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PART 2

Personality and Emotional Development

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CHAPTER

2 Personality and Emotional Development

Nathan A. Fox, Bethany C. Reeb-Sutherland, *and* Kathryn A. Degnan

Abstract

Over the past 20 years, research on the development of emotions and interest in the emotion–cognition interface has blossomed. Coinciding with this growth has been research on the neural circuitry and development of two basic motivational/emotion states: one brought on by threat and danger (i.e., fear) and one resulting from actively pursuing or receiving reward (i.e., reward/joy). The current chapter reviews traditional approaches to thinking about emotional development and temperament in infants and children. It then reviews the neuroscience work associated with fear and reward with a focus on the development of these systems. A particular emphasis will be placed on how this research and the examination of gene–environment interactions can influence research in personality and emotion development.

Key Words: emotion; personality; temperament; fear; reward; neuroimaging; gene × environment; amygdala; nucleus accumbens; prefrontal cortex

Key Points

- The study of emotion has assumed a central place in the science of human behavior over the past 50 years.
- Research on emotion has benefited from the development of measurement approaches that include assessment of facial, vocal, and postural behaviors.
- Measurement techniques for studying emotion have also been developed for infant and child populations and have led to increased study of emotional development.
- Two neural systems that have been extensively studied in rodent and nonhuman primate populations involve fear and reward circuitry.
- Fear circuitry has been studied by examining and manipulating fear conditioning in animals. The structures implicated involve amygdala, hippocampus, and ventrolateral prefrontal cortex.
- Reward circuitry has been studied by examining animal and human responses to appetitive stimuli, including drugs, food, money, and sex. The structures implicated involve basal ganglia and striatum (caudate, putamen, and nucleus accumbens).
- Genes that are involved in either the fear system or the reward system have also been identified, and the effects of environment on different polymorphisms have been extensively studied in both neural systems.
- Until recently there has been little animal (rodent or nonhuman primate) research in the neurosciences that has studied the development of either the fear or reward systems.
- Key advances in understanding the development of emotion, individual differences in emotion expressivity, and its underlying brain bases necessitate translational research, research in both human infants and animal models.
- Exciting advances in brain imaging technologies including functional neuroimaging and source localization with the electroencephalogram hold promise for identifying

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the development of underlying brain circuitry in fear and reward, avoidance and approach motivational systems.

Introduction

Emotions play a key role in human psychological life. Our languages have many words to express feeling states; emotions can be communicated through multiple avenues (face, voice, body posture, as well as language); and we believe that controlling one's emotions is an important life skill (Lewis, 2008; Saarni, Campos, Camras, & Witherington, 2006). As well, perceiving and appropriately interpreting cues that signal different emotion states is viewed as critical for adaptive social discourse (Widen & Russell, 2008). Just how infants and young children develop the skills, competencies, and behaviors necessary for expressing, experiencing, and understanding their rich emotional lives is a matter of lively debate. This debate entails discussion of what aspects of emotion appear to be present from early infancy and how socialization of emotion occurs over childhood. It also touches on questions of how emotion perception and understanding develop in infancy and childhood. Finally, it should be informed by what we have learned about emotion from research in neuroscience. The following chapter will review theories of emotion that have been influential in understanding the development of emotion in infants and young children. It also will cover theories of individual differences in emotional reactivity, often thought of as temperament, and it will address issues integral to the measurement of emotion and temperament. The emphasis of this chapter will be on what we have learned about emotion and temperament from research in neuroscience and genetics. It will review the work in two systems, fear and reward, examining the underlying neural circuitry of each system and how this research informs our thinking about emotion development in human infants and children.

Theories and Measurement

Theories of Emotional Development

A BRIEF LOOK BACK

It was not long ago that the topic of emotion was not considered a mainstream area of research in developmental psychology. Examination of the subject index of one of the most popular child development textbooks of its day, *Child Development and Personality*, 4th edition (Mussen, Conger, & Kagan, 1974), finds only a brief mention of emotion as its own subject and no mention of temperament. Rather, the study of emotion was subsumed within topics such as attachment and dependency. Infants' emotional lives were described as diffuse and reflected in variations of physiological state and arousal levels. The social life of the infant was thought to be solely the result of caregiving experiences and learning histories. Smiling and crying were behaviors that could be shaped and controlled by environmental contingencies. However, this lack of interest in emotion was not unique to developmental studies. Experimental psychology viewed emotions as interfering with an individual's cognitive processing and as such endeavored to reduce the emotional demands in experimental situations. That said, the role of emotion in behavior was always of interest to those studying personality and social psychology and resulted in numerous debates throughout the field (for reviews of these debates see Frijda, 2008; Schorr, 2001).

Among the debates in social psychology was the issue of whether the experience of emotion was the result of individual appraisal of physiological changes as a result of perception of affectively laden stimuli or whether the experience of emotion was the result of a more generalized appraisal of social context. One side of this debate was framed by the theory proposed by William James (1884), which suggested that the experience of emotion was a result of interpretation of physiological changes that occurred when perceiving stimuli of different emotional values. For example, we know we are afraid because we feel our heart beat fast and our palms get sweaty and we know we are angry because we feel our face flush and feel temperature in our periphery (hot under the collar). However, in a classic study, Schachter and Singer (1962) tested this model by manipulating subjects' autonomic arousal (some received an injection of adrenaline, some a placebo) as well as the emotional context (some interacted with a happy confederate, some with a confederate there to make the subject angry). Independent of autonomic arousal, subjects reported emotional changes as a function of context. This line of work and the work of personality psychologists such as Richard Lazarus (1982), who studied the role of appraisal and coping in emotional behaviors, oriented psychology toward a position in which cognition maintained a dominant frame: individuals' interpretations of stimuli in context are integral to their emotional experiences. The implications for a developmental approach to emotion were obvious: emotional development was contingent upon

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cognitive development and a child's emerging understanding of the social world (Lazarus, 2001).

THEORIES OF EMOTION AND EMOTION DEVELOPMENT

Despite the late start in the field of developmental psychology and the ongoing debates regarding the primacy of cognition, the study of emotion and emotional development has blossomed over the past few decades. Two of the biggest impediments for emotion research include the inconsistency of its definition and the lack of specificity in its measurement (Frijda, 2008; Kagan, 2007). Emotion theories can be distinguished by their position regarding the features that emotions share, the distinctions between individual emotions, the amount of significance awarded to each component of the emotion process, and the suggested associations between components (Frijda, 2008; Kagan, 2007). Similarly, theories of emotion development explore how emotional expression becomes organized over time, whether facial expressions are linked to specific elicitors or situations, and links between emotion expression and their related responses (Camras & Fatani, 2008; Lewis, 2008).

The importance of emotion in behavior changed radically with the writings and work of Paul Ekman (1982, 1994, 2003), who followed in the footsteps of Charles Darwin (1998) by focusing on the evolutionary history of social signals and particularly of facial expressions of emotion. His position was that these signals evolved to communicate important information about the individual's psychological state and intent. As well, these signals were universal in nature: they were identifiable by people from different cultures and shared similar meaning to individuals from varied cultures (Ekman, Sorenson, & Friesen, 1969). The face expressed emotion and the patterns of facial musculature reflected the internal psychological state of the individual. As well, patterns of muscle activity on the face could signify masking of emotion, deception, or attempts at regulation of emotion intensity. Ekman meticulously studied the muscles of the face and their actions, and created a system for coding these movements. The Facial Action Coding System (FACS; Ekman & Friesen, 1978; Ekman, Friesen, & Hager, 2002) became a standard for analyzing facial expression. It also proved useful in identifying prototypical patterns of facial expression for what Ekman was to label as specific or discrete emotions. These specific prototypes were universally recognized and could be measured on the face. The immediate advantages of Ekman's work were to provide researchers with a means to measure the presence of emotion in a subject by coding changes in facial expression. In addition, it provided a source for identifying prototypical emotion stimuli that could be presented to assess perception, identification, and reaction to different emotions. Ekman also provided conceptual definitions for emotion and contrasted it with mood (Ekman, 1984). He used both temporal and behavioral parameters to distinguish the two states. Emotion was a relatively quick psychological state reflected in changes in facial musculature; moods were longer lasting and did not have specific facial signs. Finally, Ekman's writings provided a theoretical foundation for understanding the importance of emotion as it influenced behavior.

In addition to Ekman, and around the same time, a developmental psychologist, Carroll Izard, led the way toward emphasizing the importance of emotion in early development. Izard, who also studied Darwin, developed his own coding system, specifically for infants and young children (Maximally Discriminative Facial Coding System, MAX; Izard, 1995). Izard's theory, called Differential Emotions Theory (DET), argued that during early development infants express a set of discrete emotions that reflect their cognitive abilities and motivational states (Ackerman, Abe, & Izard, 1998; Izard, 1991; Izard & Malatesta, 1987). These emotions include joy, anger, distress, disgust, and interest, each having multiple components stemming from neural, expressive, and experiential mechanisms (see Camras & Fatani, 2008, for review). Development of these emotions is said to occur as a result of the interaction between initial emotion states, enhanced motivational states, and cognitive skills, which create complex emotion–cognition interactions as more complex emotions emerge (e.g., sadness, shame, and pride). Overall, Izard articulated a developmental theory that argued for the universal nature of discrete emotions and emotion development, as well as the interface of emotion and cognition across development.

In comparison to Ekman's and Izard's theories of discrete emotions, another view of emotion development suggested that emotions go through a process of differentiation and integration across infancy. Bridges (1932) suggested that infant emotions begin as general excitement and become more differentiated into distress or delight, and then into distinct emotion states (e.g., fear, happiness). Following this idea, Sroufe (1996) described three stages of emotion development: pre-emotion reactions, precursor

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emotions, and mature emotions. The transitions between each stage are attributed to maturational processes, just as in DET; however, these differentiation theorists suggest different relations among expression, emotion-eliciting stimuli, and emotion-related behaviors at each stage (Camras & Fatani, 2008). Specifically, Sroufe posits that pre-emotion reactions are akin to reflexes (neonatal period), precursor emotions are true emotions that require simple cognitive processes (up to 6 months of age), and mature emotions involve more complex cognitions and may be differentiated through facial expressions (6 months and older). Thus, in contrast to Ekman's and Izard's theories, differentiation theorists contend that distinct expressions are not linked to emotions at all ages, but that this type of coherence and differentiation occurs after cognitive complexity and emotional maturity emerge (Camras & Fatani, 2008).

Another differentiation theorist, Michael Lewis (2008), suggested that certain emotions emerge as children's cognitive knowledge develops. For instance, Lewis suggested that infants display distress and pleasure at birth, but only display joy, sadness, and disgust around 3 months of age, anger around 4 months of age, and fear around 7 months of age (Lewis & Michalson, 1983). Each of these new developments arises as children gain an understanding of the world around them (e.g., anger requires some means–ends knowledge; Lewis, 1991). This model also suggested that there are relations between emergent emotions and the child's developing sense of self. Lewis focused specifically on the emotions of shame, guilt, embarrassment, and pride (Lewis, 1992, 2008; Lewis & Sullivan, 2005). These emotions emerge later in the second and third years of life and are associated with the child's sense of self versus other as well as the child's development of self-standards for behavior (Lewis, 1992). Thus, self-conscious emotions develop in response to either a violation of some internal or external standard (shame or embarrassment) or in response to achievement of some internal goal (pride). From this perspective, by 3 years of age, children's emotions are quite differentiated and will continue to be elaborated as new experiences and more complex cognitive skills develop (Lewis, 2008).

While DET and differentiation theories focused on the expression and emergence of the emotion or emotional expression, the functionalist framework viewed emotional development as centered on the functions that specific emotions play in the social world of the child (Barrett & Campos, 1987; Jenkins, 2002; Saarni et al., 2006). As such, emotions are inherently communication to oneself and others (Oatley & Jenkins, 1992). Anger, distress, and joy figure prominently in infancy as emotions that allow the infant to signal his or her displeasure or pleasure in response to a particular context or stimulus. They also provide signals for enhancing the proximity between an infant and the caregiver. With development, other emotions emerge with their function to signal the internal state of the infant to the caregiver. Thus, each emotion is a class of responses used to signal oneself and others in order to attain a desired goal. However, these signaling responses are not universal and emerge from the individual's repertoire of behavior toward achieving that particular goal in that particular situation (Camras & Fatani, 2008). Furthermore, Campos and others have suggested that this framework posits emotions as relational processes that allow individuals to establish, change, or maintain some aspect of their relationship to the external or internal environment (Saarni et al., 2006). Johnson-Laird and colleagues (2006) have posited that hyperintense emotions initiate the onset and maintain psychological illness by fueling one's focus on the situations that elicited the emotion in an effort to reason about its occurrence. These suppositions require multiple components of emotion and function to be ascertained: individual behaviors, goals, and success or progress toward the goals. All of these components are necessary to understand the emotion, as they are all interrelated and may change across time as more complex cognitive processes develop.

Another framework that integrated multiple components in the study of emotion is the dynamic systems perspective (Camras & Fatani, 2008; Lewis, Lamey, & Douglas, 1999). Overall, this perspective assumed that complex systems include nonlinear interactions among system components, certain periods of sensitivity or insensitivity to external factors, and numerous changes between states (Lewis, et al., 1999). More specifically, multiple psychological elements are thought to repeatedly interact with one another in a bidirectional manner throughout real time and over developmental time. By examining psychological states, such as emotional states, in this framework, investigators could observe how these states self-organize within and across situations, leading to stable tendencies across development (Lewis, et al., 1999). In addition, changes in the organization of emotion and emotion states could be observed by examining the consistency of interactions among emotional

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constituents and the changes in these interactions between periods of development (e.g., before and after 4 months). For instance, using state-space grid analysis (see Lewis, et al., 1999, for detailed explanation), mother–child interactions have been examined for the relations between infant distress and attention to the mother (Lewis et al., 1999), as well as the congruence between mother and child emotion states during an interaction (Granic, O'Hara, Pepler, & Lewis, 2007). In general, use of dynamic systems theory and state-space grid analysis provided a unique perspective on longitudinal (across real or developmental time) change within interactive processes, such as those between individuals or those between individual states such as emotional reactivity and attention allocation. The dynamic systems perspective also may assist with bridging the gap between emotion theory and neurobiology (Lewis, 2005). While emotion theory focused on linear causal assumptions about broader constructs, such as attention and emotion, neurobiology examines more complexity, focusing on the interaction between individual components. Dynamic system principles are suggested to include both the broader perspective of emotion theory and the biological detail of neuroscience (Lewis, 2005).

SUMMARY

While multiple theories of emotion and emotional development exist, much of the contemporary study of emotion derives from the early work of Ekman and Izard and the more recent writings of Joseph Campos and Michael Lewis. Throughout these various viewpoints, several components of emotion are highlighted and suggested to change in varying degrees throughout early development (Camras & Fatani, 2008; Frijda, 2008; Lewis, 2008). Lewis (2008) recently summarized emotions as integrated processes including multiple levels of analysis: emotional elicitors, emotional states, emotional expressions, and emotional experiences. Each of these features could be the independent focus of study and is independently affected by a developmental process. However, there also may be developmental influences on the relations between these individual emotion components. These might also be studied from a developmental systems perspective, as interactive processes are changing across real and developmental time (Lewis et al., 1999). In the past few decades, the study of emotion has blossomed and is now viewed as a central construct and driving force in a child's early social development (Lewis, Haviland-Jones, & Barrett, 2008; Oatley, Keltner, & Jenkins, 2006; Saarni et al., 2006).

Theories of Temperament

Basic emotion research within developmental psychology has included the development of emotions, the socialization of emotion, and such topics as perception of emotions in infancy and childhood. One of these issues addressed is temperament, or individual differences in the expression and experience of emotion. These early differences in emotional and behavioral characteristics are thought to be relatively stable traits with genetic and biological components (DiLalla & Jones, 2000; Goldsmith, Lemery, Aksan, & Buss, 2000).

THOMAS AND CHESS

The modern study of infant temperament has its origins in the work of Alexander Thomas and Stella Chess (Thomas, Birch, Chess, Hertzig, & Korn, 1964; Thomas & Chess, 1977; Thomas, Chess, & Birch, 1968, 1970). These researchers were interested in describing individual differences among infants in the first months of life and how these differences affected parent behavior. Thomas and Chess selected 100 families living in New York City and visited them on an intensive schedule after the birth of their infant. They characterized the infant's behavior with regard to the sleep–wake cycle, eating, activity level, and reactivity. On the basis of their observations and notes, Thomas and Chess created nine factors that they felt characterized infant individual differences in temperament. Each of these factors (e.g., activity level) was a continuum along which an individual infant could be placed. Thomas and Chess also recognized that certain combinations of behaviors and rankings on these factors seemed to occur at higher rates than others. They proposed that there were at least three temperamental types: slow-to-warm-up, easy, and difficult. For example, a difficult temperament reflected a child rated high in activity level and negative affect, and low in adaptability and soothability. Thomas and Chess theorized that social development was a product of both an infant's initial temperament and environmental influences, such as caregiving behavior. They reasoned that parents had expectations about the temperament of their new child (whether he or she would be active or not) and the match between their expectations and the child's temperament would either be a stressor for the family or not. They called this match "goodness of fit" and predicted that the degree of goodness of fit would

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determine the degree to which an infant's temperament predicted behavior difficulties. Although Thomas and Chess's approach to temperament included the idea that some infants expressed negative emotions more frequently than others (and other infants expressed more positive emotions), emotion itself was not a centerpiece. Rather, their conceptual view was shaped by their collaboration with Herbert Birch (a student of T. C. Schneirla). Temperament was seen as reflecting underlying differences in approach and withdrawal motivation. Indeed, approach–withdrawal was one of the nine factors created by Thomas and Chess.

CAMPOS AND GOLDSMITH

Despite the work of Thomas and Chess, it was not until the late 1980s that a theory of temperament centered itself squarely within emotion theory. That temperament theory, articulated by Hill Goldsmith and Joseph Campos (1982, 1990), viewed temperament as reflecting individual differences in the frequency and intensity of expression of basic or discrete emotions. Temperament was to be assessed in contexts that could reliably elicit specific emotions. For example, infants' temperamental anger could be assessed in situations designed to elicit frustration by blocking the infants' ability to achieve a goal (such as a barrier placed between the infant and an attractive toy). Temperamental joy could be assessed during social interaction with an adult or a caregiver. Differences in the latency to express a specific emotion and the frequency and intensity of the emotional expression characterized individuals' temperament. In addition, these individual differences in emotional reactivity would serve as cues or stimuli for the responsive caregiver (Scarr & McCartney, 1983). Measurement of temperament was accomplished by coding infant facial expression of emotion during specific stimulus-eliciting situations or contexts. In fact, along with Mary Rothbart, Goldsmith developed the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith & Rothbart, 1992, 1996), a set of tasks that were age-appropriate and could be administered in a laboratory to elicit temperamental reactivity and regulation.

ROTHBART

A third approach to the study of infant temperament was presented by Mary Rothbart (Rothbart & Derryberry, 1981). Rothbart approached temperament from a tradition introduced by Pavlov (1955) and expanded on by his students and psychologists in Europe (Strelau, 1983). Individuals were thought to differ in the strength of their nervous system, in the speed with which they could be conditioned, and in the magnitude with which they responded to stimuli in the environment. These differences in reactivity to sensory stimuli could be quantified by measuring motor behavior, affect, and physiological responses (Rothbart, Ahadi, & Evans, 2000; Rothbart, Derryberry, & Hershey, 2000). In addition to identifying differences in reactivity as one important component of temperament, Rothbart had the insight to observe that children also differed in the manner in which they modulated or regulated this initial reactivity (Rothbart, Ahadi, et al., 2000). She focused on attention processes as being the core skills necessary for adaptive regulation of reactive tendencies in individuals. Thus, from early in life, infants and children used attention to regulate their reactivity to stimuli and contexts in their world (Rothbart, 1981, 1986). Whereas other models of temperament focused on differences in approach–withdrawal tendencies (e.g., Thomas and Chess) or differences in the frequency and intensity of basic emotions (e.g., Campos and Goldsmith) as present in the child from infancy, Rothbart suggested that this second component of temperament (regulatory skills and attention) developed across infancy and early childhood (Rothbart, 1989). Therefore, individual differences exist in children's initial reactivity and in their development of these regulatory skills.

Rothbart and her collaborator, Michael Posner, identified the attention skills that are important for adaptive regulation of reactive tendencies (Posner & Rothbart, 1992, 2000). These include attention shifting and inhibitory control, both of which are subsumed under the rubric of executive attention. At first, in early infancy, children differ in their abilities to shift their attention from a particular reactive state to another (Rothbart, Posner, & Rosicky, 1994). They are also less likely able to inhibit prepotent motor responses (i.e., inhibitory or effortful control; Rothbart, 1989). These skills emerge over early childhood, and differences among children in their ability to use these skills to regulate their reactions to novelty and to stimuli in the environment constitute temperament in Rothbart's approach.

Emotional and motivational systems both play a central role in Rothbart's model of temperament (Rothbart & Bates, 1998, 2006). Infant reactive behavior is motivated by approach–withdrawal tendencies—infants differ in their reactions to novelty and to the intensity of stimulation. At the same time, Rothbart placed an emphasis on the emotions

of fear and anger as primary responses to situations of novelty or frustration. Thus, infants differed in the degree to which they reacted with fear to novel or unfamiliar stimuli or contexts or differed in the degree to which they reacted with anger to frustrating events. Although Rothbart's approach was not derivative of a discrete or basic emotions view of temperament, it did acknowledge the emotions of fear and anger as playing a central role in the child's life. Rothbart also developed a number of approaches to the measurement of temperament. As mentioned above, she developed a set of laboratory tasks in collaboration with Hill Goldsmith (Lab-TAB; Goldsmith & Rothbart, 1992, 1996). In addition, Rothbart developed a series of questionnaires that assessed reactivity and regulation (e.g., Infant Behavior Questionnaire, Rothbart, 1981; Children's Behavior Questionnaire, Rothbart, Ahadi, Hershey, & Fisher, 2001). These laboratory tasks and questionnaires have become critical measures for assessing infant and child temperament.

BEHAVIORAL INHIBITION: A FOCUSED STUDY OF TEMPERAMENT

One specific temperamental style that has received a good deal of attention is behavioral inhibition (Fox, Henderson, Marshall, Nichols, & Ghera, 2005; Kagan, Reznick, & Snidman, 1987). Using Rothbart's theory, this area of study provides a model of interdisciplinary integration for research on temperament and emotion processes (Fox, Henderson, et al., 2005). Indeed, work regarding behavioral inhibition has included basic neuroscience, social-emotional development, cognitive development, and clinical psychopathology. Kagan, for many years, studied infant responses to novelty and the state of uncertainty that was often elicited (Kagan, 1994). Uncertainty could serve as a motivating force for cognitive change or it could lead to behavioral responses of withdrawal. In a series of studies beginning in 1984, Kagan and colleagues described a group of toddlers who, when presented with novel or uncertain social or nonsocial situations, exhibited withdrawal and fearful behaviors (Garcia-Coll, Kagan, & Reznick, 1984; Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984; Kagan et al., 1987; Kagan, Reznick, Snidman, Gibbons, & Johnson, 1988; Kagan & Snidman, 1991; Kagan, Snidman, & Arcus, 1998; Reznick et al., 1986). These behaviors were often accompanied by increased autonomic reactivity, heightened startle responses, and elevated reactivity of the hypothalamic-pituitary-adrenal (HPA) axis (Kagan et al., 1987; Kagan, Reznick, & Snidman, 1988). Kagan called these toddlers "temperamentally behaviorally inhibited." He argued that the constellation of their behaviors and physiology warranted denoting them as a temperament type. The innovation in this work was the link that Kagan and others (Fox, Henderson, & Marshall, 2001; Kagan, 2001) established between this behavioral disposition and the neuroscience research at that time of Joseph LeDoux and Michael Davis that, from work in rodents, described a circuit underlying the acquisition, learning, and display of conditioned fear responses (Davis, 1986; Ledoux, Iwata, Cicchetti, & Reis, 1988). This work had identified the amygdala and nuclei within that structure as having a major role in both the acquisition of fear responses as well as the behavioral and physiological outcomes of fear learning. Thus, for example, the central nucleus of the amygdala was viewed as a primary center from which output went to other subcortical structures controlling autonomic reactivity, HPA reactivity, and freezing behavior. These were the same physiological and behavioral responses that Kagan had identified as part of the constellation of behavioral inhibition. Indeed, Kagan would argue that heightened amygdala activation underlies this disposition (Kagan, 2001).

SUMMARY

As emotion research has increased over the years, so has an interest in the individual differences in emotional reactivity, or temperament. While multiple theories have been presented, much of the current work in this area is informed by Rothbart's theories of temperamental reactivity and regulation. Throughout the field, models of temperament have focused on either differences in approach–withdrawal tendencies (e.g., Thomas and Chess) or differences in the frequency and intensity of basic emotions (e.g., Campos and Goldsmith). However, Rothbart has integrated both of these frameworks and posits that individual differences exist in children's initial reactivity and in their regulation of this reactivity. Using this viewpoint, researchers have aimed to explore both the continuity and discontinuity in temperamental reactivity, while also exploring what factors influence the patterns and outcomes of these trajectories (Degnan & Fox, 2007). Future study is needed to elucidate the multiple components of temperamental reactivity and regulation, in terms of physiology, behavior, and attributions. If emotions are integrated processes with different levels of analysis, individual differences in each of

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these levels may be examined independently or in relation to the other components.

Issues of Measurement

The measurement of emotion is a critical issue for both the study of general emotion processes and the study of temperament (Kagan, 2007; Saarni et al., 2006). Without reliable, valid means for measuring emotion it is not possible to study emotional phenomena in an objective fashion. Throughout the history of emotion research, many studies have depended upon self-report of emotion (how "angry" did you feel?) or upon physiological measurements that were themselves multiply determined (e.g., heart rate). The correspondence between the presence of an emotion and either self-report or physiological response is proximate at best. The problem is only magnified when thinking about emotions in children: infants and young children cannot report their feelings and their physiological changes are often imprecise.

REPORT MEASURES

A good deal of research has used questionnaire methods for assessing an individual's emotional response to a particular stimulus. Subjects are often asked to rate how they feel (sometimes rating on a Likert scale) toward a particular stimulus or event. Alternatively, subjects are asked to rate the affective quality of a stimulus: how angry, sad, or happy a particular individual appears to be. In the former approach, the question of interest is usually to assess the subject's experience of a particular emotion, while in the latter type of study it is an evaluation of the subject's perception of a particular stimulus. There have been numerous discussions of the veridicality or reliability of subject self-report, and the value of self-report of emotion for understanding emotion processes is still debated (for review of this debate see Kagan & Fox, 2006).

BEHAVIORAL MEASURES

An alternative to self-report or parent report is the observational coding of behavior reflecting emotion. As already described, Ekman developed a coding system for assessing changes in facial muscle activity (FACS; Ekman & Friesen, 1978; Ekman et al., 2002). These patterns of muscle activity are thought to reflect emotion signals. While Ekman did not investigate the development of facial expression of emotion, a number of investigators have modified his FACS system for use with younger samples. Oster's Baby FACS is an example of the use of the facial action coding system for examining emotion expression in infants and young children (Oster, 2006). In addition, there are now numerous studies that have used either full versions of FACS or Baby FACS with infants and children to examine emotion responses in child populations (e.g., Delgado, Messinger, & Yale, 2002; Rosenstein & Oster, 1988). Using these coding systems, individual responses to specific stimuli can be coded from videotape to provide an index of emotion expression. In addition, the Lab-TAB (Goldsmith & Rothbart, 1992, 1996) employs a standard set of tasks that may be used to elicit such emotion expression in both prelocomotor and locomotor infants, as well as preschool-age children.

A number of studies have used such batteries to assess various emotions. One example of the use of facial coding to examine emotion in 4- and 5-year-olds during an emotion-eliciting paradigm is a study by Cole and colleagues (Cole, Zahn-Waxler, & Smith, 1994), in which they used a paradigm designed to "disappoint" their subjects (subjects were led to believe they would receive a "good" toy but instead received a broken one). Cole and colleagues coded children's facial expressions of emotion to the receipt of the "bad" toy, including five basic emotions (joy, anger, sadness, fear, and disgust) and combinations of these emotions (i.e., blends), and a general emotional state of worry/distress. Using this type of approach, numerous studies have examined emotion in infants as young as 1 day of age. Studies have used facial coding of emotion to identify the presence of specific emotions. For example, there are a number of studies examining the expression of anger in infants (e.g., Field et al., 2005; Izard, Hembree, & Huebner, 1987; Lewis, Alessandri, & Sullivan, 1990) using different assessment strategies. Sternberg and Campos (1990) first coded anger expressions when they gave infants an attractive toy (or cookie) and then when they took it away. Doing this multiple times elicited anger in 5-month olds. Others have used a procedure in which a pacifier is repeatedly removed and returned to 1-month old infants, or in which 5-month-old infants' hands are held down while they are sitting upright (e.g., Calkins & Fox, 1992; Stifter & Braungart, 1995). In all instances, the frequency and intensity of anger expressions have been examined using facial coding systems that objectively identify changes in facial muscle movement and the configuration of patterns of expression denoting anger. While this type of emotion assessment may result in a more objective measure of emotion than

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self-report or parent report, it is important to realize that observational methods are vulnerable to distortion as well. If the period of observation is short, the range of behaviors observed may be constricted and provide an inaccurate view of a child's typical emotional expressions or reactivity. Therefore, many have argued for a combination of observational and parent-report measures (Calkins, Dedmon, Gill, Lomax, & Johnson, 2002; Matheny, Riese, & Wilson, 1985; Rothbart & Bates, 1998).

PHYSIOLOGICAL MEASURES

Another approach to the measurement of emotion has been the assessment of physiological responses, including heart rate, skin conductance, electromyographic (EMG) activity, and cortisol. However, unlike report-based or observational measures of emotional processes, physiological measures bring with them an additional level of complexity when examined in relation to the development of emotion. Each system must be understood in relation to the overall system, as well as in terms of the physical and psychological development of the individual (Fox, Schmidt, & Henderson, 2000). In addition, each of the physiological responses mentioned above has its own time course in the nervous system. Heart rate, measured usually in beats per minute or interbeat intervals in milliseconds, has a response course in seconds. Skin conductance, reflecting action of the sweat glands, is actually slower in its response course, while EMG activity reflecting muscle activity can be in the millisecond range. How long does an emotion last? If emotion is linked to facial expression, then emotions must be thought of as fast-acting, usually lasting no more than a few seconds. If, however, one thinks of creating a mood state rather than eliciting a particular discrete emotion, the duration is perhaps longer, allowing for the measurement of longer response systems. This is most obvious when one measures cortisol reactivity (see the chapter by Gunnar & Herrera in this handbook). Cortisol is a hormone that is the end-product of the HPA system. It is often interpreted as an indicator of stress. The latency of the HPA response and the time interval between a stressor and when a cortisol response may be picked up in the periphery are on the order of minutes—often 15 to 20 minutes. Thus, measurement of cortisol reactivity may occur long after the stressor is gone and perhaps long after the subject has evinced an emotional response. Therefore, conceptualization of what is being measured (i.e., an emotion, a mood state, a chronic or acute stressor) is critical with regard to choosing which physiological measure to examine. While it is beyond the scope of this chapter to review the methodological issues and conceptual associations between these physiological systems and emotion, it is important to understand the physiological basis for emotion, in addition to any observation or report of emotional states, expressions, or experiences (for more detailed reviews see Fox, Schmidt, Henderson, & Marshall, 2007; Larsen, Berntson, Poehlmann, Ito, & Cacioppo, 2008).

SUMMARY

The value of emotion and temperament research is predicated upon having reliable, valid means for measuring the phenomena in an objective fashion. Many studies have depended on a particular type of measurement, whether it is self-report or parent report, behavioral observations, or psychophysiology. However, typically there is moderate to low concordance among these levels of analysis (Kagan, 2007). In addition, each measurement technique has certain biases or limitations (Kagan & Fox, 2006). These problems are only magnified when trying to assess emotional processes in infants and children. Therefore, many have argued in favor of multimethod assessments, including report-based, observational, and physiological measures (Calkins & Howse, 2004; Rothbart & Bates, 1998). While these different tools may not always lead to the same results, examining them in concert may provide researchers with a more complete and clearer picture of how different emotional processes are interrelated.

Theories and Measurement: Summary

Throughout the history of emotion and temperament research, many theories and debates have ensued with regard to how infants and young children develop the skills, competencies, and behaviors necessary for expressing, experiencing, and understanding their social world. This work has also resulted in numerous measurement approaches and perspectives. However, it also should be informed by what we have learned about emotion from research in neuroscience. The link between the work in rodents and nonhuman primates on fear and reward conditioning served as an important bridge to theories of temperament and emotion in human populations (e.g., LeDoux & Phelps, 2008; Yin, Ostlund, & Balleine, 2008). Therefore, this work should continue to add to our understanding of the development of emotion processes. In addition,

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although researchers use a discrete or basic emotions approach, particularly when it comes to creating stimuli for use in neuroimaging tasks, the study of emotion has begun to take a dynamic systems perspective (Lewis, 2005), where integrated emotion processes occurring at multiple levels of analysis (i.e., emotion elicitors, emotion states, emotion expressions, emotion experiences) may be examined independently, as they are affected by the developmental process.

Currently most of this work has focused on the emotional processes involved in fear, as opposed to other discrete emotions. This focus is due to the progress in mapping the neural circuitry involved in fear behaviors and findings with animals regarding the contexts in which "fear" behaviors (i.e., freezing, defecating) are often subserved by similar neural pathways. If different behaviors are evident in the rodent, subserved by similar neural circuitry involved in fear conditioning, then observations of one particular behavior cannot represent the state of fear. Similarly, no one facial expression or behavior in the human can be said to represent fear. Rather, fear is a system of processes stemming from multiple levels of analysis, each of which becomes organized and integrated with the others throughout development. Summaries of the observational and behavioral work involved in the identification and development of emotion elicitors, states, expressions, and experiences can be found elsewhere (Kagan, 2007; Lewis, Haviland-Jones, & Barrett, 2008; Saarni et al., 2006). Therefore, the current review includes a summary of the underlying neural circuitry of the fear and the reward systems and discusses how this research informs our thinking about emotion development in human infants and children.

Neural Circuitry of Emotion

The majority of what is known about the neurobiology of the fear and reward systems stems from animal studies investigating the neural circuitry underlying aversive and appetitive learning. Because both fear and reward learning rely on structures within the limbic region of the brain, there is some overlap in the neural circuitry of these separate learning processes (for reviews, see LeDoux & Phelps, 2008; Yin et al., 2008). It has been established that the amygdala (lateral and central nucleus), hippocampus, medial prefrontal cortex (mPFC), and dorsolateral prefrontal cortex (dlPFC) play a central role in the various processes involved in Pavlovian or classical fear conditioning (LeDoux, 2000; LeDoux & Phelps, 2008). As well, the amygdala (basolateral and central nucleus), hippocampus, nucleus accumbens, dorsal striatum, and prefrontal cortex (particularly orbitofrontal cortex, mPFC, and cingulate) have been identified as brain regions involved in Pavlovian appetitive and instrumental conditioning (Everitt & Robbins, 2005). Recently, neuroimaging studies in humans have verified that similar neural circuitry for fear (Phelps & LeDoux, 2005) and reward (O'Doherty, 2004) processes have been preserved across species. In this section, the neural circuitry of the fear and reward systems will be reviewed briefly, and the remainder of this section will describe studies that have examined the development of the fear and reward systems using animal models as well as those using neuroimaging techniques in human pediatric populations.

Neural Circuitry of Fear

The amygdala is a subcortical region of the brain that is housed in the temporal lobes. Over 80 years ago, initial studies by Kluver and Bucy (1937; 1939) gave researchers the first clue that brain regions in the temporal lobe, such as the amygdala, play a significant role in the production of fear behaviors. After performing bilateral temporal lobectomy in nonhuman primates, Kluver and Bucy (1937, 1939) found that such lesions to the temporal lobes led to dramatic decreases in fear behaviors in the animals. With more precise ablation of the amygdala, Weiskrantz (1956) later reported finding similar behaviors in monkeys, thus more definitively determining the central role that the amygdala plays in the expression of fear. These findings, as well as several other studies conducted over the past 50 years, have led to the conclusion that the amygdala is necessary in the processing of emotional information (LeDoux, 2000; LeDoux & Phelps, 2008).

Much of what is known about the neural circuitry of fear has been revealed by examining classical conditioning of the fear response in rodents (LeDoux, 2000; LeDoux & Phelps, 2008). There are several processes of fear conditioning, including cue conditioning, context conditioning, and extinction, all of which involve distinct neural pathways associated with the amygdala. In initial cue conditioning, typically referred to as fear conditioning, a neutral stimulus (e.g., light, tone) is paired with an unconditioned stimulus (US), such as a shock, which naturally elicits an unconditioned fear response (e.g., freezing). Eventually,

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after several pairings of the neutral stimulus with the US, the neutral stimulus alone will come to elicit a conditioned fear response, thus establishing it as a conditioned stimulus (CS). In addition to becoming conditioned to a particular cue, individuals also learn to associate particular environmental cues with delivery of the US (i.e., context conditioning), thus demonstrating a conditioned fear response to the conditioning context. After conditioning, an individual learns to no longer associate the CS with the US through the process of extinction. During extinction, the CS is repeatedly presented in the absence of the US, therefore leading to a learned reduction in the fear response.

All processes of fear conditioning are dependent upon interconnections between various brain regions and the amygdala (Fig. 2.1; LeDoux & Phelps, 2008; Sotres-Bayon, Bush, & LeDoux, 2009). The amygdala consists of several nuclei, each with different connections within the brain (Pitkanen, Savander, & LeDoux, 1997). The most relevant nuclei to the different processes of fear conditioning are the lateral (LA), basolateral (BLA), accessory basal (AB), and central (CE) nuclei of the amygdala, all of which are differentially involved in fear conditioning processes. Cue conditioning involves the LA receiving and integrating information about the CS and US from thalamic and cortical areas and relaying this integrated information to the CE. The CE, in turn, relays this information to the brainstem to produce the behavioral, neuroendocrine, and autonomic responses associated with fear. In contrast, context conditioning involves the hippocampus, which creates a representation of the environment in which fear conditioning has taken place. This representation is transferred to the CE via the BLA and AB, which leads to the production of a fear response to the context as compared to the cue. Extinction processes involve the mPFC, which has been suggested to be involved in regulating amygdala responses to stimuli (Morgan, Romanski, & LeDoux, 1993; Quirk, Garcia, & Gonzalez-Lima, 2006; Sotres-Bayon et al., 2009). In addition, the dlPFC, a brain region that is unique in primates (Preuss & Goldman-Rakic, 1991) and necessary for higher cognitive functions (Miller & Cohen, 2001; Smith & Jonides, 1999), has been implicated in regulating the fear response (Ochsner, Bunge, Gross, & Gabrieli, 2002). However, there are few direct connections between the amygdala and the dlPFC (Stefanacci & Amaral, 2002), suggesting that the dlPFC must not mediate amygdala activation directly. Given the connections between the mPFC and both the amygdala and dlPFC, it has been suggested that the down regulation of the amygdala by the dlPFC may be mediated by the mPFC (LeDoux & Phelps, 2008).

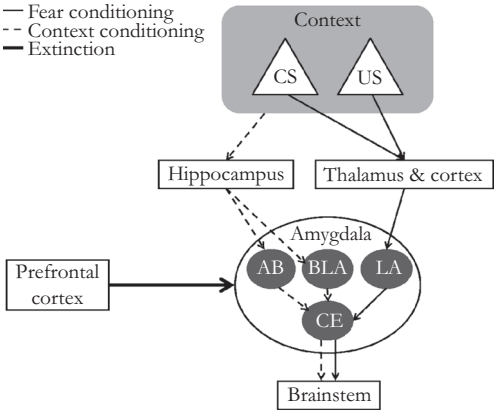

Figure 2.1. Neural circuitry of fear conditioning. During fear conditioning (thin black line), the thalamus and cortical regions receive information about the conditioned stimulus (CS) and unconditioned stimulus (US). This information is relayed to the central ~~(CE)~~ nucleus of the amygdala via the lateral nucleus (LA) and is eventually outputted to the brainstem to produce various behavioral, neuroendocrine, and autonomic responses related to fear. Conditioning of the context (dashed black line) takes place via the hippocampus, which relays information to the brainstem via the accessory basal (AB), basolateral (BLA), and CE nuclei of the amygdala. Extinction (thick black line) of the conditioned fear response occurs via regulation of the amygdala by the prefrontal cortex.

Neural Circuitry of Reward

The investigation of the neurobiology underlying emotions associated with receiving reward has primarily focused on the neural circuitry involved in both Pavlovian appetitive and instrumental conditioning (Everitt & Robbins, 2005; Yin et al., 2008). Pavlovian appetitive conditioning is conducted similarly as fear conditioning, but instead of pairing the CS with an aversive US, it is paired with an appetitive reward (e.g., food) and produces automatic goal-directed or approach responses (e.g., salivation, licking CS) rather than withdrawal or fear responses. In appetitive conditioning, the reward is presented regardless of the individual's behavior. In contrast, during instrumental conditioning, receiving a reward is contingent upon the individual's response. For example, animals learn to press a bar to receive a food reward such that the animal will receive the food reward only if it has pressed the bar.

Because fear and appetitive conditioning are both forms of Pavlovian conditioning, there is

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some overlap between the neural circuitry involved in both fear and reward. Similar to the pathways involved in fear conditioning, sensory information is received through thalamocortical pathways to the BLA, which is necessary for appetitive conditioning (Fig. 2.2; Alderson, Robbins, & Everitt, 2000; Whitelaw, Markou, Robbins, & Everitt, 1996). The BLA then transfers information about the reward to the CE, which leads to the expression of consummatory behaviors as well as autonomic and neuroendocrine responses associated with receiving reward. Information about the context in which appetitive conditioning occurs is transferred to the BLA via connections with the hippocampus (Everitt & Robbins, 2005).

Thus far, these neural pathways are similar to those described in fear conditioning. However, appetitive conditioning also relies upon additional neural processes. One key difference between the neurobiology of the fear and reward systems is that appetitive learning is dependent upon the neurotransmitter dopamine, which is projected from the ventral tegmental area (VTA)

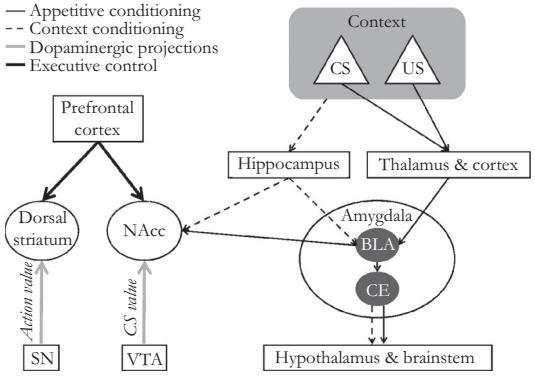

Figure 2.2. Neural circuitry of appetitive conditioning. During appetitive conditioning (thin black line), information about the conditioned (CS) and unconditioned stimulus (US) is relayed to the amygdala via the thalamus and cortical regions. The information is relayed to the central nucleus (CE) of the amygdala via the basolateral nucleus (BLA). The CE then transmits information to the hypothalamus and brainstem to produce consummatory behaviors and autonomic and neuroendocrine responses. During context conditioning (dashed black line), information about the context is transmitted to the BLA and CE of the amygdala via the hippocampus. The value of the cue and that of the context are relayed to the nucleus accumbens (NAcc) via the BLA and hippocampus respectively. The value of the CS is signaled to the NAcc via dopaminergic projections (thick gray line) from the ventral tegmental area (VTA). The value of learned action is signaled to the dorsal striatum via dopaminergic projections from the substantia nigra (SN). Consummatory behaviors are controlled via inputs from the prefrontal cortex to the dorsal striatum and NAcc (thick black line).

and substantia nigra (SN) (see Fig. 2.2; Yin et al., 2008). Dopaminergic projections from the VTA terminate in the nucleus accumbens (NAcc), while projections from the SN primarily terminate in the dorsal striatum. The NAcc mediates the effects of appetitive learning (Parkinson, Olmstead, Burns, Robbins, & Everitt, 1999) via connections from the BLA and hippocampus that transfer information about the value of the cue and context respectively (Cardinal & Everitt, 2004). The NAcc also receives inputs from the PFC (orbital, medial, and cingulate), which are involved in controlling goal-directed behaviors. The dorsal striatum also plays an important role in performance after appetitive learning has been acquired. Dopamine projections from the SN to the dorsal striatum reflect the value of the learned action as compared to the value of the CS, which is signaled by the dopaminergic projections from the VTA to the NAcc (Yin et al., 2008). In addition, this pathway between SN and dorsal striatum has been implicated in the formation of habits (Yin, Knowlton, & Balleine, 2004), which may also be controlled via inputs from the PFC to the dorsal striatum (Killcross & Coutureau, 2003). Although dopamine plays a primary role in the reward system, it should also be noted that the neurotransmitter serotonin interacts with the reward system through its projections from the raphe nucleus to the VTA and NAcc (De Deurwaerdere, Stinus, & Spampinato, 1998; Di Mascio, Di Giovanni, Di Matteo, Prisco, & Esposito, 1998) and it has recently been shown to mediate the effects of dopamine on appetitive conditioning (Alex & Pehek, 2007; Sanders, Hussain, Hen, & Zhuang, 2007).

SUMMARY

Ever since the initial studies by Kluver and Bucy (1937, 1939) and Weiskrantz (1956), there has been tremendous interest in the neural circuitry underlying emotions. Through the use of animal models of aversive and appetitive conditioning, the circuitry underlying both fear and reward has been successfully identified. There are several structures that are common to both fear and reward conditioning (i.e., amygdala, hippocampus, mPFC) as well as those that are different (i.e., NAcc, VTA). Although the majority of the studies identifying these neural circuits have been conducted with animals, recent research suggests that this circuitry is preserved across species (O'Doherty, 2004; Phelps & LeDoux, 2005). Therefore, it is likely that these neural systems play a significant role in the expression

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of human emotions related to both withdrawal and approach. Having a greater understanding of the development of such neural circuitry may help us better understand the development of emotions as well as individual differences in the expression of such emotions.

Neurodevelopmental Research of Fear and Reward

Translational Research

Given that the neural circuitry underlying the fear and reward systems has been highly conserved across mammals, it is reasonable to use animal models to map out the development of these systems. Recent research from Regina Sullivan's laboratory suggests that the neural circuitry involved in fear conditioning is not fully developed at birth (Moriceau & Sullivan, 2004a, 2004b; Moriceau, Wilson, Levine, & Sullivan, 2006; Sullivan, Hofer, & Brake, 1986; Sullivan, Landers, Yeaman, & Wilson, 2000). In these studies, rat pups were exposed to a novel odor that was paired with either an appetitive stimulus, such as tactile stroking (Sullivan, Brake, Hofer, & Williams, 1986; Sullivan, Hofer, et al., 1986), or an aversive stimulus, such as a shock (Moriceau & Sullivan, 2004a; Sullivan et al., 2000). An odor preference for the novel stimulus was observed before postnatal day 10 regardless of whether the stimulus was appetitive or aversive. However, after postnatal day 10, the rat pups exhibited no preference for the odor paired with the stroking and a showed an aversion to the odor paired with the shock. These results suggest that the neural circuitry underlying fear conditioning, particularly that involving the amygdala, is not fully developed until 10 days after birth. Interestingly, the time at which this fear system appears to come online coincides with the time at which walking is fully developed in the rat pup and greater exploration of the environment occurs (Bolles & Woods, 1965), leading Sullivan and colleagues to suggest that development of this fear circuitry is associated with the onset of behaviors that may reflect attachment in the rodent (Moriceau & Sullivan, 2005).

Research in neonatal nonhuman primates also suggests that amygdala development is important for the expression of fear behaviors as well as the appropriate expression of emotions during social interactions. Lesioning of the amygdala during the first weeks of life in macaque monkeys leads to significant decreases in fear of novel objects at 6 months of age (Prather et al., 2001), an effect that has previously been observed in lesioned adult monkeys (Weiskrantz, 1956). However, socioemotional behaviors were disrupted only in animals who received early lesions but not those who received lesions as an adult (Bauman, Lavenex, Mason, Capitanio, & Amaral, 2004a, 2004b; Prather et al., 2001). Specifically, early lesioning did not interrupt expressions of social behaviors; however, it increased the display of fear behaviors with a familiar conspecific (Prather, et al., 2001) and induced inappropriate approach behaviors toward an unfamiliar conspecific (Bauman, et al., 2004b) and indiscriminate approach behaviors toward an unfamiliar adult female (Bauman, et al., 2004a). Overall, amygdala damage early in life led to drastically inappropriate social behavior, although the expression of such behaviors was preserved. From these findings, the authors suggest that the development of the amygdala is not necessary for the direct expression of social behaviors but rather for the evaluation of potential threats in the environment that are used to produce appropriate social and emotional behavioral responses (Amaral et al., 2003).

Neuroimaging and Fear

In humans, neuroimaging techniques have been extremely useful in examining the underlying neurobiology of fear, primarily focusing on amygdala activation. Some studies have examined amygdala activation during fear conditioning while others have examined amygdala activation during the processing of emotional face stimuli. In adult populations, increased amygdala activation has been observed during presentations of a CS that predicted the presentation of an aversive stimulus (Buchel, Morris, Dolan, & Friston, 1998; LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998; Morris, Ohman, & Dolan, 1998). Similar heightened amygdala activation has been observed during the presentation of emotional faces (for review see Vuilleumier & Pourtois, 2007). Specifically, increased amygdala response has been consistently observed during presentations of fearful faces compared to neutral faces (Breiter et al., 1996; Morris et al., 1996), happy faces (Morris, et al., 1996; Whalen et al., 1998), or fixation crosses (Whalen et al., 1998). This increased amygdala response to fearful faces has been suggested to be the result of increased ambiguity of the source of threat associated with fearful expressions (Whalen, 1998).

Only within the past 10 years has fear conditioning been examined in a pediatric population (Grillon, Dierker, & Merikangas, 1998; Grillon et

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al., 1999; Reeb-Sutherland et al., 2009). These studies used an age-appropriate fear-potentiated startle paradigm in which two different colors are presented: one color is the CS that predicts the possible presentation of an aversive stimulus (e.g., airblast to the larynx) while the other color predicts no presentation of the aversive stimulus (Grillon et al., 1998, 1999). When startle probes (i.e., bursts of white noise) are presented during presentations of the CS, an increase in the EMG startle response is observed compared to presentations of startle probes during presentations of the "safety" cue. This increase in the startle response to the CS has been associated with increased amygdala activation in nonhuman animals (Davis, 2006). Similar to findings in adults (for review see Lissek et al., 2005), children showed an increased startle response to presentations of the threatening CS compared to presentations of the safety cue (Grillon et al., 1999).

Studies examining individual differences in the startle response have found that both familial risk of anxiety (Grillon et al., 1998) and behavioral inhibition (Reeb-Sutherland et al., 2009) alter the startle response. Grillon and colleagues (1998) examined fear-potentiated startle in a population of adolescents (7 to 18 years old) with a family history of anxiety. A differential startle response was found between high- and low-risk children, with different abnormalities observed in high-risk boys and girls. Specifically, high-risk girls showed an overall increase in startle response, even during presentations of the safety cue, while boys showed an increased startle only during presentations of the threatening CS (Grillon et al., 1998). Reeb-Sutherland and colleagues (2009) examined the startle response in behaviorally inhibited and noninhibited adolescents (13 to 17 years old) with and without a history of anxiety diagnoses and found that only behaviorally inhibited adolescents with a lifetime anxiety disorder showed an abnormal startle response. Specifically, anxious behaviorally inhibited adolescents displayed a heightened startle response to safety cues, while noninhibited adolescents and nonanxious behaviorally inhibited adolescents did not show such an increased response. This heightened response during safety cues has been consistently observed in adults with anxiety diagnoses (Lissek et al., 2005). Although there have not been many studies that have investigated fear conditioning in pediatric populations, the results thus far show that similar responses are observed in children as adults and that the startle response may be used as a biological marker of affective disorders such as anxiety.

Only a few studies to date have investigated amygdala response to facial expressions in normal children and adolescents. The first study to examine amygdala activation in a pediatric population was conducted by Baird and colleagues (1999). In this study, amygdala activation was examined in 12- to 17-year-old adolescents while they were presented with either fear faces or nonsense figures. It was found that adolescents display increased amygdala activation to the fear faces compared to the nonsense figures, a similar response to that displayed by adults (Whalen, 1998). However, when children were shown both fearful and neutral faces, they showed greater amygdala activation to neutral versus fearful faces, while adults showed an opposite pattern of amygdala activation (Thomas, Drevets, Whalen, et al., 2001). The authors suggested that this pattern of amygdala activation in children may reflect their interpretation of neutral faces as being more ambiguous than the fearful faces. Overall, these results suggest that the amygdala response to fear faces during childhood differs from that in adults.

A number of studies have investigated individual differences in the amygdala response to facial stimuli in relation to both temperament and anxiety. In a study investigating the amygdala response to novelty in adults who were identified as behaviorally inhibited at 2 years of age, it was found that behaviorally inhibited adults showed increased amygdala activation to novel neutral versus familiar neutral faces compared to noninhibited adults (Schwartz, Wright, Shin, Kagan, & Rauch, 2003). In a recent study by Perez-Edgar and colleagues (2007), the modulatory role of attention on amygdala activation was examined in adolescents (10 to 15 years of age) who were characterized as behaviorally inhibited during early childhood using an emotion face-rating task. In this study, adolescents were presented with happy, fearful, angry, or neutral faces and were asked to passively view the face, rate the width of the nose, report their subjective fear of the face, or report how hostile the face was. Similar to findings in adolescents with generalized anxiety disorder (McClure et al., 2007), behaviorally inhibited adolescents showed increased amygdala activation to fear faces when rating how afraid they were of the face compared to passive viewing of the fear faces. In addition, heightened amygdala activation to happy faces in behaviorally inhibited adolescents was observed when having to rate how afraid they were of the face, possibly reflecting increased uncertainty and novelty and therefore eliciting greater

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amygdala activity. Overall, these studies suggest that behaviorally inhibited individuals show heightened amygdala activation to novelty or uncertainty and that attention to emotional stimuli modulates amygdala activation among behaviorally inhibited individuals.

Individuals with anxiety disorders typically display heightened amygdala response to presentations of fearful or angry faces compared to nonanxious individuals (Phan, Fitzgeral, Nathan, & Tancer, 2006; Rauch, Shin, & Wright, 2003), and similar heightened amygdala responses have been observed in anxious adolescents (Thomas, Drevets, Dahl, et al., 2001). In addition, individuals with anxiety have been shown to have an increased attention bias to threat (for reviews, see Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Bradley, Mogg, White, Groom, & de Bono, 1999; Vuilleumier, Armony, Driver, & Dolan, 2001), which has been related to both hyperactivity in the amygdala response as well as decreased recruitment of the ventrolateral prefrontal cortex (vlPFC; Bishop, 2008). Furthermore, it has been suggested that this attention bias to threat may lead to the development of anxiety disorders (Pine, 2007). Monk and colleagues (2008) investigated the underlying neurobiology associated with attention bias to threat in adolescents with generalized anxiety disorder. In this study, the dot-probe task (Mogg & Bradley, 1999) was used to examine attention bias to threat. During the dot-probe task, both a threatening (i.e., angry or fearful) and a neutral face are presented simultaneously, followed by a target probe that appears in the location previously occupied by one of the two stimuli. The children and adolescents (mean age of 14 years) were asked to respond to the target as quickly and accurately as possible. Compared to nonanxious controls, anxious adolescents showed greater attention bias away from angry faces and displayed increased vlPFC activation to trials with angry faces (Monk et al., 2008). In addition, increased vlPFC activation was related to decreased symptom severity among anxious individuals, suggesting that increased activation of the vlPFC may help regulate symptoms in individuals with an anxiety disorder.

A recent study has investigated the development of emotion regulation using a novel emotional go/no-go task (Hare et al., 2008). During the emotional go/no-go task, children (7 to 12 years old), adolescents (13 to 18 years old), and adults (19 to 32 years old) were shown fear, happy, or calm faces and asked to press a button in response to frequent presentations of a particular emotional face (target) while inhibiting their response to a less frequently presented face (nontarget). In addition, trait anxiety was measured with the State-Trait Anxiety Inventory (STAI; Spielberger, 1983). Overall, adolescents showed greater amygdala activation compared to children and adults. Adolescents who reported being high on measures of trait anxiety showed less habituation to fear target faces than happy targets compared to nonanxious adolescents and adults. In addition, greater connectivity between ventral PFC (vPFC) and the amygdala was related to greater habituation to fear targets, suggesting that anxious adolescents have decreased connectivity between the vPFC and amygdala compared to adults. These individual differences in the vPFC–amygdala connectivity were suggested to underlie the development of affective disorders. Specifically, failing to consistently and continually regulate emotions may lead to aberrant or weaker connections between the amygdala and vPFC. Results from a study showing that amygdala–vPFC connectivity is weaker in children and adolescents with generalized anxiety disorder compared to controls give some support for this claim (Monk et al., 2008).

Neuroimaging and Reward

Within the past decade, there has been an increased interest in the neurobiology underlying reward in humans. In adult studies, it has been shown that receiving a reward increases activation in both subcortical (i.e., amygdala, striatum) and cortical (i.e., prefrontal cortex) regions, while omitting or taking away a reward decreases activation in these structures (Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001; Delgado, Nystrom, Fissell, Noll, & Fiez, 2000; Elliott, Newman, Longe, & Deakin, 2003; Knutson, Fong, Adams, Varner, & Hommer, 2001; O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001). Within the past 5 years, a number of studies have examined the development of these reward systems in children and adolescents (Bjork et al., 2004; Ernst et al., 2005; Eshel, Nelson, Blair, Pine, & Ernst, 2007; Galvan, Hare, Voss, Glover, & Casey, 2007; Galvan et al., 2006; Guyer et al., 2006; May et al., 2004). These studies have investigated neural processes underlying the receipt of a reward (Bjork et al., 2004; Ernst et al., 2005; Galvan et al., 2006, 2007; May et al., 2004), omission of or losing a reward (Ernst et al., 2005; May et al., 2004), anticipation of receiving a reward (Bjork et al., 2004; Guyer et al., 2006), and decision making about reward risks (Eshel et al., 2007).

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In an initial study by May and colleagues (2004), children and adolescents, aged 8 to 18 years, were asked to guess whether a hidden number was less than or greater than 5. If the children guessed correctly they would receive a monetary reward, but if they guessed incorrectly they would be punished by losing money. Participants displayed increased activation in the ventral striatum and orbitofrontal cortex (OFC) when receiving a reward and decreased activation in these areas when losing a reward, thus replicating findings in adults using a similar task (Delgado et al., 2000). Bjork and colleagues (2004) expanded upon these results by directly comparing reward-related neural circuitry of adolescents (12 to 17 years old) and adults (22 to 28 years old). In this study, a modified version of the monetary incentive delay (MID) task was used (Knutson et al., 2001; Knutson, Westdorp, Kaiser, & Hommer, 2000). In the Bjork study (2004), participants were required to respond with a button press as quickly as possible during the presentation of a target. The target was preceded by either a reward or loss cue that indicated whether the response to the target would result in receiving money or avoiding the loss of money. The cues also indicated the size (small, medium, or large) of the amount of monetary gain or loss. Adolescents showed similar increased activation in the NAcc as adults when they received a large reward. However, compared to adults, adolescents showed reduced recruitment of the ventral striatum and amygdala while anticipating responding for a reward (i.e., during presentation of reward cues). In a study using the MID task to examine individual differences in 10- to 15-year-olds, it was found that behaviorally inhibited adolescents showed increased striatal activation in anticipation of receiving a reward compared to noninhibited adolescents (Guyer et al., 2006). These studies demonstrate that adolescents differ from adults in activation of the reward system when anticipating a reward and that the sensitivity of this reward system differs across individuals.

Some studies have examined reward-related circuitry in relation to making decisions about risk in adolescents and adults (Ernst et al., 2005; Eshel et al., 2007). Using the wheel of fortune (WOF) task (Ernst et al., 2004), participants were asked to choose between high- and low-risk probabilities to receive a monetary reward. These studies reported that both adolescents and adults display increased amygdala and NAcc activation when receiving a reward compared to not receiving a reward (Ernst et al., 2005; Eshel et al., 2007). Ernst and colleagues (2005) reported that adolescents (9 to 17 years old) showed an increased NAcc response when receiving a reward compared to adults (20 to 40 years old), while adults showed a greater reduction in amygdala response to reward omission compared to adolescents. Using the same population, Eshel and colleagues (2007) expanded upon these results by examining differences in brain activation between adults and adolescents when making decisions about potential risks in obtaining a reward. Compared to adults, adolescents showed reduced activation of the OFC/vlPFC and dorsal anterior cingulate cortex. In addition, it was found that increased activation in these regions was related to decreased risk-taking behavior (Eshel et al., 2007). The authors suggest that these results together indicate that the more active reward-related subcortical regions in adolescents may be the result of a decreased recruitment of frontal regions used to regulate risk-taking behavior during adolescence.

Thus far, the studies described above have primarily examined differences in reward-related neural correlates between adolescents and adults without taking into consideration underlying reward systems in children prior to adolescence. However, studies by Galvan and colleagues (2006, 2007) have examined developmental differences in the neural circuitry of reward in groups of children (7 to 11 years old), adolescents (13 to 17 years old), and adults (23 to 29 years old). These studies used a matched-sample delay response task in which participants were presented one of three cues and asked to respond to the side on which the cue appeared by pressing a button. Participants who responded correctly received a predetermined amount of a monetary reward but no reward if they responded incorrectly. Each of the three cues presented was related to different amounts of reward. All participants displayed increased activation in the NAcc and OFC in relation to reward size (Galvan et al., 2006). Interestingly, adolescents showed the greatest increase in NAcc (compared to fixation) when receiving a reward compared to both children and adults, while children and adults did not differ on NAcc activation (Galvan et al., 2006). Expanding upon these findings, Galvan and colleagues (2007) found that increased changes in NAcc to receiving a reward were related to self-report of risk-taking behavior such that increased NAcc activation was related to increased engagement in risky behavior. It was also reported that OFC volume activation differed between children and adults, with adolescents displaying OFC volume activation between children

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and adults (Galvan et al., 2006). Therefore, adolescents are reported to have a hyperactive NAcc when receiving a reward; however, they do not have a mature prefrontal cortex to help regulate NAcc activity. It has been suggested that this disproportionate development between subcortical structures related to reward and cortical structures related to control functions may result in the increased risky behavior observed during adolescence (Casey, Jones, & Hare, 2008; Dahl, 2001; Giedd, Keshavan, & Paus, 2008; Hare & Casey, 2005; Steinberg, 2005).

SUMMARY

Research investigating the neurobiology underlying the fear and reward systems has primarily focused on adult populations, and only recently has the development of such systems been investigated in both animals and humans. Developmental studies with nonhuman animals suggest that the amygdala, a structure involved in both fear and reward, is not fully mature at birth and is necessary for the normal expression of socioemotional behaviors (Bauman et al., 2004b; Moriceau & Sullivan, 2005). Similarly, neuroimaging studies of pediatric populations have determined that the structures involved in the processing of fear and reward develop over time, with subcortical regions (i.e., limbic system) maturing prior to cortical regions (i.e., vlPFC, OFC). As well, subcortical regions develop in a nonlinear fashion; in contrast, the cortical regions' development is more linear. In addition, neuroimaging studies have shown that individual differences in activation of the fear and reward systems in the brain are associated with temperament as well as the development of anxiety disorders.

Gene × Environment Interactions

Throughout the literature on emotion and temperament, studies have focused on the heterotypic continuity of individual differences in expression and reactivity. However, there also are many examples of discontinuity. For example, almost half of all behaviorally inhibited toddlers do not display behavioral inhibition in later childhood, and between 30% and 80% of inhibited children do not develop internalizing disorders (Degnan & Fox, 2007). While infants' emotional tendencies or temperament may lead to certain outcomes, features of the environment are thought to have an important impact on these trajectories. Indeed, evidence from animal and human studies reveals the importance of contextual factors on the plasticity of social developmental outcomes (Hane & Fox, 2007). One recent theory of emotion development, the socialization internalization model, suggests that infants' expressions are linked to specific emotion states and behaviors through the infants' social interactions with caregivers (Camras & Fatani, 2008; Holodynski & Friedlmeier, 2006).

Environmental Influence

Multiple aspects of the environment are suggested to influence the development of emotion and temperament. Factors such as parenting behavior, personality, and the childcare context may have an impact on these developments across childhood (see Degnan, Almas, & Fox, 2010, for review). For example, maternal behavior with regard to infants' attention may dampen infants' response to distressing stimuli. From birth, adults engage and disengage infants' attention in order to alter their arousal levels. States of engaged attention are linked to infant positive affect and greater arousal in general, and adults who are sensitive to an infant's need to disengage attention help reduce this arousal before it becomes overwhelming (Gottman, Katz, & Hooven, 1997). Similarly, adults who are aware of an infant's frequent negativity or distress might help by distracting the infant from the source of distress. It has been suggested that through this cyclical process of attentional engagement and disengagement, infants learn how to use their attention to regulate their emotions and behavior (Fox, Henderson, et al., 2005). In general, maternal control or positivity has been examined as modulating the links between infant negative reactivity and internalizing or externalizing behavior problems (Calkins, 2002; Degnan, Calkins, Keane, & Hill-Soderlund, 2008; Degnan, Henderson, Fox, & Rubin, 2008; Rubin, Burgess, & Hastings, 2002; Smith, Calkins, Keane, Anastopoulos, & Shelton, 2004). In addition to parenting behaviors, there is also evidence for other contextual influences, such as placement in childcare, type of childcare settings, and maternal personality (e.g., neuroticism) and psychopathology (e.g., depression and anxiety), on the temperamental differences in emotion development (Almas et al., 2011; Calkins & Degnan, 2006; Degnan et al., 2010; Degnan & Fox, 2007; Phillips, Fox, & Gunnar, 2011; Rubin & Burgess, 2002). Despite the multiple studies showing these context-modulated effects, future work needs to explore the specific processes in which these factors are involved. Overall, investigators need to examine which contextual effects, in what situations, may truly influence children's emotions and behavior across development.

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Genes, Environment, and Psychopathology

One area of research that has illuminated possible processes by which the environment might influence emotion and personality development is that of behavioral genetics. Given that individual differences in emotion and temperament are theorized to stem from an underlying biological tendency, the role of genes in these effects is imperative to understand (Plomin, DeFries, McClearn, & McGuffin, 2001; Rothbart & Bates, 2006). Until more recently, the study of genes and constitutional factors (i.e., temperament) has been pitted against the study of environmental effects (Rutter, Moffitt, & Caspi, 2006). However, in the past decade, the acceptance of a multifactorial explanation of disorders and outcomes has led to numerous examinations of G × E interaction effects. In addition, collaborations with neuroscience have allowed researchers to explore specific processes and mechanisms involved in the environmental effects on development (Caspi & Moffitt, 2006). A review by Rutter and colleagues (2006) describes four ways that genes and environmental factors are interrelated: through the effect of the environment on gene expression, changes in genetic variation as a result of environmental circumstances, gene–environment correlations, and G × E interactions. Each type of gene–environment interplay has different implications, but combined, their results illustrate that the effects of genes and environment are inseparable. Here we focus on G × E interactions in relation to socioemotional outcomes to highlight the role of genetics and neuroscience in the study of emotion development in context. While the other types of gene–environment interplay are important, inclusion of all of them is beyond the scope of the current chapter. G × E interactions are especially relevant for the study of emotions and temperament in that there is evidence in the behavioral literature for environmental effects on emotional expression and temperament, as well as heterogeneity in children's developmental response to those contextual factors (Belsky & Pluess, 2009; Rutter et al., 2006).

ANXIETY AND DEPRESSION

A great deal of work supports the existence of G × E interaction effects in relation to anxious/depressive symptomatology or related behaviors. The majority of studies in this area have focused on the effects of the serotonin transporter (5-HTT) gene, which affects the rate of serotonin uptake (Lesch et al., 1996; Lesch, Greenberg, Higley, Bennett, & Murphy, 2002), and has been linked to the manifestation of both anxiety and depression (for review see Leonardo & Hen, 2006). Animal models using knockout mice have shown that mice lacking the 5-HTT gene exhibit greater anxiety- and depression-related behaviors than wild-type mice (Holmes, Lit, Murphy, Gold, & Crawley, 2003; Lira et al., 2003; Ramboz et al., 1998) suggesting that the 5-HTT gene is important in modulating the expression of anxiety and depression in mice. Furthermore, if pups' serotonin production was inhibited with fluoxetine, a selective serotonin reuptake inhibitor (SSRI), during the first weeks of life, they showed an increase in anxiety- and depression-like behaviors (Ansorge, Zhou, Lira, Hen, & Gingrich, 2004). Interestingly, there was a gene–treatment interaction in which mice who were heterozygous for the 5-HTT gene displayed the increased anxiety to the fluoxetine treatment, while wild-type mice did not. These results were interpreted to indicate that serotonin plays a critical role in the normal development of emotion-related neural circuitry and that individuals who naturally express low levels of 5-HTT may be at increased risk for developing a psychiatric disorder.

Examinations of the 5-HTT allele in humans have shown that adults with a short 5-HTT-linked polymorphic region (5HTTLPR) allele (*s/s* and/or *s/l*) tend to have a positive link between the level of stress in their environment and depressive symptoms in adolescence or adulthood, whereas those homozygous for the long allele (*l/l*) do not show this association (e.g., Caspi et al., 2003; Cervilla et al., 2007; Kendler, Kuhn, Vittum, Prescott, & Riley, 2005; Taylor et al., 2006; Wilhelm et al., 2006; Zalsman et al., 2006). In addition, the 5-HTTLPR has been shown to moderate the relation between maternal social support and children's behavioral inhibition at 7 years of age such that children with both a short 5-HTT allele and low maternal social support displayed increases on measures of behavioral inhibition as well as increased levels of maternally reported shyness (Fox et al., 2005). Moreover, Kochanska and colleagues found an interaction between this polymorphism and mother–child attachment security at 7 months of age in relation to children's inhibitory control in early childhood, as well as an interaction effect with maternal responsiveness in relation to attachment security itself (Barry, Kochanska, & Philibert, 2008; Kochanska, Philibert, & Barry, in press). However, there are also some studies that either show gender differences in the interaction of 5-HTTLPR and having a stressful life environment (e.g., Brummett

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et al., 2008; Eley et al., 2004; Grabe et al., 2005) or have found no evidence for the effect in their sample (e.g., Araya et al., in press; Gillespie, Whitfield, Williams, Heath, & Martin, 2005). Indeed, recent reviews suggest either that the effects of this interaction on depression have not been sufficiently replicated (see Munafo, Stothart, & Flint, 2009) or that there might be even more heterogeneity in this polymorphism that could explain these contradictory findings (see Lazary et al., 2008). Thus, more work is needed to elucidate the specific neural and environmental mechanisms that underlie these effects (Caspi & Moffitt, 2006).

The studies described above suggest that the environment, particularly the early life environment, in which an individual develops plays an important role in whether certain genes associated with risk for anxiety are manifested. However, these studies in humans lack control over the particular environment in which individuals are reared, therefore making it difficult to make definitive conclusions about the interactive effects of the genes with the environment. The use of animal models is necessary to tease apart these effects. Studies by Suomi and colleagues found that monkeys with a short 5-HTT allele showed impaired serotonergic functioning, increased stress reactivity, and excessive aggression—but only if they were reared in an adverse environment (i.e., separated from their mother and reared with other peers). In contrast, mother-reared monkeys with a short allele did not show such abnormalities. These results verify gene–environment interaction findings in human and further suggest that individuals with a short 5-HTT allele may be at increased risk for developing an affective disorder; however, the manifestation of a disorder is likely dependent upon having exposure to early adversity.

Additional research from Michael Meaney's laboratory has examined the effects of maternal care on the expression of genes that influence the development of the stress response system and subsequent expression of fear behaviors (for review Kaffman & Meaney, 2007). Specifically, they found that increased levels of maternal care (i.e., licking-grooming/arched-back nursing) were associated with an increased number of glucocorticoid receptors (GRs) in the hippocampus, leading to a more adaptive stress-response system (Liu, Diorio, Day, Francis, & Meaney, 2000; Liu et al., 1997) as well as a decreased fear of novelty in the offspring (Caldji et al., 1998; Menard, Champagne, & Meaney, 2004). In contrast, low levels of maternal care were associated with increased stress reactivity and fear of novelty. Recently, maternal care has been shown to affect the development of the stress response system at the level of the gene such that pups who received high levels of maternal care displayed increased methylation of the GR promoter compared to pups who received low levels of maternal care (Weaver et al., 2004). These findings suggest that care received early in life can alter the expression of genes that are associated with the underlying neurobiology of stress and fear. Furthermore, individual differences in the expression of fear or behavioral inhibition are supported by underlying biological factors. Thus, the research reviewed above suggests that both emotion and temperament are influenced by interactive effects of both early environmental context and gene expression.

ANTISOCIAL BEHAVIOR

Another line of G × E investigation has focused on the effects of the monoamine oxidase-A (MAO-A) gene, which affects the breakdown of serotonin, norepinephrine, and epinephrine (Shih, Chen, & Ridd, 1999). Indeed, in one of the first G × E studies reported in relation to mental health outcomes, Caspi and colleagues (2002) found that a combination of low MAO-A activity and child maltreatment (occurring between 3 and 11 years of age) was associated with higher rates of antisocial behavior, including conduct disorder, disposition toward violence, and rates of convictions in adolescence and adulthood (11 to 26 years of age). In another study, low MAO-A activity combined with the presence of an adverse childhood (8- to 17-year-olds) environment was associated with an increased risk for conduct disorder (Foley et al., 2004). However, additional studies have either failed to replicate this interaction effect or found it to have very little predictive power (Haberstick et al., 2005; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Prichard, Mackinnon, Jorm, & Easteal, 2008; Prom-Wormley et al., in press). Overall, the number of studies showing G × E interaction effects in regard to MAO-A and antisocial behavior or externalizing problems are limited. Further investigation is needed to determine why this genetic polymorphism seems less interactive with environmental effects.

Genes associated with dopamine have also been reported to interact with the environment in relation to both internalizing and externalizing types of psychopathology. Specifically, a dopamine transporter gene, DAT1, was related to levels of

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depression and suicidal ideation when adolescents (16 years of age) retrospectively reported greater maternal rejection behavior (Haeffel et al., 2008). Dopamine receptor genes, DRD2 and DRD4, are associated with externalizing or approach-motivated behavior, such as attention-deficit/hyperactivity disorder (ADHD), aggression, sensation seeking, and alcoholism. For instance, DRD2 was related to alcoholism when adults (18 to 87 years of age) reported high levels of stress (Madrid, MacMurrary, Lee, Anderson, & Comings, 2001). In addition, DRD4 has been shown to interact with positive parenting in relation to fewer externalizing behavior problems in toddlerhood (Bakermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, & Juffer, 2008; Propper, Willoughby, Halpern, Carbone, & Cox, 2007). A study by Eisenberg and colleagues (2007) also found that DRD4 interacted with the season a child was born in relation to reward-seeking personality traits in a sample of college students. Other DRD polymorphisms (DRD1 and DRD5) were found to interact with environmental factors such as maternal prenatal smoking and infant birth weight in relation to child antisocial behavior symptoms (Langley et al., 2008). Finally, Propper and colleagues (2008) found that DRD2 was related to poor vagal regulation at 3 and 6 months of age, but that when combined with greater maternal sensitivity, infants displayed better regulation by 12 months of age. Throughout the literature on dopamine-linked genes there is a wide range of findings, suggesting that there is heterogeneity in both the genetic and environmental effects associated with externalizing behaviors. However, there are also a number of studies that do not find gene × environment interactions in this domain (for review see Swanson et al., 2007).

Overall, there are few studies showing gene–environment interactions in regard to MAO-A and antisocial behavior or externalizing problems. In addition, research on dopamine-linked genes in relation to externalizing problems has shown mixed results. Given that individual differences in emotion and temperament, such as anger and frustration, are supported by biological factors, the research reviewed above is informative to the mechanisms influencing emotional development. While some of the findings suggest that environmental factors can alter the expression of genes in relation to antisocial and externalizing behavior problems, further investigation is needed to determine why this genetic polymorphism seems less interactive with environmental effects than those linked with stress and fear (e.g., 5-HTTLPR). Perhaps the underlying neurobiology of reward and anger is more variable throughout the population or more variable in the plasticity afforded in different contexts. Clarifying these questions would enhance the understanding of both emotion and temperament in relation to later antisocial and externalizing behavior problems and disorders.

SUMMARY

While the current review of gene × environment interaction effects is brief, it suggests the wide-reaching influence that this research domain has in the field. The recent influx of studies supporting the roles of both genetics and environment has come a long way since the twentieth century, but there is a lot more work to be done. As Caspi and Moffitt (2006) suggested, there is a mutually beneficial relationship between gene–environment studies and neuroscience. While neuroscience may provide the building blocks needed to formulate good hypotheses of gene–environment effects, these interaction effects then need to inform future neuroscience in order to fully explore the mechanisms behind these developmental processes (Caspi & Moffitt, 2006). In addition, investigators are beginning to bring more complexity into their analyses of both genes and environment. For instance, multiple studies have shown gene × gene × environment interactions in relation to outcome behavior. For instance, Cicchetti and colleagues (2007) found that levels of MAO-A had implications for the effect of 5-HTTLPR on depression and anxiety in the context of child maltreatment (see Wichers et al., 2008, for another example). While these few studies are not the norm, they strongly suggest that many of these outcomes may result from multiple genetic, environmental, and individual factors. Furthermore, studies of genetic effects in infant and child samples lend themselves to investigations of the effects of genes and the environment on each other and child behavior across time (e.g., Propper, et al., 2008). Recent theoretical work by Belsky and Pluess (2009) suggests that these G × E effects should influence development in both directions. A majority of the work reviewed above has focused on the vulnerability of specific types of individuals to the negative effects of adverse environments. However, these individuals may also be differentiated by their susceptibility to the positive effects of supportive, enriching environments (see Belsky & Pluess, 2009; Pluess & Belsky, 2010; Pluess, Belsky, & Neuman, 2009). Therefore, future investigations

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should focus on both ends of the environmental continuum when exploring the interplay of these genetic, environmental, and individual factors in relation to developmental patterns in emotion, temperament, and behavior over time.

Overall, individual differences in emotion and temperament are likely supported by underlying biological tendencies, which include genetic expression (Plomin et al., 2001; Rothbart & Bates, 2006). Therefore, the role of genes and the environment in the expression of various emotion components, temperament styles, and social-emotional outcomes is important to understand. Until recently, studies have focused on either genes and constitutional factors (i.e., temperament) or environmental effects (Rutter et al., 2006). Most recently, a multifactorial explanation of disorders and outcomes has been accepted and prompted an increase in the numerous examinations of gene × environment interactions in relation to outcome behavior. This collaboration between developmental psychology and neuroscience has allowed for the exploration of specific mechanisms involved in development in general and in relation to emotional expression and disorder specifically (Caspi & Moffitt, 2006).

Conclusions and Future Directions

An abundance of research has emerged in the past 20 years on the development of emotions, the role they play in children's emerging social lives, and the link between emotions and cognitive development. At the same time, much of this work has not been informed or linked to the growing corpus of data on the neurocircuitry of approach- and avoidance-related behaviors (i.e., fear and positive affect). In part, this is due to the lack of developmental neuroscience work with rodents and nonhuman primates (exceptions being the work of Hen and Sullivan) and the use of adult animals as models for examining fear and reward circuitry. However, the associations between the study of emotion development and the underlying neurocircuitry are increasing, as human developmental scientists recognize the importance of examining the neural underpinnings of emotion and as neuroscientists recognize the importance of early experience in molding neural circuits that lead to the development of adult behavior.

There are challenges on both sides of this divide that must be met in the future if progress towards a multilevel understanding of emotional development is to be made. Human developmental psychologists interested in emotion must become conversant with a large and complex neuroscience literature (Frijda, 2008). This can best be accomplished by focus on either the work on fear or the work on reward behavior (or both). In either case, as reviewed in this chapter, there is a wealth of information on the circuitry supporting these emotion/motivation states that can be helpful to developmentalists. At the same time, neuroscientists must address questions regarding the effects of early experience, and they must chart the role of experience in the development of neural circuits, instead of only studying the adult rodent or nonhuman primate. Neuroscientists must, as well, expand the repertoire of "emotions" that are studied with animal models. For example, work involving anger and disgust suggests that these are easily elicited and recognized with regard to human emotion behavior.

In addition to neural work on these emotions, there must be continued integration between cognitive processes and emotions in order to understand more complex social-emotional behaviors. Recent work in social neuroscience, examining complex social situations involving social rejection, empathy, altruism, and trust and the underlying neural correlates of these situations, should be expanded to explore their development. Developmental psychologists have examined the emergence of these complex behaviors, but less is understood about the development of the associated cognitive processes and their underlying neural correlates. Future work on the neural correlates of complex emotions, mood states, or characteristics of the individual should adopt a developmental perspective both in terms of emerging brain circuitry as well as the effects of experience on that brain architecture.

Finally, work that emphasizes the effects of early experience on developing brain circuitry involved in emotion could be the best way to understand significant individual differences in emotional life (i.e., temperament). Individual differences that can be examined from behavioral, experiential, and genetic approaches remain an important area of work that will provide greater understanding of differences in socioemotional trajectories over time.

Questions for Future Research

1. There has been a great deal of interest recently in the neuropeptide oxytocin and its role

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in social behavior, including empathy, trust, love, bonding, and attachment. What are the specific actions of oxytocin in the human brain, and how is it affected by variations in individual experience?

2. The work of Michael Meaney and others has suggested that early experience may alter gene expression in key structures of the brain associated particularly with the stress system. These alterations in gene expression, thought of as epigenetic action, may have profound effects on individual differences in emotion expressivity. Exactly how can these relations be studied in infants and young children?

3. Recent work in adult human neuroimaging studies has begun to describe the network of brain structures involved in complex emotions such as empathy, social relatedness, and social rejection. Advances in the use of functional imaging techniques with children may allow researchers to examine similar emotions in younger populations. However, will functional imaging studies take into account the development of connectivity and structures to fully understand neural development of complex emotions?

4. One of the important advances in prevention/intervention is the use of neuroscience-based training approaches. These training methods have been used to facilitate cognitive processes such as inhibitory control. Can similar approaches be used to prevent/intervene in instances of heightened anger or negative affect in children?

5. As neuroscience and developmental psychology researchers learn more about cognitive processes such as attention, working memory, and cognitive control, can this research be translated to the study of emotion and how affect perturbs basic cognitive or learning mechanisms?

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