Child Development, February 1997, Volume 68, Number 1, Pages 139–143

Temperament and the Reactions to Unfamiliarity

Jerome Kagan

The behavioral reactions to unfamiliar events are basic phenomena in all vertebrates. Four-month-old infants who show a low threshold to become distressed and motorically aroused to unfamiliar stimuli are more likely than others to become fearful and subdued during early childhood, whereas infants who show a high arousal threshold are more likely to become bold and sociable. After presenting some developmental correlates and trajectories of these 2 temperamental biases, I consider their implications for psychopathology and the relation between propositions containing psychological and biological concepts.

INTRODUCTION

A readiness to react to events that differ from those encountered in the recent or distant past is one of the distinguishing characteristics of all mammalian species. Thus, the events with the greatest power to produce both an initial orienting and sustained attention in infants older than 3 to 4 months are variations on what is familiar, often called discrepant events (Fagan, 1981; Kagan, Kearsley, & Zelazo, 1980). By 8 months of age, discrepant events can produce a vigilant posture of quiet staring and, occasionally, a wary face and a cry of distress if the event cannot be assimilated easily (Bronson, 1970). That is why Hebb (1946) made discrepancy a major basis for fear reactions in animals, why a fear reaction to strangers occurs in the middle of the first year in children growing up in a variety of cultural settings, and, perhaps, why variation in the initial behavioral reaction to novelty exists in almost every vertebrate species studied (Wilson, Coleman, Clark, & Biederman, 1993).

Recent discoveries by neuroscientists enrich these psychological facts. The hippocampus plays an important role in the detection of discrepant events (Squire & Knowlton, 1995). Projections from the hippocampus provoke activity in the amygdala and lead to changes in autonomic function and posture and, in older children, to reflection and anticipation (Shimamura, 1995). Because these neural structures and their projections are influenced by a large number of neurotransmitters and neuromodulators, it is reasonable to expect inherited differences in the neurochemistry of these structures and circuits and, therefore, in their excitability. Variation in the levels of, or receptors for, corticotropin releasing hormone, norepinephrine, cortisol, dopamine, glutamate, GABA, opioids, acetylcholine, and other molecules might be accompanied by differences in the intensity and form of responsivity to unfamiliarity (Cooper, Bloom, & Roth, 1991). This speculation is supported by research with infants and children (Kagan, 1994). This article summarizes what has been learned about two temperamental types of children who react in different ways to unfamiliarity, considers the implications of these two temperamental categories for psychopathology, and comments briefly on the relation between psychological and biological constructs.

INFANT REACTIVITY AND FEARFUL BEHAVIOR

About 20% of a large sample of 462 healthy, Caucasian, middle-class, 16-week-old infants became both motorically active and distressed to presentations of brightly colored toys moved back and forth in front of their faces, tape recordings of voices speaking brief sentences, and cotton swabs dipped in dilute butyl alcohol applied to the nose. These infants are called high reactive. By contrast, about 40% of infants with the same family and ethnic background remained motorically relaxed and did not fret or cry to the same set of unfamiliar events. These infants are called low reactive. The differences between high and low reactives can be interpreted as reflecting variation in the excitability of the amygdala and its projections to the ventral striatum, hypothalamus, cingulate, central gray, and medulla (Amaral, Price, Pitkanen, & Carmichael, 1992; Davis, 1992).

When these high and low reactive infants were observed in a variety of unfamiliar laboratory situations at 14 and 21 months, about one-third of the 73 high reactives were highly fearful (4 or more fears), and only 3% showed minimal fear (0 or 1 fear) at both ages. By contrast, one-third of the 147 low reactives were minimally fearful at both ages (0 or 1 fear), and only 4% displayed high levels of fear (Kagan, 1994).

The profiles of high and low fear to unfamiliar

 0009-3920/97/6801-0012$01.00

events, called inhibited and uninhibited, are heritable, to a modest degree, in 1- to 2-year-old middle-class children (DiLalla, Kagan, & Reznick, 1994; Robinson, Kagan, Reznick, & Corley, 1992). Further, high reactives show greater sympathetic reactivity in the cardiovascular system than low reactives during the first 2 years (Kagan, 1994; Snidman, Kagan, Riordan, & Shannon, 1995).

As children approach the fourth and fifth years, they gain control of crying to and reflex retreat from unfamiliar events and will only show these responses to very dangerous events or to situations that are not easily or ethically created in the laboratory. Hence, it is important to ask how high and low reactive infants might respond to unfamiliar laboratory situations when they are 4–5 years old. Each species has a biologically preferred reaction to novelty. Rabbits freeze, monkeys display a distinct facial grimace, and cats arch their backs. In humans, restraint on speech seems to be an analogue of the immobility that animals display in novel situations (Panksepp, Sacks, Crepeau, & Abbott, 1991), for children often become quiet as an initial reaction to unfamiliar situations (Asendorpf, 1990; Kagan, Reznick, & Gibbons, 1989; Kagan, Reznick, & Snidman, 1988; Murray, 1971). It is also reasonable to expect that the activity in limbic sites provoked by an unfamiliar social situation might interfere with the brain states that mediate the relaxed emotional state that is indexed by smiling and laughter (Adamec, 1991; Amaral et al., 1992).

When the children who had been classified as high and low reactive were interviewed at 4½ years of age by an unfamiliar female examiner who was blind to their prior behavior, the 62 high reactives talked and smiled significantly less often (means of 41 comments and 17 smiles) than did the 94 low reactives (means of 57 comments and 28 smiles) during a 1 hour test battery: $F(1, 152) = 4.51$, $p < .05$ for spontaneous comments; $F(1, 152) = 15.01$, $p < .01$ for spontaneous smiles. Although spontaneous comments and smiles were positively correlated ($r = 0.4$), the low reactives displayed significantly more smiles than would have been predicted from a regression of number of smiles on number of spontaneous comments. The high reactives displayed significantly fewer smiles than expected. Every one of the nine children who smiled more than 50 times had been a low reactive infant.

However, only a modest proportion of children maintained an extreme form of their theoretically expected profile over the period from 4 months to 4½ years, presumably because of the influence of intervening family experiences (Arcus, 1991). Only 19% of the high reactives displayed a high level of fear at both 14 and 21 months (>4 fears), together with low values (below the mean) for both spontaneous comments and smiles at 4½ years. But not one low reactive infant actualized such a consistently fearful and emotionally subdued profile. By contrast, 18% of low reactive infants showed the opposite profile of low fear (0 or 1 fear) at both 14 and 21 months together with high values for both spontaneous smiles and spontaneous comments at 4½ years. Only one high reactive infant actualized that prototypic, uninhibited profile. Thus, it is uncommon for either temperamental type to develop and to maintain the seminal features of the other type, but quite common for each type to develop a profile that is characteristic of the less extreme child who is neither very timid nor very bold.

The 4½-year-old boys who had been high reactive infants had significantly higher resting heart rates than did low reactives, but the differences between high and low reactive girls at this older age took a different form. The high reactive girls did not show the expected high negative correlation (−0.6 to −0.8) between heart rate and heart rate variability. It is possible that the greater sympathetic reactivity of high reactive girls interfered with the usual, vagally induced inverse relation between heart rate and heart rate variability (Porges, Arnold, & Forbes, 1973; Richards, 1985).

Honest disagreement surrounds the conceptualization of infant reactivity as a continuum of arousal or as two distinct categories. The raw motor activity score at 4 months formed a continuum, but the distribution of distress cries did not. Some infants never fretted or cried; others cried a great deal. A more important defense of the decision to treat high and low reactivity as two distinct categories is the fact that within each of the two categories variation in motor activity and crying was unrelated to later fearfulness or sympathetic reactivity. If reactivity were a continuous trait, then a low reactive infant with extremely low motor and distress scores should be less fearful than one who showed slightly more arousal. But that prediction was not affirmed. Second, infants who showed high motor arousal but no crying or minimal motor arousal with frequent crying showed developmental profiles that were different from those who were categorized as low or high reactive. Finally, high and low reactives differed in physical and physiological features that imply qualitatively different genetic constitutions. For example, high reactives have narrower faces than low reactives in the second year of life (Arcus & Kagan, 1995). Unpublished data from our laboratory reveal that the prevalence of atopic allergies among both children and their parents is significantly greater among high than low reactive infants. Studies of monozygotic and dizygotic same-sex twin pairs reveal significant heritability for inhibited and uninhibited behavior in the second year of life (Robinson et al., 1992). These facts imply that the two temperamental groups represent qualitatively different types and do not lie on a continuum of arousal or reactivity to stimulation.

The decision to regard individuals with very different values on a construct as members of the discrete categories or as falling on a continuum will depend on the scientists' purpose. Scientists who are interested in the relation, across families and genera, between brain size and body mass treat the two measurements as continuous. However, biologists interested in the maternal behavior of mice and chimpanzees regard these two mammals as members of qualitatively different groups. Similarly, if psychologists are interested in the physiological foundations of high and low reactives, it will be more useful to regard the two groups as categories. But those who are giving advice to mothers who complain about the ease of arousal and irritability of their infants may treat the arousal as a continuum.

IMPLICATIONS

The differences between high reactive–inhibited and low reactive–uninhibited children provoke speculation on many issues; I deal briefly with implications for psychopathology and the relation between psychological and biological propositions.

Anxiety Disorder

The high reactive infants who became very inhibited 4-year-olds—about 20% of all high reactives—have a low threshold for developing a state of fear to unfamiliar events, situations, and people. It is reasonable to expect that these children will be at a higher risk than most for developing one of the anxiety disorders when they become adolescents or adults. The childhood data do not provide a clue as to which particular anxiety profile will be most prevalent. However, an extensive clinical interview with early adolescents (13–14 years old), who had been classified 11 years earlier (at 21 or 31 months) as inhibited or uninhibited (Kagan et al., 1988), revealed that social phobia was more frequent among inhibited than among uninhibited adolescents, whereas specific phobias, separation anxiety, or compulsive symptoms did not differentiate the two groups (Schwartz, personal communication). This intriguing result, which requires replication, has interesting theoretical ramifications.

Research with animals, usually rats, suggests that acquisition of a fear reaction (e.g., freezing or potentiated startle) to a conditioned stimulus (light or tone) that had been paired with electric shock is mediated by a circuitry that is different from the one that mediates the conditioned response to the context in which the conditioning had occurred (LeDoux, 1995).

Davis (personal communication) has found that a potentiated startle reaction in the rat to the context in which light had been paired with shock involves a circuit from the amygdala to the bed nucleus of the stria terminalis and the septum. The potentiated startle reaction to the conditioned stimulus does not require that circuit. A phobia of spiders or bridges resembles an animal's reaction of freezing to a conditioned stimulus, but a quiet, avoidant posture at a party resembles a fearful reaction to a context. That is, the person who is extremely shy at a party of strangers is not afraid of any particular person or of the setting. Rather, the source of the uncertainty is a situation in which the shy person had experienced anxiety with other strangers. Thus, social phobia may rest on a neurophysiology that is different from that of specific phobia.

Conduct Disorder

The correlation between social class and the prevalence of conduct disorder or delinquency is so high it is likely that the vast majority of children with these profiles acquired their risk status as a result of life conditions, without the mediation of a particular temperamental vulnerability. However, a small proportion—probably no more than 10%—who began their delinquent careers before age 10, and who often committed violent crimes as adolescents, might inherit a physiology that raises their threshold for the conscious experience of anticipatory anxiety and/or guilt over violating community standards for civil behavior (Tremblay, Pihl, Vitaro, & Dubkin, 1994). Damasio (1994) and Mountcastle (1995) have suggested that the surface of the ventromedial prefrontal cortex receives sensory information (from the amygdala) that originates in the peripheral targets, like heart, skin, gut, and muscles. Most children and adults who think about committing a crime experience a subtle feeling that accompanies anticipation of the consequences of an antisocial act. That feeling, which might be called anticipatory anxiety, shame, or guilt, provides an effective restraint on the action. However, if a small proportion of children possessed a less excitable amygdala, or a ventromedial surface that was less responsive, they would be deprived of the typical intensity of this feeling and, as a result, might be less restrained than the majority (Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996; Zahn-Waxler, Cole, Welsh, & Fox, 1995). If these children are reared in homes and play in neighborhoods in which antisocial behavior is socialized, they are unlikely to become delinquents; perhaps they will become group leaders. However, if these children live in families that do not socialize aggression consistently and play in neighborhoods that provide temptations for antisocial behavior, they might be candidates for a delinquent career.

Biology and Psychology

The renewed interest in temperament has brought some psychologists in closer intellectual contact with neuroscientists. Although this interaction will be beneficial to both disciplines, there is a tension between traditional social scientists who describe and explain behavioral and emotional events using only psychological terms and a smaller group who believe that an acknowledgment of biological events is theoretically helpful. The recent, dramatic advances in the neurosciences have led some scholars to go further and to imply that, in the future, robust generalizations about psychological processes might not be possible without study of the underlying biology (LeDoux, 1995).

Although some neuroscientists recognize that the psychological phenomena of thought, planning, and emotion are emergent—as a blizzard is emergent from the physics of air masses—the media suggest, on occasion, that the biological descriptions are sufficient to explain the psychological events. This publicity creates a misperception that the biological and psychological are competing explanations when, of course, they are not. Vernon Mountcastle notes that although "every mental process is a brain process, . . . not every mentalistic sentence is identical to some neurophysiological sentence. Mind and brain are not identical, no more than lung and respiration are identical" (Mountcastle, 1995, p. 294).

Some neuroscientists, sensing correctly the community resistance to a strong form of biological determinism, are emphasizing the malleability of the neuron's genome to environmental events. A few neurobiologists have come close to declaring that the human genome, like Locke's image of the child's mind, is a tabula rasa that is subject to continual change. This position tempts citizens unfamiliar with neuroscience to conclude that there may be a linear cascade that links external events (e.g., loss of a loved one) directly to changes in genes, physiology, and, finally, behavior, with the psychological layer (e.g., a mood of sadness) between brain physiology and apathetic behavior being relatively unimportant. This error is as serious as the one made by the behaviorists 60 years ago when they assumed a direct connection between a stimulus and an overt response and ignored what was happening in the brain. Both corpora of evidence are necessary if we are to understand the emergence of psychological qualities and their inevitable variation. "The phenomena of human existence and experience are always simultaneously biological and social, and an adequate explanation must involve both" (Rose, 1995, p. 380).

ACKNOWLEDGMENTS

This paper represents portions of the G. Stanley Hall Lecture delivered at the annual meeting of the American Psychological Association, New York City, August 1995. Preparation of this paper was supported, in part, by grants from the John D. and Catherine T. MacArthur Foundation, William T. Grant Foundation, and NIMH grant 47077. The author thanks Nancy Snidman and Doreen Arcus for their collaboration in the research summarized.

ADDRESS AND AFFILIATION

Corresponding author: Jerome Kagan, Harvard University, Department of Psychology, Cambridge, MA 02138; e-mail: JK@WJH.HARVARD.EDU.

REFERENCES

Adamec, R. E. (1991). Anxious personality and the cat. In B. J. Carroll & J. E. Barrett (Eds.), *Psychopathology in the brain* (pp. 153–168). New York: Raven.

Amaral, D. J., Price, L., Pitkanen, A., & Carmichael, S. T. (1992). Anatomical organization of the primate amygdaloid complex. In J. P. Aggleton (Ed.), *The amygdala* (pp. 1–66). New York: Wiley.

Arcus, D. M. (1991). *Experiential modification of temperamental bias in inhibited and uninhibited children*. Unpublished doctoral dissertation, Harvard University.

Arcus, D. M., & Kagan, J. (1995). Temperament and craniofacial variation in the first two years. *Child Development, 66*, 1529–1540.

Asendorpf, J. B. (1990). Development of inhibition during childhood. *Developmental Psychology, 26*, 721–730.

Bronson, G. W. (1970). Fear of visual novelty. *Developmental Psychology, 2*, 33–40.

Cooper, J. R., Bloom, F. E., & Roth, R. H. (1991). *Biochemical basis of neuropharmacology*. New York: Oxford University Press.

Damasio, A. (1994). *Descartes' error.* New York: Putnam.

Davis, M. (1992). The role of the amygdala in conditioned fear. In J. P. Aggleton (Ed.), *The amygdala* (pp. 256–305). New York: Wiley.

DiLalla, L. F., Kagan, J., & Reznick, J. S. (1994). Genetic etiology of behavioral inhibition among two year olds. *Infant Behavior and Development, 17*, 401–408.

Fagan, J. F. (1981). Infant intelligence. *Intelligence, 5*, 239–243.

Hebb, D. O. (1946). The nature of fear. *Psychological Review, 53*, 259–276.

Kagan, J. (1994). *Galen's prophecy*. New York: Basic.

Kagan, J., Kearsley, R. B., & Zelazo, P. R. (1980). *Infancy*. Cambridge, MA: Harvard University Press.

Kagan, J., Reznick, J. S., & Gibbons, J. (1989). Inhibited and uninhibited types of children. *Child Development, 60*, 838–845.

Kagan, J., Reznick, J. S., & Snidman, N. (1988). Biological bases of childhood shyness. *Science, 240*, 167–171.

Kochanska, G., Murray, K., Jacques, T. Y., Koenig, A. L., & Vandegeest, K. A. (1996). Inhibitory control in young children and its role in emerging internalization. *Child Development, 67*, 490–507.

LeDoux, J. E. (1995). In search of an emotional system in the brain. In M. S. Gazzinaga (Ed.), *The cognitive neurosciences* (pp. 1049–1062). Cambridge, MA: MIT Press.

Mountcastle, V. (1995). The evolution of ideas concerning the function of the neocortex. *Cerebral Cortex, 5*, 289–295.

Murray, D. C. (1971). Talk, silence, and anxiety. *Psychological Bulletin, 75*, 244–260.

Panksepp, J., Sacks, D. S., Crepeau, L. J., & Abbott, B. B. (1991). The psycho and neurobiology of fear systems in the brain. In M. R. Denny (Ed.), *Fear, avoidance, and phobias* (pp. 17–59). Hillsdale, NJ: Erlbaum.

Porges, S. W., Arnold, W. R., & Forbes, E. J. (1973). Heart rate variability: An index of attention responsivity in human newborns. *Developmental Psychology, 8*, 85–92.

Richards, J. E. (1985). Respiratory sinus arrhythmia predicts heart rate and visual responses during visual attention in 14 to 20 week old infants. *Psychophysiology, 22*, 101–109.

Robinson, J. L., Kagan, J., Reznick, J. S., & Corley, R. (1992). The heritability of inhibited and uninhibited behavior: A twin study. *Developmental Psychology, 28*, 1030–1037.

Rose, R. J. 1995. Genes and human behavior. In J. T. Spence, J. M. Darley, & D. P. Foss (Eds.), *Annual review of psychology* (pp. 625–654). Palo Alto, CA: Annual Reviews.

Shimamura, A. P. (1995). Memory and frontal lobe function. In M. S. Gazzinaga (Ed.), *The cognitive neurosciences* (pp. 803–814). Cambridge, MA: MIT Press.

Snidman, N., Kagan, J., Riordan, L., & Shannon, D., 1995. Cardiac function and behavioral reactivity in infancy. *Psychophysiology, 31*, 199–207.

Squire, L. R., & Knowlton, B. J. (1995). Memory, hippocampus, and brain systems. In M. S. Gazzinaga (Ed.), *The cognitive neurosciences* (pp. 825–838). Cambridge, MA: MIT Press.

Tremblay, R. E., Pihl, R. O., Vitaro, F., & Dubkin, P. L. (1994). Predicting early onset of male antisocial behavior from preschool behavior. *Archives of General Psychiatry, 51*, 732–739.

Wilson, D. S., Coleman, K., Clark, A. B., & Biederman, L. (1993). Shy-bold continuum in pumpkinseed sunfish (*Lepomis gibbosus*): An ecological study of a psychological trait. *Journal of Comparative Psychology, 107*, 250–260.

Zahn-Waxler, C., Cole, P., Welsh, J. D., & Fox, N. A. (1995). Psychophysiological correlates of empathy and prosocial behavior in preschool children with behavioral problems. *Development and Psychopathology, 7*, 27–48.