# Confronting Social Disparities in Child Health: A Critical Appraisal of Life-Course Science and Research

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**ABBREVIATION**
HDL-C—high-density lipoprotein cholesterol

The views presented in this article are those of the author, not the organizations with which he is affiliated.

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## abstract

The utility of the life-course framework to address disparities in child health is based on its ability to integrate the science of child development with the requirements of effective and just public policy. I argue that the life-course framework is best assessed in a historical context and through 4 essential observations. First, early genetic and environmental interactions are complex and influence outcomes in different settings in very different ways. Second, these early-life interactions are themselves subject to considerable later influences and, therefore, may not be highly predictive of later outcomes. Third, the etiologic nature or timing of early-life interactions does not, per se, determine if their life-course effects are amenable to later interventions. Fourth, a highly deterministic view of early-life interactions is not supported by the science and can generate counterproductive approaches to research and policy development. Finally, an alternative approach is proposed on the basis of a "human-capacity" model of the life course that connects the search for underlying basic mechanisms with a policy-based examination of the comparative effectiveness of influences at different developmental stages. This approach suggests an expanded research and policy agenda that might be more capable of generating urgently needed strategies for reducing disparities in child health. Such an approach could ultimately define more comprehensively the power and limits of life-course effects in shaping the social distribution of health outcomes in the real world. *Pediatrics* 2009;124:S203–S211

Our understanding of social disparities in child health has always involved the relationship between science and justice. Historically, the original calculations of disparate child health outcomes were closely tied to the emerging science of disease prevention in Victorian England and led to a variety of technical reforms in sanitation and hygienic infrastructure.[1] However, this early documentation of social disparities in child health was also used by others to advocate for broader social reforms and greater societal equity.[2] This dual genealogy, drawing on both the pursuit of science and the struggle for justice, has long generated conceptual and disciplinary tensions that in many ways continue to define, and at times torture, our current approach to disparate child health outcomes.[3] It should not be surprising, therefore, that the policy-based interpretation of life-course influences on disparate health outcomes has been similarly plagued by misunderstanding and, at times, oversimplification. The purpose of this discussion is to critically assess the remarkable new science of the life course and how best to ensure that it facilitates a constructive and informed policy response.

To meet this goal, this discussion addresses 5 related arenas of inquiry. To begin, the life- course framework is placed in a historical context because it represents only the most recent approach by which the science of child development is sculpted into a public perception of children and their social claims. Second, the scientific basis of life-course influences is assessed. Of special concern here are the policy implications of complex gene-environment interactions in shaping developmental processes. Building on this respect for complexity, the third section examines the evidence regarding not whether but the extent to which early life events shape later patterns of health outcomes. Perhaps most relevant to policy is the subsequent section, which assesses the relative amenability of early-life influences to effective intervention. The elements and findings of these sections suggest that the life-course framework, although potentially of great explanatory value, could, without some caution, generate policy impulses that can distract rather than compel constructive public action. Therefore, the final section presents a revised, policy-responsive framework that better ensures that the science of the life course can confront the persistent shame of disparate child health outcomes with the analytic force and political urgency that this issue demands.

## SCIENCE AND THE SOCIAL UTILITY OF CHILD DEVELOPMENT

In assessing the utility of the life-course framework, it is useful to first recognize that the policy interpretation of child-development science has always been influenced by societal needs and currents of popular sentiment. Indeed, the field of child development enjoys a proud history as a source of empirical evidence used to support major social reforms. In the late 19th and early 20th century, the fledgling science of child development helped shift public perceptions of children in a manner that embraced child well-being as a legitimate governmental concern. Before this period, child development was largely considered a private or familial affair, not rising to the level of societal concern, much less societal action. However, the new science of child development[4] helped legitimize protective child labor laws and the enactment of a variety of health and education initiatives. This experience signaled that developmental science had the capacity to influence the public's perception of childhood and that healthy child development was, for the first time, a state interest.[5]

Scientific support for the idea that early childhood events have a lifelong impact grew dramatically in the early to mid-20th century. The work of Gesell[6] and others emphasized the early role of biological determinants, particularly adverse fetal and perinatal events, in shaping later development and social functioning. Bowlby[7] and others countered with evidence suggesting that profound social exposures, such as isolation and the lack of a nurturing environment in early childhood, could result in a variety of adverse conditions. Although this work tended to exacerbate the traditional tensions between the biological and social explanations for child development, it also underscored the central importance of early life events in shaping later child and adult outcomes.

The antagonism between biological and environmental explanations for adverse developmental outcomes began to give way as more integrative models, such as those of Piaget,[8] became more widely accepted in the 1960s. Although both empirical and theoretical evidence supported the important role of early life experiences in shaping later developmental outcomes, new studies suggested that these outcomes varied considerably and were potentially responsive to later interventions. At the same time, the civil rights movement was elevating public awareness of racial inequalities and the fact that existing policies did not facilitate, much less guarantee, equal opportunity for achieving the requirements of a fulfilled life. Together, these scientific and political trends transformed observations on the development of young children into statements on social equality and life opportunity. In the new policy discourse, justice demanded a level playing field, and the science of child development

had, for the first time, cast this policy requirement as inherently developmental in nature. What resulted was a series of public initiatives, including the creation of the Head Start program in 1964, which, significantly, was housed not in a child-development division but in the newly formed Office of Economic Opportunity.

Recent scientific breakthroughs in genetics, neuroimaging, and human development have become a regular feature of television, the Internet, and popular culture. In response, the science of development has become far more accessible to a general audience and, in turn, has provided new, highly personalized ways to account for one's own set of capabilities, behaviors, and fears. Perceptions of development have increasingly become intertwined with self-perceptions and notions of identity and, consequently, have become less the province of societal improvement than of self-improvement. It is not surprising, therefore, that personal memoirs of traumatic childhoods have become perennial best sellers and that the relationship between early life events and later adult well-being might capture the public imagination.

This rich history leads to 2 general conclusions. First, the recognition that early life events shape outcomes later in life is not new. Second, the utility of the life-course framework cannot be assessed merely by listing new evidence that documents the basic mechanisms of how early life influences later health. The daily tragedy of disparate child health outcomes demands more. What is also needed is a deep appreciation of how the science will be interpreted in public discourse, how it can guide constructive action, and, ultimately, how it can speak to questions of equal opportunity and social justice.

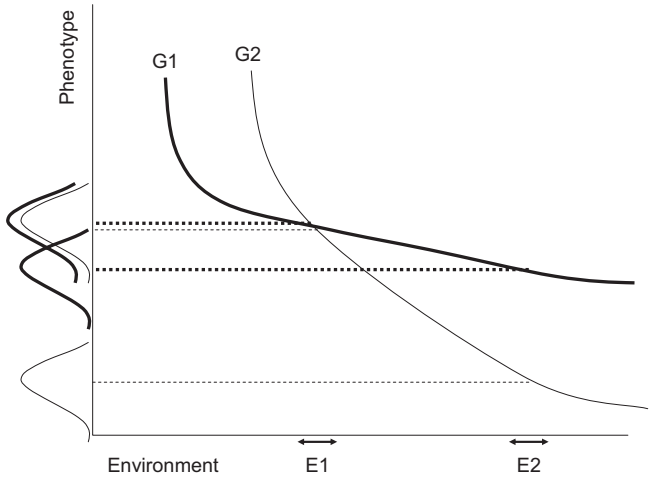


**FIGURE 1**

Norms of reaction of 2 genotypes, G1 and G2, over the same environmental range. The distributions of phenotypic expression are similar for both genotypes over the environmental range E1, but the distributions of phenotypic expression are distinctly different over the environmental range E2. (Reproduced with permission from van der Weele C. Figure 5.2. In: *Images of Development: Environmental Causes in Ontogeny.* Albany, NY: State University of New York Press; 1999:95.)

## EARLY EXPOSURES AND COMPLEXITY

Recent studies have suggested that physiologic or structural responses to early-life exposures may be adaptive in nature, altering development in ways that anticipate an environment that may exist later in life.[9] For example, there is considerable evidence that fetuses developing in a relatively poor nutritional environment, particularly those born small for gestational age, may exhibit metabolic adaptations, including insulin-related pathways, that affect the efficient use of available calories.[10] Such an adaptation would seem useful in later life if the early, calorie-poor environment persisted. However, in most instances, these infants once they are born will be exposed not to a calorie-poor but to a calorie-rich environment. This false anticipation of later exposures may prove maladaptive, potentially elevating the risk of obesity and related disorders. This erroneous anticipatory response is of particular policy concern, because it may relate to a variety of serious chronic conditions later in life. Of special relevance is the recognition that many of these anticipatory mechanisms involve intense gene-environment interactions, including epigenetic phenomena in which environmental exposures can alter the functional, phenotypic expression of a particular genetic profile.

### Complex Norms of Reaction

Although these scientific insights are providing a glimpse into what is sure to become vast new fields of inquiry, the implications of early gene-environment interactions for disparities in health, however, are likely to be exceedingly complex and demand considerable caution in interpreting the relevance of any single basic discovery in a policy context. A primary reason for this complexity is that gene-environment interactions are rarely uniform and can vary widely at different levels of exposure. These patterns of variation are generally known as "norms of reaction," which describe the continuum of phenotypic outcomes over different levels of exposure. Figure 1 depicts a gene-environment interaction in which the phenotypic expression of 2 different genotypes, G1

and G2, is equivalent under one range of exposure (E1) and markedly different under another (E2).

The evidence regarding norms of reaction for early-life exposures in humans suggests highly complex phenomena that defy simple interpretation in the policy arena. For example, a series of studies conducted by Caspi et al[11] revealed that severe maltreatment in childhood was more likely to be associated with later antisocial behavior if a genotype producing low levels of the neurotransmitter monoamine oxidase A (MAOA) was present. However, for children who did not experience maltreatment, the low MAOA genotype was associated with a reduced risk of antisocial behavior. This kind of reversal of effect (ie, a "crossover" norm of reaction) may be quite common and has been noted in such important cases as the influence of the neurotransmitter-related *5-HTT* gene in mediating the relationship between developmentally stressful life events and the probability of major depressive episodes.[12] Another striking example of crossover relationships can be seen in the role of different hepatic lipase genotypes in determining levels of high-density lipoprotein cholesterol (HDL-C) as dietary fat intake rises (see Fig 2). This high degree of phenotypic variation in the face of intense gene-environment interaction suggests that it is likely to be difficult to make sense of analyses that search for main or "average" effects of any particular genetic or epigenetic profile. Indeed, when strong crossover relationships exist, no main or average effects may even be discernable. Any given genetic or epigenetic profile may result in different outcomes depending on the nature or intensity of different environmental exposures.

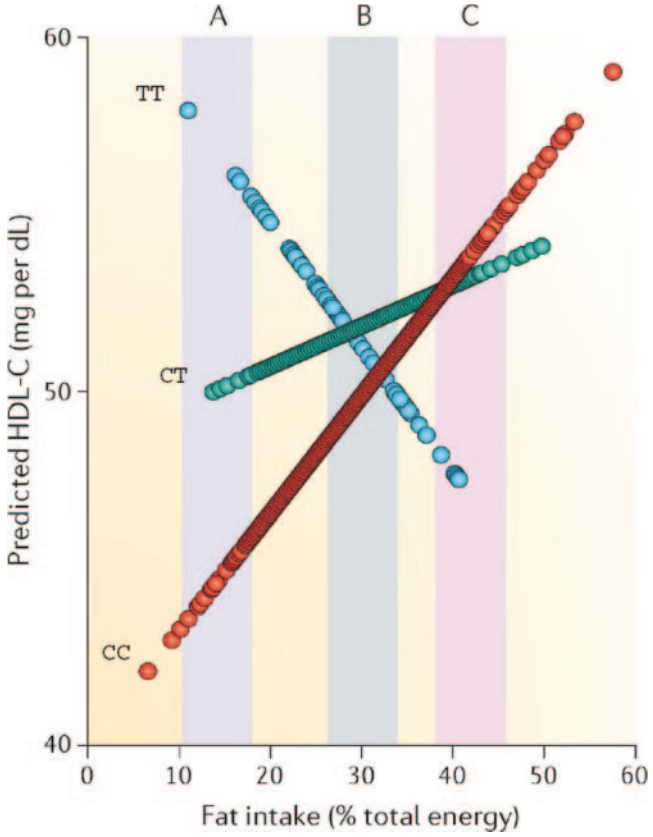


**FIGURE 2**
Crossover interactions between genotypes of hepatic lipase and dietary fat intake in producing HDL-C. At low levels of fat intake (band A), genotype TT produced the highest levels and genotype CC produced the lowest levels of HDL-C. However, at high levels of dietary fat intake (band C), genotype CC produced the highest and TT produced the lowest levels of HDL-C. At moderate levels of dietary fat intake (band B), the hepatic lipase genotypes do not appear to be related at all to HDL-C production. (Modified with permission from Macmillan Publishers Ltd: data are from Ordovas JM, Corella D, Demissie S, et. al. *Circulation.* 2002;106:2315–2321; as cited in Manolio TA, Bailey-Wilson JE, Collins FS. *Nat Rev Genet.* 2006;7(10):812–820.)

## Developmental Modulation

The nonuniform nature of many gene-environment interactions also suggests that the impact of a particular exposure may act differently at different developmental stages. Gene-environment interactions that occur at one developmental stage may lead to outcomes quite different from interactions that occur at another stage.[13] This potential for developmental modulation of gene-environment interactions not only underscores the complexity inherent in any interpretation of a singular developmental research finding but also lays the groundwork for the possibility that events that occur early in life may have special meaning, when developmental systems may be particularly sensitive or responsive. Complicating these relationships even more is the possibility that such sensitivity may act differently at the extremes of any norm of reaction than in the more prevalent middle. Although severe trauma or maltreatment in early life has been shown to tragically elevate the risk of later psychological and other health disorders, the impact of less severe trauma or maltreatment may be far less predictable. Depending on the precise exposure, the interaction with other exposures and capacities, and the possibility of different genetic susceptibilities, there may be functional nonlinearity or even thresholds of ultimate effect that operate during different developmental phases of life.

### Complexity of Stress

This complexity of effect may extend to a variety of exposures, including some, such as "stress," that have been attracting increased attention for their harmful role in determining long-term health outcomes. For example, thoughtful reviews have synthesized a growing literature suggesting an important role for stress-related influences, particularly those associated with racism, on such conditions as prematurity[14,15] and asthma.[16] However, some caution is needed when generalizing these stress-related findings to social settings in which deeply felt social injustices or serious emotional trauma are not involved. The biology of stress seems to be highly complex and may not lend itself to highly deterministic formulations of early-life exposures and later health outcomes. For example, much of what is generally accepted as appropriate child discipline implies generating some level of stress for the child. A simple punishment, a requirement to perform well in school, a parental insistence on "doing the right thing" in a difficult ethical situation, or even trying out for a varsity team, implies some level of enhanced stress. In other words, much of moral and social learning may involve enhanced levels of stress. Whether stress proves beneficial or harmful may reside less in its nature, per se, than in its severity, chronicity, and social context; the presence of compensatory or coping capabilities; and many other interactive factors. This suggests that a specific stressful exposure may be harmful in one context but beneficial in another. The public appreciation of this complex perception of stress is evident in recent recruiting strategies for the US Army. Without question, army basic training and, particularly, service in areas of major combat must be considered intensely stressful. However, unlike earlier recruiting efforts that highlighted vocational education, the current recruiting slogan, "There's strong, and then there's *Army* strong," implies that the army experience, indeed the potentially lethal conditions inherent in military service, will ultimately prove beneficial in dealing with challenges later in life.

There is no doubt that there is a societal obligation to prevent clearly harmful exposures during early life, many of which can have devastating, long-term consequences. No new scientific insight is required to support this contention. However, although the current literature provides initial glimpses into what has already become an exciting arena of research, it also underscores the importance of biological and social context in shaping early-life influences on later outcomes and the caution that should be exercised in portraying these complex interactions as simple associations in public discourse.

## INTERACTION, PLASTICITY, AND PREDICTION

A major element of many current life-course approaches is the concern that the impact of early-life exposures will persist over a lifetime. This perspective is, in turn, based on developmental modulation, the notion that the impact of certain exposures may be greater if they occur during periods of enhanced developmental vulnerability (eg, gestation or the first few months of life). The argument is that developmental processes early in life may generate windows of increased sensitivity to biological and environmental influences and that these effects could result in permanent physiologic or anatomic changes that lead to an elevated risk of long-term adverse outcomes. Often labeled "critical periods," these developmental stages of special vulnerability and lasting impact serve as important conceptual touchstones for life-course mechanisms and arguments about policy.[17]

To get a sense of just how important this perspective has become in life-course policy discussions, one must only examine the language it generates. The metaphor of "trajectories" is often invoked to convey the pivotal importance of early-life exposures on the future course of health risks and life outcomes. Although the visual image of trajectories can emphasize the inherent continuity of different developmental phases over a lifetime, it may also oversimplify the impact of early-life exposures and thereby undermine a more constructive understanding of these processes in a policy context. The concern is that both the language and imagery of trajectories serve to depict scientific studies documenting early influence as evidence for early determination of later outcomes. The language used to frame this transformation can be quite subtle and, ultimately, quite seductive. Influential interactions that occur during gestation are sometimes described with the highly deterministic language of "embedding" or "fetal programming." The visual metaphor is a trajectory diverging from the norm early in life and smoothly extending an elevated level of risk over the life course.

The primary problem with this highly deterministic perspective is that it does not accurately represent the science. Although there are a growing number of studies that have documented early-life influences on later health outcomes, very few of these influences seem to have any real predictive value. For example, Tremblay et al,[18] in a well-constructed study, reported that exposure to family dysfunction before the age of 6 months was associated with more than a doubling of the likelihood that the child would exhibit aggressive behaviors

later in early childhood. However, although it is a useful finding, what was not explicitly stated in the article was that 88% of all the infants exposed to this undesirable familial characteristic did not exhibit high levels of aggression during the later study period. The influence of early-life exposures is almost always depicted through deviations in relative risk, but the notion of a critical period and the visual imagery of trajectory are almost always understood in the policy world as reflecting attributable risk. Indeed, although relative risks may be significant, the predictive value of these risks is almost always strikingly low, as is their associated population-attributable risk.[19] Rather than rely on highly deterministic language and visual models, it seems far more useful to recognize the plasticity of developmental processes[20,21] and to seek policy guidance in more comprehensive perspectives, such as those provided by Rutter[22] and the landmark report *Neurons to Neighborhoods: The Science of Early Childhood Development*.[23]

## THE AMENABILITY TO INTERVENTION

It is a common misperception that genetic or epigenetic effects are somehow less amenable to ameliorative intervention than are the consequences of other etiologies. In general, the etiology of a disorder has little to do with whether it is amenable to intervention. Genetic diseases, such as phenylketonuria, are highly treatable; indeed, the health impact of this disorder can largely be eliminated with access to newborn screening and alteration of diet. Similarly, outcomes more generally related to environmental determinants, such as childhood trauma, are clearly amenable to both preventive and, for injured children, therapeutic interventions. Asthma is likely to have its roots in gene-environment interactions, some of which may occur early in life.[24] However, there are highly effective interventions (eg, the reduction of environmental allergens or the use of inhaled steroids) that, when accessible, can allow patients to lead a normal life. In fact, it is likely that virtually all health disorders are the product of some complex interaction between a variety of determinants, and one cannot make any judgment regarding the potential of developing an efficacious treatment or cure on the basis of the nature of these etiologic pathways. The issue for policy is the presence of an efficacious intervention, not the nature or timing of the etiologic pathways per se.[25]

Despite the use of such terms as "embedding," "programming," and "trajectories," the emerging science of the life course in no way suggests that the impact of early exposures is any less amenable to intervention than are the impacts of any other influences on human health. Meaney and Szyf[26] have shown that maternal nurturing patterns in rats can influence the behavior of their offspring through epigenetic changes. This work is rightly invoked as an important illustration of the potential role of gene-environment interactions in shaping later behaviors. What is not often noted, however, is that this same group has been able to reverse the epigenetic effects with the administration of a pharmaceutical agent, a finding that has generated the expectation that ameliorative drugs, a kind of manipulated environmental change, could be developed for humans.[27]

These observations are not intended to diminish the potential importance of early life events and interactions. Furthermore, they should not detract from the compelling obligation to address the underlying social conditions that undermine the well-being of childbearing women and their offspring. Rather, these observations suggest a developmental reality that is far more dynamic and dependent on the provision of efficacious services than is usually implied by life-course representations.

This recognition, in turn, provides a more constructive framework for applied research and effective intervention. This is because highly deterministic framings, in addition to their inaccurate portrayal of the scientific evidence, also tend to set in motion unhelpful and unnecessary antagonisms between disciplines and constituencies that should be working more closely together to reduce disparities in health over the life course. Simply put, suggestions that early-life influences are highly deterministic tend to elevate the interests of those who focus on early life while devaluing the contributions of those who do not. If we portray the science of the life course as inevitable health trajectories set in motion before the age of 3 years, what does this imply about the importance of virtually all exposures and interventions that occur after this period? A highly constrained advocacy for early-life effects has the potential to undermine the relevance of virtually all interventions implemented after early life and could, for example, transform adolescent medicine into a kind of palliative care. Such framings do not support a progressive social agenda and can often represent not a portrayal of a complex science but a confined tactical move to generate public attention or, less charitably, a public manifestation of a far less consequential struggle for disciplinary preeminence.

## CONSTRUCTING A HUMAN-CAPACITY MODEL FOR LIFE-COURSE RESEARCH AND POLICY

The dual recognition that life-course effects are both considerably plastic and potentially amenable to intervention provides the logical foundation for a far more integrative and, ultimately, a far more empowering vision of the

life-course framework. Although efforts to understand the biological underpinnings of long-term health consequences must, of course, continue, it is also important to realize that the policy relevance of this science is shaped by our potential capacity to influence whatever underlying mechanisms are at work.

For example, a critical period is generally considered to be defined by developmental processes, often neurobiological in nature, that permanently fix a physiologic or anatomic capability for a lifetime. However, if a drug were developed that could substantially and easily alter this capability later in life, would the developmental processes in question still be considered a critical period? Not from a policy perspective. What if a highly effective social intervention was developed? Would the original developmental period in question still be considered "critical" in nature? Again, not in a policy context.

The neuroanatomy or physiology may be fixed, but their impact on outcomes may not be. Moreover, compensatory skills or changes in the demands of society can diminish the functional impact of a particular developmental problem, thereby altering the relationship between the developmental period and outcomes. These examples illustrate that the logic underlying life-course concepts, such as the notion of the critical period, although informed by the neuroscience, is inherently defined by whether a later exposure or, here, a capacity exists to influence its functional impact on capability formation or outcomes. At a fundamental level, this shift in frame reflects the fact that a drug or intervention is merely a manipulated environment that, as with any other environmental exposure, is subject to the same interactions with genetic, epigenetic, or other processes that define a norm of reaction. More broadly, this policy perspective respects the importance of ensuring access to all available efficacious interventions. Here, the norm of reaction is linked to equitable provision and is seen as being subject to our human capacity for technical innovation and societal change.

The practical implication of this kind of "human-capacity" model for life-course approaches is that it elevates not one particular period of development or area of disciplinary expertise per se but, instead, reframes the analysis of life-course policies as the neutral assessment of the sensitivity of different developmental stages to specified interactions, interventions, or investments. The economist James J. Heckman and his colleagues[28–30] have pioneered this approach; their work attempts to assess the interaction between capabilities generated during one developmental stage with those generated during a subsequent stage. This "dynamic complementarity" recognizes that capabilities and risks defined during one developmental period may be enhanced or diminished by subsequent interactions. This complementarity between developmental periods may be seen as cumulative, but it is not necessarily gradual or linear as new exposures or interventions are introduced. Although the analysis of dynamic complementarity has primarily concerned economic issues, they nevertheless provide a pragmatic template for organizing insights from a variety of relevant disciplines.[29] This approach recognizes that interventions such as Head Start may be efficacious but, at the same time, emphasizes that its beneficial impact is greatest when high-quality elementary, middle, and high school education is also provided. This focus on complementarity recognizes the complexity of development and shifts the policy analysis of the life course into a kind of comparative effectiveness format.[31] The developmental stages that are found to be more effective than other stages in producing a certain capability can then be described as "sensitive" periods; when one stage alone is effective in producing a capability, it then becomes a "critical" period.[28] From this perspective, sensitive or critical periods, although rooted at some basic level in developmental biology, are, for policy purposes, actually defined by comparative effectiveness judgments. It may well be true that given our current social fabric and array of interventions, efforts to eliminate disparities in child outcomes are best directed at very young children.[30] It may also be useful at some level to document the physiologic mechanisms involved to underscore that the shamefully common, adverse early-life exposures in the United States should be the focus of urgent programmatic and policy response. However, the point here is that the call for a preferential focus on early life is a comparative effectiveness argument and is best supported by the empirical analysis of developmental complementarity. Documenting basic physiologic mechanisms or simple associations with later outcomes is not enough. A broader, more encompassing perspective is needed that emphasizes the dynamic nature of child development and respects the potential that opportunities to improve health and life chances may exist or be created during any developmental period. This approach, in turn, opens up a whole new arena for purposeful investigation, a research agenda that can use comparative effectiveness, econometric, and complex systems techniques to integrate the rapidly expanding basic science with the applied lessons of child life in the real world.

## CONCLUSIONS

From a policy perspective, the most useful way to make sense of the

emerging science of the life course is not to be preoccupied with whether etiologic phenomena are genetic, environmental, or epigenetic in nature or even whether developmental processes are confined to a particular period of life. Rather, the essential element in a life-course framework is the disciplined linkage of any and all of these processes to our human capacity to ensure that they are optimized for all children.

The basic conclusion of this discussion is that the life-course framework is at its most powerful when it respects both the complexity of the science and the requirements of effective social policy. Frameworks cannot map complex, empirical processes; rather, they serve as metaphors, language tools that can make empirical processes understandable and manageable for public deliberation and action. This process of translation depends on framing the science not only accurately but also constructively. In this respect, the life-course framework has the capacity to be extremely useful, linking long-insulated arenas of inquiry into a more integrated understanding of human development. However, like all metaphors, the life-course framework can easily and inadvertently be transformed from a tool into a weapon, acting as a fragmenting force by generating a splintered epidemiology and a series of counterproductive disciplinary tensions. Genetic, epigenetic, or other early life processes may convey important risks that extend over the life course, and one should expect that discoveries in these arenas will continue to be both illuminating and relevant to policy. However, the danger lies in elevating these early- life processes by devaluing the complex interactions and, therefore, the complex potential for intervention that define health outcomes over long periods of time. In this manner, the life-course framework can be construed, despite the best intentions, as being about limits rather than opportunities. As the history of child development and policy foretells, mapping the life-course influences on health and capability will likely extend far beyond child health alone, serving, now more than ever, as technical guidance for identifying and navigating the developmental precursors of social justice. To confine the power of human capacity to one developmental period will, in the end, only serve to diminish concomitant claims to social justice in all others. By recognizing that the struggle to bring equity to child health outcomes has always demanded a comprehensive vision and a coherent analytic voice, the life-course framework can define the critical research and inform the essential policies that, together, will best ensure that all our children enjoy a life course that is both optimal and just.

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