- 118. Shonkoff J, Hauser-Cram P: Early intervention for disabled infants and their families—a quantitive analysis. Pediatrics (in press)
- 119. Siegel L. Correction for prematurity and its consequences for the assessment of the very low birth weight infant. Child Dev 54:1176-1188, 1983
- 120. Simeonsson R, Cooper D, Scheiner A: A review and analysis of the effectiveness of early intervention programs. Pediatrics 69:635-640, 1982
- 121. Smyth J, Tabachnik E, Duncan W et al: Pulmonary function and bronchial hyperactivity in long-term survivors of bronchopulmonary dysplasia. Pediatrics 68:336, 1981
- 122. Southall D, Richards J, Rhoden K et al: Prolonged apnea and cardiac arrhythmias in infants discharged from neonatal intensive care units. Failure to predict an increased risk for sudden infant death syndrome. Pediatrics 70:844, 1982
- 123. Taft L: Cerebral palsy. Pediatr Rev 6:35-45, 1984
- 124. Taft H, Roin J: Effect of furosemide administration on calcium excretion. Br Med J 1:437, 1971
- 125. Tepper R, Morgan W, Cota K et al: Expiratory flow limitation in infants with bronchopulmonary dysplasia. J Pediatr 109:1040-1046, 1986
- 126. Tronick E, Scanlon K, Scanlon J: A comparative analysis of the validity of several approaches to the scoring of the behavior of the preterm infant. Infant Behav Dev 8:395-411, 1985
- 127. Venkataraman B, Han B, Tsang R et al: Secondary hyperparathyroidism and bone disease in infants receiving long-term furosemide therapy. Am J Dis Child 137:1157, 1983
- 128. Vohr B, Garcia Coll C: Neurodevelopmental and school performance of very low-birthweight infants: A seven-year longitudinal study. Pediatrics 76:345-350, 1985
- 129. Vohr B, Bell E, Oh W: Infants with bronchopulmonary dysplasia: Growth pattern and neurologic and developmental outcome. Am J Dis Child 136:443, 1982
- 130. Volpe J: Evaluation of neonatal periventricular-intraventricular hemorrhage. Am J Dis Child 134:1023, 1980
- 131. Weinstein M, Oh W: Oxygen consumption in infants with bronchopulmonary dysplasia. I Pediatr 99:959, 1981
- 132. Wheeler W, Castile R, Brown E et al: Pulmonary function in survivors of prematurity. Am Rev Respir Dis 129:218, 1984
- 133. Yu V, Loke H, Bajuk B et al: Prognosis for infants born at 23 to 28 weeks' gestation. Br Med J 293:1202-1204, 1986
- 134. Zeskind P. Production and spectral analysis of neonatal crying and its relation to other biobehavioral systems in the infant at risk. In Field, Sostek (eds): Infants Born at Risk: Physiological, Perceptual and Cognitive Processes. New York, Grune & Stratton, 1983

HOB 2 **Boston City Hospital** 818 Harrison Avenue Boston, Massachusetts 02118

Double Jeopardy: The Impact of Poverty on Early Child Development

Steven Parker, MD,* Steven Greer, MD,† and Barry Zuckerman, MD‡

It is clear that poverty places children at risk for a variety of adverse behavioral and developmental outcomes; it is less clear why this is so. Children growing up in similarly impoverished environments commonly have very different outcomes. Even within the same family, one child may fail while another excels. The risk factors associated with poverty frequently, but not invariably, lead to untoward outcomes, for children. In this article we shall discuss the mechanisms by which poverty impacts on early developmental functioning and suggest interventions to ameliorate these effects.

Children living in poverty experience double jeopardy. First, they are exposed more frequently to such risks as medical illnesses, family stress, inadequate social support, and parental depression. Secondly, they experience more serious consequences from these risks than do children from higher socioeconomic status. It is the synergistic double jeopardy of increased exposure to and greater sequelae from environmental risks that predisposes children living in poverty to adverse developmental outcomes.

It is important to acknowledge an important limitation of the literature in this area: an inordinate emphasis is placed on the intelligence quotient (IQ) as an outcome measure. IQ tests are easily quantified and widely used. However, there are many other aspects of early childhood functioning of equal importance. Outcomes such as a child's sense of mastery, self-esteem, motivation to learn, and joy in life are rarely addressed. Consequently, the complexity and richness of childhood functioning are notably absent from most studies. The available data provide a very limited understanding of the impact of poverty on child development and, if anything, underestimate its true cost.

POVERTY AND CHILD DEVELOPMENT: A TRANSACTIONAL MODEL

Early theories of child development implied that negative developmental outcomes could result from a single risk factor, such as an early insult to the central

^{*}Assistant Professor of Pediatrics, Boston University School of Medicine; Director, Developmental Assessment Clinic, Boston City Hospital, Boston, Massachusetts

[†]Instructor in Pediatrics, Boston University School of Medicine; Fellow in Developmental and Behavioral Pediatrics, Boston City Hospital, Boston, Massachusetts

[‡]Professor of Pediatrics, Boston University School of Medicine; Director, Division of Developmental and Behavioral Pediatrics, Boston City Hospital, Boston, Massachusetts

nervous system. This approach is called the *main effect* model and implies a linear cause-and-effect relationship between risk and outcome. It was best articulated and gained wide acceptance after a retrospective study by Pasaminick and Knobloch, in which they hypothesized a "continuum of reproductive causality" to describe the relationship between perinatal factors (e.g., perinatal asphyxia, low birthweight, delivery complications) and poor outcomes (e.g., cerebral palsy, epilepsy, mental retardation, or learning disorders).

With such a model in mind, researchers in the 1970s were confronted with a curious fact: the best predictor of long-term developmental outcomes for infants born at risk was parental socioeconomic status (SES), rather than the type or degree of neonatal illness. For example, in a landmark followup study of 26,760 infants enrolled in the National Collaborative Perinatal Project, SES and maternal education were the factors most predictive of children's intellectual performance at 4 years. This finding was unexpected to the researchers, who included 158 biomedical and only 11 sociobehavioral independent variables in their analysis.

Perhaps the most elegant and sustained longitudinal study of developmental outcomes for children has been conducted by Werner. Her group has reported data at 18 years on 88 per cent of all 698 children born in 1955 on the island of Kauai, Hawaii. A significant interaction between the quality of the caretaking environment and the amount of perinatal stress has been apparent at each stage of followup. For example, 2-year-old children from high SES who had experienced perinatal complications had mean IQ scores 5 to 7 points lower than children from the same social class with no perinatal problems. In contrast, 2 year olds from low SES with perinatal complications had IQs 19 to 37 points less than their unstressed counterparts. Here

Werner found that children from high SES with the most severe perinatal complications had mean IQ scores similar to children with no perinatal complications from poor homes. The children with the most significant delays had experienced severe perinatal complications and grew up in the poorest homes. By 18 years of age, ten times as many children with poor behavioral or developmental outcomes lived in poverty than had been exposed to significant perinatal stress. Other studies^{6, 49} have since confirmed that poverty places children at greater developmental risk from perinatal insults.

In a recent study of 215 full-term children, Sameroff et al. ⁶⁷ demonstrated the cumulative nature of social risk factors in predicting IQ at 4 years. These risk factors included: maternal mental health problems, maternal anxiety, impaired motherchild interactions, low maternal education, negative parental attitudes and values, unemployment, minority group status, inadequate social support, large family size, and stressful life events. Although these risk factors tended to cluster in poor families, a cumulative deleterious effect was evident regardless of SES. In the highest SES group, for example, the mean IQ of 4 year olds with zero to one risk factor was 120, compared to a mean IQ of 100 for children exposed to four or more risk factors. The lowest SES group demonstrated a similar trend, with a mean IQ of 113 associated with zero to one risk factor and a mean IQ of 91 with 4 or more risk factors.

These data demonstrate that SES is a marker for potential psychosocial risk factors that may lead to developmental and behavioral morbidity. These factors additively or synergistically interact with the child's inherent strengths and vulnerabilities to shape outcomes. This viewpoint was articulated by Sameroff and Chandler in 1975 and called a *transactional* model of child development. It evolved out of the studies that demonstrated developmental outcomes to be largely unexplainable solely by the presence or degree of a biologic insult such as perinatal asphyxia, Leurologic insults, and prematurity, and prematurity, and unless these insults were of the most severe variety with clear organic

sequelae. In this model of development, outcomes can only be understood by considering the transaction between the *content* of the child's behaviors and the *context* in which they are manifested.

The transactional model addresses the dynamic interplay between the environment and the child. Characteristics of the child (e.g., genetic endowment, temperament, health) shape his or her responses to the environment. These interactions, in turn, transform environmental responsivity. Just as the child is shaped by his environment, so is the environment actively modified by the child. The child brings a host of attributes to the transaction. Three that have been particularly well studied are genetic endowment, temperamental style, and health status. These characteristics in part determine how the child will respond to the environment.

The environment likewise brings specific attributes to the transactions with the child. (In referring to environmental characteristics, the term low SES will be used interchangeably with poverty, although there are many other ways to define SES.) In a low SES environment, more risk factors for adverse developmental and behavioral outcomes are likely to be present. The most pertinent of these include increased stress, diminished social support, and maternal depression. These risk factors, in turn, exert their influence on the child through the quality of the home environment and the parent-child interactions, to name two of the most widely studied mechanisms.

As an example of a transactional view of child development, consider a child born at 34 weeks' gestation to a single mother living in poverty who received minimal prenatal care. Following a 3-week hospitalization, the infant was mildly hypotonic and had difficulty maintaining an alert state. The mother felt overwhelmed, depressed, and bereft of emotional support. The child's passivity engendered maternal feelings of inadequacy that resulted in a deepening of her depression. Positive interactions with her child were rare. The child did not look to the environment for stimulation and rarely vocalized. This further heightened the mother's feelings of inadequacy and depression. By 2 years the child was clearly delayed in his language and cognitive development.

What is the etiology of this child's developmental delays? Is it biologic vulnerability secondary to prematurity? Maternal depression? Temperamental passivity? Inadequate environmental stimulation? Insufficient social support? A transactional analysis considers all of these factors as operating together to shape this outcome. Each factor modifies and potentiates the other. Together they weave a complex pattern that cannot be understood by examining the thread of only a single risk.

While adding considerable complexity to the determinants of child outcomes, such a model also suggests practical strategies for intervention. Changes in any aspect of the ecology of the child's world (e.g., maternal depression) can create positive transformations in another (e.g., environmental stimulation). Although characteristics of the child and the environment are discussed separately in this article, these distinctions are arbitrary and for purposes of clarity only.

CHILD CHARACTERISTICS

Genetic Endowment

An analysis of the interaction between the child and her environment must begin with an understanding of the child's role in that transaction. No variable has been demonstrated to be a more powerful predictor of cognitive outcomes than the child's genetic endowment.

The most compelling data concerning the heritability of intelligence derives

from comparisons of identical twins raised together or apart. ¹⁰ In one study the correlation for cognitive development at age 36 years for monozygotic twins raised apart was 0.58, compared to 0.66 for identical twins raised together. In comparison, a summary of 11 studies found the average IQ correlation for adoptive siblings from different biologic parents to be 0.30. ¹⁰ Although studies of adopted children demonstrate IQ scores about 6 to 10 points higher than their biologic parents due to the enriched environment of the adoptive home, their IQs are still more closely correlated to the biological than the adoptive parents. ⁷¹ Thus, individuals with similar genotypes raised apart are far more alike in intellectual functioning than are individuals with disparate genotypes raised in similar environments.

Estimates of the contribution of genotype to the variance of IQs range from 40 to 60 per cent. This means that genotypes account for one half of the observed differences in IQs between individuals. Such data do not support strict nativists who believe genes are destiny, nor strict environmentalists who believe in the limitless potential for intellectual achievement in every person. Intelligence is mallcable, but only within the limits circumscribed by a child's genotype.

Scarr and McCartney have proposed a transactional resolution to this nature/nurture controversy. They believe that the child's genetic endowment drives development by determining early responsivity to the environment. These genotypically determined responses not only shape the interaction with the environment but also influence the kind of experiences sought by the child. This active influence of the child's genotype explains the variability of outcomes for children raised in similar environments and the startling similarities in personalities and intelligence of identical twins reared apart. Poverty's risk factors may be attenuated by a resilient constitution or accentuated by a child who requires more environmental stimulation to achieve his or her true potential.

Temperament

Every infant is born with a distinct temperamental style. The importance of temperament in the developmental process was first highlighted by the pioneering work of Thomas and Chess. They describe temperament as the "how" of behavior, as compared to motivations (the "why" of behavior) and abilities (the "what" of behavior). Nine temperamental dimensions are identified in their model: activity level, regularity of biologic functions, approach/avoidance tendencies, adaptability, responsivity to stimuli, intensity of reactions, quality of mood, distractibility, and persistence.

Thomas and Chess followed a cohort of 133 middle-class children from birth to young adulthood to relate early temperamental characteristics to long-term outcomes. Forty per cent of their sample were described as having an "easy" temperament, characterized by a predominantly positive mood, high adaptability to change, and a positive approach to unfamiliar stimuli. Children with irregular biologic functions, withdrawal from novel stimuli, and intense expressions of mood were characterized as "difficult" and constituted 10 per cent of the sample. A third group, labeled the "slow-to-warm-up child," constituted 15 per cent of the sample. The remainder of the children did not fall into any category.

Children with difficult temperaments were more likely to have behavioral problems in the first 5 years of life. 79 By early adulthood, however, this relationship was not seen. In analyzing the development of each child, Thomas and Chess found the nature of the interaction between the child's temperament and the environment to be the best predictor of long-term outcomes. They rated this interaction by its "goodness of fit." If the environmental expectations were compatible with the child's style, healthy development occurred. When the environment made inappropriate demands on the child, especially in relation to his innate temperament, a

poor fit resulted, which left that child vulnerable to behavioral dysfunction in young adulthood.

Temperamental characteristics have not been directly associated with SES. However, an easy disposition may moderate the impact of stress on a child. In the Kauai Longitudinal Study, for example, temperamental traits considered rewarding to caretakers, such as social responsiveness and a normal activity level, were associated with fewer learning and behavioral problems at age 10 years. Children who are more adaptable, less intense, and more responsive are less likely to manifest behavioral problems when stressed. Additionally, children with a positive mood, high regularity, and high adaptability are less likely to be the target of parental hostility, criticism, and irritability. Difficult temperaments are associated with a higher incidence of physical abuse when other family stresses are also present. A child with a difficult temperament is a source of added stress, especially to parents who already feel overwhelmed and unsupported.

Biologic Insults

Children born into poverty are at greater risk for a host of biologic insults that can affect outcomes. The data support both a higher prevalence and greater sequelae of illnesses for poor children.²⁷ In the prenatal period, for example, low SES increases the risk of contracting a cytomegalovirus (CMV) infection.² Infants infected with CMV from poor families have lower IQ scores and 2.7 times more school failure than do matched controls.³⁶ In contrast, these outcomes were not seen for infected infants of middle or upper class backgrounds. Poverty exposes children to double jeopardy from CMV infections: more exposure and greater developmental morbidity.

There are numerous other examples of increased biologic risks for children in poverty that are detailed in this volume. Prenatal insults to the developing nervous system from maternal drug use, malnutrition, intrauterine infections, or other medical illnesses are more common. The incidence of low birthweight is two to three times higher and developmental morbidity greater in low SES groups. Poor children are also far more likely to experience lead poisoning, failure to thrive, otitis media, and other infectious diseases. In reviewing the effects of biologic insults on child development, Shonkoff concludes that children in poverty carry a disproportionate burden of biologic vulnerability that is largely related to the increased health risks of poverty. . . . Their developmental outcomes will be determined by a highly complex series of transactions among a great number of biological and environmental facilitators and constraints." In the next section some of these environmental constraints will be discussed.

ENVIRONMENTAL RISKS

Stress

The concept of stress was introduced by Hans Selye⁷² in 1950 to explain a number of physiologic disorders caused by hypersecretion of the adrenal gland. The psychogenic causes of stress have since been catalogued,²⁵ but there is still no consensus as to its definition. Most recently, Garmezy and Rutter³³ have defined stress as a stimulus "requiring a change in adaptation (strain), mental state (distress), and bodily reaction or response."

People living in poverty experience stress more frequently and more chronically than do middle and upper class families. For example, Roghmann et al. 61 examined the rates of stressful life events in different social classes. The incidence of major stressors (e.g., housing problems, financial shortfalls, death of a relative or friend,

school difficulties) was two to four times greater for mothers with incomes of less than \$6000 (in 1969) than for those with more financial resources. Stress appears to be especially high for poor women with children under 6 years of age. 15

In addition to a higher frequency of stressful events, there is evidence that stress begets stress. For example, inadequate financial resources greatly exacerbate the problems experienced by children and parents in divorced families. 20 Equivalent levels of stress also engender more clinical depression among women from lower SES. 15 Chronic stress, in the form of unemployment, lack of material goods, and so forth is also more prevalent in poor families and far more likely to have negative consequences than acutely stressful events.8

Stress is associated with adverse consequences for parents and, directly or indirectly, for children. Children from highly stressed environments are at increased risk for a variety of developmental and behavioral problems, including poorer performance on developmental tests at 8 months, 30 lower IQ scores and impaired language development at 4 years,7 and poorer emotional adjustment and increased school problems at school age. 69 This last relationship is strongest for children in low SES/high-stress families and much less apparent for children in high SES/highstress families. These findings suggest that the psychological and material resources associated with higher social class buffer families from the vicissitudes of stress.

Increased stress interferes with the mother's ability to respond appropriately to her infant. It has been associated with impaired bonding behaviors between mothers and their premature infants³⁵ and less positive interactions at 4 months.²³ Toddlers in families exposed to high stress appear to be less secure in their attachment to their mothers regardless of social class. 80 There is also less consistency of attachment behaviors over time by these infants. 82 It is well established that insecure attachments are associated with an increased risk for subsequent behavioral and emotional problems. 42 The data suggest that stress causes negative outcomes by inhibiting positive interactions and the attachment between parent and child.

Inadequate Social Support

Social support is defined as "the availability of meaningful and enduring relationships that provide nurturance, security and a sense of interpersonal commitment."74 The benefits of social support fall into three categories: material supports (e.g., day care, nutritional supplements, availability of emergency help), emotional supports (e.g., friendships, counseling), and information/referral services (e.g., community resource availability, child-rearing techniques). Social support may derive from formal networks (e.g., health care providers, educational services, or peer groups) or informal networks (e.g., family, friends, or the media).81

Families living in poverty are at greater risk for experiencing inadequate social support. The most common problem mentioned by poor families living in hotel rooms, for example, is the lack of emotional support. 18 Pascoe et al. 54 demonstrated an association between low SES and low social support. Single parents are especially susceptible to social isolation.85 Since the absence of social support is particularly damaging to families under stress,23,48,74 their children are again placed in double jeopardy. For example, low levels of social support are associated with decreased cognitive abilities at 8 months, 30 more behavior problems among 5 to 8 year olds, 60 lower IQ and receptive language skills at 4 years,7 and a higher incidence of child abuse.38

Social support exerts its influence on childrens' development by direct and indirect means.17 Indirect effects occur by providing parents with access to emotional support, material assistance, external monitoring of their child rearing practices, and positive role models. These are especially important for infants and young children, who frequently have little contact with anyone other than their primary caretakers. The direct benefits of providing the child with cognitive and social

stimulation, emotional support, positive role models, and a widened social network may play a larger role, especially when they are lacking in the child's home

Adequate social support has been associated with enhanced parental functioning. Mothers with high social support appear to be more satisfied with their lives in general²³ and feel more positively about their maternal role. Additionally, the protective role of social support in reducing the incidence of maternal depression, 51 anxiety,46 and other psychiatric problems15 has been well established.

More positive parent-child interactions are seen in families with helpful support networks. Mothers who feel supported, for example, use less punishment and are more responsive to their 8-month-old infants³⁰ and are more actively involved with their infants in general.⁸¹ They continue to exhibit more optimal interactions with their children at 2 to 4 years. 45 This improvement in mother-child interactions helps to explain the positive relationship found between social support and secure attachment behaviors of toddlers who had been irritable infants.24

In addition to enhancing parent-child interactions, the presence of adequate social support is associated with a more stimulating and appropriate home environment for the child. Pascoe et al.54 demonstrated that, regardless of stress levels, mothers who reported more social support provided more stimulation to their 3year-old infants. Strong associations also were seen between social support and a more organized physical environment, the provision of appropriate play materials, and a wider range of available stimulation for the child. Numerous studies have shown the quality of the home environment to be one of the most powerful predictors of developmental outcomes for children. 11 By enabling stressed parents to fashion a more stimulating and responsive environment, social support can improve outcomes for children.

Maternal Depression

Depression is defined as a mood characterized by sadness, helplessness, gloom, loss of interest, emotional emptiness, and a feeling of "flatness." Inadequate financial resources, 43, 52 lower educational attainment, 84 recent immigrant status, 92 race,26 dissatisfaction with housing,47 stressful life events,22 and inadequate social support¹⁶ can all contribute to an increased incidence of depression. With these risk factors in mind, it is not surprising that numerous studies report an association between low SES and depression.^{3, 66} Brown et al., ¹⁵ for example, described the prevalence of depression in a London borough as 5 per cent for middle class women and 25 per cent for lower class women.

Mothers of young children are at much higher risk for becoming depressed. The prevalence of depression in such circumstances has been estimated from 12 per cent (when strict diagnostic criteria are used)12 to 52 per cent (when selfreported symptoms are used).47 The younger the mother at the time of her first child⁴⁵ and the greater the number of young children, ¹⁶ the greater the risk for depression. Low SES mothers of young children are therefore the most vulnerable for depression and, in conjunction with poor social support and increased stress, may experience the most severe consequences.

Maternal depression has been linked to adverse health outcomes for their children such as lower birthweights, ⁵⁰ more accidents, ¹⁴ failure to thrive, ⁵⁷ complaints of headaches and stomachs, ³⁶ and more surgical procedures. ⁸⁷ Maternal depression also is associated with a number of negative behavioral and developmental outcomes for children, such as sleep problems, 60, 97 depression, 5 attention deficit disorder, 87 socially isolating behaviors at school age, so and withdrawn and defiant behaviors

A number of studies have examined the mechanisms underlying these associations. Three-month-old infants will respond negatively when their mothers simulate a depressed mood. 19 Depressed mothers have been shown to display less spontaneity, more unhappy affect, fewer vocalizations, and diminished physical contact with their 4 month olds. 4 These infants already manifest fewer vocalizations and happy expressions toward their mothers.29 By the toddler stage, maternal depression is associated with infants demonstrating anxious and avoidant behaviors toward their mothers following a brief separation. 58, 77 The level of negative child outcomes engendered by maternal depression appears to vary with the degree to which depressive symptoms are evidenced in the parent-child interaction.

Maternal depression, like stress and diminished social support, places children living in poverty at double jeopardy for poor outcomes. These risk factors are highly intercorrelated and their effects are synergistic. Stress is exacerbated by a lack of support. Depression inhibits seeking adequate supports. The cycle becomes selfperpetuating because stress causes more depression, which elicits less support, which causes more stress. Ultimately, through the parent-child relationship and the quality of the home environment, these risks are passed on to the child.

PROTECTIVE MECHANISMS: STRESS-RESISTANT CHILDREN

We have discussed the world of children living in poverty as filled with potential risk factors that through their transactions with the child, lead to negative outcomes. However, as Garmezy noted over 15 years ago:

In the study of high risk and vulnerable children, we have come across another group of children whose prognosis could be viewed as unfavorable on the basis of familial or ecological factors, but who upset our prediction tables and in childhood bear the visible indices that are the hallmarks of competence: good peer relations, academic achievement, commitment to education and to purposive life goals, early and successful work histories. . . . Were we to study the forces that move such children to survival and to adaptation, the long range benefits to our society might be far more significant than our many efforts to construct models of primary prevention designed to curtail the incidence of vulnerability.31

Some view the issue of vulnerability versus resiliency as more semantic than real. Rather than designating "risk factors," one can as easily call their opposite "protective factors." If low social support is a risk factor, then high social support must be a protective factor. However, protective mechanisms are more than merely the absence of risk factors. The process by which resiliency occurs appears to be qualitatively different than that of vulnerability. 63 For example, a shy personality protects against delinquency, but an outgoing personality does not predispose one to antisocial behaviors. Focusing only on vulnerabilities prevents an understanding of how protective mechanisms shield children from risk.

A constructive approach is to identify the factors that promote successful adaptation in children. In the past decade, efforts to understand "invulnerable" children have begun. Garmezy has proposed three categories of protective factors:32 (1) the personality characteristics of the child; (2) a supportive, stable, and cohesive family unit; and (3) external support systems that enhance coping and project positive values.

Recently, investigators have looked to the child's self-concept as a key determinant of successful outcomes. 63 It is suggested that children with positive feelings of self-esteem, mastery, and control can more easily negotiate stressful experiences. These children elicit more positive experiences from their environment. They show initiative in task accomplishment and relationship formation. Even in stressed families, the presence of one good relationship with a parent reduces psychiatric risk for children. 62 For older children, the presence of a close, enduring relationship with an external support figure (e.g., schoolteacher) may likewise serve a protective

function. A child with a positive self-concept seeks, establishes, and maintains the kind of supportive relationships and experiences that promote successful outcomes. These successes enhance the child's self-esteem and sense of mastery, which leads to further positive experiences and relationships. The cycle of success can be as self-perpetuating as that of failure. One goal of social support and therapeutic interventions must be to establish environments and relationships for the child that

IMPLICATIONS FOR INTERVENTION

The developmental costs of poverty for children are excessively high. Approximately two thirds of all children who test as mildly retarded have grown up in poverty.73 The cost to society can be measured in terms of school dropout, unemployment, delinquency, unwanted pregnancies, and the intergenerational perpetuation of failure. Successful interventions require both the amelioration of risk factors and the enhancement of protective mechanisms.

The care of adolescent mothers and their infants is an example of the necessity of such an approach. It is known that the adolescent mother's nutritional status plays an important role in the increased incidence of low birthweight among their infants.98 The child's subsequent developmental and social functioning will be related to birthweight, as well as social support from the maternal grandmother, 99 the level of involvement of the baby's father, and the mother's mental health status. 44 Optimal interventions for adolescent mothers should address all of these factors by providing ongoing nutritional, emotional, and psychological support for

The most ambitious and comprehensive intervention of this type has recently begun in Chicago. This program, called the Beethoven Project, is providing pregnant women living in poverty and their children with nutritional, medical, educational, and social support from the prenatal period through the first 5 years of the child's life. It is an exciting effort that addresses child health and the ecology of family functioning in an early, continuous, and comprehensive fashion. If it proves successful and cost effective, it will serve as a model program in the future.

Transactional theory implies that even partial interventions may benefit other areas of functioning. For example, improving the health status of children can lessen their vulnerability to a disorganized home environment. Interventions also have benefits beyond their intended scope. The first studies of the Head Start programs, founded in the 1960s, revealed only short-term gains in IQ scores. More creative researchers then looked at other areas of functioning and discovered marked longterm benefits in such areas as diminished special education services and less grade retention. 40 Subsequent studies have demonstrated that early intervention services decrease juvenile delinquency, teenage pregnancy, and unemployment during young adulthood.9 These studies have lead the House Select Committee on Children, Youth, and Families to estimate that every \$1.00 spent on preschool education saves at least \$4.75 in later educational and social costs.³⁷

Early intervention is effective for children at biologic 76 or environmental risk. 59 Current research is examining the necessary and sufficient interventions to achieve long-term benefits. Until this question is resolved, current wisdom dictates that interventions start early, be comprehensive, and continue for as long as possible.

The first level of intervention must address basic needs for children and their parents living in poverty. Such interventions are best accomplished by public health measures such as the provision of material support (e.g., food, shelter, and money) and accessible medical care. The emphasis on improved prenatal care for women at risk, for example, has reduced the incidence of low birth weight deliveries and

saved \$3.38 in medical costs for every \$1.00 invested.37 The developmental benefits of these interventions, although more difficult to quantify, may be even more impressive.

Pediatricians are comfortable with assessment and intervention for medical risks. However, the prevention of developmental and behavioral morbidity secondary to poverty requires a broader focus. During a health care visit the family's material resources for nutrition, housing, and other finances should be addressed. The clinician can evaluate the child's temperamental characteristics and the "goodness of fit" with the environment. The earlier a mismatch is identified, the more successful can be the interventions.

The clinician should also attend to potential social risks for the child in the environment. Is there significant family stress? Do the parents appear depressed and overwhelmed? What is the level of social support available for the family? What is the quality of the parent-child interaction seen in the office? What sort of home environment awaits the child after the visit? Any or all of these questions can be addressed in the medical setting. The answers will determine the appropriate interventions. For one family, social support in the form of a visiting nurse may be helpful. For another, advocacy for adequate housing is necessary. A third may require a comprehensive early intervention program to provide the child with adequate stimulation and emotional nurturance.

Finally, the clinician should try to identify these risks to developmental and behavioral outcomes as early as possible. Developmental screening tests alone, however, poorly predict long-term problems unless the context of the child's environment also is considered. Using such an approach at Boston City Hospital, early childhood educators have been integrated into medical settings such as the failure to thrive, lead poisoning, adolescent mother, and neurology clinics, as well as the inpatient wards. The goal has been to identify children in need of services to prevent developmental morbidity. The assessment includes not only developmental testing but an evaluation of the child's learning style, self-esteem, sense of mastery, the parent-child interaction, "goodness of fit," and health and social risk factors. Using this approach, 23 per cent of the first 853 children were judged to be in need of new or additional services. Health care settings provide the earliest and best opportunity to intervene with children at risk. The challenge for us all is to recognize risk early and draw on available resources to eliminate the double jeopardy for children living in poverty.

ACKNOWLEDGMENT

Support was provided by Academic Training Program in Behavioral Pediatrics funded by Bureau of Health Care Delivery and Assistance, Maternal and Child Health Branch (Grant #MCJ-009094), the Boston Foundation, and the Jessie B. Cox Charitable Trust. We thank Ms. Margaret Stanhope and Ms. Jeanne McCarthy for their help in preparing the manuscript for this article. Bill Harris's tireless efforts and commitment to improve the health and wellbeing of children living in poverty has been a special source of support and inspiration.

REFERENCES

- 1. Abernathy V: Social network and response to the maternal role. Intl J Soc Family 3:86-92, 1973
- 2. Alford C: Prenatal infections and psychosocial development in children born into low socioeconomic settings. In Mittler P (ed): Research in Mental Retardation. Baltimore, University Park Press, 1977, pp 251-259
- 3. Aneshensel C, Clark V, Fredrichs R: Race, ethnicity and depression: A confirmation analysis. J Personality Soc Psych 44:385-398, 1983

- 4. Bates J: Temperament in infancy. In Osofsky J (ed): Handbook of Infant Development. New York, John Wiley and Sons, 1987
- 5. Beardslee W, Keller M, Klerman G: Children of parents with affective disorders. Int J Fam Psychiatry 6(3):283-299, 1985
- 6. Beckwith L, Parmelee A: EEG patterns of preterm infants, home environment, and later IQ. Child Dev 57:777-789, 1986
- 7. Bee H, Hammond M, Etres S et al: The impact of parental life changes of the early development of children. Res Nurs Health 9:65-74, 1986
- 8. Belle D: The social network as a source of both stress and support to low income mothers. Presented at the Society for Research in Child Development, 1981
- 9. Berrueta-Clement J, Schweinhart L, Barnett W et al: Changed Lives: The Effects of the Perry Preschool Project on Youths Through Age 19. Ypsilanti, Michigan, High Scope Educational Research Foundation, 1986
- 10. Bouchard T, McGue M: Familial studies of intelligence: A review. Science 212:1055-
- 11. Bradley R, Caldwell B: Pediatric usefulness of home assessment. Adv Behav Pediatr 2:61-
- 12. Bromet E, Solomon Z, Dunn L et al: Affective disorders in mothers of young children. Br J Psychiatry 140:30-36, 1982
- 13. Broman S, Nichols R, Kennedy W: Preschool IQ: Prenatal and Early Developmental Correlates. Hillsdale, New Jersey, Laurence Erlbaum, 1975
- 14. Brown G, Davidson S: Social class: Psychiatric disorder of mothers and accidents to children. Lancet 1:378, 1978
- 15. Brown G, Bhrolchain M, Harris T: Social class and psychiatric disturbance among women in an urban population. Soc 9:225-254, 1975
- 16. Brown G, Harris T: The Social Origins of Depression. London, Tavistock Publications,
- 17. Cochran J, Brassard J: Child development and personal social networks. Child Dev 50:601-616, 1979
- 18. Cohen C, Adler A: Assessing the role of social network interventions with an inner city population. Am J Orthopsychiatry 56:278-288, 1986
- 19. Cohn J, Tronick E: Three-month-old infants' reaction to simulated maternal depression. Child Dev 54:185-193, 1983
- 20. Colletta N: Divorced mothers at two income levels: Stress, support and child rearing practices. Dissert Abstr Intl 38(12-B):6114, 1978
- 21. Corah N, Anthony E, Painter P et al: Effects of perinatal anoxia after seven years. Psychol Mongr 79:3, 1965
- 22. Costello C: Social factors associated with depression: A retrospective community study. Psychol Med 12:329-339, 1982
- 23. Crnic K, Greenberg M, Ragozin A et al: Effects of stress and social support on mothers and premature and full term infants. Child Dev 54:209-219, 1983
- 24. Crockenberg S: Infant irritability, mother responsiveness, and social support influences on the security of infant-mother attachment. Child Dev 52:857-865, 1981
- 25. Drillien C: A longitudinal study of the growth and development of prematurely and maturely born children: Mental Development at 2-5 years. Arch Dis Child 36:233,
- 26. Eaton W, Kessler L: Rates of symptoms of depression in a national sample. Am J Epidemiol 114:528-538, 1981
- 27. Egbuonu L, Starfield B: Child health and social status. Pediatrics 69:550-557, 1982
- 28. Escalona S: Babies at double hazard: Early development of infants at biologic and social risk. Pediatrics 70:343-350, 1982
- 29. Field T: Early interactions between infants and their postpartum depressed mothers. Infant Behav Dev 7:517-522, 1984
- 30. Garcia-Coll C, Vohr B, Hoffman J et al: Maternal and environmental factors affecting developmental outcome of infants of adolescent mothers. Dev Behav Pediatr 7:230-236, 1986
- 31. Garmezy N: Vulnerability research and the issue of primary intervention. Am J Orthopsychiatry 41:101-116, 1971
- 32. Garmezy N: Stress, competence and development: Continuities in the study of schizo-

- phrenic adults, children vulnerable to psychopathology, and the search for the stressresistant child. Am J Orthopsychiatry 57:159-174, 1987
- 33. Garmezy N, Rutter M: Stress, Coping and Development in Children. New York, McGraw Hill. 1983
- 34. Ghodsian M, Zajicek E, Wolkind S: A longitudinal study of maternal depression and child behavior problems. J Child Psychol Psychiatr 25:91-109, 1984
- 35. Grossman P: Prematurity, poverty related stress, and the mother-infant relationship. Dissert Abstr Intl 40(4-B):1954, 1979
- 36. Hanshaw J, Scheiner A, Moxley A et al: School failure and deafness after silent congenital cytomegalovirus infection. N Engl J Med 295:468-470, 1976
- 37. House Select Committee on Children, Youth, and Families: Yearly Report, 1986
- 38. Hunter F, Kilstrom N: Breaking the cycle in abusing families. Am J Psychiatry 136:1320-
- 39. Illsley R: Early prediction of perinatal risk. Proc Royal Soc Med 59:181-184, 1966
- 40. Lazar I, Darlington A: Lasting effects after preschool. Monogr Soc Res Child Dev 47 (2 and 3):1982
- 41. Lerner R, East P: The role of temperament in stress, coping and socioemotional functioning in early development. Infant Ment Health I 5:148-159, 1984
- 42. Main M, Kaplan K, Cassidy J: Security in infancy, childhood and adulthood. Monogr Soc Res Child Dev 50:66-104, 1985
- 43. Makosky V: Sources of stress: Events or conditions? In Belle D (ed): Lives in Stress: Women and Depression. Beverly Hills, Sage Publications, 1982, pp 35-53
- 44. Maziade M, Boudreault M, Thivierge I et al: Infant temperament: SES and gender differences and reliability of measurement in a large Quebec sample. Merrill-Palmer Q 30:213-226, 1984
- 45. McGee R, Williams S, Kaskani J: Prevalence of self-reported depressive symptoms and associated social factors of mothers in Donedin. Br J Psychiatry 143:473-479, 1983
- 46. Miller P, Ingham J: Friends, confidents and symptoms. Soc Psychiatry 11:51, 1976
- 47. Moss P, Plewis I: Mental distress of preschool children in inner London. Psychol Med 7:614-652, 1977
- 48. Mueller D: Social networks: A promising direction for research on the relationship of the social environment to psychiatric disorder. Soc Sci Med 40:147-161, 1980
- 49. National Center for Health Statistics: Factors associated with low birthweight: 1976. Pub. # 80-1915. Washington, D.C., U.S. Dept. of Health and Human Services, 1980
- 50. Niswander K, Gordon M (eds): The Collaborative Perinatal Study of the National Institute of Neurologic Diseases and Stroke. Philadelphia, W.B. Saunders, 1972
- 51. Nuckolls K, Cassel J, Kaplan B: Psychosocial assets, life crisis and the prognosis of pregnancy. Am J Epidemiol 95:431-441, 1972
- 52. Orr S, James S: Maternal depression in an urban pediatric practice: Implications for health care delivery. Am J Public Health 74:363-365, 1984
- 53. Parmelee A, Michaelis R: Neurologic Examination of the Newborn. In Hellmuth J (ed): Exceptional Infants: Studies in Abnormalities. New York, Brunner/Mazel, 1971
- 54. Pascoe J, Loda F, Jeffries V, Earp J: The association between mothers' social support and provision of stimulation to their children. [Dev Behav Pediatr 2:15-19, 1981
- 55. Pasaminick B, Knobloch H: Retrospective studies on the epidemiology of reproductive casuality: Old and new. Merrill-Palmer Q 12:7-26, 1966
- 56. Plomin R: Behavioral genetics and intervention. In Gallagher J, Ramey C (eds): The Malleability of Children. Baltimore, Brookes, 1987, pp 15-24
- 57. Pollitt E, Eichler A, Chan C: Psychosocial development and behavior of mothers in failure to thrive syndrome. Am J Orthopsychiatry 45:525, 1975
- 58. Radke-Yarrow M, Cummings E, Kuczynski L, et al: Patterns of attachment in two- and three-year-olds in normal families and families with parental depression. Child Dev 56:884-893, 1985
- 59. Ramey C, Campbell F: Preventive education for high-risk children: Cognitive consequences of the Carolina Abecedarian Project. Am J Ment Defic 88:515-523, 1984
- 60. Richman N: A community survey of characteristics of one- to two-year-olds with sleep disruptions. J Am Acad Child Psychiatry 20:281-291, 1981
- 61. Roghmann K, Hecht P, Haggerty R: Coping with stress. In Haggerty R, Roghmann K, Pless I (eds): Child Health and the Community. New York, John Wiley and Sons, 1975, pp 54-66

- 62. Rutter M: Early sources of security and competence. In Bruner J, Garten A (eds): Human Growth and Development. London, Oxford University Press, 1978
- 63. Rutter M: Psychosocial resilience and protective mechanisms. Am J Orthopsychiatry 57:316-331, 1987
- 64. Sameroff A: Environmental context of child development. J Pediatr 109:192-199, 1986
- 65. Sameroff A, Chandler M: Reproductive risk and the continuum of caretaking casualty. In Horowitz F, Hetherington M, Scarr-Salanatek S et al (eds): Review of Child Development Research. Chicago, University of Chicago Press, 1975, pp 187-244
- 66. Sameroff A, Seifer R: Familial risk and child competence. Child Dev 54:1254-1268, 1983 67. Sameroff A, Seifer R, Barocas et al: Intelligence quotient scores of 4-year-old children: Social environmental risk factors. Pediatrics 79:343-350, 1987
- 68. Sameroff A, Seifer R, Zax M: Early development of children at risk for emotional disorders. Monogr Soc Res Child Dev 47:1-71, 1982
- 69. Sandler W, Block M: Life stresses and maladjustment of poor children. Am J Commun Psychol 8:41-52, 1980
- 70. Scarr S, McCartney K: How people make their own environments: A theory of genotypeenvironmental effects. Child Dev 54:424-435, 1983
- 71. Scarr S, Weinberg R: The influence of "family background" on intellectual attainment. Am Soc Rev 43:674-692, 1987
- 72. Selye H: The physiology and pathology of stress. Montreal, Acta, 1950
- 73. Shonkoff J. Biologic and social factors contributing to mild mental retardation. In Heller K, Holtzman W, Messiuk S (eds): Placing Children in Special Education: A Strategy for Equity. Washington, National Academy Press, 1982
- 75. Shonkoff J. Social support and vulnerability to stress: A pediatric perspective. Pediatr Ann 14:550-554, 1985
- 76. Shonkoff J, Hauser-Cram P: Early intervention for disabled infants and their families: A quantitative analysis. Pediatrics 80:650-658, 1987
- 77. Spieker S: Patterns of very insecure attachment found in samples of high-risk infants and toddlers. Top Early Child Spec Educ 6:37-53, 1986
- 78. Thomas A, Chess S: Temperament and Development. New York, Brunner/Mazel, 1977 79. Thomas A, Chess S: Genesis and evolution of behavioral disorders from infancy to early adult life. Am J Psychiatry 141:1-9, 1984
- 80. Thompson R: Stability of infant-mother attachment and its relationship to changing life circumstances in an unselected middle class sample. Child Dev 53:144-148, 1982
- 81. Unger D, Powell D: Supporting families under stress: The role of social networks. Fam Relat 29:566-574, 1980
- 82. Vaughn B, Egeland B, Sroufe L et al: Individual differences in infant-mother attachment at 12 and 18 months: Stability and change in families under stress. Child Dev 50:971-975, 1979
- 83. Vietze P, Falsey S, Sandler H et al: Transactional approach to prediction of child maltreatment. Inf Ment Health J 1:248-261, 1980
- 84. Warren L, McEachern C: Psychosocial correlates of depressive symptomology in adult women. J Abnorm Psychol 92:151-160, 1983
- 85. Weinraub M, Wolf B: Éffects of stress and social supports on mother-infant interactions in single and two parent families. Child Dev 54:1297-1311, 1983
- 86. Weintraub S, Neale J, Liebert D: Teachers' ratings of children vulnerable to psychopathology. Am J Orthopsychiatry 45:839-849, 1975
- 87. Weissman M, John K, Merikangas K et al: Depressed parents and their children: General health, social and psychiatric problems. Am J Dis Child 140:801-809, 1986
- 88. Weissman M, Prusoff B, Gammon G et al: Psychopathology in the children (age 6-18) of depressed and normal parents. J Am Acad Child Psychiatry 23:78-84, 1986 89. Weissman M, Siegal R: The depressed woman and her rebellious adolescent. Social
- Casework 53:563-570, 1972 90. Werner E, Smith R: Kauai's Children Come of Age. Honolulu, Hawaii, University of
- Hawaii Press, 1977
- 91. Werner E, Simonian K, Bierman J, et al: Cumulative effect of perinatal complications and deprived social environment on physical, intellectual, and social development of preschool children. Pediatrics 39:480-505, 1967
- 92. Williams H, Carmichael A: Depression of mothers in a multiethnic urban industrial municipality. J Child Psychol Psychiatr 26:277-288, 1985

 Wolkind S: Prenatal emotional stress: Effects on the fetus. In Wolkind S, Zajicek E (eds): Pregnancy: A Psychological and Social Study. Orlando, Florida, Grune Stratton, 1981, pp 177–193

94. Zuckerman B, Amaro H, Beardslee W: Mental health of adolescent mothers: The implications for depression and drug use. Dev Behav Pediatr 8:111-116, 1987

95. Zuckerman B, Beardslee W: Maternal depression: A concern for pediatricians. Pediatrics 79:110-117, 1987

 Zuckerman B, Stevenson J, Bailey V: Stomachaches and headaches in a community of sample of preschool children. Pediatrics 79:677–682, 1987

97. Zuckerman B, Stevenson J, Bailey V: Sleep problems in early childhood: Continuities, predictive factors, and behavioral correlates. Pediatrics 80:664-671, 1987

98. Zuckerman B, Hingson R, Alpert J et al: Neonatal outcome: Is adolescent pregnancy a risk factor? Pediatrics 71:489-495, 1983

Zuckerman B, Walker D, Frank D et al: Adolescent pregnancy: Biobehavioral determinants of outcome. J Pediatr 105:857

–862, 1984

Division of Developmental and Behavioral Pediatrics Department of Pediatrics Boston City Hospital Boston, Massachusetts 02118

Foster Care

Edward L. Schor, MD*

Our society has always been faced with the problem of providing care for children without living parents or whose parents are unable or unwilling to care for them. In preindustrial America there was incentive for taking such children into a family because they could be expected to materially contribute to the welfare of the family. Seen as important resources and necessary to the economy, dependent children were apprenticed or "bound out" to other families until the age of majority. This provided the child with some sense of permanency while he or she was expected to pay his or her own way. It also led to abuses of child labor. As the country developed, childhood became a more protected life stage, in which expectations of children to contribute materially were limited. The late 1800s also were characterized by immigration from both outside and within the country or region. Industrialization created a dispersion of extended families and isolated individuals from established systems of social support. These circumstances contribute to family dysfunction and in the extreme case the inability of more parents to parent their children adequately. As a consequence, society responded by developing institutions for the care of children, foundling homes and orphanages.10 The minority of children were truly orphans, and most came from homes that were unable to care adequately for them.

At the 1909 White House Conference on Children, the principles of supporting families so that they could continue caring for their children, or alternatively, placing children in foster families to be cared for until their biologic families were able to resume care, were adopted. 10 This recommendation was formalized in law when in 1935, Congress enacted Title IV-A, Aid to Dependent Children, as a component of the Social Security Act. The objective of this Title was to provide financial assistance to widows and widowers to avoid the breakup of families from economic hardship. The title was subsequently amended to include assistance to the parent and certain specified relatives and the name was changed to its current one, Aid to Families with Dependent Children (AFDC). The program has changed considerably since its inception, especially in the decade from 1967 to 1976 when the number of supported children increased from 3.6 to 8.1 million. The upward trend was accompanied by a marked decrease in children who were paternal orphans to a rapid increase in children with living fathers absent from the home. Despite programs to support the family, the number of children in need of substitute parenting increased. In 1961, Title IV-A was amended to provide Federal matching

^{*}Clinical Associate Professor, Department of Pediatrics, Stanford University School of Medicine; Program Officer, The Henry J. Kaiser Family Foundation, Menlo Park, California