Risky Families: Family Social Environments and the Mental and Physical Health of Offspring

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Risky families are characterized by conflict and aggression and by relationships that are cold, unsupportive, and neglectful. These family characteristics create vulnerabilities and/or interact with genetically based vulnerabilities in offspring that produce disruptions in psychosocial functioning (specifically emotion processing and social competence), disruptions in stress-responsive biological regulatory systems, including sympathetic-adrenomedullary and hypothalamic–pituitary–adrenocortical functioning, and poor health behaviors, especially substance abuse. This integrated biobehavioral profile leads to consequent accumulating risk for mental health disorders, major chronic diseases, and early mortality. We conclude that childhood family environments represent vital links for understanding mental and physical health across the life span.

Good health begins early in life. In the first years of childhood, the family is charged with responsibilities for the care and development of the child. In healthy families, children learn that they can count on the environment to provide for their emotional security and their physical safety and well-being, and they acquire behaviors that will eventually allow them to maintain their own physical and emotional health independent of caregivers. From this vantage point, a healthy environment for a child is a safe environment; it provides for a sense of emotional security and social integration and it offers certain critical social experiences that lead to the acquisition of behaviors that will eventually permit the child to engage in effective self-regulation (Basic Behavioral Science Task Force of the National Advisory Mental Health Council, 1996). Poor health also begins early in life. Research consistently suggests that families characterized by certain qualities have damaging outcomes for mental and physical health. These characteristics include overt family conflict, manifested in recurrent episodes of anger and aggression, and deficient nurturing, especially family relationships that are cold, unsupportive, and neglectful.

Families with these characteristics are risky because they leave their children vulnerable to a wide array of mental and physical health disorders. In this article, we propose a synthetic model of these links, focusing on the pathways through which risky families may not only hinder healthy development in childhood, but influence physical and mental health into adolescence and adulthood. As indicated in Figure 1, risky family characteristics create a cascade of risk, beginning early in life. Families with these characteristics may create vulnerabilities and may exacerbate certain genetically based vulnerabilities, which not only put children at immediate risk for adverse outcomes (such as is the case with abuse), but lay the groundwork for long-term physical and mental health problems. Specifically, we will show that risky families create deficits in children's control of and expression of emotions and in social competence, and also lead to disturbances in physiologic and neuroendocrine system regulation that can have cumulative, long-term, adverse effects. We especially focus on disruptions in sympathetic-adrenomedullary (SAM) reactivity, hypothalamic–pituitary–adrenocortical (HPA) reactivity, and serotonergic functioning. Children who grow up in risky families are also especially likely to exhibit health-threatening behaviors, including smoking, alcohol abuse, and drug abuse; the risk for promiscuous sexual activity in these children is also high. These forms of behavioral or substance abuse may represent a method of compensating for deficiencies in social and emotional development, as well as a self-medication process whereby adolescents manage the biological dysregulations produced or exacerbated by risky families. Taken together, these behavioral and biological consequences of risky family environments represent an integrated risk profile that is associated with mental health disorders across the lifespan, including depression and aggressive hostility, major chronic illnesses including hypertension and cardiovascular disease, and early death. As indicated in Figure 1, the cascade of accumulating risk is also heavily influenced by the social context in which children and families live, including factors such as chronic stress, neighborhood violence, and poverty (Taylor, Repetti, & Seeman, 1997). In the conclusions, we identify critical issues raised by our analysis, address links in the model for which evidence is currently
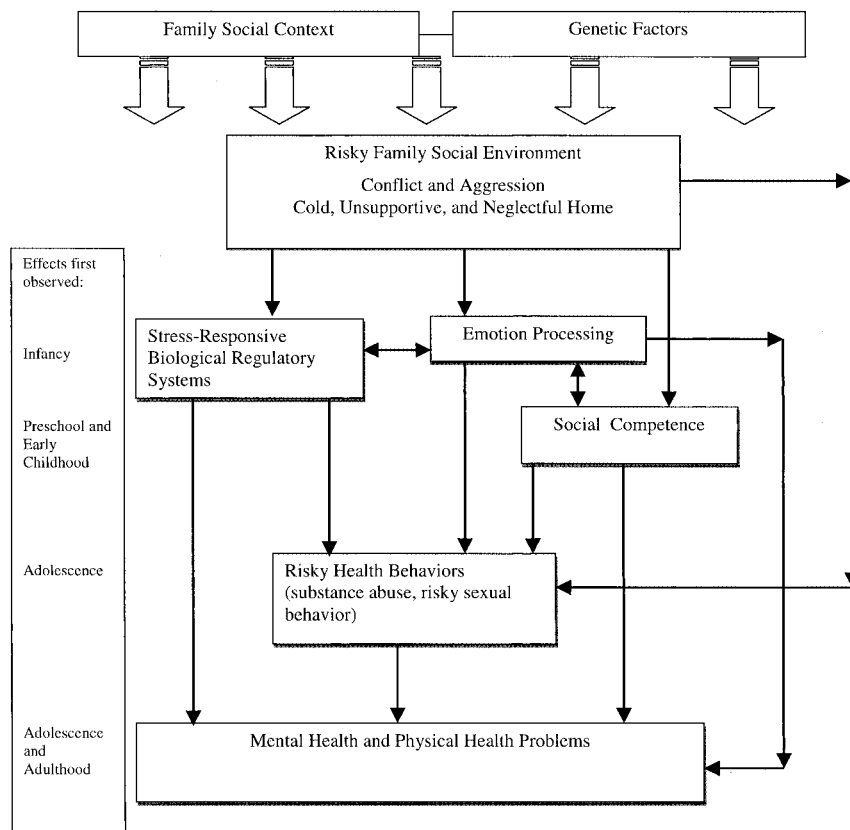
Figure 1. Risky families model. A time line on the left provides a rough idea of the point in development when an effect of the family environment is first observed. The social context of the family and genetic factors may directly and indirectly influence all of the variables in the model: the family environment, the sustaining factors, and health outcomes.

insufficient, propose a research agenda for the future, and discuss the potential role of intervention in offsetting the cascade of risk that is generated by risky families.

Risky Family Environments and Mental and Physical Health Outcomes

The backbone of the model depicted in Figure 1 is an association between growing up in a risky family environment and poor mental and physical health outcomes, with the most serious consequences manifested in adulthood. In this section, we describe these adverse family characteristics and present empirical evidence of their connections to mental and physical health outcomes.

Mental Health

Anger and aggression are highly noxious agents in a family environment. Conditions ranging from living with irritable and quarreling parents to being exposed to violence and abuse at home show associations with mental and physical health problems in childhood, with lasting effects into the adult years. There is overwhelming documentation in the research literature that overt conflict and aggression in the family are associated both cross-sectionally and prospectively with an increased risk for a wide variety of emotional and behavioral problems in children, includ-

ing aggression, conduct disorder, delinquency and antisocial behavior, anxiety, depression, and suicide (Emery, 1982, 1988; Grych & Fincham, 1990; Kaslow, Deering, & Racusia, 1994; R. J. Reid & Crisafulli, 1990; Wagner, 1997). Empirical efforts to tie different types of maltreatment and abuse in the home to different forms of psychopathology reveal only a general association of family violence and child psychopathology (Emery & Laumann-Billings, 1998). Parenting that constrains, invalidates, and manipulates children's psychological and emotional experience and expression is also related to both internalizing and externalizing symptoms[1] (Barber, 1996). A meta-analysis of 47 studies found

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[1] Behavioral problems in childhood are often discussed in terms of two broad symptom categories that relate to self-control or self-regulation (although the two types of symptoms tend to co-occur). Externalizing symptoms involve aggression and hyperactivity and are sometimes referred to as problems of *undercontrol*. Internalizing symptoms, which involve social withdrawal and negative emotions such as anxiety, are sometimes referred to as problems of *overcontrol*. Thus, in the case of externalizing symptoms, the child has difficulty successfully inhibiting socially prohibited behavior and controlling impulses and, in the case of internalizing symptoms, on the other hand, there often appears to be an extreme level of behavioral inhibition (Eisenberg, Fabes, Guthrie, et al., 1996).

that higher levels of coercive control in the family were related to problems of undercontrol on the part of children, particularly more aggressive and noncompliant behavior (Rothbaum & Weisz, 1994), a relation that is argued to be bidirectional.

Families characterized by high levels of conflict, aggression, and hostility are often lacking in acceptance, warmth, and support. However, there is evidence that inadequate emotional nurturance is independently associated with poor mental health outcomes. Our use of the adjectives *cold*, *unsupportive*, and *neglectful* covers a wide range of family characteristics and measures in the research literature, including emotional neglect of children; parenting that is unresponsive or rejecting; a lack of parental availability for, involvement in, and supervision of child activities; a lack of cohesiveness, warmth, and support within the family; and an experience of alienation, detachment, or feelings of lack of acceptance by children. Research studies that assess these characteristics of family life report reliable associations between them and a broad array of mental health risks, including internalizing symptoms such as depression, suicidal behavior, and anxiety disorders (Chorpita & Barlow, 1998; Kaslow et al., 1994), and externalizing symptoms such as aggressive, hostile, oppositional, and delinquent behavior (Barber, 1996; Rothbaum & Weisz, 1994; Steinberg, Lamborn, Darling, Mounts, & Dornbusch, 1994).

Although genetic predispositions appear to account for some of the observed relations between risky family social environments and child mental health (Plomin, 1994), evidence also suggests that parenting practices have both direct and indirect effects. A longitudinal adoption study found that children judged to be at genetic risk for behavioral problems were more likely to receive negative parenting from their adoptive parents than were children not at risk. However, the genetic risk did not explain the association of negative parenting and children's externalizing behavior, suggesting that environmentally mediated parenting effects on children's behavior was a plausible pathway (O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). This conclusion is supported by 16–18-year longitudinal data showing a significant association between maladaptive parental behavior and an increased risk of psychiatric disorder in offspring during late adolescence and early adulthood, even after controlling for parental psychopathology and earlier offspring characteristics (i.e., psychiatric disorders during early adolescence and difficult temperament in childhood; Johnson, Cohen, Kasen, Smailes, & Brook, 2001). In addition, genetic predispositions may act to increase vulnerability to risky family environments. For example, a research review suggests that families with high levels of conflict may not be able to provide the extra support needed by a child who is at a biological risk for depression (Kaslow et al., 1994). In fact, many of the family environmental effects represented in Figure 1 may well assume the form of gene–shared environment interactions. With very few exceptions, the dominant research design in the behavioral genetics field, twin studies, does not model gene–environment interactions; any variance due to those interactions is attributed to genetic effects.[2] The assumption made in twin studies, that these gene–environment interactions are negligible, and therefore can be ignored, is unwarranted.

Although they may live in the same home, siblings do not experience their family environment in precisely the same way. Most obviously, in families with more than one child, each child has a unique constellation of siblings. In addition, parenting not only varies between families but also within families, because the same parent may use different child-rearing techniques and behaviors with different children. Thus, in the review of research that follows, assessments of parent–child interaction or parenting cannot necessarily be understood as family-level variables. For example, in a study of genetic and environmental determinants of adolescent depression and antisocial behavior, 60% of the variance in adolescent antisocial behavior and 37% of the variance in depressive symptoms could be accounted for by nonshared environmental factors, specifically conflictual and negative parental behavior directed to an adolescent; when harsh parental behavior was directed at a sibling, it appeared to have protective effects for an adolescent (a phenomenon known as the *sibling barricade*; Reiss et al., 1995).

Whether the unit of analysis is the shared family environment or the parent–child relationship, comprehensive reviews of the research literature associate family relationships that are marked by high levels of anger and aggression or that are cold, unsupportive, or neglectful, with mental health problems in childhood and adolescence.

Physical Health

There also is growing evidence that offspring of risky families have increased rates of a wide variety of physical health problems throughout life. For example, short of permanent disability or death, a history of abuse is associated with chronic problems resulting from injuries and, in adulthood, with a broad array of physical symptoms and medical diagnoses (Walker et al., 1999). A study of 13,494 adults found a strong, graded relationship between breadth of exposure to abuse or household dysfunction during childhood and risk for certain adult diseases, including ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease (Felitti et al., 1998). Because there are no reviews of this literature, Table 1 summarizes research findings that illustrate a link between growing up in a risky family environment and physical health outcomes. The table is restricted to studies that were based on a longitudinal or follow-up research design,[3] or studies that used data from different sources, or studies with both characteristics. Many of the studies included observational or objective measures to ensure that respondent bias did not inflate the association between the measure of the family environment and the health outcome.

Support for a link between family conflict and physical health was found in a representative sample of the Swedish population in which reports of "serious dissention" in the family during childhood were associated with a variety of self-reported illnesses 13 years later, after controlling for psychological distress and mental illness (O. Lundberg, 1993). Other research, also summarized in

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[2] That is because interactions between genetic predispositions and family characteristics that are experienced by all siblings (referred to as *shared family environments* in the behavioral genetics literature) act to increase the similarity of monozygotic twins relative to dizygotic twins and are, thus, automatically included as part of the heritability estimates in twin studies.

[3] We use the term *follow-up research design* for studies that have multiple data collection points but that do not assess change over time in the outcome. We reserve the label *longitudinal research design* for studies in which change over time in the outcome variable is assessed.

Table 1
Risky Family Characteristics and Physical Health

Study and design	Sample characteristics	Family environment measure	Physical health outcome	Key finding
		Conflict and aggression		
O. Lundberg, 1993 GC/FLW-UP SELF	Representative sample of Swedish population (*n* = 4,216). Time 1 (1968) age range: 17–62 years, Time 2 (1981) age range: 30–75 years.	Time 1: individual responses in 1968 to "Was there any **severe dissention** in your family during your upbringing?" (10% reported "yes" or "uncertain").	Time 2, **health status**: self-reports in 1981 of physical illness (e.g., aches and pains, high blood pressure) and distress (e.g., tiredness, anxiety).	Severe dissention in the family during upbringing was associated with an increased risk, 13 years later, of self-reported illnesses and other indicators of distress. The increased risk of health problems at follow-up was found even after controlling for psychological distress and mental illness at Time 1.
Mechanic & Hansell, 1989 CORR/LONG SELF	7th, 8th, & 9th graders, *N* = 1,067	Time 1: adolescent report of **quarrelling or fighting** in home.	Time 2: adolescent report of **common physical** symptoms.	More quarreling and fighting at home was associated with more physical symptoms 1 year later (controlling for the earlier level of symptoms). Conflict was also associated with increases in depression and anxiety.
Montgomery et al., 1997 GC/FLW-UP IND RPRTS OBJ	Representative sample of over 6,500 participants in a national longitudinal study of a British birth cohort. Time 1 age: 7 years, Time 2 age: 33 years.	Time 1: **family conflict and difficulties** (4.5% of cohort) based on a health visitor's report of family difficulties (due to domestic tension, divorce, separation, desertion).	Time 1 and Time 2: assessment of **height.**	Exposure to family conflict was associated with less height attainment at ages 7 and in adulthood. Those who had been exposed to family conflict were more likely to be in the lowest 5th percentile of height distribution (31% in childhood and 24% in adulthood).
Stein et al., 1994 CORR/CS OBS & OBJ	Mothers with (*n* = 34) or without (*n* = 24) eating disorders and their children. Child age range: 12–14 months.	**Mother–child conflict**: based on an at-home observation of mother–infant interaction during mealtime.	**Infant weight.**	More conflict was associated with lower infant weight attainment, even after controlling for birth weight and maternal height, and for mothers' concerns about her body shape and whether she had an eating disorder.
Walker et al., 1999 GC/CS IND RPRTS	*N* = 1,225 women enrolled in a large HMO. *M* age = 42 ± 12 years.	Self-reported maltreatment in childhood. Women in the **nonsexual maltreatment** group reported a history of one or more of the following: physical abuse, physical neglect, emotional neglect, or emotional abuse. Women in the **sexual maltreatment** group reported a history of sexual abuse.	**Physician-coded diagnoses** during 18-month period prior to the study, of **minor infectious diseases** (e.g., urinary tract infections, upper respiratory infections) and **other diseases** (e.g., hypertension, diabetes). Self-reported **physical symptoms** during the previous 6 months.	Women who reported a history of either sexual or nonsexual maltreatment during childhood also reported more physical symptoms, and their medical records showed that they suffered from a greater number of minor infectious diseases and other diseases, when compared to women who did not report histories of childhood maltreatment.
Weidner et al., 1992 CORR/CS OBJ	Children and their parents, *N* = 64. Child age range: 6–18 years.	**Family conflict**: parent report of the degree to which open expressions of anger and aggression and conflictual interactions characterize the family.	**Risk of coronary heart disease** based on sample of child's blood: ratio of total plasma cholesterol divided by high density lipoprotein cholesterol.	A high-conflict family environment was associated with an unfavorable plasma lipid profile in sons. There was no association between family conflict and daughters' cholesterol.

(*table continues*)

Table 1 (*continued*)

Study and design	Sample characteristics	Family environment measure	Physical health outcome	Key finding
		Cold, unsupportive, and neglectful homes		
Gil et al., 1987 CORR/CS IND REPORTS	Children being treated for severe atopic dermatitis (*n* = 44). Child age range: 2–21 years. *M* child age = 6.9 years.	Parent or child (if > 12 years of age) report of levels of **family organization and cohesion.**	**Symptom severity**: specialty nurse's report of the percentage of body area affected by atopic dermatitis; records of periods of remission.	Less family cohesiveness was associated with more severe symptoms.
Gottman & Katz, 1989 CORR/CS OBS	Parents and children (*n* = 56 families). Age range of child: 4–5 years.	**"Negative" or poor parenting composite**: based on observations of parent behavior during interactions with child in the lab, a style that was unstructured, cold, unresponsive, angry, etc.	**Child illness**: based on mothers' reports on a health scale.	Children of parents with a negative parenting style had higher rates of illness.
Gottman et al., 1996, 1997 CORR/FLW-UP	Parents and children (*n* = 53 families). Age range of child Time 1: 4–5 years, Time 2: 6.8–9.2 years.	Time 1, parent **"emotion coaching" composite**: 11 ratings, based on an interview with the parent, of the degree to which the parent offers acceptance and assistance when the child feels anger and sadness (such as talking, intervening, comforting, teaching about emotion expression and coping strategies).	Time 2, **childhood illness**: parent report of the child's overall health and proneness to illness.	Children whose mothers were poor at "emotion coaching" had higher rates of illness and poor health 3 years later. "emotion coaching" was not related to child health.
Lissau & Sorensen, 1994 GC/LONG IND RPRTS	Representative sample of Danish population (*n* = 756). Time 1 (1974) child age range: 9–10 years, Time 2 (1984–1985) child age range: 20–21 years.	Time 1: **parent support** based on teacher rating (7% of parents described as providing "no support," 57% described as "harmonious"). **Neglect** based on a child hygiene rating provided by the school medical service (4% described as "dirty & neglected," 21% "well-groomed," and 75% "averagely groomed").	Time 2: **obesity in adulthood** indicated by self-reported weight and height; a body mass index (weight/height) at or above the 95th percentile.	Children of nonsupportive parents were at greater risk of obesity in early adulthood (18% vs. 3% of children with "harmonious" support), even after controlling for body mass index in childhood and child sex. Neglected children were also more likely to be obese: 29% versus 3–4%.
Martin et al., 1998 CORR/CS OBS & OBJ	Parent (mostly mother)–child dyads (child under treatment for diabetes; *N* = 74). Child age range: 8–18 years, *M* age = 13.0 years.	**Parenting** based on observations of parent–child interaction: parental emotional support, dyadic conflict resolution, parent expressions of warmth, anger, sadness.	Metabolic control of the diabetes assessed by glycosylated hemoglobin, a metabolic measure based on blood tests, assessed over several months.	Children and adolescents whose parents were less nurturing had poorer metabolic control over diabetes.
Russek & Schwartz, 1997 GC/FLW-UP	Men (*N* = 85). Time 1: Harvard undergraduates from classes of 1952, 1953, & 1954, Time 2: 35 years later.	Time 1: student description of **relationship with each parent** coded as **positive,** "very close" or "warm and friendly," or **negative,** "tolerant" or "strained and cold" (12% of relationships with mothers and 20% with fathers were "negative").	Time 2: **health status** based on in-person interviews and review of available medical records. Diagnosed conditions included cardiovascular disease, duodenal ulcer, and alcoholism.	Men who described a negative relationship with either their mother or their father were more likely to have a diagnosed disease 35 years later: 85–91% who had described negative relationships with a parent compared with 45–50% of those who had described positive relationships.

Table 1 (*continued*)

Study and design	Sample characteristics	Family environment measure	Physical health outcome	Key finding
		Cold, unsupportive, and neglectful homes (*continued*)		
Shaffer et al., 1982 GC/FLW-UP SELF	White male physicians (*n* = 913) who graduated from medical school between 1948 and 1964 and whose health status was assessed annually.	Medical students' descriptions of **family members' attitudes toward each other** (with respect to both parent–child and marital relationships) in positive terms (warm, close, understanding, confiding) and negative terms (detached, dislike, hurt, high-tension).	Physicians' self-reports of a **diagnosed cancer** in annual follow-up questionnaires and interviews during the years following graduation (25 of 913 participants had a diagnosed cancer).	Men who described more negative and less positive family relationships were at increased risk of future cancer, even after controlling for health risk factors such as age, alcohol use, cigarette smoking, being overweight, and serum cholesterol levels.
M. Valenzuela, 1997 CORR/CS OBS/OBJ	Mother–infant dyads (*n* = 85). Full-term, healthy, normal birth weight babies. Urban, poor socioeconomic status mothers in Santiago, Chile. Child age range: 17–21 months.	**Maternal sensitivity** based on observations during a home visit. Maternal behavior during a problem-solving task used to assess **support** (ability to elicit and maintain child's interest and prevent frustration) and **quality of assistance** (ability to help and guide child by scaffolding instructions to solve problems).	**Infant weight for age** (compared with norms); **chronic undernourishment status** based on longitudinal data on health, height, and weight.	Infants with less sensitive mothers were not growing as well (lower weight for age) as other infants. Infants whose mothers were less supportive and less able to assist the child were more likely to be chronically undernourished.
Wickrama et al., 1997 CORR/LONG OBS	White adolescents and their families living in a rural area (*N* = 310). Time 1 child age: 7th grade, Time 5 child age: 11th grade.	Time 1, **affective quality of parent behavior toward the adolescent**: (a) observer ratings of two family discussions (amount of derogatory, insulting, contemptuous behavior minus the amount of affectionate and supportive behavior and compliments) (b) adolescent report of the frequency of negative parent behavior (anger, criticism, threats, ignoring) minus the frequency of positive behavior (care, affection, appreciation, listening).	Time 1–Time 5: **annual assessments of adolescent health**, based on self-reports of 12 common physical symptoms (e.g., headaches, sore throat, muscular aches, stomach aches, congested nose, skin rash, allergies).	In a latent growth curve analysis, more hostile and less supportive parent behavior during family interactions, as well as adolescents' reports of more negative and less positive parent behavior, predicted increases in the adolescents' physical health complaints over the next 4 years.

Note. The key family and outcome variables in each study are highlighted in bold type. For each study, design (GC = group comparison; CORR = correlational), timing of assessments (FLW-UP = multiple data collection points, but change over time in the outcome is not assessed; LONG = longitudinal; CS = cross-sectional), and sources of data for primary variables (SELF = all self-report data; IND RPRTS = assessment of predictor and outcome variables based on data from separate sources; OBJ = objective measures; OBS = observational data) are reported.

the first half of Table 1, indicates that family conflict and aggression has adverse effects on health in childhood and adulthood, and on physical growth and development. Two of these studies used a longitudinal or follow-up research design (Mechanic & Hansell, 1989; Montgomery, Bartley, & Wilkinson, 1997), and three others used objective measures of health outcomes (Stein, Woolley, Cooper, & Fairburn, 1994; Walker et al., 1999; Weidner, Hutt, Connor, & Mendell, 1992).

Findings summarized in the second part of Table 1 show that growing up in a cold, unsupportive, or neglectful home is also associated with poor physical health and development. Investigations based on longitudinal and follow-up research designs have tied a lack of support and deficient nurturing during childhood to higher rates of illness and physical complaints several years later (Gottman, Katz, & Hooven, 1996, 1997; Wickrama, Lorenz, & Conger, 1997), to obesity in early adulthood (Lissau & Sorensen, 1994), and to more serious medical conditions in midlife (Russek & Schwartz, 1997; Shaffer, Duszynski, & Thomas, 1982). Cross-sectional studies that included independent assessments of a child health outcome and the family environment have shown links to poorer growth during infancy (M. Valenzuela, 1997), poorer general health (Gottman & Katz, 1989), and, among children with a diagnosed medical problem, less control over or more severe symptoms of the disease (Gil et al., 1987; M. T. Martin, Miller-Johnson, Kitzmann, & Emery 1998).

Summary

In summary, diverse research literatures consistently point to adverse developmental effects of the two general characteristics of a risky family social environment, conflict and aggression and a cold, unsupportive, or neglectful home. Besides direct effects on health, such as can be the case with physical abuse, the effects may be mediated and sustained by disruptions in the child's ability to mount a successful physiologic/neuroendocrine and/or behavioral response to stress, and to acquire appropriate emotional and behavioral self-regulatory skills, issues to which we now turn.

Mediating and Sustaining Factors

How are the long-lasting, even permanent, health consequences of growing up in a risky family environment sustained over the lifetime? Our model proposes that children from risky families experience disruptions in their physiologic/neuroendocrine functioning, especially in response to stress, and also develop deficits in emotion processing, social competence, and behavioral self-regulation. Although there is no essential mapping across these systems, the emotional, social, and biological disruptions appear to be linked to each other in a cascade arrangement. Figure 1 depicts a succession of developmental processes whose course can be influenced by risky family environments, so that early disruptions continue to have an impact on development in future stages. Not all of the connections in Figure 1 have been convincingly made, but emerging evidence suggests the value of considering these self-regulatory and biological dysfunctions as aspects of an integrated profile of risk.

Alterations in Physiological/Neuroendocrine Responses to Stressful Situations

The bulk of damage done to physical health in risky families may come from the initiation of biologically dysregulated responses to stress, the effects of which may be cumulative over the lifespan. As a result, the trajectories of major causes of morbidity and mortality in developed countries, namely the chronic diseases of hypertension, cardiovascular disease, diabetes, and some cancers, may begin as early as childhood in these biological dysregulations. The evidence currently suggests that constant or recurrent exposure to the stressful circumstances created by risky families may lead to alterations in the SAM system and HPA axis responses to stress and to disruptions in serotonergic functioning.[4] Deficiencies in the ability to mount a parasympathetic nervous system (PNS) response to stress may be affected as well. Given the interdependence of different components of the neuroendocrine system, we would anticipate disruptions in other systems as well, for example in dopamine regulation, but at present these links are not documented.

Dysregulation in vital biological regulatory systems may occur in utero or in infancy, periods that are thought to be critical in the normal development of biological regulatory systems. Dysregulation may also result from damage due to repeated activation of these systems. In making this case, we draw on the concept of allostatic load (McEwen & Stellar, 1993), arguing that repeated social challenges in a child's environment can disrupt basic homeostatic processes that are central to the maintenance of health. The consequences of exposure to these sources of family stress early in childhood may be cascading, potentially irreversible interactions between genetic predispositions and these environmental factors that, over time, can lead to large individual differences in susceptibility to stress, in biological markers of the cumulative effects of stress, and in stress-related physical and mental disorders. The fact that risky families appear to fuel such a wide range of mental and physical health disorders lends credence to the role that such families may play in exacerbating a broad array of genetic predispositions. Accumulating allostatic load is believed to lead to accelerated aging, which may include, over the long term, chronic hypertension, slower cardiovascular recovery from stress, hippocampal atrophy, and certain cognitive dysfunctions, signs of dysregulation of the HPA axis (such as flat or prolonged cortisol response to stress), dysregulation of the PNS, and dysregulation of serotonergic functioning, among other deleterious biological changes (see McEwen, 1998; Seeman & Robbins, 1994; Seeman, Singer, Horwitz, & McEwen, 1997; for reviews). We confine our coverage here to system dysregulations that have been related both to the antecedent conditions of risky families and to the deleterious consequences for mental and physical health outcomes.

SAM Functioning

The chronic stress of a risky family may produce repeated or chronic SAM activation in children, or both, which in turn leads to wear and tear on the cardiovascular system that, over time, may

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[4] A caution needs to be kept in mind that not all indicators of the functioning of these systems show these effects.

lead to pathogenic changes in sympathetic or parasympathetic functioning, or both. Such a model has been put forth by several investigations as the developmental underpinnings of essential hypertension (e.g., Ewart, 1991) and coronary heart disease (e.g., Woodall & Matthews, 1989). A secondary pathway to the same adverse cardiovascular outcomes argues that the impact of risky families on the SAM functioning of offspring routes through the development of a hostile interpersonal style, which itself increases the frequency of conflictual social interactions, recurrent SAM activation, and the likely development of risk factors for coronary heart disease (CHD). There is empirical evidence suggestive of both models.

Most children show increases in sympathetic arousal in response to exposure to angry adult interactions (El-Sheikh, Cummings, & Goetsch, 1989), and children in families marked by conflict are exposed to this physiological activation on a recurrent basis. Such repeated exposure may lead to alterations in SAM reactivity to stress. In support of this reasoning, Woodall and Matthews (1989) found that boys (but not girls) from families characterized as less supportive by parents had stronger heart rate responses to a series of laboratory stressors, compared with boys from more supportive families. These family characteristics were also associated with higher anger and hostility, providing suggestive evidence for the pathway to adverse CHD outcomes via hostility, but this mediational hypothesis was not directly tested in the study. Luecken (1998) similarly found that college students who reported poor family relationships in childhood had higher blood pressure, both at resting level and in response to a laboratory challenge. Early studies relating so-called Type A behaviors in children to heightened cardiovascular responses to stress (e.g., Brown & Tanner, 1988; Lawler, Allen, Critcher, & Standard, 1981; U. Lundberg, 1983; Matthews & Jennings, 1984; T. H. Schmidt, Thierse, & Eschweiler, 1986) are also consistent with this link. Such changes in reactivity have, in turn, been tied to traditional risk factors for CHD. For example, cardiovascular reactivity to stress among boys as young as 8 to 10 years old has been associated with increased left ventricular mass, a risk factor for CHD (Allen, Matthews, & Sherman, 1997). A genetic basis for heightened or prolonged sympathetic reactivity to stress may also be implicated in the phenotypic expression and exacerbation of these responses. In support of this reasoning, Ballard, Cummings, and Larkin (1993) found that children of hypertensive parents showed greater systolic blood pressure reactivity to interadult anger than children of non-hypertensive parents. They suggested that familial transmission of essential hypertension may be mediated, in part, by recurrent exposure to such hostile episodes (see also Ewart, 1991).

In sum, there is evidence that a risky family environment is associated with heightened cardiovascular reactivity to stress, which is linked with cardiovascular disease risk factors in children. Studies that track all of these links within a single population are lacking, however, and should be a focus of future work. The second pathway, namely that risky families increase a propensity for an angry, hostile interpersonal style, which, in turn, heightens risk for cardiovascular disease is discussed further in the section on social competence.[5] Alterations in SAM reactivity in children exposed to risky family environments may also reflect contributions from the counterregulatory parasympathetic nervous system. Negative emotions such as anxiety and hostility have been associated with lower heart rate variability, a marker of reduced para-

sympathetic response (Kawachi, Sparrow, Vokonas, & Weiss, 1994; Sloan et al, 1994), and lower heart rate variability has in turn been linked to increased health risks (Kristal-Boneh, Raifel, Froom, & Rivak, 1995). These findings suggest that children in risky families may experience reductions in PNS activity, an important counterregulatory "brake" on SAM activity, thereby contributing to dysregulation of the SAM system.

HPA Functioning

Alterations in HPA reactivity have also been tied to risky family characteristics. In response to stress, the hypothalamus releases corticotrophin-releasing hormone (CRH), which influences the pituitary gland to secrete adrenocorticotropic hormone, which, in turn, stimulates the adrenal cortex to release corticosteroids. This integrated pattern of HPA activation modulates a wide range of somatic functions, including energy release, immune activity, mental activity, growth, and reproductive function. Appropriate cortisol regulation (an indicator of HPA responses to stress) allows the body to respond to stress by preparing for short-term demands. However, persistent activation of the HPA system is associated with immune deficiencies, inhibited growth, delayed sexual maturity, damage to the hippocampus, cognitive impairment, and certain forms of psychological problems, such as depression (Chrousos & Gold, 1992). As such, chronically elevated corticosteroids have potentially deleterious effects on developing competent cognitive and emotional functioning (Gunnar, 1998).

The cortisol response may be adversely affected in the offspring of families characterized by conflict, anger, and aggression. J. Hart, Gunnar, and Cicchetti (1996) found that children who had been physically abused in their families had somewhat elevated afternoon cortisol concentrations; maltreated children who were also depressed had lower morning cortisol concentrations compared with nondepressed maltreated children and were more likely to show a rise, rather than the expected decrease in cortisol, from morning to afternoon. HPA functioning also appears to be affected by parental nurturing behavior. Studies of rhesus monkeys have found that ventral contact between offspring and mother following a threatening event promotes rapid decreases in HPA activity (Gunnar, Gonzalez, Goodlin, & Levine, 1981; Mendoza, Smotherman, Miner, Kaplan, & Levine, 1978). Paralleling the animal results, a study of 264 infants, children, and adolescents found that a family environment characterized by few positive affectionate interactions and a high level of negative interactions, including irrational punishment and unavailable or erratic attention from parents, was associated with abnormal cortisol response profiles, diminished immunity, and frequent illnesses (Flinn & England, 1997). Chorpita and Barlow (1998) noted that in families characterized by low levels of warmth and high levels of social control,

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[5] Noradrenergic functioning may also be implicated directly in the health consequences of risky families. For example, a compelling animal model of stress-induced behavioral symptoms of depression (Weiss 1991; Weiss, Bailey, Pohorecky, Korzeniowski, & Grillione, 1980) links exposure to uncontrollable stress to norepinephrine depletion in the locus coeruleus; this depletion has, in turn, been linked to depressive symptomatology including declines in motor activity and appetitive behavior, weight gain, and decreased responding for rewarding brain stimulation. Whether risky families give rise to this pattern is unknown at present.

HPA axis functioning may be disrupted in response to stress, leading to increased CRH and hypercortisolism. Two retrospective studies suggested that these biological dysregulations can persist into adulthood. In one, poor family relationships, assessed by college students' ratings of their early family environment, were associated with elevated cortisol responses to a laboratory challenge (Luecken, 1998). In the other, women who reported having been abused in childhood showed greater HPA responses to laboratory stress than did women without such histories; these responses were greater for abused women who also had symptoms of anxiety and depression (Heim et al., 2000; see also Kaufman, Plotsky, Nemeroff, & Charney, 2000).

The child's attachment to parents also affects cortisol responses.[6] For example, children with disorganized/disoriented attachment patterns exhibit higher cortisol levels following brief separations from their mothers (Hertsgaard, Gunnar, Erickson, & Nachmias, 1995). In other research, attachment patterns moderated cortisol responses of babies in stressful circumstances (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). The protective effects of a secure attachment relationship were especially evident for socially fearful or inhibited children—a finding that parallels previous research by Levine and Wiener (1988). To explain these results, Gunnar and her associates argued that temperamentally fearful children are especially vulnerable to stress, and an insecure attachment is implicated in their elevated cortisol responses to new situations, serving both as a marker of the parent–infant relationship and as a potential predictor for later anxiety disorders. This underscores the interactive role that parenting style may play with other risk factors, especially genetic risks, in the etiology of biological and psychological stress responses.

Although evidence is currently lacking on the longer term stability of child patterns of HPA reactivity, animal data suggest that early experiences can permanently alter adrenocortical activity in rat pups in response to stress, responses mediated by maternal attention. Research by Meaney and associates found that rat pups handled in early infancy were the recipients of more maternal licking and grooming; in turn, these rat pups had more hippocampal glucocorticoid receptors, lower hypothalamic corticosteroid releasing factor, and less glucocorticoid secretion during a stressor and faster recovery to baseline afterward. They also showed more open field exploration (an indicator of less anxiety) and more receptors in the brain for benzodiazepines (anxiety-reducing tranquilizers; Liu et al., 1997; Meaney, Aitken, van Berkel, Bhatnagar, & Sapolsky, 1988; see also Caldi et al., 1998). The handled rats were also less likely to show age-related onset of HPA dyregulation in response to challenge and less likely to exhibit age-related cognitive deficits. This compelling animal model suggests that nurturant stimulation by the mother modulates responses of offspring to stress in early life in ways that have permanent effects on the offspring's HPA response.

Serotonergic Functioning

Dysregulations in the serotonergic system have been tied to several of the health outcomes related to risky families, including depression, suicidality, aggression, and as will be seen, substance abuse. Research on serotonergic function in children remains relatively sparse, in part because of the invasive nature of assessments of central serotonergic functioning, but the existing evidence does suggest that children from risky families experience serotonergic dysregulation. Kaufman et al. (1998) identified depressed abused and depressed non-abused children from clinic records, and compared them with normal children on a serotonin challenge (L-5-HTP) administered intravenously. Prolactin responses to the challenge, an indicator of serotonergic functioning, were highest in the abused children, and those responses were correlated both with clinical ratings of aggressive behavior and with family history of suicide attempt[7] (see also Petty, Davis, Kabel, & Kramer, 1996; Risch, 1997).

The dysregulation in serotonergic functioning associated with abuse may be due both to genetic and to experiential factors. Suggestive evidence for a genetic link comes from the fact that first- and second-degree relatives of depressed abused children have elevated rates of depression, suicidality, and aggressive behavior (Kaufman et al., 1998), and serotonin dysregulation is highest in people with the greatest familial loading for these disorders (Coccaro, Silverman, Klar, Horvath, & Siever, 1994; Halperin et al., 1997; Linnoila, DeJong, & Virkkunen, 1989; Pine, Shaffer, Davies, & Schonfeld, 1997). However, deficient nurturing may also contribute to these dysfunctions. For example, Pine and colleagues (Pine, Coplan, et al., 1997; Pine et al., 1996) found that children from families characterized by deficient nurturing produced boys at risk for delinquency who also showed evidence of serotonergic dysfunction. Primate studies provide the most comprehensive data linking nurturant family and maternal environments to biobehavioral profiles characterized by better serotonergic regulation and less behavioral dysregulation. Such studies have found that monkeys raised in poor early rearing conditions show long-term alterations in serotonergic functioning (Kraemer & Clarke, 1990; Rosenblum et al., 1994). Studies by Suomi (1987, 1991, 1997) also have suggested a role of maternal behavior in moderating genetic risk for serotonergic dysfunction. One study compared monkeys with two forms of the 5-HTT allele, a gene related to serotonin transport; the short form of the gene confers decreased serotonin function, whereas the long form is associated with normal serotonin functioning (Suomi, 1999). In addition, some of the monkeys were raised by their mothers and some were raised by peers. Being raised by peers is a risk factor for the development of a reactive, impulsive temperament, and other deficits in social behavior, including more aggressive exchanges

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[6] According to attachment theory, children use an adult caregiver as a secure base for exploration and as a haven of safety in times of stress. Individual differences in secure base behavior are thought to reflect the extent to which the attachment figure is a source of security for the infant. On the basis of their emotional and behavioral responses to brief separations and reunions with the attachment figure, typically the mother, children are classified either as *secure* or into one of several *insecure* categories (Waters, Vaughn, Posada, & Kondo-Ikemura, 1995). Evidence suggests that maternal sensitivity and other dimensions of parenting behavior are significant components of infant attachment security (De Wolff & van IJzendoorn, 1997).

[7] Although lower levels of serotonergic activity are associated with aggressive behavior in adults, in preadolescent samples, the relation is often the reverse with especially high serotonin levels correlated with aggression, depression, and other adverse outcomes (Kaufman et al., 1998).

and less grooming.[8] Monkeys with the short 5-HTT allele who were raised by peers showed lower concentrations of the primary central serotonin metabolite (5-HIAA) than was true for monkeys with the long allele. But for monkeys raised by their mothers, primary serotonin metabolite concentrations were identical for monkeys with either allele. This pattern clearly suggests a protective effect of maternal behavior on expression of genetic risk for low levels of serotonin.

Additional evidence for the genetic–experiential interaction is provided by Suomi (1987), who randomly assigned rhesus monkey neonates selectively bred for differences in temperamental reactivity to foster mothers who were either unusually nurturant or within the normal range of mothering behavior. Infants whose pedigrees suggested normative patterns of reactivity exhibited normal patterns of biobehavioral development, independent of the relative nurturance of the foster mother. In contrast, highly reactive infants cross-fostered to normal mothers exhibited deficits in early exploration and exaggerated behavioral and physiological responses to minor environmental perturbations. In adulthood, they tended to drop to and remain low in the dominance hierarchy (Suomi, 1991). Highly reactive infants cross-fostered to exceptionally nurturant females, in contrast, appeared to be behaviorally precocious. They left their mothers earlier, explored the environment more, and displayed less behavioral disturbance during weaning than both control (low-reactive) infants reared by either type of foster mother and highly reactive infants cross-fostered to normal mothers. In addition, when permanently separated from their foster mothers and moved into larger groups, the highly reactive animals cross-fostered to nurturant mothers became adept at recruiting and retaining other group members as allies, and most achieved high dominant status.

The primate studies by Suomi (1987, 1991, 1997) are significant for the present argument, not only because they suggest an important role of parenting for modifying the expression of genetically based temperamental differences, but also because they tie serotonergic dysfunction directly to aggression and other deficits in social behavior that are similar to behavioral problems found in children from risky families (see for review, Suomi, 1997). Causal links between serotonin levels and aggressive behaviors are further indicated by research showing that lowering levels of serotonin through pharmacologic treatment with fenfluramine hydrocholoride leads to increases in aggression (Raleigh et al, 1986). Peer-raised adolescent monkeys also show certain propensities for substance abuse, for example, requiring larger doses of the anesthetic ketamine to reach a state of sedation and consistently consuming more alcohol and developing a greater tolerance for alcohol, relative to mother-raised monkeys, a pattern predicted by their central nervous system's serotonin turnover rates (Higley et al., in press). Difficulty in moderating aggressive impulses, problems in developing and maintaining social relationships, and risk for substance abuse are among the outcomes most consistently seen in response to risky family characteristics of hostility, conflict, and deficient nurturing.

The potential intergenerational transmission of these behavior patterns also warrants attention. Female monkeys who are raised by peers (rather than by their mothers) are significantly more likely to exhibit neglectful or abusive treatment of their own offspring (especially firstborns) as compared with their mother-reared counterparts (Champoux, Byrne, Delizio, & Suomi, 1992; Suomi &

Levine, 1998). Exposure to effective parenting reduces these behaviors. In the study of females bred for high reactivity mentioned earlier, when females became mothers they adopted the maternal style of their foster mothers, independent of their own reactivity profile (Suomi, 1987). Highly reactive females raised by especially nurturant foster mothers not only showed lower reactivity themselves, but learned effective parenting, which they passed on to the next generation, thus providing clear evidence of behavioral intergenerational transmission of nurturance.

Other System Dysregulations

The interaction of biological systems virtually demands that other system dysregulations also occur as a result of exposure to a risky family environment. The HPA axis, for example, influences the norepinephrine system in ways that may affect anxiety and depression (Rosen & Schulkin, 1998). Serotonin and dopamine functioning influence each other (Kostrzewa, Reader, & Descarries, 1998), and it is possible that serotonin dysregulation may be related to a heightened risk for substance abuse via its effect on dopamine regulation.[9] SAM, PNS, HPA, and serotonergic functioning are all interrelated and, in turn, affect the immune system (Schleifer, Scott, Stern, & Keller, 1986), the metabolic system (Raikkonen, Keltikangas-Jarvinen, Hautanen, & Adlercreutz, 1997), and other physiologic regulatory systems of the body. As such, a risky family situation might produce or exacerbate a seemingly minor dysregulation in one system, which could, in turn, produce a small dysregulation in another system, and so on. Over time, small but accumulating perturbations in the body's regulatory systems may lead to multiple vulnerabilities, which may further help to explain the association of risky family characteristics with the wide range of documented mental and physical health risks.

Summary

In summary, alterations in SAM, HPA, and serotonergic functioning appear to result from exposure to risky family environ-

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[8] Monkeys raised with peers have difficulty moderating behavioral responses to rough-and-tumble play, sometimes escalating those bouts into full-blown aggressive exchanges. Rearing with peers is also associated with other social deficits, such as diminished social competence and low levels of grooming (Coccaro 1992; Coccaro, Kavoussi, Cooper, & Hauger, 1997; Higley, Mehlman, et al., 1996; Higley, Suomi, & Linnoila, 1996a, 1996b; Mehlman et al., 1994; Raleigh & McGuire, 1994; Virkkunen & Linnoila, 1993).

[9] Dopamine pathways are consistently implicated in substance abuse including opiates, cocaine, amphetamines, nicotine, and alcohol (Koob, Sanna, & Bloom, 1998), as well as in neuropsychiatric disorders, such as bipolar disorder (Depue & Iacono, 1989). In animal studies, early social isolation or maternal deprivation are associated with alterations in dopamine concentrations in the nucleus accumbens and the caudate-putamen (Lewis et al., 1990; Kehoe, Shoemaker, Arons, Triano, & Suresh, 1998), with permanent alterations in dopamine function (Hall, Wilkinson, Humby, & Robbins, 1999) and receptor sensitivity (Lewis et al., 1990), with ambivalent responses to novelty, cognitive impairment, and altered responses to stress (Robbins, Jones, & Wilkinson, 1996), and with stereotyped behavior sequences (e.g., Lewis et al., 1990). As yet, however, specific qualities of parenting have not been directly implicated in this process.

ments characterized by conflict, anger, and aggression and by deficient nurturing. The evidence for disruptions in SAM functioning comes primarily from boys at risk for cardiovascular disease, suggesting a potential exacerbation of preexisting vulnerabilities in risky families. Offspring from risky families are also more likely to show abnormal cortisol reactivity; these patterns appear especially common in children predisposed to a fearful, inhibited temperament. Evidence for the mediating role of serotonergic functioning in explaining the association between risky families and multiple outcomes stems largely from studies of abused children and from studies on offspring (and especially nonhuman primates) raised in situations characterized by deficient nurturing. The significance of this link stems from the associations between serotonin dysregulation and other adverse outcomes associated with risky parenting, including aggression, depression, social incompetence, and substance abuse. In light of the growing evidence of extensive "cross-talk" among biological regulatory systems, other system dysregulations are expected to emerge, as research on these issues progresses.

Emotion Processing

In Figure 1, risky family environments are associated with the way that offspring process emotions, a factor that may also be implicated in the development of mental and physical health disorders. By emotion processing we mean the experience, control, and expression of emotion, particularly in emotionally arousing situations. Despite recent advances in emotion research, emotion processing is still poorly understood, and we do not attempt here to further clarify or specify such a complex phenomenon. Our more modest goal is to present research on those components of emotion processing that have been studied for their association with risky family environment variables. Three aspects of emotion processing meet this criterion: emotional reactivity in emotionally arousing situations, emotion-focused coping, and emotion understanding.

Risky Family Characteristics and Emotion Processing

Empirical findings supporting a link between risky family environments and the three aspects of emotion processing mentioned above are summarized in Table 2. The first group of studies listed in Table 2 are *short-term reaction studies*, in which a child's immediate emotional reaction in an emotion-arousing situation is observed. There are several methodological advantages to these studies: The investigator can control the situation in which the child is observed, the child's behavior can be rated by independent observers, and information about the family environment is separated from the laboratory assessment of the child's emotional reaction. One drawback to this approach to studying emotion processing is that it is difficult to distinguish processes involved in controlling an emotional state from an individual's propensity to experience intense emotions that are difficult to regulate. Because of the different methodologies involved, Table 2 distinguishes these short-term reaction studies from other studies, discussed below, that assess emotion understanding and coping.

Conflict and aggression. Most of the short-term reaction studies in Table 2 focus on children as they listen to or observe angry and conflictual interactions (which either are staged in laboratory settings or are naturalistic interactions in the home). The emotional and behavioral responses of children whose home lives are characterized by conflict and aggression are then compared with the responses of children from homes with less aggression and happier marriages. The findings indicate that high levels of conflict at home sensitize children to anger. They react with greater distress, anger, anxiety, and fear (Ballard et al., 1993; E. M. Cummings, Zahn-Waxler, & Radke-Yarrow, 1981; Davies & Cummings, 1998; O'Brien, Margolin, John, & Krueger, 1991). Increased reactivity may result from chronic stress levels in conflictual and violent homes. According to the allostatic load model, a period of recovery following physiological arousal is essential for the proper functioning of homeostatic processes in the body (McEwen, 1998; McEwen & Stellar, 1993). Periods of relief or respite from aroused emotional states may also be critical to the proper regulation of emotion dynamics. Chronic or repeated stressors in the environment, such as high levels of violence and family conflict, may not allow for sufficient recovery from heightened emotional arousal. Sustained states of emotional arousal may, over time, increase reactivity. This is consistent with a model proposed by Perry and his colleagues that suggests that chronic stress affects neurobiological development and creates a sensitized stress-responsive system that influences arousal, emotion regulation, behavioral reactivity, and cardiovascular regulation (Perry & Pollard, 1998). Thompson and Calkins (1996) suggested that hypervigilance in children from aggressive and violent homes may also contribute to increased reactivity.

Emotion processing is also assessed by tasks that measure children's understanding of emotions and by self-reports of methods used to cope with stressful experiences in the past. These are listed as *emotion understanding and coping studies* in Table 2. As operationalized in research studies, emotion understanding includes the ability to recognize emotional states (both in self and others), the skills to express emotions in a culturally acceptable manner, and the knowledge of causal antecedents of different emotions, factors that are integral to the processing of emotions in stressful or arousing situations. Because emotion understanding shapes social perception, we only included emotion understanding studies in Table 2 that used information from independent sources to assess the family. In two investigations of young children, those who were maltreated or whose homes were marked by high levels of anger and distress had a less accurate understanding of emotions, compared with their peers (Camras et al., 1988; Dunn & Brown, 1994). This may be because families with high levels of negative affect are less likely to engage in conversations about feelings (Dunn & Brown, 1994), and more talk about feeling states in the home is associated with better emotion understanding in children (Dunn, Brown, Slomkowski, Tesla, & Youngblade, 1991).

Although there are few studies of emotional reactivity and emotion understanding in adolescents, there is a research literature on coping in adolescence. We focus here on strategies for controlling emotional states in stressful situations, which are often conceptualized as emotion-focused coping strategies. Most of these investigations are correlational studies that rely on self-report measures. To limit the impact of self-report biases, we restrict our analysis to studies in which the assessment of coping was made years after the description of the family had been provided. In

Table 2
Risky Family Characteristics and Emotion Processing

Study and design	Sample characteristics	Family environment measure	Child/adolescent emotion processing variable	Key findings
		Short-term reaction studies (immediate emotional reactions and coping responses in stressful situations): Conflict and aggression		
Ballard et al., 1993 GC/CS IND RPRTS OBS	Children and their parents ($n = 48$). Distressed homes ($n = 21$). Nondistressed homes ($n = 27$). M child age = 12.5 years ($SD = 1.4$ years).	Child was categorized as living in a **"maritally distressed" home** if either parent reported poor marital adjustment or the use of physical conflict tactics during marital disputes.	Children videotaped while "overhearing" a staged angry interaction between 2 adult strangers in the next room. **Nonverbal reactions** coded (e.g., freezing, postural distress, facial distress, smiling, laughing). Children also **reported emotional responses** (i.e., by choosing an emotion face that represented how they felt and indicating how much they felt that way).	Early adolescents from maritally distressed homes reported more fear and showed greater nonverbal reactivity.
Camras & Rappaport, 1993 GC/CS IND RPRTS OBS	Maltreated & nonmaltreated children were paired by the experimenter ($n = 18$ pairs). The children in each pair were from same daycare or social service center. Age range: 3.33–7.25 years. M age = 4 years 11 months.	**Maltreated** children identified by state department of child and family services.	During a play session with a peer, **child attempts to negotiate sharing of a gerbil box** (e.g., attempts to obtain box and resistance responses) were coded.	Maltreated children appeared reluctant to either engage or resist the peer. They smiled more (signs of placation) and used fewer negative upper face expressions, such as "brow frowns" (signs of determination), when resisting a peer's attempts to obtain the box. They made fewer attempts to obtain the box and, after encountering resistance, waited longer before renewing an attempt, and they used "pulls" less often to obtain the box.
J. S. Cummings et al., 1989 CORR/CS IND RPRTS OBS	Children & their mothers ($n = 48$). Child age range: 2–6 years.	**Physical aggression** in parents' marriage (average of both parents' reports on the Conflict Tactics Scale)	Child's response to lab simulation in which the experimenter expressed anger toward the mother. **Social responsibility** was indicated by responses to mother, such as provisions of physical and/or verbal comfort, by expressions of concern (such as asking about feelings), and by defense of mother.	Physical conflict in the parents' marriage correlated with reactions suggesting greater social responsibility (e.g., children were more solicitous, took actions to comfort and protect their mothers).

(table continues)

Table 2 (*continued*)

Study and design	Sample characteristics	Family environment measure	Child/adolescent emotion processing variable	Key findings
		Short-term reaction studies (immediate emotional reactions and coping responses in stressful situations): Conflict and aggression (*continued*)		
E. M. Cummings et al., 1981 Cross-sequential, daily monitoring	Children from intact, middle-class families (*n* = 24). Child age range: 10–20 months, at start of study.	Total number of incidents of **interparent fighting,** based on mothers' daily reports over a 9-month period.	Maternal description of **child emotional responses** to each episode of interparent fighting as it occurred.	More interparent fighting associated with greater sensitization to individual episodes of fighting (more anger, distress, and affectionate/prosocial behavior; fewer instances of an unemotional reaction).
Davies & Cummings, 1998 CORR/CS IND RPRTS OBS	Children & mothers from maritally intact families (*n* = 56). Child age range: 6–9 years. *M* age = 7.5 years.	Latent variable based on maternal report of **"marital discord,"** which included measures of disagreements, adjustment, hostility, and conflict in the marriage.	**Child emotional reactions** to a simulated conflict between mother and the experimenter, measured by observer ratings of child distress and/or vigilance (watchful attention and/or preoccupation with the possibility of danger), and by self-reported feelings of anger.	More marital discord associated with more emotional reactivity (signs of vigilance and distress, and self-reported anger).
Gordis et al., 1997 CORR/CS IND RPRTS OBS	Family triads (*n* = 90). Child age range: 9–13 years. *M* age = 11.3 years.	**Physical marital aggression:** both spouses' reports of frequency of violence during the past year.	Child responses during a family discussion (about aspects of the child's behavior that act as a source of conflict between the parents). Child responses coded for amount of **withdrawal, anxiety, and signs of distraction.**	Physical marital aggression correlated with more attempts to distract during the discussion (silly/irrelevant behavior that draws attention away from the discussion).
O'Brien et al., 1991 GC/CS IND RPRTS	Mother–son dyads (*n* = 35). Child age range: 8–11 years.	Dyads classified into groups based on the amount of **marital conflict** reported by mother and son: (a) physically aggressive marriages (*n* = 12), (b) verbally aggressive marriages (*n* = 11), (c) low-conflict marriages (*n* = 12).	Sons' reports of their thoughts and feelings as they listened to audiotaped (simulated) family conflicts. **Self-distraction**—unique and tangential comments (reflecting an inability to deal directly with the conflict situation); **interference**—reports that they would attempt to verbally or physically intervene in the conflict; **arousal**—reports of increased heart beat, breathing faster, feeling sweaty.	Sons of verbally or physically aggressive parents were more likely to self-distract. Sons of physically (as opposed to verbally) aggressive parents were more likely to self-distract, interfere, and report physiological arousal.

Table 2 (*continued*)

Study and design	Sample characteristics	Family environment measure	Child/adolescent emotion processing variable	Key findings
		Short-term reaction studies: Cold, unsupportive, and neglectful homes		
Nachmias et al., 1996 GC/CS OBS	Mother–toddler (all White) dyads (*n* = 77). Child age = 18 months (+/− 2 weeks).	**Mother–child attachment security** assessed in the Strange Situation (securely attached vs. insecurely attached toddlers).	**Child coping with novel arousing stimuli**, such as a live boisterous clown who invited the child to play while the mother was present. More competent coping was indicated by child efforts to use mother to become engaged in the situation (such as through social referencing behaviors), fewer attempts to escape the situation, fewer infantile self-soothing behaviors (such as stroking or sucking on a toy).	Less securely attached toddlers were less competent copers. They were less likely to use their mothers to become engaged and more likely to attempt to escape the situation.
		Emotion understanding and coping studies: Conflict and aggression		
Camras et al., 1988 GC/CS IND RPRTS	Abused (*n* = 20) & nonabused (*n* = 20) child–mother dyads. Age range: 3.33–7.25 years. *M* age = 4 years, 11 months.	**Child abuse** status identified by state agency.	**Facial emotion expression posing task** (posing of six emotions). **Expression recognition task** (several weeks after the production task): Child chooses expressions to match emotions described in stories.	Abused children were less accurate in recognizing emotional expressions, and posed less recognizable expressions. Mothers' posing scores correlated with their children's recognition scores; mothers of abused children posed less recognizable expressions.
Dunn & Brown, 1994 CORR/FLW-UP IND RPRTS OBS	Mother–child dyads (*n* = 50). Child age: 33 months at Time 1; 40 months at Time 2.	Time 1: coding of **negative affect (anger and distress) during family observations** at home on two separate occasions.	Time 2 (7 months later): two **emotion understanding** tasks, identifying emotions in facial expressions and identifying emotional response in puppet vignettes.	More expressions of maternal negative affect at home was associated with less emotion understanding.
Johnson & Pandina, 1991 CORR/FLW-UP SELF	Adolescents (*n* = 1,308). Ages: 12, 15, 18 years at Time 1.	Time 1: adolescents' descriptions of **parent hostility**.	Time 2 (3 years later): adolescents' reports of how they generally cope with problems. Dysfunctional methods of **coping** included use of alcohol and drugs and emotional outbursts.	Maternal and paternal hostility was associated with more dysfunctional coping among sons (a pattern not consistently observed in the girls' data, except for cross-sectional analyses).

(*table continues*)

Table 2 (*continued*)

Study and design	Sample characteristics	Family environment measure	Child/adolescent emotion processing variable	Key findings

Emotion understanding and coping studies: Conflict and aggression (*continued*)

| Valentiner et al., 1994
CORR/FLW-UP
SELF | College students (*n* = 175; 124 women, and 51 men). *M* age at Time 2 = 20 years (*SD* = 0.4). | Time 1 (fall of freshman year): composite measure based on students' descriptions of amount of **marital conflict** at home and the general **emotional quality of their relationships with their parents** (e.g., supportiveness of each parent). | Time 2 (2 years later): self-reported coping with the most important problem experienced in the past year. Coping was scored as the percentage of all coping responses that represented **"approach" coping** (i.e., positive reappraisal of the problem and problem solving attempts). | In LISREL models, there was an association between a more negative family environment and less frequent use of approach coping strategies (with controllable problems). The association between a negative family environment and psychological distress was mediated by less approach coping. |

Emtion understanding and coping studies: Cold, unsupportive, and neglectful homes

Hardy et al., 1993 CORR/CS IND RPRTS	Children and their mothers (*n* = 60). Age range: 9–10 years. *M* age = 10.2 years.	**Family support** rating based on maternal descriptions of family cohesion, her own nurturance and responsiveness to child input, her monitoring or awareness of her child's life, and family adaptability.	Interview-based child descriptions of responses to, and attempts to cope with, stressful situations that had been identified by the mother as having occurred during the past 2 months. **Avoidant strategies:** removing self from the situation, giving up trying, use of distraction to avoid thinking about the situation.	A less supportive family environment associated with fewer different coping strategies and with less use of avoidant coping strategies in uncontrollable situations.
Laible & Thompson, 1998 CORR/CS IND RPRTS	Young children and their mothers (95% White; *n* = 40). Age range: 2.5–6 years. *M* age = 50 months.	**Security of child's attachment** assessed by *Q*-sort in which mother described her child's current behavior with primary caregivers.	**Emotion understanding composite:** recognition of emotions corresponding to depicted facial expressions; identification of emotions portrayed by a puppet in a vignette; naturalistic assessment of appraisals of emotion events as they occurred at school (identification of emotion expressed by another child and description of its causal antecedents).	Children with less secure attachments to their primary caregivers demonstrated less understanding of negatively valenced emotions, such as sadness, anger, and fear.
van den Boom, 1994 EXP/FLW-UP OBS	Mother–infant pairs (*n* = 100). All infants were rated as "irritable." Child age, start of treatment: 6 months; end of treatment: 9 months; follow up: 12 months.	Time 1: A parenting skills training program was provided during home visits to half of the pairs over a 3-month period. The intervention succeeded in enhancing **maternal responsiveness, stimulation, visual attentiveness, and control.**	Time 2: postintervention observation of infant behavior during home visits (**mother–infant interaction** in a family context and in a free play situation to assess **quality of infant exploration**) and a 12-month follow-up assessment of attachment security.	Infants whose mothers received the intervention were more sociable, better able to soothe themselves, and engaged in more cognitively sophisticated exploration (at end of treatment). At follow-up, more of the intervention infants were securely attached.

Note. The key family and outcome variables in each study are highlighted in bold type. For each study, design (GC = group comparison; CORR = correlational; EXP = experimental; timing of assessments (CS = cross-sectional; FLW-UP = multiple data collection points, but change over time in the outcome is not assessed), and sources of data for primary variables (IND RPRTS = assessment of predictor and outcome variables based on data from separate sources; OBS = observational data; SELF = all self-report data) are reported.

research summarized in Table 2, relating measures of conflict and hostility at home to adolescents' descriptions (provided 2–3 years later) of how they coped with different kinds of problems and stressors, the strategies favored by teens from the risky families emphasized a desire to reduce tension and escape the situation (V. Johnson & Pandina, 1991; Valentiner, Holahan, & Moos, 1994). This pattern was also found in the short-term reaction studies reported in Table 2; in the studies of preteens and teens, those from high-conflict homes tried to distract their own and others' attention away from interpersonal conflict (Gordis, Margolin, & John, 1997; O'Brien et al., 1991). It is interesting that in the short-term studies of younger children, those from high-conflict homes sometimes engaged in solicitous or placating behavior (Camras & Rappaport, 1993; E. M. Cummings et al., 1981; J. S. Cummings, Pellegrini, Notarius, & Cummings, 1989). It is possible that after repeatedly failing to change stressful events in the family, perhaps through behaviors such as appeasement and placation, children growing up in angry and aggressive homes gradually abandon efforts to control difficult situations and focus, instead, on simply trying to escape and recover from heightened emotional arousal. In short, the legacy of growing up with high levels of overt anger and aggression at home may be not only a stronger emotional reaction in situations that involve conflict, but also a particular set of behaviors for responding in those situations.

Cold, unsupportive, and neglectful homes. Table 2 also summarizes research findings relating deficient nurturing to emotion processing, effects that have been observed very early in development. Babies begin to regulate their emotional responses soon after birth, engaging in behaviors such as sucking to soothe themselves (Campos, 1988). Parental nurturing appears to facilitate the development of these primitive coping behaviors. For example, in an experimental study listed in Table 2, an intervention for mothers of irritable newborns that improved maternal responsiveness, attentiveness, and control also resulted in an increase in infant self-soothing behaviors. The irritable infants whose mothers did not receive the intervention actually showed a slight decrease in self-soothing from 6 to 9 months, and they were judged as having less secure attachments to their mothers (van den Boom, 1994). A short-term reaction study of toddlers (Nachmias et al., 1996) and two coping studies also indicate that an insecure parent–child attachment or little cohesion and support in the family are associated with less adaptive coping across a wide age range (Hardy, Power, & Jaedicke, 1993), and with deficits in emotion understanding among preschoolers (Laible & Thompson, 1998).

Summary. The findings summarized in Table 2 indicate that growing up in a risky family environment interferes with the development of means for processing emotions. In particular, the data point to high emotional reactivity, deficits in emotion understanding, and a reliance on unsophisticated coping responses to stressful situations. Across studies using different methodologies and age groups, findings indicate that children living in risky family environments are more likely than their peers to focus on tension reduction, distraction, and escape in stressful situations, a relation that is also found in cross-sectional studies (e.g., Stern & Zevon, 1990).

Emotion Processing and Mental and Physical Health Outcomes

Figure 1 depicts emotion processing as a link from risky family characteristics to adverse mental and physical health outcomes. Poor regulation of emotions is implicated in more than one half of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.) Axis I and in almost all of the Axis II psychiatric disorders (American Psychiatric Association, 1994). A small but growing research literature has tied indicators of emotion regulation to both internalizing and externalizing symptoms in children and adolescents (Eisenberg, Fabes, & Murphy, 1996; Southam-Gerow & Kendall, 2002; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). In addition to acting as a mediator of the link between negative family environments and mental health (Valentiner et al., 1994), emotion processing may also moderate a child's vulnerability to risky family characteristics. In one study, for example, the link between parents' marital hostility and subsequent externalizing behavior in offspring was observed only among children with inadequate emotion regulation (Katz & Gottman, 1995).

Emotion processing is also implicated in physical health, first, because of interrelations between the regulation of emotional and physiological responses to stress. For example, children who are emotionally reactive in certain situations (such as angry social interactions) are also more likely to be physiologically reactive (El-Sheikh et al., 1989). High vagal tone, which is based on an index of heart rate and heart rate variability and is a marker of parasympathetic functioning, indicates both better homeostatic capacity and better regulation of emotional responses to daily stressors (Fabes & Eisenberg, 1997). In one study, high vagal tone in 4 to 5 year olds predicted more effective emotion regulation 3 years later, and was cross-sectionally correlated with more parent "emotion coaching" (i.e., discussions of effective ways to cope with anger or distress; Gottman et al., 1996). In another study, high vagal tone buffered the impact of marital conflict on physical health; there was a weaker association between conflict in the home and health problems among the children with high vagal tone (El-Sheikh, Harger, & Whitson, 2001).

One emotion in particular, anger, appears to play a significant role in the development of coronary artery disease and hypertension, at least among some individuals (e.g., Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; Jorgensen, Johnson, Kolodziej, & Schreer, 1996; Julkunen, Salonen, Kaplan, Chesney, & Salonen, 1994; Smith, 1992). Emotion processing may also be indirectly implicated in the onset and course of certain diseases through its link with psychopathology, particularly with respect to mental health problems that involve chronic or recurrent negative emotional states. Depression and anxiety appear to play a significant role in numerous health risks, including all-cause mortality (L. R. Martin et al., 1995). Epidemiological, psychological, and experimental evidence point to a clear dose–response relation of anxiety and coronary heart disease (Kubzansky, Kawachi, Weiss, & Sparrow, 1998). Major depression, depressive symptoms, history of depression, and anxiety have all been identified as predictors of cardiac events (Frasure-Smith, Lesperance, & Talajic, 1995), and depression is a risk factor for mortality following a myocardial infarction, independent of cardiac disease severity (Frasure-Smith et al., 1995). State depression and clinical depres-

sion also have been related to sustained suppressed immunity (Herbert & Cohen, 1993).

Emotions are pivotal in our model. As depicted in Figure 1, disruptions in emotion processing occur early in the cascade and are directly linked to each of the other factors that mediate the effects of risky families.

Social Competence

As Figure 1 shows, emotion processing ultimately blends into social competence, that is, how skilled children are at managing the often frustrating and challenging experiences they have with family and peers. For example, in order to negotiate difficult social interactions, such as peer conflicts, children must learn how to respond in a socially appropriate manner while feeling frustrated and angry. The importance of emotion regulation for children's social functioning has been extensively studied, and research consistently shows that emotionally intense children who are poor regulators of their emotions are liked less by their classmates and viewed as less socially competent by observers (e.g., Cassidy, Parke, Butkovsky, & Braungard, 1992; Eisenberg et al., 1993; Eisenberg et al., 1997; Gottman et al., 1996; Krevans & Gibbs, 1996). Popular and socially competent children are better able to control their angry and excited emotions in arousing situations, and they tend to display less overt negative emotion than other children (Hubbard & Cole, 1994).

Risky Family Characteristics and the Development of Social Competence

Table 3 summarizes findings from investigations that have related risky family characteristics to indicators of social competence, in particular the quality of social behavior and relationships outside of the home. The child participants in these studies ranged in age from infants to adolescents. Two long-term studies, in which adults were recontacted 20–30 years after an initial assessment when they were in their 20s, are also included (Graves, Wang, Mead, Johnson, & Klag, 1998; Klohnen & Bera, 1998). Each of the 16 studies cited in Table 3 has one or more of the following characteristics: a longitudinal or follow-up research design (7 studies) and information obtained from independent sources or observational data, or both (13 studies), ensuring that correlations between the family environment and social competence were not inflated by individual respondent bias.

Conflict and aggression. The first group of studies in Table 3 addresses the social behavior and social standing of children living in homes with high levels of conflict and aggression. Those living with hostile and aggressive parents had fewer of the positive skills that facilitate successful interactions with peers (Crockenberg & Lourie, 1996; Pettit, Dodge, & Brown, 1988) or were more likely to behave in an aggressive or antisocial manner (C. H. Hart, Nelson, Robinson, Olsen, & McNeilly-Choque, 1998; Schwartz, Dodge, Pettit, & Bates, 1997). Other studies found that sons from aggressive families were more likely to be rejected and victimized by peers (Dishion, 1990; Schwartz et al., 1997), and women who had grown up in troubled and conflictual homes had more avoidant attitudes and feelings about closeness and intimacy (Klohnen & Bera, 1998).

Cold, unsupportive, and neglectful homes. The second group of studies in Table 3 is part of a growing literature pointing to the negative effects that a lack of warmth and nurturance can have on the ability to form and maintain social relationships. Most of these studies have examined links between quality of the mother–child bond (focusing in particular on attachment security) and children's relationships with peers. The results indicate that children whose parents were less responsive, warm, and sensitive were less likely to initiate social interactions and were more aggressive and critical (Brody & Flor, 1998; C. H. Hart et al., 1998; Kerns, Klepac, & Cole, 1996; Landry, Smith, Miller-Loncar, & Swank, 1998). In addition, when parents were cold, unsupportive, or neglectful, their offspring's social relationships throughout life were more problematic and less supportive (Booth, Rose-Krasnor, McKinnon, & Rubin, 1994; Bost, Vaughn, Washington, Cielinski, & Bradbard, 1998; Graves et al., 1998; Kerns et al., 1996; Larose & Boivin, 1998; MacKinnon-Lewis, Starnes, Volling, & Johnson, 1997).

Overall, the findings summarized in Table 3 suggest that the development of social competence and supportive relationships outside of the family are compromised by growing up in a risky family environment. Next, we argue that risky families have this effect because they shape the way that offspring come to think about and behave in relationships.

Social skills. There are several ways that risky families can hinder the early acquisition of social skills for initiating and maintaining friendships and for managing difficult interpersonal situations, such as those involving conflict and anger. First, young children model the social behavior that they observe in the family. Empirical evidence points to a close correspondence between social skills observed in the family and a child's behavior when interacting with peers. Children growing up in families in which complex social skills are rarely demonstrated (e.g., sensitivity to the child's feelings or needs) demonstrate fewer conflict management skills and are less sensitive and responsive with peers (Herrera & Dunn, 1997; Lindsey, Mize, & Pettit, 1997; Putallaz, 1987). Similarly, children who are the recipients of anger, aggression, and hostility from siblings and parents are, in turn, described by their teachers as less socially competent and more aggressive (Carson & Parke, 1996; Stormshak et al., 1996). In addition to acting as role models, parents engage in active efforts to shape their children's relationships and social skills through discussions of social problems and advice giving (Laird, Pettit, Mize, Brown, & Lindsey, 1994). Mothers who suggest fewer constructive techniques to solve social problems have children who themselves have fewer social skills, engage in more aggressive and less prosocial behavior, and are less likely to generate prosocial solutions to problems (Eisenberg, Fabes, & Murphy, 1996; Mize & Pettit, 1997; Pettit et al., 1988).

Social cognition. Social relationships in adolescence and adulthood are also shaped by aspects of social cognition first developed in childhood. On the basis of the same risky family experiences that shape social skills, children may develop and store in memory "social algorithms," "relationship schemas," or "working models" of self and others in close relationships that are activated and applied in new situations throughout life (Andersen & Berk, 1998; Bugental, 2000). For example, evidence suggests that growing up in a violent household shapes the development of the basic cognitive structures that guide social behavior and rela-

Table 3
Risky Family Characteristics and Social Competence

Study and design	Sample characteristics	Family environment measure	Child/adolescent social competence variable	Key finding
		Conflict and aggression		
Crockenberg & Lourie, 1996 CORR/FLW-UP IND RPRTS OBS	Mothers and their children (*n* = 42). Child age: 2 years at Time 1, 6 years at Time 2.	Time 1, **maternal negative control strategies** (based on lab and home observations): frequency with which mother used negative, highly power-assertive, child-control strategies, including maternal expressions of anger, annoyance, and criticism, and use of threats, punishment, and physical control.	Time 2 (4 years later): children described **peer conflict-resolution strategies** in actual and hypothetical situations. **Unskilled avoidance of conflict**: child gives up own goals and does nothing overt to resolve the problem; does not seek help of an authority figure. **Manipulative behavior**: covert power assertion, such as "finagle," and manipulation of friendship (e.g., tells friends not to play with the other child).	Maternal negative control strategies predicted less competent conflict-resolution strategies: Daughters described more unskilled avoidance, sons described more manipulative strategies.
Dishion, 1990 CORR/CS IND RPRTS OBS	Boys and their families (99% White, mostly working class and lower SES, 32% of parents unemployed, 35% single-parent homes; *n* = 206).	**Negative and aggressive parenting composite**: (based on home observations) *nattering*— noncontingent aversiveness with the child, parent negative verbal (e.g., commands) and physical behavior regardless of the child's behavior; *punishment density*— relative frequency of parent-to-child verbal and physical aggressive behavior in relation to positive parent–child interaction; observer impressions that parents were ineffective, unfair, and inconsistent in discipline practices.	Sociometric assessment, based on classmates' nominations of child (a) as "a friend you like," and someone "who makes friends easily" (**peer acceptance**) and, (b) as someone who doesn't "get along with most other kids," and "you don't want to be friends with" (**peer rejection**). **Aggressive and antisocial behavior composite** (argues, disobedient, lies, steals, etc. across multiple settings) based on parent questionnaire and 6 daily telephone interviews, teacher questionnaire, and child self-report (structured interview, and daily telephone interviews).	More negative and aggressive parenting (particularly discipline that was unfair and inconsistent) associated with more peer rejection and less peer acceptance, an effect mediated by aggressive and antisocial behavior.
Hart et al., 1998 CORR/CS IND RPRTS	Parent–child dyads (children attending nursery school in Russia; *n* = 207). Child age range: 3.5–6.5 years. *M* age = 5.1 years.	**Maternal coercion**: self-reported slapping, grabbing, yelling; use of physical punishment and shouting as discipline. **Paternal responsiveness**: self-reported patience, being easy-going and relaxed, joking and playing, being responsive to child feelings/needs, giving comfort and understanding when child is upset. **Marital conflict**: mother's and father's reports of overt conflict observed by the child.	Teacher ratings of **relational aggression**: causing harm through damage to relationships, such as social exclusion; **overt aggression**: causing harm through damage to physical or psychological well-being, such as bullying, threatening, and use of physical force.	Coercive parenting tactics associated with more relational and overt aggression (with controls for other aspects of parenting, child sex and age, parent education). Sons (but not daughters) who observed more marital conflict were more aggressive (both overt and relational aggression).

(*table continues*)

Table 3 (*continued*)

Study and design	Sample characteristics	Family environment measure	Child/adolescent social competence variable	Key finding
		Conflict and aggression (continued)		
Klohnen & Bera, 1998 GC/FLW-UP SELF	1958 and 1960 college seniors (all women; $n = 85$). Time 1: senior year in college (age 21). Time 2: age 52.	Time 1: retrospective description of "troubled" home life during childhood and adolescence (e.g., whether there was open conflict, a lack of family closeness, and whether the participant had thought about running away from home).	Time 2 (32 years later): attachment style measure—attitudes and feelings about closeness, intimacy, and interdependence in relationships. Responses used to categorize midlife women as having secure versus avoidant attachment styles.	Women with secure versus avoidant attachment styles in midlife were compared. An avoidant attachment style in midlife was predicted by a more troubled and conflictual home in childhood.
Pettit et al., 1988 CORR/CS IND RPRTS	Preschoolers and their mothers (most Caucasian, lower SES, single parents; $n = 46$). Child age range: 4–5 years.	Maternal hostile/aggressive attitudes: endorsement of aggressive solutions to interpersonal problems. Hostile attributions: interpretation of negative child behavior as having a hostile intent (as assessed by stories describing an ambiguous provocation directed toward the mother).	Deficient social problem solving: child responses, to hypothetical social situations, that suggest a tendency to generate fewer solutions (especially effective, prosocial solutions) to social problems, and a propensity to respond with aggression. Social competence composite: teacher ratings and classmates' nominations of children who are cooperative and "nice to play with."	Mothers' hostile and aggressive attitudes associated with less social competence, an effect mediated by deficient social problem solving.
Schwartz et al., 1997 GC/FLW-UP IND RPRTS OBS	Boys and their mothers (majority from lower to middle SES backgrounds; $n = 198$). Time 1: summer before kindergarten. Time 2: 3rd grade (M age = 8 years) or 4th grade (M age = 9 years).	Time 1, maternal hostility: observers' ratings, during a home visit, of physical and verbal hostility and annoyance directed toward the child. Derived from interview with mother: (a) maternal restrictive discipline—use of severe, strict, physical discipline with child, (b) parental aggression—own and partner's use of overtly aggressive strategies with child during conflict, (c) marital aggression—own and partner's use of overtly aggressive strategies toward each other during conflicts, and (d) physical harm—interviewer rating of "probable" or "definite" harm to child.	Time 2 (4–5 years later): victim/ aggressor status assessed by sociometric data from classmates. Victim score: sum of nominations received for three items ("gets picked on," "gets teased," "gets hit or pushed"). Aggressor score: sum of nominations received for three items ("starts fights," "says mean things," "gets mad easily"). Aggressive victims: boys whose victim and aggressor scores were both well above the mean ($n = 16$).	Boys who were "aggressive victims" in 3rd/4th grade were compared with all other boys. Being both a victim and aggressor was predicted by a more hostile and aggressive family life.
		Cold, unsupportive, and neglectful parenting		
Booth et al., 1994 CORR/LONG: OBS	Children and their mothers (87% White; $n = 79$). Child mean age Time 1: 4.3 ($+/-$ .1) years, Time 2: 8.0 ($+/-$ .15) years.	Time 1, attachment security: coding of child's behavior upon reunion with the mother following a brief (approximately 15 min) separation.	Social engagement, Time 1: child behavior during a play session with an unfamiliar same-sex peer, coded for proportion of positive or neutral conversation and play. Time 2 (4 years later): composite based on child behavior during a play session with three unfamiliar same-sex peers, coded for proportion of positive or neutral conversation and play, and likeability ratings made by the three other children.	A less secure maternal attachment predicted a decline in positive engagement during play.

Table 3 (*continued*)

Study and design	Sample characteristics	Family environment measure	Child/adolescent social competence variable	Key finding
		Cold, unsupportive, and neglectful parenting (*continued*)		
Bost et al., 1998 CORR/CS OBS	Mother–child dyads (*n* = 69). Child age range: 3–4 years.	**Attachment security**: *Q*-sort rating made by two observers, after a home visit, on the basis of child behavior with a specific caregiver. Rating based on child's use of caregiver as secure base for exploration and as a haven when threatened or otherwise distressed.	**Social competence composite**: observers' *Q*-sort ratings of child classroom behavior; frequency of attention from classmates; sociometric ratings from classmates (nomination and social preference). **Social network composite**: child report of satisfaction with social support and the number of individuals available for recreation, emotional support, and child care tasks.	Less attachment security associated with less social competence, an effect mediated by a less satisfying and smaller social network.
Brody & Flor, 1998 CORR/CS OBS	Mother–child dyads from mostly poor, rural, Black, single-mother households (*n* = 156). Child age range: 6–9 years.	Observers' ratings of **"harmony" in the mother–child relationship** (warm, supportive, relaxed, positive affect) based on mother–child interaction during three tasks (game playing, story telling, model building).	**Social competence composite**: teacher ratings of child social competence, behavioral conduct problems, anti-social behavior. **Child self-regulation composite**: mother and teacher ratings of child self-control (plans ahead, persists, focuses attention).	Less mother–child harmony correlated with less social competence; an effect mediated by deficient self-regulation.
Graves et al., 1998 CORR/FLW-UP SELF	Time 1: students (93% male) enrolled in medical school (1948–1964; *n* = 589). *M* age = 22 years. Time 2: *M* age = 56 years.	Time 1, **emotional closeness to parents**: self-report adjective checklist measure of emotions characterizing mother–child and father–child relationships (warmth, understanding, detached, etc.) assessed while respondents were in medical school (1948–1964).	Time 2 (over 20 years later). **Social connectedness**: self-reported number of close friends and family members that respondent feels "at ease with, can talk to about private matters, can call for help, or would lend you money," as well as the number of people for whom respondent provides support functions like these.	Less closeness to parents predicted less social connectedness (with controls for respondent age, race, and parent education).
Hart et al., 1998 CORR/CS IND RPRTS	See above.	See above.	See above.	Less paternal responsiveness associated with more relational and overt aggressive child behavior (with controls for other aspects of parenting, child sex and age, parent education).
Kerns et al., 1996 Study 1 CORR/CS IND REPORTS	Study 1: 5th graders (*n* = 76);	**Child–mother relationship security**: child report of maternal responsiveness, availability, reliability, interest in communication with child (secure attachments = top 2/3 of the distribution of scores).	Study 1, **sociometric ratings**: classmates' ratings of how much they like to play with the child, and of close friendships. Self-reported feelings of **loneliness**.	Study 1: Less secure maternal attachments associated with being less well liked by classmates, fewer reciprocated close friendships, and feeling lonely.
Study 2 GC/CS OBS	Study 2: best friend dyads (5th and 6th graders; *n* = 55).		Study 2: Videotaped discussion between best friends coded for **criticisms** voiced (i.e., negative evaluations of another, hostile comments, complaints) and **responsiveness** (degree to which they attended to, acknowledged, and responded to each other's social cues). Best friends also rated the degree of **companionship** in their relationship.	Study 2: Compared with best friend dyads in which both children had secure maternal attachments, dyads in which one child had an insecure attachment were more critical and less responsive to each other's social cues and felt less companionship in their relationship.

(*table continues*)

Table 3 (*continued*)

Study and design	Sample characteristics	Family environment measure	Child/adolescent social competence variable	Key finding
		Cold, unsupportive, and neglectful parenting (*continued*)		
Landry et al., 1998 CORR/LONG OBS	Infants and their mothers (*n* = 299). Ages: 6 months, 12 months, 24 months, 40 months.	**Warm sensitivity composite:** mother's sensitivity to her child's cues (promptness and appropriateness of reactions, acceptance of the child's interests, amount of physical affection, positive affect, and tone of voice).	**Infant social skills:** growth, over 34 months, in social behaviors (gestures, positive affect, eye gaze, vocalizations/ words) observed while at home with mother during daily activities. **Initiations of social interaction:** social behaviors when the mother had not interacted with the infant for at least 3 s. **Responsiveness:** social behaviors that followed within 3 s of a maternal attention-directing event. Plus, orienting to the focus of mother's attention directing behavior (coded at 6 and 12 months) and compliance to maternal requests (coded at 24 and 40 months).	A lack of maternal sensitivity to infant social cues predicted slower growth in social skills, particularly with respect to initiations of social interactions and responsiveness.
Larose & Boivin, 1998 CORR/LONG: SELF	Adolescents (*n* = 285). Age range: 15–20 years. *M* age: 17 years (at both data collection points).	Time 1 (end of high school), **security in the parent-adolescent relationship:** degree of mutual trust, quality of communication, prevalence of anger and alienation with mothers and fathers.	Time 2 (middle of the first semester at college): **expectations of social support** to cope with worrisome events related to late adolescence and to college transition; **feelings of loneliness** experienced in interpersonal relationships over the last month.	A less secure parent–adolescent relationship predicted a decline in expectations of social support and greater feelings of loneliness during the transition to college.
MacKinnon-Lewis et al., 1997 CORR/CS (all variables assessed over the course of a single year). OBS IND REPORTS	Triads: 3rd–5th grade boys, their siblings, and their mothers (*n* = 71). Target child age range: 8–10 years. *M* = 9.2 years. Sibling age range: 4–14 years. *M* = 8.75 years.	**Maternal rejection composite:** target child report, sibling report, and mother's observed negativity with both children during a play session (board game).	**Sibling aggression:** negative behavior by target child and sibling during a 15 min play session (board game), includes verbal and physical behavior and expression of affect. **Aggression with peers:** teacher and classmate ratings. **Social acceptance:** sociometric nominations by classmates.	Maternal rejection was associated with less social acceptance, an effect mediated by aggressive behavior (with sibling and peers).

Note. The key family and outcome variables in each study are highlighted in bold type. For each study, design (CORR = correlational; GC = group comparison), timing of assessments (FLW-UP = multiple data collection points, but change over time in the outcome is not assessed; CS = cross-sectional; LONG = longitudinal), and sources of data for primary variables (IND RPRTS = assessment of predictor and outcome variables based on data from separate sources; OBS = observational data; SELF = all self-report data) are reported. SES = socioeconomic status.

tionships in childhood and adulthood. In a study of college students, the negative effect of childhood exposure to physical aggression at home on current relationships was mediated by heightened rejection sensitivity (worries about social acceptance; S. Feldman & Downey, 1994). During childhood, the link between physical abuse at home and aggressive behavior with peers is partially mediated by patterns of social information processing, such as tendencies to attribute hostile motives to others, to pay less attention to relevant social cues, and to think of fewer effective behavioral responses to problematic social situations (Dodge, Bates, & Pettit, 1990).

Hostility. Hostility is an oppositional orientation toward people stemming from feelings of insecurity about oneself and negative feelings toward others (Houston & Vavak, 1991). Early family environments characterized as unsupportive, unaccepting, and conflictual contribute to the development of hostility (Houston & Vavak, 1991; Smith, Pope, Sanders, Allred, & O'Keefe, 1988; Woodall & Matthews, 1989), a link that has been documented in longitudinal studies (Matthews, Woodall, Kenon, & Jacob, 1996; Woodall & Matthews, 1993). In addition to its origins in the family environment, hostility may also have biological origins, specifically representing a psychological response to high levels of physiological reactivity (Fukudo et al., 1992; Krantz & Manuck, 1984). To the extent that hostility has a genetic basis in physiological reactivity, parents and children who share genes that predispose them to this reactivity may create and respond to the family environment in ways that foster, rather than counteract, the development of hostility. Probably no single construct better reflects the complex intertwining of biological regulatory systems, emotion processing, and social competence that is at the core of our concept of an integrated risk profile. Biopsychosocial constructs such as hostility are ideally suited for models with multiple developmental processes that have cascading effects over time; they are precisely the type of outcomes our model predicts, manifesting themselves most frequently later in the cascade of developmental processes.

Summary. Parents and siblings in risky families are poor models of prosocial behavior, and they do not provide other kinds of active socialization that would facilitate the early development of complex social skills. Social experiences in risky families may also contribute to social information-processing rules and biases, and to mental representations of self and others, that interfere with positive social interaction and the maintenance of healthy relationships. Moreover, the early and continuing impact of warped emotion processing, such as increased reactivity to anger and conflict, place added demands on the social skills of children from risky families and further impede the development of social competence.

Social Competence and Mental and Physical Health Outcomes

Social competence is an integral component of mental health at all ages. Longitudinal studies show that school-age children who are rejected or neglected by their peers are at an increased risk for behavioral and emotional problems a few years later (Hymel, Rubin, Rowden, & Le Mare, 1990; Kupersmidt & Patterson, 1991). A lack of social integration among adults, particularly with respect to primary ties with supportive significant others, such as a spouse or children, is associated with an increased risk of depression (George, 1989). There is also a long-term association

between childhood social competence and adult mental health; rejected children are at an increased risk for adult psychopathology (Bagwell, Newcomb, & Bukowski, 1998; Parker & Asher, 1987).

In terms of physical health risks, social competence most clearly translates into the ability to attract and sustain social support. In over 100 investigations, social support has been documented to reduce health risks of all kinds, affecting the likelihood of illness initially, the course of recovery among people who are already ill, and mortality risk more generally (House, Umberson, & Landis, 1988; Seeman, 1996; Uchino, Uno, & Holt-Lunstad, 1999). Some of the social behaviors associated with risky family environments, such as a hostile interpersonal style, can also generate stress. Research suggests that conflictual social interactions may contribute as much to illness and poor health as supportive social contacts contribute to good health (e.g., Rook, 1984; see Taylor, 1999). For example, hostility has been tied to high levels of low-density lipoprotein cholesterol, high levels of triglycerides, and a higher ratio of total cholesterol to high-density lipoprotein cholesterol in women (Suarez, Bates, & Harralson, 1998), as well as to the likelihood of developing coronary heart disease in adulthood (Dembroski et al., 1985). The association between hostility and cardiovascular health may be multidetermined, partly mediated by the elevated physiological reactivity that appears to be a component of hostility and partly mediated through the added stress of interpersonal conflict.

Through their impact on social competence, and the skills and cognitions it entails, childhood family environments influence the kind of interpersonal relationships that offspring have throughout life. It is through this channel, particularly because relationships can act as sources of social support and social stress, that the development of social competence in the family has a lasting impact on mental and physical health. Although no single study has addressed this entire model—which links the family environment to social competence, the quality of subsequent relationships, and ultimately, health—there is empirical support for several of the steps. Ewart (1991) reviewed evidence that hostile parenting produces deficits in social competence that foster vulnerability to emotionally charged negative interpersonal events, which, in turn, is associated with heightened cardiovascular reactivity and disease risk. With respect to mental health outcomes, evidence indicates that chronic interpersonal stress in adulthood is one of the conditions that connects exposure to family violence during childhood to recurrence of depression in adulthood (Kessler & Magee, 1994).

Substance Abuse and Risky Sexual Behavior

Adolescence brings increasing autonomy to make decisions about how, and with whom, to spend time. The range of options for teens in the United States includes behaviors that pose serious threats to health. Although parents can exert some direct control over teenagers' behavior and activities, our model suggests that the family's indirect effects are even more significant. As depicted in Figure 1, the cascade of influences impinging upon the behavior of the adolescent offspring of risky families includes all of the earlier disruptions in biological regulation, emotion processing, and social competence. In this section, we discuss how a risky family environment might increase the likelihood that an adolescent will engage in behaviors that threaten health. We focus on two classes of behavior—substance use (including alcohol, cigarettes, and

illicit drugs) and risky sexual behaviors (such as early, promiscuous, and unprotected sexual intercourse)—because of the short- and long-term risks they pose for mortality, severe illness, or serious life disruption.

The substance abuse treatment literature indicates that repair of the family social environment reduces adolescent drug use. For example, family therapy is more effective than individual or peer group counseling (Stanton & Shadish, 1997), and improvements in parenting practices over the course of family therapy are associated with reductions in drug use (S. E. Schmidt, Liddle, & Dakof, 1996). These findings highlight the important part played by the family in changing patterns of drug use, although they do not rule out the possibility that the same characteristics of a child may both undermine the development of supportive family relationships and also increase his or her propensity to abuse substances (Wills, DuHamel, & Vaccaro, 1995).

Risky Family Characteristics and Substance Abuse and Risky Sexual Behavior

The specific characteristics of risky families have been tied to increased rates of smoking, alcohol abuse, drug use, and risky sexual behavior in adolescence and adulthood. For example, some research suggests that the experience of abuse in childhood is a risk factor for these behaviors (Malinosky-Rummell & Hansen, 1993; Small & Luster, 1994). The evidence for this association is based primarily on cross-sectional analyses of data provided by a single respondent, often retrospective descriptions of abuse in childhood (Anda et al., 1999; R. M. Cunningham, Stiffman, Doré, & Earls, 1994; Dietz et al., 1999; Felitti et al., 1998; Harrison, Hoffmann, & Edwall, 1989). Studies with stronger research designs have found increased rates of alcohol abuse among adult women who grew up in conflictual or abusive homes. In one study, interpersonal conflict in the adoptive homes of female adoptees interacted with a biologic background of alcoholism (i.e., having at least one alcoholic biological parent) to increase the probability of alcohol abuse or dependence, although neither factor alone predicted problems with alcohol (Cutrona, Cadoret, et al., 1994). In another study, individuals with documented histories of abuse or neglect, or both, during childhood were interviewed approximately 20 years later. Women (but not men) who had been abused or neglected, or both, as children were more likely than were matched control persons to qualify for clinical diagnoses of substance abuse/dependence, particularly alcohol abuse; the effects of a history of abuse/neglect were observed even after controlling for parent alcohol/drug problems, as well as other background characteristics, such as race/ethnicity, childhood social class and neighborhood of residence, and parental arrest (Widom & White, 1997).

There is a larger body of prospective research supporting an association between growing up in a cold, unsupportive, or neglectful home and health-risk behaviors. The prospective studies summarized in Table 4 found increased rates of substance abuse or risky sexual behaviors among offspring of families lacking in cohesion or offspring of parents who were neglectful and unsupportive. In two long-term investigations, a less structured and organized home or a parenting style that was observed to be unsupportive, uninvolved, or less demanding in early childhood predicted more drug and alcohol use in adolescence (Baumrind, 1991; Shedler & Block, 1990). Similar results were obtained when

children were followed over 6 years (Barnes, Reifman, Farrell, & Dintcheff, 2000; Fisher & Feldman, 1998). In other longitudinal studies, a less nurturing family environment predicted future cigarette smoking, increased drug and alcohol use, and involvement in a pregnancy (Doherty & Allen, 1994; Mounts & Steinberg, 1995; Scaramella, Conger, Simons, & Whitbeck, 1998). There is a large body of corroborating cross-sectional data suggesting that teens who abuse substances or engage in risky sexual behavior are more likely to live in homes in which interpersonal relationships, particularly with parents, tend to be hostile, distant, unsatisfying, nonsupportive, or lacking in cohesion (Barrera, Chassin, & Rogosch, 1993; Campo & Rohner, 1992; Denton & Kampfe, 1994; DiBlasio & Benda, 1990; Miller, Forehand, & Kotchick, 1999; Newcomb, Maddahian, & Bentler, 1986; Resnick et al., 1997; Small & Luster, 1994; Spoth, Redmond, Hockaday, & Yoo, 1996; Turner, Irwin, Tschann, & Millstein, 1993; Wills & Cleary, 1996).

Direct and Mediated Effects of Risky Families

Why are children who grow up in risky families more likely to abuse substances and engage in risky sexual behaviors? Figure 1 suggests several avenues through which risky families may directly and indirectly influence these behaviors. First, research indicates that some of the most potent ingredients in a neglectful home are inadequate parental knowledge about and supervision of adolescents' activities. In homes with less parental monitoring and more permissiveness, adolescents engage in more frequent sexual activity and more risky sexual behavior (Jemmott & Jemmott, 1992; Metzler, Noell, Biglan, Ary, & Smolkowski, 1994; Miller et al., 1999; Romer et al., 1994; Small & Luster, 1994), and they smoke more (Biglan, Duncan, Ary, & Smolkowski, 1995). In a 6-year longitudinal study summarized in Table 4, the association between a lack of support and nurturance at home and adolescents' increased use of alcohol was mediated by the extent to which teens told their parents about their whereabouts and activities (Barnes et al., 2000). This is consistent with the suggestion that most measures of parental monitoring assess open channels of communication between parent and child that reflect child disclosure at least as much as parental efforts to control and manage children (Stattin & Kerr, 2000).

The direct impact of neglectful parents on opportunities for alcohol and drug use may be compounded through a concomitant increase in the influence exerted by peers. For example, longitudinal data suggest that the effect of peer drug use is much weaker when parenting is authoritative (i.e., when parents are involved, make demands, and supervise while demonstrating acceptance and warmth; Mounts & Steinberg, 1995) and that the impact of inadequate parental monitoring on problem behaviors, such as substance abuse and risky sexual behavior, is partially mediated by increased association with peers who engage in antisocial and deviant behavior (Ary, Duncan, Biglan, et al., 1999). Perhaps children in supportive families are motivated to please and to imitate their parents, which imposes internal barriers to engaging in behaviors that would disappoint them (Andrews, Hops, & Duncan, 1997). One study found that support from parents lowered teen tolerance for deviant behavior, which had both a direct impact on substance use and an indirect impact by decreasing affiliation with peers who use substances (Wills & Cleary, 1996).

Table 4
Cold, Unsupportive, and Neglectful Homes and Health-Risk Behaviors

Study and design	Sample characteristics	Family environment measure	Health risk behavior variable	Key finding
Barnes et al., (2000) CORR/LONG SELF	506 adolescents (58% female; 29% Black). Wave 1 age: 13–16 years (*M* age = 14.5 years). Wave 6 age: 18–22 years (*M* age = 19.9 years).	Parenting variables assessed at Wave 1. **Maternal support and nurturance**: adolescent report of maternal behavior that communicates the adolescent is valued and loved (including praise/encouragement, advice/guidance, affection). **Parental monitoring**: adolescent report of how often he or she tells parents where he or she is going.	**Alcohol misuse index** composed of (a) ounces of alcohol per day, (b) times drunk in past year, and (c) frequency of five or more drinks at a time in past year. The outcome variable in this study was a latent factor representing increases in alcohol misuse (i.e., the slope) over 6 years.	Less maternal support and nurturance was associated with an increase in adolescent misuse of alcohol over 6 years, an effect that was mediated by poor parental monitoring.
Baumrind, 1991 GC/FLW-UP OBS	139 children and their parents. White, affluent population. Age at Time 1: 4 years old. Age at Time 3: 15 years.	**Parenting** assessed at Time 1 by a team of observers during a home visit and a structured family-interaction task.	**Drug and alcohol use** based on a 2 hr interview (at Time 3) with the adolescent about dosage, frequency, and context of use of alcohol, tobacco, marijuana, and other illicit drugs.	The Time 1 ratings of the homes of children who went on to become heavy or dependent users of alcohol, drugs, or both were compared with the Time 1 home ratings of the children who were nonusers at age 15. The childhood homes of the heavy or dependent users had been described as **less structured and organized** 11 years earlier. In addition, the **parents were more often absent from the home and made fewer maturity demands** when the children were 4 years old.
Doherty & Allen, 1994 CORR/LONG IND RPRTS	Nonsmoking adolescents at Time 1 (*n* = 312), plus 304 of their mothers and 286 of their fathers. Age at Time 1: 11–13 years.	Parent reports of **family cohesion** at Time 1 (1982).	Self-reported **smoker status** at Time 2 (1988) defined as one or more cigarettes in the past month.	Lower family cohesion was associated with an increased likelihood that the child would have become a smoker during the subsequent 6 years, after controlling for parental smoking status.
Fisher & Feldman, 1998 CORR/FLW-UP SELF	*N* = 116 adolescents. Age at Time 1: 13–18 years. Age at Time 2: 19–25 years.	Adolescent rating at Time 1 of **cohesion, orderliness, and clarity of roles and rules** in the family.	Self-reported **risk behaviors** at Time 2 based on a composite of 11 behaviors (e.g., alcohol use, sexual behavior, tobacco use, violence-related behavior).	Greater organized cohesiveness in the family at Time 1 was associated with less risk behavior 6 years later.

(*table continues*)

Table 4 (*continued*)

Study and design	Sample characteristics	Family environment measure	Health risk behavior variable	Key finding
Mounts & Steinberg, 1995 CORR/LONG SELF	Two samples of 500 students (all significant findings were replicated in both samples). Time 1: 9th, 10th, and 11th graders. Time 2: 1 year later.	Time 1 rating of parental **authoritativeness** based on the adolescent's report of parenting practices. Authoritativeness was indicated by high ratings on strictness-supervision and on parental acceptance and involvement.	Assessed at Time 1 and at Time 2: adolescent's **self-reported frequency of use of alcohol, marijuana, and other drugs.**	Among adolescents whose parents were not authoritative, higher levels of peer drug use predicted an increase in the adolescent's own use of drugs and alcohol. This was not the case in the high-authoritativeness group, however.
Scaramella et al., 1998 CORR/FLW-UP OBS	6 waves collected over a 7-year period. *N* = 368 adolescents and their parents. Wave 1: 7th grade. *M* age = 12 years, 7 months (all-White rural sample).	Observer ratings made during Wave 1 of **parental warmth and involvement** directed toward the target adolescent during a family-interaction task.	**Substance use** (assessed in 9th and 10th grades): self-reported frequency of using various substances such as alcohol, tobacco, and illegal drugs (e.g., marijuana, crack). **Involvement in a pregnancy** (assessed in 9th, 10th, 11th, and 12th grades): self-reported pregnancy status (females); for males, belief that he was the father of a child conceived during this time period.	Observer ratings of less parental warmth and involvement in the 7th grade was associated with greater use of substances in the 9th and 10th grades and with an increased likelihood of being involved in a pregnancy by the 12th grade.
Shedler & Block, 1990 GC/FLW-UP OBS	*N* = 101 children who were studied both at age 5 and age 18.	A parent's manner of interacting with the child was observed during parent–child interaction tasks when the child was 5 years old and was then described by the observers with a *Q*-sort measure.	Drug use was assessed at age 18 during individual interviews. **Frequent users** were teens who reported using marijuana once per week or more and who had tried at least one other substance (*n* = 20).	The mothers of frequent users were more likely to be described as **cold, unresponsive, and underprotective**, when the children were 5 years old.

Note. The key family and outcome variables in each study are highlighted in bold type. For each study, design (CORR = correlational; GC = group comparison), timing of assessments (LONG = longitudinal; FLW-UP = multiple data collection points, but change over time in the outcome is not assessed), and sources of data for primary variables (OBS = observational data; IND RPRTS = assessment of predictor and outcome variables based on data from separate sources; SELF = all self-report data) are reported.

Perhaps most important, risky sexual behavior and substance abuse may compensate for deficiencies in the biological, social, and emotional functioning of adolescents from risky families. Adolescents who engage in risky sexual behavior are more likely to smoke cigarettes and abuse substances, and there is an association between drinking, smoking, and other drug use (Biglan et al., 1990; Capaldi, Crosby, & Stoolmiller, 1996; Donovan & Jessor, 1985; Kandel & Yamaguchi, 1993; Millstein & Moscicki, 1995; Shiffman et al., 1994; Tubman, Windle, & Windle, 1996).[10] A clustering of behaviors often suggests that they share common origins or serve common functions. Risky sexual behaviors, smoking, and substance abuse may represent adaptations to some of the negative consequences of having grown up in a risky childhood environment. As discussed in previous sections, these offspring may have more social problems than their peers, they may be more reactive to stress (particularly interpersonal stress), and they may have fewer coping strategies and sources of social support on which to draw. In one study, the association between unsupportive parents and adolescent substance use was partially mediated by teens' use of anger to cope and a lack of self-control (Wills & Cleary, 1996). Early and promiscuous sexual behavior and substance use may help adolescents manage negative emotions and gain social acceptance in the absence of adequate emotion coping strategies or social skills (e.g., Aloise-Young, Hennigan, & Graham, 1996; Mayne & Buck, 1997; see Taylor, 1999, for a review).

Some of the risky health behaviors discussed here may also act as methods of self-medication in response to biological dysregulations, in particular to the serotonergic dysfunctions that may occur in risky families. There is significant evidence relating smoking, alcohol abuse, and drug abuse to enhancement of serotonergic activity (Balfour & Fagerstrom, 1996; Jaffe, 1990; Ribeiro, Bettiker, Bogdanov, & Wurtman, 1993; Stahl, 1996; C. F. Valenzuela & Harris, 1997). Abuse of substances such as these may function, in part, to alleviate feelings of depression or hostility that arise in conjunction with serotonergic dysfunction (e.g., Stahl, 1996). Thus, substance abuse may help offspring with dysregulated serotonin functioning by facilitating maintenance of higher levels of serotonin through increasing release or impeding uptake of the neurotransmitter, or both.

Summary

By adolescence, the offspring of risky families must adapt to the cumulative consequences of years spent in a damaging home environment. Substance abuse and risky sexual behavior may help these adolescents compensate for their biological, emotional, and social deficiencies. These processes may be compounded by parents' inadequate knowledge about and supervision of teens' activities and by the adolescents' weak internal barriers to certain risky behaviors and their greater susceptibility to peer influence. Patterns of substance abuse and problems in behavioral self-regulation during adolescence as a result of growing up in a risky family are, no doubt, influenced by the dramatic hormonal changes that occur during puberty. Exactly how these changes might exacerbate behavior problems and substance abuse is not currently known, but investigation of this issue represents the kind of biobehavioral research suggested by our analysis.

Socioeconomic Status, Family Characteristics, and Mental and Physical Health Risks

A detailed examination of the larger social ecology within which family environments emerge is beyond the scope of this article. Yet, this sociocultural context is undeniably important. Peers, schools, and neighborhoods act on the sustaining factors that are at the core of our model and thereby have indirect effects, in addition to direct effects, on children's health and development (Gump, Matthews, & Raikkonen, 1999; Leventhal & Brooks-Gunn, 2000). The present analysis is fundamentally compatible with a macrolevel analysis of psychological and biological dysfunction that takes into account socioeconomic differences, as well as other aspects of a family's social context that may give rise to risky family social environments and to adverse mental and physical health outcomes (e.g., Aneshensel & Sucoff, 1996; Brody & Flor, 1998; Flinn & England, 1997; McLoyd, 1998; van IJzendoorn & Bakermans-Kranenburg, 1996).

Socioeconomic status (SES), an important dimension of a family's social ecology, is significantly related to early mortality, and an extensive, highly consistent literature documents the negative physical and mental health outcomes that result as one moves lower on the SES gradient (Adler, Boyce, Chesney, Folkman, & Syme, 1993; Chen, Matthews, & Boyce, 2002; Williams & Collins, 1995). Children growing up in poverty also show early signs of allostatic load, including elevated secretion of cortisol and epinephrine, and higher resting blood pressure. Moreover, the effects of poverty appear to be mediated through the child's exposure to multiple chronic stressors, such as violence and family turmoil (Evans & English, in press).

Low SES has also been tied to all of the risky family characteristics described here (e.g., Dodge, Pettit, & Bates, 1994), and loss of SES has been associated with an increase in these family characteristics. Poor children are at heightened risk for physical mistreatment or abuse (McLoyd, 1998; J. Reid, Macchetto, & Foster, 1999) and exposure to family violence (Emery & Laumann-Billings, 1998; Garbarino & Sherman, 1980; U.S. Department of Justice, 1994) and are also more likely to be in family relationships lacking in warmth and support (Bradley, Corwyn, McAdoo, & Coll, 2001; McLeod & Shanahan, 1996). Both sustained poverty and descent into poverty appear to move parenting in more harsh, punitive, irritable, inconsistent, and coercive directions (Wahler, 1990). McLoyd (1998) reviewed evidence that descent into poverty precipitates marital and parent–child conflict that, in turn, alters parental behavior in a hostile and coercive direction, leading to the development of internalizing and externalizing symptoms in young children (e.g., Conger, Ge, Elder, Lorenz, & Simons, 1994; Duncan, Brooks-Gunn, & Klebanov, 1994; see also Elder, 1974; Elder, Nguyen, & Caspi, 1985). These parenting characteristics may evolve, in part, as a result of deficient coping strategies for managing the stressors associated with low SES. Poor families living in high-crime neighborhoods must accommodate to persistent, multiple, uncontrollable demands that require constant effort to meet immediate physical and psycholog-

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[10] These behaviors tend to also coexist with other health-compromising behaviors, such as poor sleep and eating habits and little physical activity (Donovan, Jessor, & Costa, 1991), and with academic failure (Ary, Duncan, Duncan, & Hops, 1999).

ical needs. Such heavy chronic burdens favor reactive coping skills and can exacerbate the negative effects of other family vulnerability factors (Aspinwall & Taylor, 1997; Repetti & Wood, 1997).

Low SES may act not only as a contextual factor for understanding the development of risky families, but also as an outcome of growing up in a risky family environment. Research literatures in developmental psychology, sociology, and public health relate the characteristics of risky families to a wide range of adverse adult outcomes that cluster with low SES, including low school achievement, low educational attainment (in years), low adult income, high likelihood of divorce in adulthood, low occupational status, and poor status on other indicators of life success (Power & Hertzman, 1997). For example, in a study mentioned above, adults with documented histories of abuse or neglect or both during childhood were less likely to have completed high school or to be employed in managerial or professional occupations; they were also more likely to have arrest records (even excluding drug-related crimes). Moreover, the effects of the childhood family environment were observed after controlling for background characteristics, such as race/ethnicity, childhood social class and neighborhood of residence, and parental arrest and alcohol/drug problems (Widom & White, 1997). The impact of risky family environments on these other indicators of social success may represent additional routes by which risky families adversely affect the functioning of offspring over the lifetime.

Of course, low SES is not inevitably associated with risky family environments. Just as parenting characteristics may be an important mediator of the effects of low SES on children's mental and physical health, effective parenting may buffer children from the adverse effects of low SES. Cowen, Wyman, Work, and Parker (1990) found that so-called *stress resilient* children, who had successfully weathered a variety of chronic problems (including poverty, family turmoil, illness, and violence) were characterized by nonseparation from the primary caregiver during infancy, positive parent–child relations during preschool and elementary years, a strong sense of parenting efficacy by the parents, and parental use of reasoned, age-appropriate, consistent disciplinary practices (see also Masten, Morison, Pelligrini, & Tellegen, 1990; Rutter, 1990; Werner & Smith, 1982).

When SES is measured and appropriate analyses are reported, risky family characteristics continue to be associated with the sustaining factors in our model and with mental and physical health even after adjustments are made for SES differences in the sample. Nonetheless, the social context within which a family lives can shape the interpersonal environment at home, and these effects represent an important pathway through which socioeconomic factors influence health. In addition, social success in adulthood, evidenced by indicators such as educational and occupational attainment, may represent an additional pathway through which the effects of risky family characteristics are maintained over the lifetime.

Conclusions, Limitations, and Implications

Risky families are characterized by conflict, anger, and aggression, by relationships that lack warmth and support, and by neglect of the needs of offspring. These families are risky in multiple ways. First, several of these characteristics, most notably physical abuse and neglect, represent immediate threats to the lives and safety of children. Second, the fact that children's developing physiological and neuroendocrine systems must repeatedly adapt to the threatening and stressful circumstances created by these family environments increases the likelihood of biological dysregulations that may contribute to a buildup of allostatic load, that is, the premature physiological aging of the organism that enhances vulnerability to chronic disease and to early mortality in adulthood (McEwen & Stellar, 1993; Seeman et al., 1997). Third, risky families fail to provide children with important self-regulatory skills, leaving the child unable to effectively create and enlist social support or to deal with emotion-engaging interpersonal situations (as well as a wide array of other stressful events that may require effective coping skills). Finally, risky families increase children's vulnerability to behavior problems and substance abuse, including smoking, alcohol, drugs, and promiscuous sexual activity. These risks are multiple and pervasive, and they are related to each other through common biological and psychosocial pathways. Separately and in concert, they place a child not only at immediate risk, but also at long-term and life-long risk for adverse mental and physical health outcomes.

Of course, the processes that appear to be maladaptive in our model may, in fact, reflect a process of adaptation within the context of a risky family environment. For example, in a violent home, vigilence, attributions of hostile motives to others, and a coping style in which escape is prioritized may be highly adaptive. However, a price is paid for these adaptations; the costs may include heightened emotional and physiological reactivity, social problems with peers, deficient problem solving, and a lifestyle that poses health risks. Questions about the adaptiveness or maladaptiveness of a response can be addressed only with respect to the specific characteristics of the outcome in question, such as type of outcome (e.g., physical vs. psychological well-being), time frame (e.g., outcomes observed immediately vs. years later), setting (e.g., school vs. home), and unit of analysis (e.g., individual outcome vs. family or community well being; Repetti & Wood, 1997).

Children who are genetically predisposed to problems (such as those with overly reactive or overly inhibited temperaments) may be more adversely affected by risky family characteristics than are children without these preexisting vulnerabilities. Genes may influence the cascade of risk at multiple levels. A genetic factor may have direct effects on physiological reactivity, on emotional processing, and on social competence. Genes may also have indirect influences via the family environment, because of overlap in genetic risk between parent and child and the effect of the child's behavior on the social environment. Phenotypic expression of a genetic vulnerability to a particular mental or physical disorder, whether an inhibited temperament, a propensity for hostility, or a risk factor for a chronic disease, may, in turn, be influenced by the family environment. In families that possess risky characteristics, exacerbation of a genetic risk may lead to a more rapid and debilitating development of a problem. In families that lack these pernicious characteristics, the genetic expression may not occur or may be delayed. For example, growing up with parents who provide consistent and effective guidance about the regulation of emotional states may counteract the impact that heightened physiological and emotional reactivity would otherwise have on peer relationships and social competence. As noted above, the fact that the same risky family characteristics appear to fuel such a diverse array of adverse physical and mental health outcomes suggests that

their effects may be to exacerbate preexisting risks, as well as (or instead of) creating risks that would not otherwise exist.

In certain respects, the family environment contribution to risky profiles may be underestimated because these same family characteristics are also associated with a broad array of adverse educational and social outcomes that themselves represent risk factors for lower SES and poor health outcomes in adulthood. For example, child maltreatment, nonsupportive parenting practices, and poor parental monitoring also predict poor performance in school (Ary, Duncan, Biglan, et al., 1999; Cutrona, Cole, Colangelo, Assouline, & Russell, 1994; Eckenrode, Rowe, Laird, & Brathwaite, 1995; Pettit, Bates, & Dodge, 1997; Steinberg, Lamborn, Dornbusch, & Darling, 1992). Moreover, school failure is itself associated with other health risks. In a large nationally representative sample, academic problems in youth were associated with cigarette smoking, alcohol use, and involvement in weapon related violence (Blum, Beuhring, & Rinehart, 2000). Some evidence suggests that poor academic competence mediates the association between unsupportive parenting and substance abuse (Wills & Cleary, 1996). In addition, our review has focused primarily on significant clinical outcomes, both mental and physical, in making the case for pernicious family environments. Yet, subclinical manifestations of the problems associated with adverse early family characteristics are also noteworthy (Felitti et al., 1998; Walker et al., 1999). Low social competence and difficulties with the control and expression of emotions, for example, are significant dysfunctions and liabilities in their own right (see, e.g., Goleman, 1996; Mayer, Salovey, Caruso, & Sitarenios, 2001). Thus, regardless of whether the problems created or exacerbated by risky family characteristics lead to diagnosable forms of psychopathology or specific chronic diseases, they can cause significant disruption in adolescent and adult life.

Limitations

There are several limitations of the current analysis that merit consideration. First, our review has focused on a wide range of mental health and physical health disorders, and each of these disorders may have a unique risk profile and trajectory. On the basis of the evidence presented in this article, we believe that the adverse family characteristics identified will be distinctively present in those diverse trajectories. But exactly how they figure into each of the disorders in question must be identified by empirical investigation.

Second, we have focused our analysis only on the adverse characteristics of conflict, deficient nurturing, and neglect. Other family characteristics, such as sexual abuse, divorce, and parental psychopathology, may also be implicated in health and mental health disorders. For example, sexual abuse in childhood has been related to adult mental health problems, such as antisocial behavior (Pakiz, Reinherz, & Giaconia, 1997); to health risk behaviors, such as smoking, alcohol and drug abuse, and risky sexual behavior; to adult medical problems, such as breast disease, gastrointestinal disorders, pelvic inflammatory disease, and bladder infections (see Golding, 1999; Leserman, Toomey, & Drossman, 1995, for reviews); and, among men, to increased prevalence of HIV infection (Springs & Friedrich, 1992; Zierler et al., 1991). We have not systematically addressed sexual abuse independent of physical abuse. Similarly, a substantial literature relates parental loss, sep-

aration, or divorce to adverse outcomes in offspring. For example, parental divorce has been associated with physical and mental health problems in childhood, with factors indicative of low social status in adulthood or less successful performance of life tasks, or both, as well as with premature mortality (Gottman, 1998; Tucker et al., 1997; Wadsworth, MacLean, Kuh, & Rodgers, 1990; Webster, Orbuch, & House, 1995). However, this literature is heterogeneous as to the reasons underlying the loss or separation, and so the impact of separation of child from parent in the etiology of risk profiles is difficult to calculate.

Parental psychopathology has been linked both to risky family characteristics and to poor health outcomes in offspring. For example, depressed mothers have been found to be more critical, negative, and irritable, and less responsive to their children's needs (Downey & Coyne, 1990; Nolen-Hoeksema, Wolfson, Mumme, & Guskin, 1995), and children of depressed mothers are at increased risk for depression and suicidal behavior, as well as a variety of other psychological problems (Kaslow et al., 1994). We elected to omit coverage of the research literature on parental psychopathology because the elevated risks observed in offspring are due to a poorly understood combination of genetic factors and parenting practices. With few exceptions, it is impossible to use these studies to isolate the specific effects of parenting behaviors that are associated with conflict, deficient nurturing, or neglect on child development. Nonetheless, parental psychopathology is an undeniably important input to risky family environments.

Because of space limitations, we have been unable to cover the extensive literature on resilience in children and the factors that protect against the adverse effects of risky environments (Haggerty, Sherrod, Garmezy, & Rutter, 1994). Nonetheless, our model is consistent with findings from this literature. Resilience is predicted by close and warm relationships with a parent or parent figure, high expectations and structure in the family, high SES, and individual characteristics that may be, at least in part, based on genetic factors, such as good intellectual functioning and an appealing, sociable disposition (Masten & Coatsworth, 1998). A supportive family environment may also contribute to the development of dispositional resources that successfully regulate emotional and behavioral functioning across the lifespan and represent additional routes that protect mental and physical health. Such resources include optimism, psychological control, and other personality characteristics (Aspinwall & Taylor, 1997).

Other gaps in our analysis include the fact that the magnitude of evidence for different points varies substantially. Some areas, such as the relation of physical abuse to serotonergic dysfunction and deficiencies in emotion understanding implicate specific family behaviors, whereas in other cases, the evidence is more conjectural. There has been little consideration of the role of gender in the patterns discussed. Studies have focused disproportionately on mothering (over fathering) and on outcomes in sons (over daughters). The literature hints that risky families are more likely to produce internalizing symptoms and depressive symptomatology in girls and externalizing symptoms and problem behavior in boys, but the evidence is not yet definitive (e.g., E. M. Cummings, Ballard, & El-Sheikh, 1991; Davis, Hops, Alpert, & Sheeber, 1998; Steinberg, 1987).

Implications for Future Research

What research agenda for the future does the risky families analysis suggest? A chief priority will be longitudinal investigations, beginning in early childhood, that chronicle how families influence the physical and mental health of offspring across the lifespan. Short of such ambitious long-term undertakings, investigations that focus on comorbidities of physical and mental health outcomes are needed. Past investigations have often conceived of mental health and behavioral variables, such as hostility, depression, and substance abuse, as potential moderators of health trajectories, such as course of CHD; the present analysis suggests, in contrast, that adverse mental health, behavioral, and physical health consequences of risky families may be comorbid outcomes of common underlying biological and psychosocial processes. A focus on these common processes has the potential to further elucidate the underlying dysregulations produced in risky families, as well as clarify how these dysregulations influence such a broad array of outcomes.

Accordingly, a third priority will be focused scientific investigations of the links among the sustaining factors we have identified, namely the biological pathways, emotion processing, and social competence. Although all three sets of factors have been linked to risky family characteristics as antecedents and to adverse physical and mental health outcomes as consequences, fewer empirical investigations have focused on the links among them. For example, responses to stress have biological, emotional, cognitive, and behavioral components; this calls for an integration of the research literature on emotion with the literatures on coping and biological responses to stress. Any effort in this direction must proceed from the assumption that each of the sustaining factors in our model receives multiple inputs from genes, environments, and the interactions between them, which result in different outcomes within each system. Therefore, simple, uniform mappings across the biological, emotional, and behavioral systems should not be expected. We suspect that individual differences in the interrelationships among the different sustaining factors help to explain why such a diverse set of physical and mental health outcomes have been linked to the same two core risky family characteristics. Nonetheless, it may be possible to identify constructs, such as hostility, that define a specific set of biopsychosocial processes that place some individuals at risk for particular health outcomes.

A fourth priority is made possible by the mapping of the genome and addresses our suspicion that a chief effect of risky families is to exacerbate certain genetic risks. As the polymorphisms of genetic risk factors are identified, it may become possible to see how parenting practices exacerbate such risk; existing animal models suggest the usefulness of such an approach (e.g., Higley et al., 1996a, 1996b). Studies are also needed that assess genetic risk in conjunction with parenting characteristics, as both human adoption studies and studies of non-human primates cross-fostered to females who provide different rearing experiences suggest that either genetic risk, poor parenting, or a combination of the two predispose offspring to certain of the problems typically associated with risky families (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995; Suomi, 1991).

An overarching conclusion of our analysis is that progress in understanding the effects of parenting on children's mental and physical health will not come unless we integrate psychosocial investigations of these processes with an understanding of the underlying biology. As part of this agenda, we recommend several foci: Research should identify potential critical or sensitive periods throughout childhood and adolescence during which particular biological dysregulations and dysfunctional psychological processes may be most likely to develop and have lasting effects. Unaddressed in this article is the critical prenatal period during which biological dysregulations may also develop, and these, too, merit exploration. Recent research suggests that the social environment of pregnant women is related to prenatal development; less social support for the mother predicts poorer fetal growth (P. J. Feldman, Dunkel-Schetter, Sandman, & Wadhwa, 2000). The adverse consequences of inadequate prenatal development may act as developmental vulnerabilities, particularly when the first few years of life are spent in a risky family. For example, a mother in the midst of the stress of poverty or in a nonsupportive or conflictual family environment may give birth to a low birth-weight baby, who may then be at greater risk for psychological problems and biological dysregulation in a risky family environment than a normal birth-weight baby would be. Parallel to this thrust should be efforts to see if there is biological or behavioral plasticity, or both, in the adverse effects of risky families, such that appropriate interventions might reverse or offset their deleterious effects. Such efforts will require experimental investigations using either animal models or comparative intervention studies with children and their families.

Implications for Interventions

The problems associated with risky families are serious ones. Chronic diseases, such as coronary heart disease, are among the chief causes of death in most developed countries, and the origins of behavioral risk factors associated with them appear to arise, in part, in risky families. Over the past 30 years, there has been a two to threefold increase in suicide and homicide rates among children, outcomes reliably tied to adverse family characteristics (Malinosky-Rummell & Hansen, 1993; Sedlak & Broadhurst, 1996; Wagner, 1997). Rates of depression, especially among women, are at high levels, and risky family environments are implicated here, too. Behavioral regulation skills and social competence are vital to managing situations that arise in high-stress environments, yet these skills are compromised by risky families.

Risky families are an important piece of the puzzle represented by these rampant social and public health problems. Moreover, they may be especially so with respect to intervention. Focusing on family characteristics that represent risk factors for (the exacerbation of) major physical and mental health disorders can provide the basis for early interventions that may at least partially offset the potential for cascading risk that may accumulate over the lifetime. Even if many of the self-regulatory problems and propensities for adverse health outcomes reviewed here are subsequently found to have genetic origins, families are the first environment within which these predispositions are expressed, and, as our review has shown, parenting appears to exacerbate or restrain the progression of these proclivities. For example, family interventions that succeed in reducing family conflict and anger and increasing cohesion and warmth have demonstrable beneficial effects for children who are aggressive or diagnosed with other externalizing problems (Barkley, Guevremont, Anastopoulos, & Fletcher, 1992; Kazdin,

1994; Sayger, Horne, Walker, Passmore, 1988; Serketich & Dumas, 1996).

Interventions that help parents learn the kinds of behaviors that may shape effective behavioral and self-regulatory skills in children, or that are specifically tailored to compensate for preexisting problems in children, are potentially valuable. A number of interventions have been developed to reduce violent behavior in families, including individual, group, and couples therapy, parent training, and home visits. In mild to moderate abuse situations, such programs have shown modest success in teaching parents better skills and reducing the likelihood of further abuse; in families marked by serious and chronic abuse, results have been less successful (Albee & Gullotta, 1997; McLoyd, 1998). Other examples include social service interventions to teach nurturant parenting behaviors, which have also shown some degree of success (e.g., increasing parents' emotionally supportive behavior, provision of intellectual stimulation, and time spent on parenting; Besherov & Laumann, 1996; Black, Dubowitz, Hutcheson, Berenson-Howard, & Starr, 1995). As the health outcomes of adverse family characteristics become better known, we may see more such family interventions. For example, efforts to modify temperamental cardiovascular risk factors, such as cynical hostility, are usually undertaken after an acute coronary event or other basis for diagnosing cardiovascular disease (e.g., English & Baker, 1983; Siegman, Dembroski, & Crump, 1992). If longitudinal evidence continues to suggest that these characteristics begin to develop in early childhood, attempts to offset their development may be pushed farther back into childhood.

Research, policy, and interventions must consider the social context within which parenting characteristics are expressed. Parents can learn to become more effective and nurturing, but if they remain in high-stress environments marked by grinding poverty, high crime rates, inadequate employment opportunities, and insufficient support systems, then the success of such efforts will be compromised (Zaslow & Eldred, 1998). The adverse effects of low SES on mental and physical health outcomes are as close to a universal truth as social science has offered. Without attention to the social contexts in which families develop, and in which risk and resilience are transmitted from generation to generation, scientists will remain the helpless chroniclers of the outcomes described in this article.

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