## INDIVIDUAL CHARACTERISTICS AND ANTISOCIAL BEHAVIOR: COGNITIONS, EMOTIONS, AND SELF-REGULATION

Social learning theory has relied heavily on observable family and peer social processes as proximal causes of antisocial behavior. As such, the theory might be accused of taking an empty organism or "black box" perspective. While we assert that such processes are the core causes, "person" variables work in concert with social–environmental experiences to determine antisocial development. A wide array of person variables have been offered as risk factors for antisocial behavior (Coie & Dodge, 1998; Rothbart & Bates, 1998). Of this array, three interrelated, organismic self-regulation variables are relevant to antisocial development and compatible with social learning theory: executive attentional control, motivational inhibition, and

negative emotional reactivity (Barkley, 1997; Nigg, 2000; Mezzacappa, Kindlon, Saul, & Earls, 1998). These forms of self-regulation hold considerable promise for several reasons. First, neuropsychological research suggests that they are tied to activity in specific neural networks. Second, objective, psychometrically sound, behavioral marker tasks independent of self-report and parent or teacher ratings are available to ascertain individual differences in each of these self-regulatory capacities (Kindlon, Mezzacappa, & Earls, 1995). Third, though such capacities come "on line" at an early age and show considerable temporal continuity, they are malleable and affected by social experience into adolescence (Davidson, Jackson, & Kalin, 2000).

These child self-regulatory capacities and their hypothesized contribution to antisocial behavior are briefly described. Executive attentional control is associated with activity in the midline prefrontal neural network, and ties emotions, cognitions, and attention together to facilitate planful action and goal-directed behavior. This network functions as a "top-down" cognitive system involving effortful inhibition of irrelevant responses, and is requisite to sustained task orientation in the face of stimulus or resource competition (Posner & Rothbart, 2000; Nigg, 2000). Performance deficits in marker tasks for executive attentional control are related to externalizing disorders and reduced social competence, even after controlling for IQ, age, sex, and reading level (Kindlon et al., 1995). Motivational inhibition is mediated by the limbic system, and entails the suppression of responses under contingencies for punishment and extinction. It is a "bottom-up" form of behavioral inhibition critical to passive avoidance learning (Nigg, 2000). Antisocial behavior has been associated with reduced sensitivity to aversive feedback, especially in the presence of reward (Mezzacappa et al., 1998). Negative emotional reactivity reflects the frequency and intensity with which negative emotions are experienced and expressed. Normally adaptive emotions contribute to disordered behavior when their experience and expression do not fit the context, are out of proportion to events, or unduly persist (Davidson et al., 2000). Negative emotional reactivity or emotion dysregulation increase vulnerability for externalizing behavior problems (Bates, 2000). The limbic system is implicated in emotional experience and expression, in part via modulation of peripheral nervous (resting heart rate and vagal tone), motor (facial expression), and endocrine (cortisol secretion) functions. Prefrontal networks associated with executive attentional control are also involved in emotional anticipation and in preparatory behavioral approach and withdrawal. Negative emotionality, deployment of attention, and sensitivity to punishment involve functionally overlapping neural systems and behavioral functions.

Although there is some empirical support for the role of these self-regulatory capacities in the development of antisocial behavior, additional research using prospective longitudinal and field-experimental designs are

needed to more stringently test their causal status. First, the degree to which self-regulatory capacities are associated with antisocial behavior must be examined in prospective longitudinal designs, using measurement methods that minimize overlap in source variance. This association should be examined during the elementary school years because it is during this period that children are progressively exposed to new environments, activities, and people while adult tracking and contingencies simultaneously diminish. Given these conditions, antisocial development, especially in its covert form, is likely to reflect children's capacity to manage emotional distress in response to challenge, inhibit behavioral choices driven by immediate environmental contingencies, and attend to relevant information in order to formulate and execute plans consistent with goal-directed behavior under delayed reinforcement contingencies. Second, it is important to test how and how much children's self-regulatory capacities are shaped by social processes in the family during early child development, and thereafter are elaborated in less supportive and more challenging experiences in school and peer settings. The degree to which such capacities are malleable in response to environmental manipulation is critical to examining their causal status and to their incorporation as targets of intervention. Third, the mediator and moderator relationships among child self-regulatory capacities and social–environmental influences should be examined. Parents, peers, and teachers are impacted by a child's capacity to self-regulate behavior and emotions. Self-regulatory capacities may influence how the ambient social environment is experienced by the child. A more complete understanding of the reciprocal and conjoint roles of child self-regulatory and social–environmental processes in the development of antisocial behavior may facilitate more precise targeting and adaptation of standard medical and psychosocial interventions. Child self-regulatory and family environmental profiles may provide information about what works well for whom (in contrast to a "one size fits all" method), and guide prioritizing and efficient resource allocation in primary and secondary prevention efforts. We anticipate that efforts to alter self-regulatory mechanisms using interventions that target the child in the absence of changes in natural environmental contingencies will have modest effects on antisocial behavior, but may be useful adjuncts to such contingency-based programs.

## INDIVIDUAL CHARACTERISTICS AND ANTISOCIAL BEHAVIOR: CHILD GENDER

Gender differences in the rate of opposition are observed as early as 18 months of age (Shaw & Winslow, 1997). Gender differences in aggressiveness are well in place by age 5 (McFadyen-Ketchum, Bates, Dodge, & Pettit, 1996) and persist throughout childhood and adolescence. Moffitt, Caspi, Rutter, and Silva (2001) report that fewer girls (1%) than boys (5–

10%) evidence persistent and serious antisocial behavior associated with early-onset or life-course-persistent trajectories. Males account for more adolescent and adult crimes (especially those involving violence), and males have a higher lifetime prevalence for antisocial disorders. An exception to this general developmental pattern occurs in early adolescence, during which time females' offending approaches that of males. Females' increasing display of antisocial behavior during this period is congruent with later onset, life-course-limited trajectories (Moffitt, 1994). The adolescent burst in antisocial activity is tied to early timing of pubescence in females but not in males. Thus, causal explanations for two types of gender differences in antisocial behavior may be sought. One refers to mean-level group differences. The other concerns gender differences in variability and distribution of antisocial behavior—particularly at the high end of the distribution. The two types of differences may or may not share common causes.

The early origins of gender differences in opposition and aggression may reflect average gender-related variation in rates of nervous system maturation (Maccoby, 1998). Male infants lag behind females in behavioral inhibition, emotion regulation, attention deployment, and verbal development. These regulatory differences extend into the second and third years of life. As a result, boys and girls may evoke different responses from parents, and may respond differently to the same parenting conditions (Martin, 1981). Boys and girls are exposed to different social contingencies. Mothers are more coercive toward boys than girls, and this difference is even more pronounced for highly aggressive boys and girls (McFayden-Ketchum et al., 1996). Variation in the frequency of coercive exchanges may reflect gender differences in child self-regulation, parents' gender-biased attitudes about how to socialize children, acquired differences in boys' and girls' responsiveness to aversive social stimuli, or some combination thereof. We hypothesize that girls display less antisocial behavior because they are less frequently involved in coercive parent–child interaction, and also less frequently reinforced for oppositional and countercoercive responding. We hypothesize that, on the average, parents value and more frequently reinforce the positive social behavior of girls than boys.

Peer socialization processes contribute even more powerfully to gender differences in opposition and aggression as children move into preschool and kindergarten. Boys and girls show a robust preference for interaction with same-gender children beginning at age 3. The dyadic and group play of boys and girls have markedly different behavioral characteristics. There is more verbal challenge, noncompliance, and rough-and-tumble play, plus a more clearly articulated dominance ranking, in male groups. There is more cooperation, verbal exchange, compliance, and mutual accommodation in girls' groups (Maccoby, 1998). Peer reactions to male and female aggression differ. Based on extensive observation in preschool groups, Fagot, Hagan, Leinbach, and Kronsberg (1985) found that boys' physical

aggression led to negative peer responses 40% of the time, and were ignored 15% of the time. In contrast, girls' physical aggression led to negative peer responses 15% of the time and were ignored 48% of the time. Boys also ignore girls who attempt to enter their play groups. Boys receive substantially more peer playground training for aggression than girls. A girl who is socialized to engage in frequent opposition and aggression by her family is not likely to find such behavior very functional in the peer group. She would have difficulty finding other highly coercive females with whom to interact, and also would be relatively unable to access boys' groups (Offord, Boyle, & Racine, 1991). In summary, average gender differences in social contingencies and in self-regulation operate conjointly to increase boys' risk relative to girls' risk for persistent antisocial behavior during the elementary school years (i.e., for early-starter or life-course-persistent trajectories).

Less is known about gender differences in the origins, emergence, and growth of covert antisocial behavior. High rates of overt antisocial behavior during childhood increase risk for early onset and growth in covert antisocial behavior for males. We hypothesize that the same risk-transformation process applies to females, implying more delayed emergence and slower growth in covert antisocial behavior as a result of their lower average rates of overt antisocial behavior during childhood. There are also fewer highly antisocial, same-sex peers with whom younger girls can associate and exchange deviant talk. Gender differences in emergence and growth in covert behavior are the result of the different environmental experiences and contingencies encountered by boys and girls.

There are two developmental periods or settings in which the typical gender differences in antisocial behavior are dramatically diminished. One occurs in early adolescence. Contingencies change as preference for same-gender peer associates diminishes, a broad array of potential peer affiliates becomes available, association with opposite-gender individuals increases (especially for early maturing females), and monitoring decreases. We hypothesize that these transitions result in systematic changes in social contingencies and experiences that diminish previous gender-differentiated environmental support for antisocial behavior, and that these changes are responsible for the growth burst in antisocial behavior by females in adolescence. Similar gender-leveling social processes may explain the reciprocal involvement of both men and women in partner-directed violence (Capaldi, Dishion, Stoolmiller, & Yoerger, 2001).

The same core, causal variables that account for individual differences in aggression and antisocial behavior more generally also account for gender differences in that behavior, including its more extreme and persistent forms. Aggressive antisocial behavior is displayed insofar as it is socially functional in a specific setting and in a specific development period. It varies across settings and over time in predictable ways, changing with ambi-

ent social environmental contingencies. The moderating effect of child self-regulatory capacities on social experience is similarly involved in individual and gender differences in antisocial behavior. The causes of antisocial behavior in females have been less well specified than antisocial behavior in males. An important research agenda is to ascertain whether the same causal variables operate across gender, or operate in the same way or to the same degree. Field-experimental and prospective longitudinal research are both relevant to this agenda. Gender, even as an ascribed social classification, is not very malleable. The differential effectiveness of interventions for boys and girls and the need for gender-sensitive interventions should be ascertained.