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Commentary

Framework as Metaphor: The Promise and Peril of MCH Life-Course Perspectives

Paul H. Wise, MD, MPH

Life-course analytic frameworks expressly link the determinants of health and illness across the lifespan. Such frameworks could serve as a foundation for integrating child and adult health policies by emphasizing the potential that social and biologic processes early in life can find clinical expression as adult-onset disease. However, there are elements of these frameworks that can be misinterpreted in ways that obscure scientific processes and fragment rather than integrate health policies. First, casting early life influences as determining rather than merely influencing adult health obscures the complexity of social and biologic etiologies over a lifetime and diminishes the impact of events in adolescence and adult life. Second, oversimplifying the impact of early influences on adult disease tends to imply that such processes are particularly unamenable to clinical and public health interventions, a suggestion without an empirical basis and likely to undermine pleas for enhanced access to such interventions. Third, exaggerating early life events as being highly deterministic of adult illness in order to shift societal resources from the elderly towards children can generate unnecessary antagonisms between potentially allied constituencies. Together, these considerations suggest that the utility of life-course frameworks will depend upon cautious interpretation and an ongoing process of active refinement.

KEY WORDS: life-course; health status; pregnancy; maternal and child health; policy.

Analytic frameworks are merely metaphors. Although technical in nature, they seek insight not through original experimentation but through reframing, through the redescription of causal relationships in ways that create new coherence out of the chaos old frameworks have left behind. This means that the central requirement of a good analytic framework is not so much knowledge as imagination. Like good poetry, good analytic frameworks seek clarity through the redescription of the traditional, through the construction of metaphors that can transform the commonplace, the routinely accepted, into moments of startling insight and deep emotion.

Recently in the *Maternal and Child Health Journal* (2003;7(1):13–30), Lu and Halfon presented a cogent articulation of a life-course framework, an analytic approach directed at the causal linkages of health and illness across the lifespan (1). Kotelchuck in the same issue underscored the many attributes and contributions such a framework could make to the maternal and child health community (2). This commentary was triggered by these two articles but in no way is constructed as a challenge or even as a critique of these important contributions to the field. Rather, the issues addressed here are concerned with the larger interpretation of life-course frameworks in the world of policy and, in many ways, their ultimate translation into the lexicon of justice.

Correspondence should be directed to Dr. Paul H. Wise, Department of Pediatrics, Boston Medical Center, 91 East Concord Street, MAT 4, Boston, Massachusetts 02118; e-mail: pwise@bu.edu.

EARLY LIFE EXPERIENCES AND THE FALLACY OF TRAJECTORIES

A basic proposition of life-course approaches is that experiences in utero or early in life can be

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expressed symptomatically later in life, both as childhood or adult disease (3). This concept has drawn upon a growing base of empirical evidence documenting the influence of early life exposures on the development of a variety of illnesses and adverse behaviors. There is evidence, for example, that in utero exposures can affect later patterns of growth, glucose metabolism, and reproductive capacity. Experiences early in childhood may affect later patterns of health-related behaviors, psychosocial pathology, and mental illness. Indeed, a growing variety of studies suggesting a link between early life experiences and later health outcomes have in many ways generated the need for a broad life-course framework (4).

Often the metaphor of "trajectories" is invoked to signal the impact of early events on the life-course. Although the visualization of a trajectory underscores the linkage of health across different ages and periods of development, it can also oversimplify this linkage by implying that the life-course path is determined by early events, like a cannonball being fired out of a cannon. If you know the power of the cannon and its angle of deflection off the horizon, you can visualize its trajectory, its apogee, and ultimately, its return to earth.

The problem here is not that a trajectory is not the proper metaphor for visualizing what many see in a life-course approach. The problem is that it probably is. Both the language and graphical depictions of life-course frameworks tend to transform data suggesting early *influence* into a theoretical embrace of early *determination* of later health. The language can be seductive, as in recasting in utero influence as embedding or "fetal programming." The graphical visualization of early effects on life trajectories appear graceful, with departures from the norm early in life leading invariably to graceful departures later in life. Although some advocates of life-course frameworks recognize the danger of deterministic interpretations, these cautions are, in reality, no match for the deterministic impulse of life-course rhetoric and visuals.

It is useful to examine an illustrative case. In a pioneering paper that remains oft-quoted, Klebenoff and colleagues documented an almost 2:1 elevated risk for delivering small for gestational age (SGA) newborns if the mothers themselves were born SGA (5). Because of the quality of the data set and the methodologic rigor of the authors, this study provides some of the best evidence of an early influence on subsequent reproductive health events. However, while the 2:1 elevation in risk appears well documented, more than three-quarters of the women born SGA would actually go on to deliver newborns of normal birth weight. Similarly, of all the SGA newborns in the study, approximately three-quarters were delivered to women who were themselves born at normal birth weights. Multivariate adjustment suggested that maternal birth weight probably had an even smaller influence on the birth weight of their offspring, and therefore, on patterns of birth outcomes in general populations. Although this study provided an important insight into potential etiologic mechanisms of SGA and potentially other adverse birth outcomes, it does not support frankly deterministic relationships between maternal birth weight and that of their offspring, and the authors made no such claims. However, this paper is highly representative of empirical analyses used to stabilize trajectory arguments. Most troublesome is the use of case-control studies with no sense of population prevalences to speculate upon the causes of population-based problems (6). What would the "trajectories" look like associated with maternal birth weight given that the vast majority of women born SGA had normal birth weight babies and that the vast majority of SGA babies had mothers who were born at normal birth weights? Fettuccine comes to mind. The concern here is not confined to the misinterpretation of one paper or this one reproductive issue, but rather that the translation of relative risk into trajectories has characterized the theoretical interpretation of a variety of empirical studies documenting early influences on later health events.

DEVELOPMENTAL MODULATION AND CRITICAL PERIODS

Life-course frameworks are strongest when they elevate the dynamic developmental cadences of childhood (3). The notion that biologic or social influences may have a different or more profound effect at certain developmental moments as compared to others is an important etiologic and policy-based insight. This developmental modulation of influence is framed by concerns regarding selected periods in which interference with normal processes can have an elevated risk of leading to long-term adverse outcomes. Often labeled "critical periods" these points in development are elevated as central potential modifiers of life-course pathways. Of particular concern are periods during gestation or early childhood when anatomic processes and functional interactions with the environment have been shown to influence cognitive and other physiologic capacities. Critical periods, therefore, are viewed, virtually by definition, as inherently deterministic. There is, of course, growing evidence

that certain critical developmental processes will be concentrated at different developmental periods and that this may confer differential vulnerability to influence at different times. However, the nature of this vulnerability or sensitivity is likely to be highly complex and it requires some caution when extrapolating the influence of severe, highly anomalous exposures on later outcomes to the mild or moderate experiences which are likely to characterize the vast majority of actual experiences in most settings. Protective and adaptive responses to these more common experiences may be expressed as nonlinearity and threshold effects on outcomes. Although life-course frameworks are extremely useful in focusing attention on the need for further research on critical periods, some caution seems warranted that sensitive periods not be deemed critical periods prematurely.

PUNCTUATED ALLOSTASIS

Life-course frameworks also provide a strong conceptual basis for the important contributions of cumulative effects on health. This basis draws upon a growing literature on the health impact of long-term, physiologic burdens accumulated in response to life's multiple stresses and demands. Often termed "allostatic load," this cumulative challenge to health has become an important element of lifecourse frameworks and is generally depicted as gradual, sustained accumulations of risk (7). However, there are virtually no data to suggest that these cumulative processes are in fact slow and gradual or necessarily sustained. Life is rarely gradual. Indeed, the life-course framework itself emphasizes the differential vulnerabilities of different developmental moments to adverse events or influences. The acute onset of asthma may influence later patterns of health greatly. An acute injury or family tragedy may similarly influence long-term health outcomes. Even the clinical product of cumulative processes, such as a heart attack, could acutely punctuate a life-course pattern of accumulated health burden. Good things also happen to people and this could theoretically reduce cumulative patterns of illness and risk. Isolated individuals can find new, supportive social networks. People stop smoking and lose weight. The point is that we really do not know the cadence of allostatic load. Influences may be cumulative as they exert their influence over time. However, they are not necessarily gradual or even sustained. Life-course accumulations of morbidity and risk are, in fact, likely to be highly punctuated, with graphical depictions of jumps, bumps, and plateaus rather than graceful, gradually rising curves.

AMENABILITY TO CLINICAL AND PUBLIC HEALTH INTERVENTIONS

With a strong focus on early determination and trajectories, life-course frameworks do not emphasize the amenability of accumulated risk to clinical or public health interventions. Early life influences, such as possible in utero effects, may biologically determine certain anatomic or physiologic mechanisms. However, this does not mean that they biologically determine what these anatomic or physiologic mechanisms convey to health outcomes.

Efficacious health interventions are defined by their capacity to alter what risk ultimately conveys to outcome. It makes no difference whether the risk is cumulative or acute, whether its etiology is biologic or social in origin, or even whether the mechanisms of risk have been elucidated. What is important is whether the intervention is efficacious. For example, there may be allostatic mechanisms that underlie the emergence of hypertension in some individuals. In others, there may be a strong genetic component. In still others, nongenetic but biologic influences related to fetal exposures may be operative. Of course, interactions between all of these mechanisms may also influence the expression of hypertension. However, highly efficacious interventions that control blood pressure do exist. Indeed, people with hypertension may reduce their blood pressure in a variety of ways such that their long-term prognosis approximates that of their normotensive counterparts. Similarly, there is evidence that attributes of the autonomic nervous system in young infants, potentially genetic in origin, could affect respiratory responses and ultimately the incidence of Sudden Infant Death Syndrome (SIDS). However, if children are placed to sleep on their backs, their risk of SIDS is reduced such that autonomic factors appear to be largely meaningless. When infants sleep in the prone position, biologic, or even genetic factors may be highly important to patterns of outcome. However, when they sleep on their backs, such factors may become relatively unimportant. Amenability to intervention is not based on biologic or social pathways of effect. Indeed, the "Back to Sleep Campaign," implemented in the United States in the early 1990s to encourage nonprone infant sleeping positions has had a dramatic impact on SIDS rates; this occurred without actually understanding the precise etiology of SIDS.

For the vast majority of children with mild to moderate asthma, a new series of highly efficacious medications has meant that these children need not experience any significant impact on their daily activities or well-being.

There is nothing to suggest that the impact of in utero exposures, even those characterized as "fetal programming" are inherently less amenable to intervention than is the impact of any other kind of exposure over the life-course. Barker's groundbreaking hypothesis that fetal exposures could influence a variety of adult-onset diseases similarly implies nothing about their relative susceptibility to clinical or public health interventions (8). Obesity, diabetes, cardiovascular disease are all amenable to intervention.

While the efficacy of these interventions is far from complete, the theoretical point is that they need not be complete to alter the link between early exposures and later health outcomes. Moreover, cumulative effects occurring over long periods of time are not inherently any less amenable to intervention then are recent conditions. Pointing out that women's health prior to pregnancy can influence birth outcomes (1) and that greater attention must be paid to women's health in general (9, 10) are always important contributions. However, this does not suggest that interventions during pregnancy will never be developed that cannot interrupt this conveyance of risk, risk that might have its origins in earlier health status far before pregnancy occurs.

THE LIFE-COURSE AND POLICY

The arguments above questioning the legitimacy of health trajectories, the scope of critical periods, the reality of graceful allostasis, and the depreciation of efficacious interventions, are all made in rebellion to the deterministic impulse of life-course frameworks. It should be noted once again that life-course frameworks do not need to be presented as deterministic to be interpreted as deterministic. Thoughtful presentations and qualifications regarding overly deterministic representations are helpful (1, 3) but they are likely to be insufficient without major revision. The reasons for the deterministic tendencies of life-course frameworks are both epistemological and disciplinary in nature.

The Threat of Complexity

It is useful to recognize that the greatest threat to any overarching analytic framework is complexity. An analytic model with scores of bidirectional arrows may reflect reality but is not likely to be very useful. Indeed, mapping the details of nature is precisely what analytic models are trying to avoid. The purpose of frameworks is coherence. Because our minds can only grapple with so much, coherence almost always demands simplicity. The simplification of a life-course approach to health—perhaps the most overarching analytic challenge there is—will almost always gesture towards a deterministic interpretation. Relative risks become trajectories, in utero processes become fetal programming, sensitive periods become critical periods, and health services are pretty much ignored altogether. A nuanced appreciation of the determinants of health over a lifetime can be both the objective and the enemy of life-course constructs.

The concern here is that life is too complicated, the human organism is too adaptive, society is too dynamic, and clinical and public health interventions too innovative, to plot with any precision life-course health trajectories on the basis of early life experiences. The reality is that health trajectories are retrospective in nature. To date, even the highly informative, prospective, longitudinal studies of health plot trajectories retrospectively, only once all the data are in (11). This characteristic makes these experiences of historical interest. Of course, history can teach and such experiences offer very real insights into current and future patterns of health. However, they are history nevertheless, and a deep respect for the interaction of biologic processes, social change, and clinical progress, should convey some humility to the prediction of long-term health outcomes on the basis of early life experiences. To plot prospective trajectories is somewhat like drawing a map of a place not yet explored.

Depicting the Life-Course and the Search for Solidarity

There are disciplinary reasons for the deterministic interpretation of life-course frameworks. Simply put, a focus on the importance of early life influences elevates the interests of those who work in this area. An embrace of fetal programming as a major cause of adult-onset disease would necessarily focus attention on improving fetal well being. Critical periods scream out for attention since their impact is, well, critical. It should not be surprising that the maternal and child health community might gravitate toward such framings.

The problem here is that the stated objective of life-course frameworks is to integrate our understanding of health and our policies over the lifespan (2, 3). Ironically, without revision, the depiction of early influences as highly deterministic elevates pregnancy and early childhood in ways that separate interests between generations not integrate them. As a pediatrician, I am highly sympathetic to any framing that might leverage more resources for pregnant women and children. However, I have also been highly concerned with the elevation of gestation and early childhood on the basis of deterministic arguments because in the policy world this almost always functionally devalues the claims of other age groups, particularly those of young adults, particularly women. If trajectories are deemed to be determined very early, this is not likely to convey a sense of high effectiveness to interventions and programs for adolescents. Similarly, framing fetal exposures as deterministic always puts a heavy burden on the behavioral autonomy of pregnant women (12). The contention that even mild intrauterine cocaine exposure would create life-long behavioral and cognitive damage led to a tragically counterproductive public rage against poor, drug using women (12, 13). This abated only when the evidence of longstanding and severe impact were questioned. In this manner, life-course frameworks could without revision, speak to the policy world in ways that inadvertently pit one generation against another and undermine one of the very real practical utilities of an integrated life-course approach.

The relative devaluation of clinical and public health interventions also must be addressed. It is easy to devalue such intervention in an effort to elevate the social causation of disease. While underscoring the fundamental nature of social causation is always useful, suggesting that health interventions are thereby irrelevant is both unnecessary and harmful. The result of disrespecting the efficacy of health interventions is always to undermine pleas for access to them. At a time when the political debate over access to health care is gaining new intensity, the devaluation of maternal and child health interventions is particularly destructive. Elevating social causation by depreciating the value of health services is not progressive, and should not be tolerated by an active maternal and child health community. Life-course strategies must strengthen their concern for the role of health services in shaping patterns of health. The data and the politics of the life-course demand such a revision. The requirements of collective progressive action demand such a revision.

Confronting the policy implications of a deterministic life-course framework is not a clash between science and politics, not an attempt to hold back the truth because of its implications for policy. It is a clash between the good interpretation of science and the bad interpretation of science. My concern for the political ramifications of the life-course approach as currently presented is not a product of my distrust of "*p*" values but of the translation of "*p*" values into societal values. Sen has provided important guidance in this respect by converting different currencies of social and health effects into functional, human capabilities (14). This framing respects the role of health in shaping the practical meaning of freedom and opportunity but yet also embraces the enormous plasticity of life. The life-course perspective's central focus on the impact of social engagement on lifelong health requires more than the documentation of psychosocial processes. It must also embrace analytically the exercise of power. It is impossible to talk about life-course patterns of health without talking about justice. We still have a lot to learn about such things and Sen, in many ways, could be our best teacher.

Lu and Halfon must be commended not only for their insights but also their courage. Analytic frameworks are always difficult to introduce into an applied field, particularly one with strong traditions. Kotelchuck must also be applauded for guiding their paper into publication and helping the reader to understand its importance. I, like the rest of the maternal and child health community, will continue to look to these authors for leadership in moving the life-course framework forward, reshaping its elements, and significantly, guiding more clearly its interpretation and representation.

Analytic frameworks are not about truth. Analytic frameworks are metaphors and metaphors are tools. Like language, frameworks are instrumental and have meaning only to the extent that they are useful. For the maternal and child health community, an inherently applied field, a life-course framework must do more than depict empirical relationships. It must depict empirical relationships in a way that moves us to constructive action. Such action requires building linkages between diverse constituencies. Such action has always required solidarity and solidarity is not discovered but created (15). The genius lies in imagining the language, the metaphors, the poetry, that can show us the way.

REFERENCES

1. Lu MC, Halfon N. Racial and thnic disparities in birth outcomes: A life-course perspective. *Mater Child Health J* 2003;7:13–30.
2. Kotelchuck M. Building on a life-course perspective in maternal and child health. *Mater Child Health J* 2003;7:5–11.
3. Halfon N, Hochstein M. Life Course health development:an integrated framework for developing health, policy, and research. *Milbank Quart* 2002;80:1–37.
4. Smith GD, Egger M. Understanding it all-health, metatheories, and mortality trends. *BMJ* 1996;313:1584–5.
5. Kebanaoff MA, Meirik O, Berendes HW. Second-generation consequences of small-for-dates birth. *Pediatrics* 1989;84:343–7.
6. Keller G, Zimmer G, Mall G, Ristz E, Amann K. Nephron number in patients with primary hypertension. *New Engl J Med* 2003;348:101–8.
7. Seeman TE, McEwen BS, Rowe JW, Singer BH. Allostatic load as a marker of cumulative biological risk. *Proc Natl Acad Sci* 2001;98:4770–5.
8. Barker DJP, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* 1986;8489:1077–81.
9. Chavkin W, Breitbart V, Wise PH. Finding common ground: the necessity of an integrated agenda for women's and children's health. *J Law Med Ethics* 1994;22:262–9.
10. Wise PH, Brett M. Prenatal care and women's health. In: McCormick M and Siegel J, editors. *Prenatal care*. Cambridge: Cambridge University Press, 2000.
11. Richards M, Hardy R, Kuh D, Wadsworth MEJ. Birth weight and cognitive function in the British 1946 Birth Cohort:longitundinal population-based study. *BMJ* 2001;322:199–203.
12. Chavkin W, Wise PH, Elman D. Policies towards pregnancy and addiction. Sticks without carrots. *Ann NY Acad Sci* 1998;846:335–40.
13. Zuckerman B, Frank DA. Mayes L. Cocaine-exposed infants and developmental outcomes: "crack kids" revisited. *JAMA* 2002; 287(15):1990–1.
14. Sen A. *Inequality reexamined*. Cambridge: Harvard University Press, 1992.
15. Rorty R. *Contingency, Irony, and Solidarity*. Cambridge: Cambridge University Press, 1989.