Eds.), *Frontiers of developmental psychopathology* (pp. 5–43). New York, NY: Oxford University Press.
- Goldsmith, H. H., Gottesman, I. I., & Lemery, K. S. (1997). Epigenetic approaches to developmental psychopathology. *Development and Psychopathology*, *9*(2), 365–387.
- Gosch, A., & Pankau, R. (1997). Personality characteristics and behaviour problems in individuals of different ages with Williams syndrome. *Developmental Medicine & Child Neurology*, *39*(8), 527–533.
- Gottesman, I. (1963). Heritability of personality: A demonstration. *Psychological Monographs*, *77*(9), 1–21.
- Gottesman, I., & Shields, J. (1972a). *Schizophrenia and genetics: A twin study vantage point*. New York: Academic Press.
- Gottesman, I., & Shields, J. (1973a). Genetic theorizing and schizophrenia. *British Journal of Psychiatry*, *122*, 15–30.
- Gottesman, I. (2004). Postscript: Eyewitness to the maturation of behavioral genetics. In L. F. DiLalla (Ed.), *Behavior genetics principles: Perspectives in development, personality, and psychopathology* (pp. 217–223). Washington, DC: American Psychological Association.
- Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry*, *160*(4), 636–645.
- Gottesman, I., & Hanson, D. R. (2005). Human development: Biological and genetic processes. *Annual Review of Psychology*, *56*, 263–286.
- Gottesman, I. I., & Shields, J. (1972b). A polygenic theory of schizophrenia. *International Journal of Mental Health*, *1*(1–2), 107–115.
- Gottesman, I. I., & Shields, J. (1973b). Genetic theorizing and schizophrenia. *British Journal of Psychiatry*, *122*(566), 15–30.
- Guralnick, M. J. (1996). Future directions in early intervention for children with Down's syndrome. In J. A. Rondal, J. Perera, L. Nadel, & A. Comblain (Eds.), *Down's syndrome. Psychological, psychobiological, and socio-educational perspectives* (pp. 147–162). Philadelphia, PA: Whurr Publishers, Ltd.
- Guralnick, M. J. (2005). Early intervention for children with intellectual disabilities: Current knowledge and future prospects. *Journal of Applied Research in Intellectual Disabilities*, *18*(4), 313–324.
- Hagerman, R. J. (2006). Lessons from fragile X regarding neurobiology, autism, and neurodegeneration. *Journal of Developmental & Behavioral Pediatrics*, *27*(1), 63–74.
- Hariri, A., Mattay, V., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., et al. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science*, *297*, 400–403.
- Harris, P. L. (1994). The child's understanding of emotion: Developmental change and the family environment. *Journal of Child Psychology and Psychiatry*, *35*(1), 3–28.
- Hauser-Kram, P., Warfield, M. E., & Shonkoff, J. P. (1999). Family influences on adaptive development in young children with Down syndrome. *Child Development*, *70*(4), 979–989.
- Heath, A. C., Eaves, L. J., & Martin, N. G. (1998). Interaction of marital status and genetic risk for symptoms of depression. *Twin Research: The Official Journal of the International Society for Twin Studies*, *1*(3), 119–122.
- Hughes, C., & Cutting, A. L. (1999). Nature, nurture, and individual differences in early understanding of mind. *Psychological Science*, *10*(5), 429–432.
- Hughes, C., Deater-Deckard, K., & Cutting, A. L. (1999). 'Speak roughly to your little boy?' Sex differences in the relations between parenting and preschoolers' understanding of mind. *Social Development*, *8*(2), 143–160.
- Hughes, C., & Ensor, R. (2005). Executive function and theory of mind in 2 year olds: A family affair? *Developmental Neuropsychology*, *28*(2), 645–668.
- Hughes, C., Jaffee, S. R., Hapke, F., Taylor, A., Caspi, A., & Moffitt, T. E. (2005). Origins of individual differences in theory of mind: From nature to nurture? *Child Development*, *76*(2), 356–370.
- Hughes, C., & Plomin, R. (2000). Individual differences in early understanding of the mind: Genes, nonshared environment and modularity. In P. Carruthers & A. Chamberlain (Eds.), *Evolution and the human mind: Language, modularity and social cognition* (pp. 47–61). Cambridge, England: Cambridge University Press.
- Iidaka, T., Ozaki, N., Matsumoto, A., Nogawa, J., Kinoshita, Y., Suzuki, T., et al. (2005). A variant C178T in the regulatory region of the serotonin receptor gene HTR3A modulates neural activation in the human amygdala. *Journal of Neuroscience*, *25*(27), 6460.
- Jones, W., Bellugi, U., Lai, Z., Chiles, M., Reilly, J., Lincoln, A., et al. (2000). II. Hypersociability in Williams syndrome. *Journal of Cognitive Neuroscience*, *12*(Suppl 1), 30–46.
- Knowles, J. A., Mannuzza, S., & Fyer, A. J. (1995). Heritability of social anxiety. In M. B. Stein (Ed.), *Social phobia: Clinical and research perspectives* (pp. 147–161). Washington, DC: American Psychiatric Association.
- Korenberg, J. R., Chen, X. N., Hirota, H., Lai, Z., Bellugi, U., Burian, D., et al. (2000). VI. Genome structure and cognitive map of Williams syndrome. *Journal of Cognitive Neuroscience*, *12*(Suppl 1), 89–107.
- Landry, S. H., Garner, P. W., & Pirie, D. (1994). Effects of social context and mothers' requesting strategies on Down's syndrome children's social responsiveness. *Developmental Psychology*, *30*(2), 293–302.
- Lemery, K. S., & Goldsmith, H. H. (2002). Genetic and environmental influences on preschool sibling cooperation and conflict: Associations with difficult temperament and parenting style. *Marriage & Family Review*, *33*(1), 77–99.
- Loehlin, J. C., McCrae, R. R., Costa, P. T. J., & John, O. P. (1998). Heritabilities of common and measure-specific components of the big five personality factors. *Journal of Research in Personality*, *32*(4), 431.
- McGuire, S. (2002). Nonshared environment research: What is it and where is it going? *Marriage & Family Review*, *33*(1), 31–56.
- Mervis, C. B., Robinson, B. F., & Bertrand, J. (2000). The Williams syndrome cognitive profile. *Brain and Cognition*, *44*(3), 604–628.
- Moffitt, T. E. (2005). The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. *Psychological Bulletin*, *131*(4), 533–554.
- Molko, N., Cachia, A., Riviere, D., Mangin, J. F., Bruandet, M., LeBihan, D., et al. (2004). Brain anatomy in Turner syndrome: Evidence for impaired social and spatial-numerical networks. *Cerebral Cortex*, *14*(8), 840–850.
- Morton, J., & Johnson, M. H. (1991). CONSPEC and CONLERN: A two-process theory of infant face recognition. *Psychological Review*, *98*(2), 164–181.
- Nelson, C. A. (2001). The development and neural bases of face recognition. *Infant and Child Development*, *10*(1–2), 3–18.

- Nichols, P. L. (1984). Familial mental retardation. *Behavior Genetics*, 14(3), 161–170.
- Nijhout, H. F. (2003). On the association between genes and complex traits. *The Journal of Investigative Dermatology*, 8(2(Print)), 162–163.
- Olino, T. M., Klein, D. N., Durbin, C. E., Hayden, E. P., & Buckley, M. E. (2005). The structure of extraversion in preschool aged children. *Personality and Individual Differences*, 39(2), 481–492.
- Pears, K. C., & Fisher, P. A. (2005). Emotion understanding and theory of mind among maltreated children in foster care: Evidence of deficits. *Development and Psychopathology*, 17(1), 47–65.
- Perner, J. (1991). *Understanding the representational mind*. Cambridge, MA: The MIT Press.
- Petrill, S. A. (2002). Examining social behavior and relationships using genetically sensitive designs: An introduction. *Marriage & Family Review*, 33(1), 3–10.
- Phillips, K., Fulker, D. W., & Rose, R. J. (1987). Path analysis of seven fear factors in adult twin and sibling pairs and their parents. *Genetic Epidemiology*, 4(5), 345–355.
- Piaget, J. (1929). *The child's conception of the world*. Oxford, England: Harcourt, Brace.
- Piaget, J. (1960). *The child's conception of the world*. London: Routledge.
- Plomin, R. (1999). Genetic research on general cognitive ability as a model for mild mental retardation. *International Review of Psychiatry*, 11, 34–36.
- Plomin, R. (2001). Genetic factors contributing to learning and language delays and disabilities. *Genetic contributions to learning and language delays and disabilities*, 10(2), 259–277.
- Plomin, R. (1994). The Emanuel Miller Memorial Lecture 1993: Genetic research and identification of environmental influences. *Journal of Child Psychology and Psychiatry*, 35(5), 817–834.
- Plomin, R., Asbury, K., & Dunn, J. (2001). Why are children in the same family so different? Nonshared environment a decade later. *Canadian Journal of Psychiatry*, 46(3 Print), 225–233.
- Plomin, R., & Colledge, E. (2001). Genetics and psychology: Beyond heritability. *European Psychologist*, 6(4), 229–240.
- Plomin, R., & Craig, I. (2001). Genetics, environment and cognitive abilities: Review and work in progress towards a genome scan for quantitative trait locus associations using DNA pooling. *British Journal of Psychiatry*, 178(Suppl40), s41–s48.
- Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences*, 10(1), 1–16.
- Plomin, R., Fulker, D. W., & Corley, R. (1997). Nature, nurture, and cognitive development from 1 to 16 years: A parent–offspring adoption study. *Psychological Science*, 8(6), 442–447.
- Plomin, R., & McClearn, G. E. (1993). *Nature, nurture & psychology*. Washington, DC: American Psychological Association.
- Plomin, R., McClearn, G. E., Smith, D. L., Vignetti, S., Chorney, M. J., Chorney, K., et al. (1994). DNA markers associated with high versus low IQ: The IQ quantitative trait loci (QTL) project. *Behavior Genetics*, 24(2), 107–118.
- Plomin, R., & Rutter, M. (1998). Child development, molecular genetics, and what to do with genes once they are found. *Child Development*, 69(4), 1223–1242.
- Pollak, S. D., & Sinha, P. (2002). Effects of early experience on children's recognition of facial displays of emotion. *Developmental Psychology*, 38(5), 784–791.
- Posamentier, M. T., & Abdi, H. E. R. (2003). Processing faces and facial expressions. *Neuropsychology Review*, 13(3), 113–143.
- Ristic, J., Friesen, C. K., Kelland, C., & Kingstone, A. (2002). Are eyes special? It depends on how you look at it. *Psychonomic Bulletin & Review*, 9, 507–513.
- Ronald, A., Happé, F., Hughes, C., & Plomin, R. (2005). Nice and nasty theory of mind in preschool children: Nature and nurture. *Social Development*, 14(4), 664–684.
- Rose, R. J., & Ditto, W. B. (1983). A developmental-genetic analysis of common fears from early adolescence to early adulthood. *Child Development*, 54(2), 361–368.
- Rose, S. (2001). Moving on from old dichotomies: Beyond nature–nurture towards a lifeline perspective. *British Journal of Psychiatry*, 178(Suppl. 40), S3–S7.
- Rose-Krasnor, L. (1997). The nature of social competence: A theoretical review. *Social Development*, 6(1), 111–135.
- Ross, J., Zinn, A., & McCauley, E. (2000). Neurodevelopmental and psychosocial aspects of Turner syndrome. *Mental Retardation and Developmental Disabilities Research Reviews*, 6(2), 135–141.
- Ruffman, T., Perner, J., Naito, M., Parkin, L., & Clements, W. A. (1998). Older (but not younger) siblings facilitate false belief understanding. *Developmental Psychology*, 34(1), 161–174.
- Rutter, M., Simonoff, M., & Plomin, R. (1996). Genetic influences on mild mental retardation: Concepts, findings and research implications. *Journal of Biosocial Science*, 28(1), 509–526.
- Sameroff, A. J., & Chandler, M. (1975). Reproductive risk and the continuum of caretaking casualty. In F. Horowitz (Ed.), *Review of child development research*. Chicago, IL: University of Chicago Press.
- Sameroff, A. J., Seifer, R., & Bartko, W. T. (1997). Environmental perspectives on adaptation during childhood and adolescence. In S. S. Luthar, J. A. Burack, D. Cicchetti, & J. R. Weisz (Eds.), *Developmental psychopathology: Perspectives on adjustment, risk, and disorder*. New York, NY: Cambridge University Press.
- Sangrigoli, S., & de Schonen, S. (2004). Effect of visual experience on face processing: A developmental study of inversion and non-native effects. *Developmental Science*, 7(1), 74–87.
- Saudino, K. J., Plomin, R., & Pedersen, N. L. (1994). The etiology of high and low cognitive ability during the second half of the life span. *Intelligence*, 19(3), 359–371.
- Scarr-Salapatek, S. (1973). Unknowns in the IQ equation: A review of three monographs. In F. Reber & L. Dorman (Eds.), *Child development and behavior* (2nd ed.). Oxford, England: Alfred A. Knopf.
- Schmidt, L. A., Fox, N. A., Perez-Edgar, K., Hu, S., & Hamer, D. H. (2001). Association of DRD4 with attention problems in normal childhood development. *Psychiatric Genetics*, 11(1), 25–29.
- Schmidt, L. A., Fox, N. A., Rubin, K. H., Hu, S., & Hamer, D. H. (2002). Molecular genetics of shyness and aggression in preschoolers. *Personality and Individual Differences*, 33(2), 227–238.
- Schmidt, L. A., Fox, N. A., Schulkin, J., & Gold, P. W. (1999). Behavioral and psychophysiological correlates of self-presentation in temperamentally shy children. *Developmental Psychobiology*, 35(2), 119–135.
- Skuse, D. H. (2001). Endophenotypes and child psychiatry. *The British Journal of Psychiatry: The Journal of Mental Science*, 178, 395–396.
- Skuse, D. H., James, R. S., & Bishop, D. V. M. (1997). Evidence from Turner's syndrome of an imprinted X-linked locus affecting cognitive function. *Nature*, 387(6634), 705–708.
- Slater, A. (1989). Visual memory and perception in early infancy. In A. Slater & G. Bremner (Eds.), *Infant development* (pp. 43–71). Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.
- Sroufe, L. A., & Jacobvitz, D. (1989). Diverging pathways, developmental transformations, multiple etiologies and the problem of continuity in development. *Human Development*, 32(3), 196–203.
- Stein, M. B., Jang, K. L., & Livesley, W. J. (2002). Heritability of social anxiety-related concerns and personality characteristics: A twin study. *Journal of Nervous and Mental Disease*, 190(4), 219–224.
- Stemberger, R. T., Turner, S. M., Beidel, D. C., & Calhoun, K. S. (1995). Social phobia: An analysis of possible developmental factors. *Journal of Abnormal Psychology*, 104(3), 526–531.
- Striano, T., Brennan, P. A., & Vanman, E. J. (2002). Maternal depressive symptoms and 6-month-old infants' sensitivity to facial expressions. *Infancy*, 3(1), 115–126.
- Scourfield, J., Neilson, M., & Lewis, G. (1999). Heritability of social cognitive skills in children and adolescents. *British Journal of Psychiatry*, 175, 559–564.
- Symons, D. K. (2004). Mental state discourse, theory of mind, and the internalization of self-other understanding. *Developmental Review*, 24(2), 159–188.

- Tanaka, J. W. (2001). The entry point of face recognition: Evidence for face expertise. *Journal of Experimental Psychology: General*, 130(3), 534–543.
- Topping, K., Bremner, W., & Holmes, E. (2000). Social competence: The social construction of the concept. In R. Bar-on & J. D. A. Parker (Eds.), *The handbook of emotional intelligence: Theory, development, assessment, and application at home, school, and in the workplace* (pp. 28–40). San Francisco, CA: Jossey-Bass.
- Turkheimer, E., & Waldron, M. (2000). Statistical analysis, experimental method, and causal inference in developmental behavioral genetics. *Human Development*, 43(1), 51–52.
- Vygotsky, L. S. (1934). *Thought and language*. Cambridge, MA: MIT Press.
- Wahlsten, D. (1994). The intelligence of heritability. *Canadian Psychology*, 35(3), 244–260.
- Waters, E., & Sroufe, L. A. (1983). Social competence as a developmental construct. *Developmental Review*, 3(1), 79–97.
- Wertsch, J. (1999). Cognitive development. In M. Bennett (Ed.), *Developmental psychology: Achievements and prospects* (pp. 309–322). New York, NY: Psychology Press.
- Wyman, P. A., Sandler, I., Wolchik, S., & Nelson, K. (2000). Resilience as cumulative competence promotion and stress protection: Theory and intervention. In D. Cicchetti, J. Rappaport, I. Sandler, & R. P. Weissberg (Eds.), *The promotion of wellness in children and adolescents*. Washington, DC: Child Welfare League of America.