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A developmental psychopathology perspective on child and adolescent treatment policy

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Abstract
This article considers the implications of theory and research in developmental psychopathology for existing and emerging social policy concerning treatment of child and adolescent psychopathology. Based on the concept that all psychopathology is a process, four core principles of a developmental psychopathology perspective are discussed. In turn, each is applied to an evaluation of policy issues with implications for intervention, including mental health parity, treatment guidelines, mental health reimbursement, and level of intervention. It is concluded that social policy on treatment must be based on an empirically supported theory of developmental psychopathology and on evidence-based interventions that link treatments to pathogenic processes.

Over 15 years ago, Sroufe and Rutter (1984) defined developmental psychopathology as "the study of the origins and course of individual patterns of behavioral maladaptation" (p. 18). They distinguished the field from clinical child psychology and child psychiatry by noting that differential diagnosis and treatment techniques were of "secondary interest to developmental psychopathologists" (p. 18). Over time, however, research in developmental psychopathology has been identified as the starting point for the design of interventions (Kazdin, 1997; Shirk & Russell, 1996; Toth & Cicchetti, 1999), especially for preventive interventions (Cicchetti, 1993; Luthar & Suchman, 1999).

The benefits of grounding interventions in developmental psychopathology are evident in prevention programs such as the Prenatal and Infancy Home Visiting by Nurses program developed by Olds and his colleagues (Olds, Henderson, Chamberlin, & Tatelbaum, 1986). The program seeks to modify specific risks associated with negative life outcomes, including poor birth outcomes, child health and developmental problems (including injuries and child abuse), welfare dependence, and poor maternal life course in low-income, first-time parents (many of whom are unmarried and adolescent). The nurses have three major goals: (a) to improve the outcomes of pregnancy by helping women improve their health-related behaviors during pregnancy; (b) to improve the health and development of the child by helping parents provide more responsible and competent care of the infant; and (c) to improve families' economic self sufficiency by helping parents plan future pregnancies, complete their educations, and find work. The program has been tested in a series of randomized controlled trials conducted over the past 22 years in Elmira, New York (Olds, Eckenrode, Henderson, Kitzman, Powers, Cole, Sidora, Morris, Pettitt, & Luckey, 1997), Memphis, Tennessee (Kitzman, Olds, Henderson, Hanks, Cole, Tatelbaum, McConnochie, Sidora, Luckey, Shaver, Engelhardt,

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James, & Barnard, 1997), and Denver, Colorado (Korfmacher, O'Brien, Hiatt & Olds, 1998).

Findings from the home-visitation program indicated that while the program was in operation (through the child's second birthday), nurse-visited, high risk-women had fewer subsequent pregnancies, fewer health care encounters for child injuries, and fewer verified cases of child abuse and neglect than did their counterparts in the control group (Olds et al., 1986). Results from the 15-year follow-up of this sample demonstrate a reduction in child abuse and neglect, fewer subsequent pregnancies and births, months of welfare use, arrests, convictions, and behavioral problems due to substance abuse for nurse-visited, low-income, unmarried women in comparison to their counterparts in the control group (Olds et al., 1997). At the age of 15, children of poor, unmarried nurse-visited women had a lower incidence of arrests, convictions, and risk behaviors (e.g., sexual promiscuity and emergent substance use) than their counterparts in the control group (Olds, Henderson, Cole, Eckenrode, Kitzman, Luckey, Pettitt, Sidora, Morris, & Powers, 1998). Most of these findings have now been replicated with African Americans living in a major urban area through the first child's 5th year of life (Kitzman, Olds, Sidora, Henderson, Hanks, Cole, Luckey, Bondy, Cole, & Glazner, 2000).

The success of this program can be attributed, in large part, to a tight link between developmental epidemiology and intervention design. In brief, the design and delivery of the intervention is based on an understanding of specific modifiable risks and protective factors associated with developmental outcomes (Mrazek & Haggerty, 1993). This type of targeted approach to the selection of well-defined populations and specific behaviors and contexts that contribute to maladaptive life trajectories increases the likelihood that interventions will be effective. We hold that such principles should be extended to the development and design of child and adolescent treatments. Further, we propose that empirically supported principles derived from developmental psychopathology entail important implications for evaluating existing and emerging policy on the treatment of child and adolescent psychopathology.

Core Principles of a Developmental Psychopathology Perspective

Perhaps the most fundamental principle of a developmental psychopathology perspective is that "all pathology is, strictly speaking, a process" (Cicchetti, 1984, p. 2). This view can be contrasted with the classic *disease model* of psychopathology with its emphasis on diagnostic *entities* that are used to both describe and explain maladaptive behavior (Sroufe, 1997). For example, highly aggressive adolescents are assumed to *have* a disorder, specifically conduct disorder, and this, in turn, is offered as an explanation for their socially disruptive behavior. In contrast, when the focus shifts from diagnostic entities to developmental processes, children's maladaptive behavior is conceptualized as a system of interdependent transactions that occur over time.

The claim that all psychopathology is a process actually entails three distinct, but highly interrelated, conceptualizations of process, including dynamic systems processes, longitudinal processes, and transactional processes. When considered collectively, they provide the outlines for a developmental psychopathology perspective on intervention and social policy.

Dynamic systems processes

Many early approaches to intervention were based on simple models of dysfunction that isolated single pathogenic factors for a wide range of psychopathology (Shirk & Russell, 1996). Perhaps the clearest example comes from the play therapy literature where low self-regard was viewed as a pervasive pathogen for varied emotional and behavioral problems (cf. Axline, 1969). Such simplistic, "main-effects" models assumed a direct, linear relation between a pathogenic process and a maladaptive outcome. A developmental psychopathology approach begins with the assumption that most dysfunction results from the complex interplay of multiple psychological, social, and biological processes (Cic-

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chetti & Toth, 1998). For example, in the case of adolescent depression, genetic, affect regulation, representational, and familial processes contribute to varying degrees to the emergence of depressive symptoms. "These varied systems do not exist in isolation" but are interrelated as part of dynamic system (Cicchetti & Toth, 1998, p. 224).

A corollary of this view is that isolated risk factors or pathogens are unlikely to produce the same outcome across individuals (Kazdin & Kagan, 1997). A growing body of evidence on resilience indicates that not all individuals are diverted from adaptive developmental paths by exposure to serious psychosocial risks (Masten, 1994; Masten, Best, & Garmezy, 1990). Such findings underscore the need to move beyond single variable models of dysfunction. Multifinality essentially means that a particular adverse event or risk factor may *not* lead to the same pathological outcome across individuals, in part because pathogens or risk factors typically do not operate in isolation.

Given the interaction of multiple factors in the development of psychopathology, it is possible that individuals who develop the same disorder do so for different reasons (Shirk, 1999a). Children who are phenotypically similar (i.e., who share similar symptoms) may show distinctive pathogenic profiles (Shirk & Russell, 1996). Among depressed youth, for example, some may be dysphoric because of unsupportive relationships, whereas others may evince similar symptoms because of a failure to attain important goals (Harter & Whitesell, 1996). The principle of equifinality, the idea that "the same end state can be reached from different initial conditions or through different processes" (Cicchetti & Rogosch, 1996, p. 597), complements the principle of multifinality. Consequently, a developmental psychopathology perspective directs attention to the interplay of multiple risk and protective factors in the development of psychopathology.

Longitudinal processes

A central concern of developmental psychopathology involves the origins and course of patterns of maladaptation. Psychopathology is conceptualized as the *cumulative* interplay of risk and protective factors *over time* (Cicchetti, Toth, Bush, & Gillespie, 1988). This perspective stands in sharp contrast to longitudinal models that primarily focus on the continuity or discontinuity of specific symptoms or disorders (Sroufe, 1997). Instead, the scope of developmental psychopathology encompasses the processes, events, or conditions that increase or decrease the probability that a child will remain on an adaptive developmental path. As such, developmental psychopathology is concerned with children's adaptations to normative developmental tasks, and with the conditions that facilitate or undermine adaptation over time.

Some years ago, Sameroff and Chandler (1975) identified continua of reproductive and caretaking risks that could undermine adaptive development. These hazards included biological (e.g., birth complications), psychological (e.g., parental rejection), and social risks (e.g., economic disadvantage). Children who encounter such hazards are at increased risk for becoming developmental casualties, in part because adaptive development depends so heavily on a facilitating environment.

Fortunately, longitudinal evidence indicates that adaptive outcomes are not foreclosed by early deviation (Sroufe, 1997). Later events or experiences can maintain or deflect a child from a maladaptive path established by exposure to early developmental risks or hazards. For example, failure to establish a secure attachment with caregivers has been shown to be an important risk for subsequent disruptive behavior problems (Hertsgaard, Gunnar, Erickson, & Nachimias, 1995; Shaw, Owens, Vondra, Keenan, & Winslow, 1996). However, engagement in subsequent supportive relationships may offset the impact of this early risk (Olds et al., 1997; Werner & Smith, 1982). From a developmental psychopathology perspective, deviance and resilience do not reside exclusively in the child but reflect the conditions that maintain or redirect individual developmental trajectories (Luthar, Cicchetti, & Becker, in press). Accordingly, continuity in maladjustment is not viewed as a property of the child, but reflects the character of subsequent events or experiences that maintain a child on a maladaptive

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pathway (Sroufe, 1997). Interventions and social policy, then, must target not only the individual child but also the conditions that maintain maladjustment.

Transactional processes

Although many recent models of dysfunction point to interactions among multiple pathogenic factors (e.g., genes and stressors in schizophrenia), a developmental psychopathology approach views "multiple transactions among environmental forces, caregiver characteristics, and child characteristics as dynamic, reciprocal contributions" that may increase or decrease the likelihood of psychopathology (Cicchetti & Toth, 1998, p. 226). Transactional processes imply that individuals are in constant exchange with their environment in a mutually influential way. For example, research on stress and depression has indicated that depressed individuals are both adversely affected by stress *and* contribute to their own stressful environments by generating stress through interpersonal conflicts (Hammen, 1992). As Sameroff and Chandler (1975) observed, "the child alters his environment and in turn is altered by the changed world he has created" (p. 234).

Moreover, the environment is viewed as being comprised of multiple, co-occurring levels, some more proximal and some more distal to the individual (Bronfenbrenner, 1979). Typically, research, intervention, and policy focus on proximal influences on maladaptive development such as family and peer interactions. However, a developmental psychopathology approach involves consideration of wider contexts that potentially impact such proximal processes. Peer and family interactions are embedded in the community and neighborhood context, which, in turn, are part of broader cultural system of beliefs and values.

Thus, a developmental psychopathology approach draws attention to contexts that support or obstruct development at levels both proximal and distal to the individual. As Sroufe (1997) has noted, this approach represents the antithesis of the classic disease model of psychiatric dysfunction with its emphasis on endogenous pathogens for particular disorders. Further, interventions or social policies that focus exclusively on the treatment of individual children are based on a decontextualized model of development or on what might be called the *myth of individual development* (Shirk, 1999b). From this perspective, intervention policy must consider multiple contexts that influence individual development.

Social Policy and Principles of Developmental Psychopathology

As an alternative to the classic disease model of mental disorders with its emphasis on diagnostic entities, a developmental psychopathology approach reorients the field to *processes* that contribute to adaptive or maladaptive development. This process perspective brings into focus a number of core principles with important implications for intervention and social policy. Although by no means exhaustive of the conceptual richness of developmental psychopathology, four principles, and their implications for treatment policy, will be considered. These principles are summarized as follows:

1. Most mental disorders result from the interplay of multiple psychological, social, and biological processes.
2. Most, if not all, mental disorders can be reached from different developmental pathways or through different pathogenic processes.
3. Exposure to early developmental hazards increases the probability of developing mental disorders, but continuity depends on subsequent events or conditions that maintain maladjustment.
4. Mental disorders are embedded in multiple contexts that are mutually influential.

It is noteworthy that some of the most successful *prevention* programs are guided by these principles. For example, the Prenatal and Infancy Home Visiting by Nurses prevention program (Olds, Henderson, Kitzman,

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Eckenrode, Cole, Tatelbaum, Robinson, Pettitt, O'Brien, & Hill, 1998), described earlier, is built on a model that (a) embraces multiple biological and psychosocial influences on adaptive outcomes, (b) recognizes that maladaptive outcomes can be reached through different processes (e.g., poor prenatal care or child abuse), (c) identifies specific developmental opportunities that can offset early risk, and (d) acknowledges multiple contexts that influence long-term adaptation (e.g., maternal employment and partnerships). It is our contention that the application of these principles should inform social policy on the *treatment* of child and adolescent disorders as well. Thus, against the backdrop of these four principles, existing and emerging policy on child and adolescent treatment will be considered.

Development or Disease: Implications for Mental Health Parity

Embedded in policy controversies regarding insurance coverage for mental disorders are a number of substantial issues that could be informed by a developmental psychopathology perspective. In 1996 Congress passed the Domenici–Wellstone Mental Illness Parity Provision in an effort to equalize health insurance coverage for the treatment of physical and mental illness. In brief, if insurance plans provide any mental health benefits, then they are required to provide group health benefits with equal annual and lifetime limits for mental and physical illnesses (Oss, 1998). Note that under the law employers or health plans are not required to provide coverage for mental disorders. Instead, they must meet parity requirements only if they provide some form of mental health coverage.

Individual states are legally permitted to design specific parameters for delimiting mental health coverage (Birch, 1998). Several states, including Maryland, Minnesota, and Vermont, have comprehensive laws that include all forms of mental illness, emotional disturbance, and substance abuse problems. In contrast, a growing number of states limit parity law to specific *biological brain diseases* (Birch, 1998). In Colorado, for example, six disorders are covered under parity law, including schizophrenia, schizoaffective disorder, bipolar disorder, major depressive disorder, panic disorder, and obsessive–compulsive disorder. Many highly prevalent disorders are not included under parity provisions, such as generalized anxiety disorder, conduct disorder, and attention deficit hyperactivity disorder (ADHD), presumably because they fail to meet criteria as biological brain diseases. Thus, at the heart of mental health parity policy is a controversy over what constitutes a mental illness.

A review of emerging policy suggests that diagnostic entities with endogenous pathogens typically are included under mental health parity provisions. Such an approach appears to be consistent with the classic disease model of psychopathology insofar as specific mental disorders are reduced to biological illnesses. From a policy perspective, the boundary between mental and physical disorders is erased by linking mental illnesses with biological processes. In essence, parity is based on a conceptualization of mental disorders as physical illness.

Although this approach to parity policy appears to level the playing field for mental and physical disorders, it is not without problems. First, on the surface it is difficult to discern the criteria for what constitutes a biological brain disease, in part because a number of psychological disorders with core biological mechanisms (e.g., generalized anxiety disorder or developmental reading disorder) or genetic etiologies (e.g., attention deficit hyperactivity disorder) fail to meet criteria. ADHD is perhaps the best example in that it is biologically mediated by deficits in brain activity in the frontal cortex (Taylor, Sandberg, Thorley, & Giles, 1991; Sonuga–Barke & Taylor, 1992), highly heritable (Taylor, 1995), and of chronic course (Hechtman, Weis, & Perlman, 1984). Moreover, untreated ADHD engenders risk for the development of other forms of psychopathology such as dysthymic disorder and substance abuse through peer rejection, family conflict, and school failure (Barkley, 1998). Although there may be other developmental pathways to phenotypically similar behavior problems (cf. Jacobvitz & Sroufe, 1987), it is clear that the criterion of *biologi-*

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cal brain disease is not consistently applied in policy decisions about mental health parity.

At a deeper level of analysis, the reduction of mental disorders to brain diseases stands in sharp contrast to a developmental psychopathology perspective. This is not to say that biology does not play an important role in many disorders; rather, in keeping with one of the core principles of developmental psychopathology, virtually all disorders involve the contribution of biological factors *in conjunction with* psychological and social factors. In this respect, the bifurcation of mental disorders into those that represent brain diseases and those that do not appears quite arbitrary.

A continuum of disorders

A number of examples from the research literature highlight the interplay among biological, psychological, and social factors across various forms of psychopathology and reveal the shortcomings of the standard of *biological brain disease* as the primary inclusion criterion for defining mental illness. In keeping with a developmental psychopathology perspective (Sameroff & Chandler, 1975), disorders will be arrayed on an etiologic continuum from biological to environmental, but in each case the interplay of multiple pathogenic factors will be highlighted.

There is no doubt that biological processes play a central etiologic role in schizophrenia. Although there is controversy about the specific mechanisms by which genes produce the pronounced cognitive and social problems found in schizophrenia (Pogue–Geile, 1991), investigators have argued for a schizophrenia-specific genetic mechanism (Hanson, Gottesman, & Heston, 1990). However, concordance rates of approximately 50% for monozygotic twins suggests that other nonhereditary factors contribute to the development of schizophrenia (Gottesman, 1991). It appears that individuals inherit a genetic risk or predisposition for schizophrenia, but not the disease itself (Walker, Neuman, Baum, Davis, DiForio, & Bergman 1996). The emergence of the manifest disorder appears to depend on the combined effect of biological predisposition and psychosocial stress, or what is commonly referred to as a *diathesis–stress* interaction.

Furthermore, evidence indicates that patterns of family interaction are pivotal for predicting relapse in schizophrenia. Research on expressed emotion, a psychosocial construct involving expressed hostility, criticism, and overinvolvement, has shown that families high on expressed emotion have approximately twice the relapse rate for impaired relatives compared to families low on this construct (Butzlof & Hooley, 1998). Such findings point to the interplay of biological and social processes in the emergence and course of schizophrenia.

Near the middle of the etiologic continuum one might reasonably place major depressive disorder. Evidence for moderate levels of heritability (McGuffin & Katz, 1989), and discernible differences in brain function between depressives and controls (e.g., difficulties in hypothalamic–pituitary–adrenal [HPA] regulation), indicate that biological processes figure prominently in this disorder (Cicchetti & Toth, 1998). On the other hand, some investigators have found limited genetic but substantial shared environmental influence on severe depressive symptomatology (Rende, Plomin, Reiss, & Hetherington, 1993). Among proximal environmental influences are family process variables such as high levels of family conflict (Lewinsohn, Roberts, Seeley, Rohde, Gotlib, & Hops, 1994), low parental warmth mixed with high levels of control (Blatt & Homann, 1992), and high parental levels of criticism (Asarnow, Thompson, Hamilton, Goldstein, & Guthrie, 1994). Furthermore, limited socioeconomic resources have been linked to increased risk for depression (Garrison, Schlucter, Schoenbach, & Kaplan, 1989). Taken together these findings suggest that biological processes constitute only a subset of pathogenic mechanisms in depressive disorders.

Interestingly, conduct disorder, one of the most prevalent and costly disorders of childhood, often is not included among mental health problems considered for parity. In part, the exclusion of conduct disorder may reflect the perception that it is principally a *behavior* disorder of environmental origin with little or

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no biological involvement. In fact, an argument can be made that conduct disorder, like major depression, involves the interplay of multiple etiologic processes, including biological processes.

Although there is strong evidence that family processes, such as harsh or inconsistent discipline, low monitoring, and poor problem solving (Patterson, Forgach, Yoerder, & Stoolmiller, 1998), and peer processes, such as socialization by deviant peers (Dishion, Patterson, & Greisler (1994), contribute to the initiation and escalation of conduct problems, genetic differences have been shown to be one of the primary contributors to individual differences in antisocial behavior in early and middle adolescence (O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998). In particular, there appears to be a high level of heritability for aggressive behavior (Edelbrock, Rende, Plomin, & Thompson, 1995).

Moffitt (1993a) posited two pathways to adolescent antisocial behavior that differ in terms of etiology and developmental course. Life-course-persistent (LCP) antisocial behavior is marked by early onset, continuity, severity, and frequency of antisocial behavior across time and context. Individuals with LCP antisocial behavior are characterized by congenital deficits in neuropsychological abilities such as verbal and executive functions (e.g., cognitive flexibility, planning, novel problem solving, and response inhibition) and a difficult temperament (Moffitt, 1993b; Pennington & Bennetto, 1993; Seguin, Pihl, Harden, Trembley, & Boulerice, 1995; Speltz, DeKlyen, Calderon, Greenberg, & Fisher, 1999). In contrast, adolescence-limited (AL) antisocial behavior is characterized by lower severity and cross-contextual inconsistency, is limited to adolescence, and does not involve neuropsychological deficits (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996).

The role of biological factors in the development of conduct disorder has been deepened with the publication of a series of papers in the last decade linking conduct disorder and violent criminality to the children's exposure to mothers' cigarette smoking during pregnancy (Rantakallio, Laara, Isohanni, 1992; Wakschlag, Lahey, & Loeber 1997; Brennan, Grekin, & Sarnoff, 1999; Weissman, Warner, Wickramaratne, & Kandel, 1999). Most of these studies have controlled for many of the potentially confounding influences and have been corroborated by animal studies that demonstrate impaired neurological and behavioral development in animals exposed to carbon monoxide and nicotine (Olds, 1997), even at levels of nicotine exposure so small that they do not lead to fetal growth restriction (Navarro, Seidler, Schwartz, et al., 1989).

Olds (1997) has hypothesized that subtle neurological damage created by prenatal tobacco exposure is likely to be exacerbated by the child's exposure to dysregulated caregiving in the early years of life, leading to compromised behavioral adjustment. It is noteworthy, then, that at least one of the longitudinal studies of humans (Brennan et al., 1999) found a unique role for prenatal tobacco exposure in the development of antisocial behavior consistent with what Moffit (1993a) has referred to as the LCP pattern. Thus, an important form of conduct disorder appears to involve biological processes that may stem from genetic liabilities and/or environmental toxicity.

One might anchor the environmental end of the continuum with posttraumatic stress disorder (PTSD). By definition, the etiology of PTSD involves exposure to an extreme environmental stressor. On the face of it, the contribution of biology appears to be minimal. However, retrospective studies of brain functioning among trauma victims show abnomalities in hormone regulation, modulation of startle response, and neurophysiological arousal (Pollak, Cicchetti, & Klorman, 1998; Pynoos, Steinberg, Ornitz, Genjian, 1997). Although such research does not directly address whether these patterns represent a posttraumatic brain alteration or a genetic–biological risk factor, in either case they point to a role for brain function in disorders likely of environmental origin. It is noteworthy that longitudinal evidence reveals that prior exposure to trauma alters subsequent neurobiologic responses to extreme stress (Resnick, Yehuda, Pitman, & Foy, 1998), and that animal studies indicate that exposure to intense stress

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and the direct application of stress hormones can produce changes in brain structure (McEwen, 1994). These findings suggest that psychosocial toxins can leave their mark on brain functioning just as clearly as physical toxins.

As these examples indicate, the bifurcation of disorders on the basis of biological brain involvement is overly simplistic and is not commensurate with emerging evidence from developmental psychopathology. The standard of biological brain disease rests on the assumption of one-to-one correspondence between manifest disorder and underlying pathogen. As the foregoing examples show, disorders that have been included or excluded from the parity list involve the dynamic interplay of biological, psychological, and social factors. Even among disorders with well-established psychosocial etiologies, impairments or alterations in brain functioning are increasingly evident. As such, policy on mental health parity, especially policy decisions on the definition of "mental illness," must be informed by empirical evidence on the development and course of psychopathology. These policies have an important bearing on the types of problems for which treatment will be financially supported.

Implications for intervention

Of equal concern, classification of disorders in terms of biological brain diseases may carry implications for treatment prescriptions. That is, if one begins with the premise that mental disorders are biological diseases, then it may seem reasonable to conclude that appropriate interventions are biological as well. However, if one acknowledges that multiple pathogenic processes contribute to the onset and course of most disorders, then interventions are more likely to be conceptualized as integrated treatment packages that attend to biological, psychological, and social contributors to dysfunction. For example, in the case of schizophrenia there is little doubt that biological interventions are critical for improved functioning. However, outcomes are improved by family-based psychosocial interventions aimed at reducing hostility and overinvolvement (Falloon & Pederson, 1985). Similarly, a large majority of children with ADHD show substantial reductions in core symptoms when treated with stimulant medication; however, changes in collateral social deficits are offset by social skills and other psychosocial interventions (Hinshaw, 1999).

In summary, there is growing evidence that many, if not all, mental disorders involve biological processes. However, it does not necessarily follow that the most effective treatments will be pharmacological. To the degree that multiple pathogenic processes contribute to a disorder, treatments will need to be multifaceted and directed toward both the biological and psychosocial underpinnings of psychopathology. Increasing evidence from developmental psychopathology indicates that most, if not all, disorders involve the interplay of biological, psychological, and social processes. Such evidence should be used for the development and evaluation of integrated treatment approaches to child and adolescent psychopathology.

Patterns of Maladaptation: Implications for Treatment Guidelines

In recent years, a growing number of treatment guidelines have been advanced by various professional associations, government agencies, and managed care entities (Pelham, 1999). Such guidelines have the potential to significantly impact funding for the delivery of mental health services to children and adolescents. For example, some managed care organizations have set treatment guidelines that prescribe short-term therapy for virtually all childhood emotional and behavioral disorders. Similarly, recent policy changes concerning allocation of medicaid funds (e.g., capitation of funds for services) typically lead to new guidelines for the distribution of mental health services.

Interestingly, these policy decisions appear to have been made without strong evidence for the relative effectiveness of various interventions. In the cost effectiveness equation, it seems that the containment of *cost* has been weighed far more heavily than the *effectiveness* of treatment. Our aim is to rebalance this

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equation by considering treatment guidelines from a developmental psychopathology perspective. It is our contention that treatment guidelines should be based not only on evidence for treatment effectiveness but on principles and evidence drawn from developmental psychopathology.

Developmental trajectories and treatment models

Given the emphasis on cost containment in the health delivery system, perhaps it should not be surprising to find increasingly restrictive treatment guidelines for the delivery of mental health services. Within the field of child psychotherapy research, approaches to treatment have moved well beyond a "one-size-fits-all" models of intervention (Shirk & Russell, 1996; Shirk, 1999a). In fact, the dominant research paradigm involves the evaluation of specific treatments for specific disorders (Shirk, 1999b). The basic operating assumption is that different treatment approaches will be needed to address variations across types of emotional and behavioral disorders. Yet practitioners often encounter restrictive practice guidelines promulgated by managed care entities that run counter to this basic assumption. These guidelines may involve limits on treatment duration or intensity or prescribe a single approach to treatment across disorders (e.g., solution-oriented therapy). In essence, while the field has moved toward greater specificity of treatments (Shirk, 1999b), emerging policy appears to be reembracing the uniformity myth of psychotherapy (Kiesler, 1966).

In addition to emerging evidence from clinical trials (cf. Lonigan, Elbert, & Johnson, 1998), evidence from developmental psychopathology supports the need for multiple treatment models in clinical practice. Perhaps the most important evidence for this issue comes from research on the developmental course of different forms of psychopathology. Recent evidence indicates relative stability for both internalizing and externalizing problems (Achenbach, Howell, McConaughy, & Stanger, 1995; Verhulst & van der Ende, 1992). However, such indices of stability mask important differences in developmental patterns for different disorders. Among the most important are the chronic and the cyclical or remitting–relapsing patterns. The former refers to disorders that remain consistent or intensify over time, whereas the latter refer to problems that emerge, abate, and reemerge over time.

LCP antisocial behavior (i.e., conduct disorder) is typically a pervasive disorder of long duration (Moffitt, 1993a, 1993b). For example, Olweus (1979) has shown that the average correlation between early aggression and later aggression, one component of conduct problems, rivals the stability of intelligence over time. When covert antisocial behavior is considered, stability coefficients are even higher (Patterson, 1992).

The protracted nature of conduct disorder, and the fact that it can intensify with age (Loeber & Stouthamer–Loeber, 1998), suggest that low-intensity treatments are unlikely to produce enduring positive effects. As Kazdin (1997) has observed, conduct disorder may be likened to diabetes melitus. Because diabetes is a chronic illness, continuous treatment with insulin and diet usually is required. Similarly, conduct disorder is likely to require some form of continuous treatment in order to ensure sustained effects. One model advocated by Kazdin (1997) involves an initial phase of high intensity treatment followed by maintenance treatment at a lower dose.

It is worth noting that even among youth presenting with conduct disorder there is substantial variation in developmental pattern. A number of investigators have found subtypes of conduct disorder based on different developmental trajectories (Loeber, 1988; Moffitt, 1993a; Patterson, Capaldi, & Bank, 1991). The fact that different individual, familial, and contextual factors are associated with different developmental trajectories (e.g., late vs. early starter patterns) indicates that a uniform treatment approach is unlikely to be equally beneficial across groups.

In contrast to chronic disorders, remitting–relapsing disorders, such as major depression, might be best served with a different treatment model. One possibility is what Kazdin (1997) calls the "dental model" (p. 124). Here

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systematic case monitoring follows the initial phase of treatment. That is, after recovery from the initial depressive episode, children are monitored regularly for signs of relapse. Consistent with a developmental psychopathology perspective, monitoring would involve both the assessment of depressive symptoms *and* psychosocial stressors (triggers) that could precipitate relapse. Treatment could be provided based on periodic checkup, much like dental care in the United States (Kazdin, 1997).

As these two examples suggest, models of treatment should be congruent with patterns of developmental psychopathology. Treatment guidelines that prescribe a uniform treatment model are inconsistent with evidence showing variations in developmental pattern across disorders. Effective treatment will require models of intervention that fit with different developmental trajectories and the characteristics associated with different developmental patterns.

Equifinality and prescriptive treatments

Consider, for example, three children who share a similar maladaptive outcome, in this case dysthymic disorder. All three present with depressed mood, low energy, and diminished interest in previously appealing activities. The first child comes from a stable but highly critical family. The second is rejected by peers and complains of loneliness. The third was removed from her family because of chronic neglect and has shuttled between foster homes for several years. Although each of these children present with the same diagnostic outcome, the pathways and the corresponding developmental processes that led to this outcome are strikingly different. Self-criticism, social isolation, and relationship disruption represent core processes, but not in equal measure for each child. In turn, the appropriate treatment for each child would focus on different pathogenic processes.

Unfortunately, many of the current approaches to treatment guidelines fail to differentiate manifest symptoms (phenotypes) from underlying processes (genotypes) that give rise to the symptoms. In fact, the development of treatment guidelines (e.g., APA Task Force) has been based on the assumption that "there are specific discernible and homogeneous diagnostic categories to which identified and validated treatments can be applied" (Kendall & Clarkin, 1992, p. 833). The problem with this approach is that homogeneity is defined exclusively in terms of phenotypic similarity (i.e., in terms of shared diagnoses based on similar manifest symptoms).

Equifinality creates problems for treatment guidelines that are exclusively driven by diagnosis. Grouping on the basis of phenotypic similarity, as found in the DSM, often masks heterogeneity at the genotypic level. That is, children who share the same diagnosis are not likely to be homogeneous in terms of underlying pathogenic processes (Kazdin & Kagan, 1994; Shirk & Russell, 1996). Thus, guidelines that prescribe a uniform treatment for a heterogenous group resurrect the uniformity myth of psychotherapy at the diagnostic level. It is highly unlikely that children with the same diagnosis but different core pathogenic processes will respond equally to the same treatment.

The implications for treatment guidelines are clear. Although a treatment may be shown to be efficacious for a disorder *in general*, it is necessary to ask the additional question of whether the treatment will be useful for a particular *individual*. In this context, one must consider the degree to which specific prescribed treatment procedures address the assessed pathogenic processes in a particular case. Such considerations imply that treatment guidelines must be reasonably flexible. Treatment packages for specific disorders should be "modularized" in a way that enables clinicians to select relevant sets of procedures for specific cases. Of course, this implies that treatment research must begin to address not just the efficacy of whole treatments but the impact of specific sets of procedures on specific pathogenic processes (Shirk & Russell, 1996). In turn, treatment guidelines should be based on empirically supported treatment procedures for particular pathogenic processes. Guidelines based on diagnosis alone are insuf-

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ficient to account for the diversity of pathogenic processes within diagnostic groups.

Disorder or Condition: Implications for Mental Health Reimbursement

In recent years, there has been considerable controversy about the definition and conceptual parameters of *mental disorder* (Richters & Cicchetti, 1993; Wakefield, 1992). Despite alternative conceptualizations, the most recent edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; APA, 1994) has retained the definition of "a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g., a painful symptom) or disability (i.e., an impairment in one or more important areas of functioning) or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom" (p. xxi). At the core of this definition is the assumption that mental disorders reside *within the individual*, and that the syndrome is "a manifestation of a behavioral, psychological, or biological dysfunction in the individual" (DSM-IV, pp. xxi–xxii).

In contrast to this view, a developmental psychopathology perspective, with its emphasis on developmental pathways and longitudinal processes that maintain children on adaptive or maladaptive paths, does not conceptualize "maladaptation or disturbance as something a child either has or does not have in the sense of a permanent condition" (Sroufe, 1997, p. 254). Neither resilience nor deviance resides in the child alone but is a function of ongoing exchanges between child and facilitating or debilitating environment. If one is to account for temporal stability in a disorder (a defining feature of a syndrome), then one must consider the longitudinal processes that maintain a child on a deviant pathway. In most cases, these processes do not reside exclusively *in the child*.

This conceptual distinction carries important implications for treatment policy. Increasingly, the DSM along with its assumptions about mental disorder has become a legitimizing system for third-party reimbursement (Richters & Cicchetti, 1993). As Richters and Cicchetti (1993, p. 23) observe, "the reimbursibility issue is particularly problematic because federal and private funding for mental health treatment and services often require that a condition qualify as a disorder with the DSM system to qualify for payment." Consequently, conditions that do not reside within the child but that could pose serious risk for subsequent distress or disability (e.g., maternal alcohol consumption during pregnancy) fall outside the parameters of the DSM, and beyond the scope of typical funding for mental health services.

Of equal importance, treatment of conditions that maintain or intensify clinical disorders often are not eligible for reimbursement given the DSM-based system. A common de facto policy is that individuals who are treated by practitioners must "have" a disorder themselves if the service is to be reimbursed. For example, serious family conflict typically will not be reimbursed if the condition is diagnosed as a "parent–child problem," a V code in the DSM, despite the fact that a persistent pattern of interaction causes distress for more than one party. Because the condition in not within an individual, but between individuals, it does not constitute a disorder. Reimbursement typically is restricted to treatment of a patient with a disorder, or to treatments that include the diagnosed patient in the session.

Such a policy is at odds with emerging evidence on efficacious psychosocial interventions for a variety of child and adolescent disorders. For example, both conduct disorder and ADHD behavioral interventions that target parenting practices have been shown to improve functioning and reduce symptoms (Barkley, 1998; McMahon & Wells, 1998). In essence, effective treatment is not directly delivered to the patient with the disorder. Although the child may show a persistent pattern of maladaptive behavior and "have" a DSM disorder, interventions that target the parenting context appear to be more beneficial than many psychological treatments (e.g., psychotherapy) that target the individual child. Yet DSM-based reimbursement policy

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will cover the latter but not the former unless the clinician can diagnose the parent with some additional disorder.

As treatments increasingly address multiple contexts that contribute to or maintain child and adolescent psychopathology, an alternative framework for delineating reimbursable services will need to be developed. We propose that the DSM-based system with its assumption that disorders reside within individuals confuses diagnostic entities with pathogenic processes. Treatments do not target diagnoses; instead they address the pathogenic processes that contribute to onset or to maintenance of a clinical problem (Shirk & Russell, 1996). Such conditions may reside within the individual or between the individual and the social environment.

As an alternative, we propose that reimbursement or funding for clinical services should be based on evidence that treatment procedures address pathogenic processes that have been shown to be implicated in the development or continuation of a clinical problem. This criterion involves two components: first, that the target of intervention has been demonstrated to be linked to the presenting problem; second, that the intervention has been shown to modify the target of intervention. The distinction between pathogenic processes that reside within the individual and those that do not appears to be arbitrary and an unnecessary consequence of a narrow definition of mental disorder. Treatments that effectively reduce symptoms or improve functioning should be funded whether they are directed to the patient with the "disorder" or to the conditions that contribute to it.

Individuals and Context: Implications for Level of Intervention

The fourth principle states that mental disorders are embedded in multiple contexts that are mutually influential. This principle raises questions about the appropriate *level* of intervention and social policy. If systems are interrelated and mutually influential, then neither targeting individuals nor targeting broad systems can be the sole approach. On the one hand, interventions and social policy geared only at individuals fail to attend to contextual influences on emotional and behavioral disorders. On the other hand, interventions and policies targeting macrolevels may not have a sufficient impact at the individual level. Furthermore, because contexts interact, an attempt to impact a problem at one level may create new difficulties or have unintended consequences at another level.

Advancing our understanding of contextualism, Boyce, Frank, Jensen, Kessler, Nelson, Steinberg, and The MacArthur Foundation Research Network on Psychotherapy and Development (1998) detail five theoretically derived propositions: (a) contexts are nested and multidimensional; (b) contexts broaden, differentiate, and deepen as development progresses, becoming more specific in their effects; (c) contexts and children are mutually determining; (d) a context's meaning to the child determines its effects on the child and arises from the context's ability to provide for fundamental needs; and (e) contexts should be selected for assessment in light of specific questions or outcomes. These principles apply directly to interventions and social policy. First, clinicians, researchers, and policymakers must examine interactions among contexts as well as consider how contexts are embedded in a broader social milieu. Second, it is important to examine changes in context over time and the effect of such changes on the course of normative development and psychopathology. Third, these propositions suggest that we need to explore how and under what conditions developmentally meaningful interactions take place in order to design effective interventions.

Numerous interventions and research programs examine and utilize information regarding the mutual influences of contexts and individuals. For example, Kellam, Ling, Merisca, Brown, and Ialongo (1998) found that a classroom intervention in first grade decreased aggression among more aggressive males by reducing overall classroom aggression. For first-grade boys, poverty level was associated with higher risk of aggression, disruption, and vulnerability to classroom level of aggression. The increased risk of aggression for more aggressive boys persisted

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through elementary school to the transition to middle school. Importantly, males and females attending schools in economically disadvantaged communities were at an increased risk of being highly aggressive in middle school regardless of level of first grade aggression. The intervention affected children differently depending on the socioeconomic status of their neighborhoods. These findings suggest that over the course of development, individual characteristics such as aggression are impacted by proximal contexts like the classroom and by more distal contexts like community socioeconomic status.

Another example illustrates the importance of carefully assessing contextual influences. Korbin, Coulton, Chard, Platt–Houston, and Su, (1998) examined the impact of neighborhood structural factors (i.e., impoverishment, childcare burden, instability, and geographic isolation) on child maltreatment reports. They found that impoverishment and child-care burden had less impact on maltreatment in African American neighborhoods than in European American ones. In African American neighborhoods, perceived quality of social connectedness mediated the effects of structural characteristics. That is, some African American neighborhoods with large numbers of poor, unemployed, and female-headed households were able to maintain their strength and solidarity. In European American communities, noneconomic indicators of community social organization were less predictive of maltreatment reports. These findings indicate that context effects (e.g., impoverishment) are not uniform but rather should be examined using comparative and multimethod analyses in lieu of aggregating data to better understand the dynamics between individuals and the contexts in which they are embedded.

A number of recent social policies and government initiatives have been designed with contextual influences and the interactions between individuals and their environments in mind. However, these policies and initiatives often fail to fully examine the role of context and use the propositions advanced by Boyce et al. (1998) as guidelines. One highly visible social policy initiative is the welfare-to-work program. This initiative ties welfare benefits to participation in programs that are designed to help people develop the requisite skills to provide financially for themselves and their families. Thus, the policy is an effort to impact the work context with the intention of making meaningful differences at the individual level. By improving employability and, thereby, the financial resources of families, policymakers hope to impact the lives of individuals living in poverty. At the individual level, policymakers expect to see improvement in parenting skills and child development, a reduction in child abuse and neglect, and a decreased dependence on government assistance (Aber, Brooks–Gunn, & Maynard, 1995).

As mentioned, policies aimed at more distal contexts (e.g., the domain of work) may not be sufficiently potent at the individual level to make meaningful differences. An evaluation of the Teenage Parent Welfare Demonstration, a program in which teenage mothers were randomly assigned to regular services or a group that involved mandatory school and work requirements and received support services, illustrates the shortcomings of the welfare-to-work program (Aber et al., 1995). The program increased teenagers' attendance at school and job-training programs and modestly increased employment rates of participants. As employment increased, welfare grants decreased. However, the overall economic well-being of families did not change. Further, at the individual level, subsequent childbearing did not decrease and the program did not affect parenting behaviors of teenagers or enhance the development of their children.

The shortcomings of welfare-to-work programs stem, in part, from a primary focus on the employment and educational contexts in which teenage parents operate with too little attention paid to individual difficulties and to other contexts, such as the environment in which they live. Even with additional support services in the enhanced program, competing needs in other areas may have outweighed the need to participate fully in order to get welfare benefits. For example, practical problems such as needing to care for younger siblings

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in addition to their own children may prevent teenagers from attending programs, classes, and their jobs. Furthermore, to the extent that a program targets employment and education, it is not likely to ameliorate individual and family problems sufficiently to reduce subsequent childbearing or enhance parenting behaviors and child development. The work of Burton and her colleagues (Burton, 1990; Merriwether–de Vries, Burton, & Eggeletion, 1996) in African American communities suggests that early childbearing is an adaptive developmental strategy in certain contexts. For example, it may be advantageous for adolescents to have children when their mothers (i.e., the infant's grandmothers) are still rearing their own children and are able to help care for the babies. As such, a goal of reducing subsequent pregnancies is unlikely to follow directly from work-related interventions.

The welfare-to-work program provides an example of a social policy that intended to influence individual development by targeting broader contexts in which individuals operate. While the program demonstrates some gains with respect to employment and education, few gains have been made at the individual level. Interventions and social policies targeting broad contexts may be too diluted by the time they reach the individual to have a meaningful impact. In the case of the welfare-to-work program, a number of difficulties at the individual level (e.g., substance abuse, domestic violence, and emotional and behavioral disorders) may interfere with the program's ability to substantially improve the lives of individuals living in poverty. Thus, aiming interventions and social policies at broad contextual factors with the hope of impacting individuals is unlikely to be sufficient.

In contrast to the welfare-to-work program, the Juvenile Justice and Delinquency Prevention Act of 1974 (JJDP) was aimed primarily at individuals and did not adequately consider the extent to which transactional processes across multiple contexts impact influence on psychopathology. The act intended to divert and deinstitutionalize juveniles from the traditional juvenile justice system. This act mandated that status offenders, juveniles who have "engaged in conduct that would not be criminal if committed by an adult but is legally not permissible for a youngster" (O'Neil, 1987, p. 140), were not subject to juvenile court jurisdiction, adult jails, or institutions for delinquents. Examples of status offenses include truancy, repeated running away from home, and repeated use of intoxicating beverages.

One of the major problems with this act is that it diverts juveniles from federal and state systems without mandating a specific system in which status offenses are handled. States in compliance with the act are eligible to receive funds from the Office of Juvenile Justice and Delinquency Prevention for diverting status offenders (Swanger, 1988). However, diversion is left to the particular state's discretion, as these cases are not processed in juvenile court and there is no other authority directly responsible for handling them. Thus, the JJDP, a social policy intended to distinguish individuals that committed status offenses, essentially left these individuals floating through various systems without specifying the manner in which these cases should be handled.

An unintended consequence of the JJDP is that in order to get services for young people who commit status offenses, parents or guardians are often forced to relinquish custody of their children to the state. Residential treatment facilities and other services for young people with serious emotional and behavioral difficulties are prohibitively expensive and not fully covered under private health insurance policies. In some states, the Mental Health Parity Act does not provide coverage for diagnoses like conduct disorder. Therefore, families that wish to obtain services for young people must sometimes make the difficult choice between staying together as a family or relinquishing custody to the state in order to receive health care benefits for appropriate treatment.

Developing appropriate social policies to serve young people committing status offenses requires an understanding of the needs of these young people and the factors that distinguish them from juvenile delinquents. Antisocial behavior, ranging from criminal activity and aggression to violating family and

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social rules, is disruptive to the individual and the various contexts in which an individual exists (Dishion, French, & Patterson, 1996). Noncompliance, avoidance, and difficulties with interpersonal relationships appear to be at the core of antisocial behaviors. As discussed previously, with respect to development course, age of onset appears to be most prognostic. Adverse environments exacerbate neuropsychological deficits found in LCP antisocial behavior (Moffitt, 1993a), leading to the development of an intractable and recidivistic antisocial behavior pattern. In contrast, problematic behaviors in the AL antisocial type appear more normative and limited to adolescence.

Taken together, this research clearly illustrates the importance of developmental psychopathology for informing appropriate interventions and, consequently, shaping policy. As an example, perhaps one of the distinctions between status offenders and juvenile delinquents can be made based on the two trajectories described by Moffitt (1993a). That is, status offenders fit the description of AL antisocial behavior, while juvenile delinquents appear to follow a more LCP antisocial behavior trajectory.

Research in developmental psychopathology also informs us about contextual influences on development of delinquency and crime. Sullivan's (1996) ethnographic studies of the development of delinquency and crime suggest that engagement in more serious delinquent behaviors that set individuals on a course of criminality depends upon contextual factors such as the cost of criminal penalties and the availability of legitimate jobs. In these studies, in contrast to the White youths, minority youths who did not live in neighborhoods with tight social control (e.g., the presence of adult males who disciplined youth), and who did not have access to networks through which they could obtain jobs became involved in serious criminal activities as a means of making money. However, within each group including the White youths, young people were able to identify peers that took "irrational" risks for highly uncertain rewards. Sullivan's contextual analysis suggests that even though the environment has a strong influence on the development of crime and delinquency, within each environment individual characteristics influence the propensity of certain people to engage in extremely high-risk behaviors or more severe criminal activity. Using both qualitative and quantitative methodologies, Sullivan (1998) provides a refined understanding of the role of context in influencing the development of delinquency.

The research on types of antisocial trajectories and on contextual influences on the development of crime and delinquency suggests that social policy and interventions aimed at individuals exhibiting such difficulties must take into account environmental influences on the development of these behaviors in addition to individual characteristics. For instance, growing up in a "socially toxic" environment (Garbarino, 1997) may lead to accurate negative perceptions of community that may be adaptive for survival. Negative perceptions of the community, or hostile attributions, have been associated with increased antisocial behaviors (Seidman, Yoshikawa, Roberts, Chesir–Teran, Allen, Friedman, & Aber, 1998). To be effective, social policies must reach beyond the individual, extending to the family context, enhancing neighborhood resources, and linking young people who have committed status offenses to systems in which they can obtain help.

More generally, given evidence that contexts interact to influence developmental outcomes, interventions must target individuals and the multiple contexts in which they develop. Aiming interventions and social policies at one level without considering the influences of multiple contexts is likely to limit their efficacy.

Conclusion

The 20th century was marked by the rapid growth of interventions for child and adolescent psychopathology. Many interventions emerged or persisted with very limited empirical support. As a new century opens, practitioners and policymakers are faced with difficult choices. Not all treatments are equally effective with children and adolescents (Shirk & Russell, 1996). Given the potential

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of such interventions to redirect youngsters onto adaptive developmental paths, the selection of treatments cannot be based on loyalty to theory, familiarity, cost containment, or political compromise. Instead, we propose that treatment policy should be based on an empirically supported theory of developmental psychopathology and evidence-based interventions that are conceptually linked to models of pathogenic process.

As mentioned at the start, some of the most successful prevention programs have been built from linkages between developmental psychopathology and clinical intervention. Treatment approaches, in contrast to preventive interventions, only recently have identified developmental psychopathology as the starting point for the development of interventions (Kazdin, 1997; Shirk & Russell, 1996; Toth & Cicchetti, 1999). It is our view that the design of therapeutic interventions, like their preventive counterparts, will benefit from close ties to research in developmental psychopathology. Treatments grounded in developmental psychopathology should then be evaluated in randomized clinical trials followed by evaluations under clinically representative conditions. Unless interventions shown to be efficacious in randomized trials can be reproduced with fidelity in clinics, it is not reasonable to expect comparable effects once disseminated (Olds, O'Brien, Racine, Glazner, & Kitzman, 1998). Public investment in such treatments, like their prevention counterparts, should be based on evidence for relicability, sustained effects, and estimates of economic impact (Olds et al., 1998).

We have identified a number of existing and emerging treatment policies that should be rectified with evidence from developmental psychopathology. Table 1 summarizes the social policy implications of the empirically based principles of developmental psychopathology discussed in this paper. These principles involve important implications for the design, delivery, and reimbursement of treatments for child and adolescent psychopathology.

First, movement away from simplistic "main-effects" models of dysfunction to models that recognize the complex interplay of multiple factors in psychological dysfunction undercuts efforts to classify mental disorders on the basis of a single criterion (e.g., biological vs. social etiology). In turn, the design of treatments *and their reimbursement* should not be based on such a restrictive and misguided criterion. Many so-called "biological disorders" involve psychosocial factors in the development, maintenance, and treatment response of such disorders. Conversely, many disorders believed to be principally behavioral involve some degree of genetic liability or neurological impairment.

Second, growing evidence for multiple pathways to specific disorders directs attention to the developmental course, trajectory, and underlying pathogenic processes involved in forms of dysfunction. All three carry important implications for the selection, timing, and delivery of treatments. Perhaps most importantly, the current effort to target treatments to specific diagnostic groups should be reevaluated in terms of the degree to which pathogenic heterogeneity characterizes a particular disorder. Failure to do so resurrects the uniformity myth of psychotherapy at the level of pathogenic process (Shirk, 1999a).

Third, deviance and resilience do not reside exclusively in the child. Although early developmental hazards might divert a child onto a maladaptive pathway, continuity in maladjustment depends on the impact of subsequent life events and experiences. One important implication of this perspective is that policymakers should invest in treatments for children who are at risk but who do not meet diagnostic criteria. In fact, such "subclinical" children should not have to be given a diagnostic label in order to receive funded treatment. Interventions that have the potential to offset early developmental risks should be reimbursable as part of a child's "well-care" program. However, interventions must focus on points and people in the child's development where there may be opportunities for change. Such interventions clearly must draw on research in developmental psychopathology that identifies critical turning points or life events that maintain or disrupt maladaptive trajectories.

Fourth, mental disorders do not occur in a vacuum. Social context can influence the ex-

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Table 1. *Treatment policy implications of a developmental psychopathology perspective*

Developmental Psychopathology Principle	Policy Implications
1. Most mental disorders result from the interplay of multiple psychological, social, and biological processes	Insurance reimbursement and clinical funding for mental disorders should be based on other factors in addition to the biological etiology of the disorder
	Even with biologically based disorders, interventions and best practices targeting mental health problems should be conceptualized as integrated treatment packages rather than focusing exclusively on biological treatments (i.e., medication)
2. Most, if not all, mental disorders can be reached from different developmental pathways or through different pathogenic processes	Choice of treatment modality and duration should be based on empirical evidence regarding the course of particular disorders and on efficacy studies of various treatments
	Developmental trajectories of mental disorders should be used to determine appropriate and effective times to intervene, thereby enhancing the effect of treatment
	Effective treatment should address underlying processes in addition to manifest symptoms; diagnosis alone cannot dictate which treatment should be provided
3. Exposure to early developmental hazards increases the probability of developing mental disorders, but continuity depends on subsequent events or conditions that maintain maladjustment	Reimbursement or funding for clinical services should be based on evidence that treatment procedures address pathogenic processes implicated in the development or continuation of a mental disorder
	Prevention efforts should target biological and psychosocial influences on adaptive outcomes, identify specific opportunities that offset early risk, and address multiple contexts that influence long-term adaptation
4. Mental disorders are embedded in multiple contexts that are mutually influential	Classification of mental disorders should include contextual factors that influence an individual's ability to function
	Reimbursement or clinical funding should cover treatments that target conditions posing serious risk for subsequent distress or disability rather than focusing exclusively on conditions that reside within individuals
	Understanding the influence of context on an individual's functioning is essential to designing effective treatments and social policy that target appropriate levels in the ecological system

pression and course of specific disorders as well as their response to treatment. For a variety of disorders, interventions that target individuals and social institutions in the identified patient's life can be more useful than treatments that focus on the child (cf. Henggeler, Melton, Brondino, Scherer, & Hanley, 1997). Current reimbursement policy penalizes creative interventions that target context insofar as funding is restricted to direct contact with the identified patient. For example, parents should not have to be diagnosed with a disorder in order to be reimbursed for parent management training of their hyperactive child. Similarly, treatments that focus on school consultation for disruptive or anxious children

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should be reimbursed even though there is no "face-to-face" contact with the identified patient. Linking reimbursement to direct treatment of the identified patient, and not including the modification of contributing context, represents an unjustified extension of the classic medical model into the domain of psychological intervention.

None of these policy recommendations should be construed as a carte blanche for treatments of child and adolescent psychopathology. Far too many treatments have been proposed, and marketed, with far too little evidence for their efficacy. Ultimately, funding or reimbursement of treatments should be based on empirical evidence that the treatment has demonstrated efficacy in clinical trials. This is a tall task, and by no means has the field reached this goal at the turn of the century. But it is a critical goal for a science-based approach to clinical intervention, and research in developmental psychopathology provides the starting point for the design of potentially effective interventions. As bridges are built among basic research in developmental psychopathology, clinical intervention, and treatment policy, troubled children and adolescents will be better served in the next century.

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