Building a Framework for Global Surveillance of the Public Health Implications of Adverse Childhood Experiences

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Background

In May 2009, the WHO and the National Center for Chronic Disease Prevention and Health Promotion (CDC, Atlanta GA) met in Geneva, Switzerland, to begin a collaborative effort to build a framework for public health surveillance that can be used to define the global health burden of adverse childhood experiences (ACEs). In addition to WHO and CDC staff, meeting participants included people working in the fields of public health and early child development from Canada, China, the former Yugoslav Republic of Macedonia, Philippines, Saudi Arabia, South Africa, Switzerland, and Thailand. Participants articulated their goal of forming a network aimed at advancing global understanding and measurement of ACEs through the exchange of information and the provision of technical expertise and support.[1]

Viewing child maltreatment and related experiences as a set of exposures that have broad implications for human development and prevention of public health problems is a relatively new concept. Only since 2004 has child sexual abuse been included in the global burden of disease estimates of death and disability attributable to particular avoidable risk factors,[2] and medical journals have recently begun to acknowledge the concept of abuse and related it as a major public health issue by raising awareness of the body of literature that supports this concept.[3,4]

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Why Adverse Childhood Experiences?

We use the term *adverse childhood experiences* (ACEs) as a way of moving toward understanding the public health implications of childhood maltreatment and related experiences. The experiences referred to herein include (but should not be conceptually limited to) abuse (emotional, physical, sexual); neglect (emotional, physical); and growing up in households where domestic violence is witnessed, members abuse alcohol or drugs or have mental illnesses, there is relational stress (such as separation or divorce), or members exhibit criminal behaviors. An important recommendation from the May 2009 expert consultation in Geneva was to expand these sets of experiences to include the ACEs that occur in both developing and developed nations. Specifically, to ensure that the contributions of criminality and organized violence to childhood adversity are adequately addressed, additional questions on forced marriage, witnessing criminal and collective violence in the community, and early conscription were added. Acknowledging that other children can be a notable source of adversity, exposure to bullying, other forms of peer-to-peer violence, and sibling physical and emotional violence were also added. Expansion of the sets of experiences not only adds new concepts and measures to consider but also requires thought on the relationships among multiple social dimensions that may be involved.

This choice of terminology and measures of childhood experience is largely based on the findings from the ACE Study, an ongoing collaboration between CDC and Kaiser Permanente (San Diego CA). The present study combines retrospective reports of ACEs at baseline and prospective follow-up of the study cohort to examine the prevalence and incidence of diseases, healthcare utilization, premature mortality, and causes of death.[5–8] Table 1 summarizes the breadth of the published findings.[9]

In 2008, the CDC developed questions similar to those used in the ACE Study for incorporation into the behavioral risk factor surveillance system (BRFSS). The BRFSS

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Table 1. Health and social problems and the ACE score

Problems from the baseline data	Outcomes associated with the ACE score
Prevalent diseases	Ischemic heart disease, cancer, chronic lung disease, skeletal fractures, sexually transmitted diseases, liver disease
Risk factors for common diseases/poor health	Smoking, alcohol abuse, promiscuity, obesity, illicit drug use, injection drug use, multiple somatic symptoms, poor self-rated health, high perceived risk of AIDS
Mental health	Depressive disorders, anxiety, hallucinations, panic reactions, sleep disturbances, memory disturbances, poor anger control
Sexual and reproductive health	Early age at first intercourse, sexual dissatisfaction, teen pregnancy, unintended pregnancy, teen paternity, fetal death
General health and social problems	High perceived stress, impaired job performance, relationship problems, marriage to an alcoholic, risk of perpetrating or being a victim of domestic violence, premature mortality in family members
Problems from the longitudinal follow-up of the study cohort	
Prescribed medications	Total prescriptions, prescribed multiple classes of drugs, psychotropics, bronchodilators
Diseases	Chronic obstructive pulmonary, autoimmune, lung cancer
Mortality	Premature mortality, lung cancer

Note: A complete bibliography of ACE Study publications listed by topic area is available online at www.cdc.gov/nccdphp/ace/.
ACE, adverse childhood experience

is the largest ongoing health survey in the world and has proved useful to define the prevalence of health behaviors and other determinants of health in the U.S.[10] Currently (in 2009), five state health departments are using these questions (www.cdc.gov/brfss/questionnaires/pdf-ques/2009brfss.pdf) in their BRFSS to determine the prevalence of ACEs and their relationship to major public health problems. The states, funded by the CDC to collect these new data, were chosen because of their sociodemographic diversity. Initial findings from ACE BRFSS data will be available in winter/spring of 2010. Additional states will be collecting ACE data in 2010. The BRFSS ACE data will serve as a frame of reference for constructing similar measures in other countries.

The ACE Concept and Primary Prevention

The emergence of ACEs as topic of research in public health is a natural evolution in the field of health promotion and disease prevention. This brief background provides a short historical and conceptual framework for understanding this evolution.

The seminal work of McGinnis and Foege, titled "Actual Causes of Death in the United States,"[11] quantified the contribution of alcohol, smoking, and other health risk behaviors to mortality in the U.S. In addition, the growth in popularity of the BRFSS[10] as a tool to monitor the prevalence of health-related behaviors reflects the momentum generated by studies documenting the influence of behavior on health. However, these important sources of health information do not tell us *why* the behaviors are present.

The U.S. Surgeon General's Report on Smoking and Health[12] and the attendant decline in the prevalence of smoking over the ensuing decades provide an example of both the success and shortcomings of health information and promotion campaigns. The success is obvious, but if information about the risk of smoking was sufficient, why do so many Americans continue to smoke?[13] And why is the prevalence of smoking so high in developed countries in Europe and Asia?[14] If the "addictive" properties of nicotine are the major determinant of continued smoking, why can some smokers quit whereas others cannot? A partial explanation for the latter questions can be found in the evidence linking depression—a common noncognitive factor in many people's lives—to smoking. People who are depressed are more likely to be smokers, and when followed prospectively, have been less likely to quit.[15,16] A convincing explanation for this finding is that the properties of nicotine that reduce anxiety and depressed affect lead to the use of cigarettes as a logical, but probably unconscious, adaptation to depression. Affect regulation is an important human capacity; apparently depressed people will use smoking as an adaptive response to facilitate this capacity— despite the negative well-known long-term health consequences.[17]

As this example illustrates, cognitive approaches to behavioral change are limited. In this paradigm, what is the *actual* cause of death for depressed smokers dying from emphysema? Is it smoking or depression? *Why* are they depressed? These questions represented the scientific gap in understanding the "root" origins of common health risk factors that the ACE Study addresses.[18]

In the case of child maltreatment, historically, most studies have focused on single types of childhood abuse, such as sexual or physical abuse, in relation to a limited number of outcomes. The ACE Study examines a broad range of early childhood traumatic stressors and their relationship to numerous clinical, public health, and social problems throughout the life span.[5–9] Measuring this wide array of adversities and their public health outcomes is suited to the broad public health missions of CDC and WHO.

The concept in using ACEs as a framework for the primary prevention of public health problems is that stressful or traumatic childhood experiences such as abuse, neglect, or forms of household dysfunction are a common pathway to social, emotional, and cognitive impairments that lead to increased risk of unhealthy behaviors, violence or revictimization, disease, disability, and premature mortality. Breakthroughs[3,19] in neurobiology show that ACEs disrupt neurodevelopment and have lasting effects on brain structure and function—the biologic pathways that likely explain the strength of the findings from the ACE Study. Importantly, research[20–23] on the perpetration of sexual violence and intimate partner violence in impoverished settings of low- and middle-income countries suggest that being a victim of child sexual and physical abuse, and witnessing intra-parental violence, are positively associated with both the perpetration of such violence by men and the likelihood of victimization in women.

This suggests that childhood adversities, though in all likelihood more frequent and intense in impoverished settings, make their own specific contribution to the developmental trajectory of individuals growing up in such environments. However, the likely bidirectional relationship between poverty and adverse childhood experiences remains poorly researched and represents a knowledge gap that the current international initiative is well placed to fill.

Moving Beyond "Measurement" Controversy and Limitations

A healthy controversy exists about the strengths and weaknesses of using retrospective self-reports of ACEs versus reports validated by child protection services for studying effects of exposure to childhood maltreatment.[24,25] A tendency to focus on the differences in findings between studies using one versus the other of these two methods has overshadowed the fact that both methods have shown substantial effects in multiple areas and are frequently concordant in terms of finding negative health effects of maltreatment.[4]

In addition, confusion surrounding terminology and assumptions about the strengths and weaknesses of "retrospective versus prospective" designs (in terms of how the ACEs are measured)[24,25] has further clouded the fact that studies in a wide variety of settings with varying designs and measures of ACEs have demonstrated negative health, behavioral, and social effects.[4] For example, the ACE Study measured experiences retrospectively, yet it has an ongoing longitudinal (prospective) follow-up component that has demonstrated increased risk for negative outcomes—including hospitalization for diseases (autoimmune, chronic obstructive pulmonary), prescription drug use prevalence, and premature mortality—that are not subject to concerns about temporality of exposure and outcome or to biases in reporting of outcomes.[6–9]

Maltreatment validated by child protection reports greatly underestimates exposure to these types of experiences, and self-reports or parent reports are probably closer to the true (unobserved and unreported) prevalence of maltreatment, although they might still be underestimates.[4] So, as a practical matter, self-reports of ACEs are suited to population-based sampling to estimate their prevalence and public health burden,[26] and they have been successfully used to examine relationships among lifetime exposures to violence, health-risk behaviors, and health outcomes in a large sample of African school children.[27]

Defining Exposure and Outcomes

A second conceptual hurdle in the assessment of the public health impact of ACEs is the tendency to view reported maltreatment "events" as the public health outcome. Although such events constitute a key target for preventive attention, only a small fraction have acute consequences of sufficient severity to bring them to the attention of public authorities. By far, the largest proportion of the burden of disease due to ACEs arises from the cumulative effect of chronic exposure to multiple adversities whose lifelong consequences may often start to become apparent only many years after exposure. Understanding these broader implications of ACEs is necessary to document their lifetime consequences and to highlight the public health importance of investing in primary prevention programs aimed at reducing them. A public health approach should thus include the short-term consequences of ACEs (death, injury, emotional, social) while also assessing the wide array of emotional, behavioral, social, and health problems that occur as longer-term (up to decades later) consequences.[28]

A Cumulative Stressor Approach

The ACE Study has shown that stressors such as abuse, neglect, witnessing domestic violence, and other forms of household dysfunction are common and frequently co-occur during childhood.[29] Review of the literature on the public health importance of child abuse emphasizes this pattern.[4]

The co-occurring nature of ACEs led to the use of an "ACE score," which is an integer count of the number of categories of ACEs. The ACE score has repeatedly shown a positive graded relationship to a wide variety of health and social problems (Table 1).[9] These findings suggest that studies of the relationship of single types of ACEs to public health outcomes are likely to overestimate the contribution of these single exposures to outcomes, miss the broader context in which they occur, and underestimate the public health impact of a wider array of ACEs.

Biologic Plausibility

Use of the ACE Score as a measure of the cumulative exposure to traumatic stress during childhood is consistent with recent understanding of the effects of traumatic stress on neurodevelopment.[19,30] Neuroscientists, using experimental animal models as well as case–control studies with humans, have linked childhood maltreatment to long-term changes in brain structure and function involving several interconnected brain regions.[31–36] Early stress is also associated with lasting alterations in stress-responsive neurobiological systems, and these lasting effects on the developing brain would be expected to affect numerous human functions into adulthood, including emotional regulation, somatic signal processing, substance abuse, sexuality, memory, arousal, and aggression.[37–42] The ACE Score appears to capture cumulative exposure of the developing brain to the activated stress response, which is likely the primary pathway by which ACEs exert their broad public health impact.

Genetics, Epigenetics, and Childhood Adversity

Inclusion of genetic and biological evidence is necessary for understanding the effects of ACEs and their intergenerational transmission.[43] A growing body of epidemiologic evidence suggests that genotypes can modify sensitivity to environmental adversity. Promising avenues of research in this arena include gene–experience interaction,[44] the influence of early life experience on genomic expression (epigenetics),[45] and the role of inflammation.[46]

The complex interplay of gene–environment interactions has been reviewed by an earlier study.[47] It concluded that the effects of genes and environments are not as separate as was once supposed and epigenetic effects, through influences on gene expression, can moderate the effects of genes in important ways. Another study[44] found that effects of life stress on depression were moderated by a polymorphism in the 5-HTT gene. It was also reported[48] that stressful life events may interact with a serotonin transmitter polymorphism to modify the likelihood of experiencing depression as a response to such life events. Elegant studies[45,47] demonstrate that the epigenetic effects of the rearing behavior of maternal rats on the subsequent behavior of their pups operates via DNA methylation of a promoter gene related to a glucocorticoid receptor in the hippocampus of the pups.

The inclusion of emerging genetic, epigenetic, and biological evidence will provide insights into the intergenerational transmission of ACEs and enhance understanding of the pathways by which they lead to negative health and social outcomes. Studies of gene–adversity interaction and epigenetic mechanisms by which life experience can modify behavior and physiologic responses to early-life stressors[46] are promising. However, these mechanisms are complex.[47] Care is needed to avoid oversimplifying this nascent research to conclude that the interaction of adversity with the genome is simple and direct and that "X is a gene for Y."[49] Nonetheless, the growing body of research revealing the relationships among genetic polymorphisms, genetic expression, and adversity are likely to lead to more effective methods to prevent and treat the effects of ACEs.[50]

Conclusion

Sufficient amounts of data exist to show that ACEs are common and are associated with many public health problems. Although the bulk of these data are from studies conducted in developed countries, a growing body of research from developing countries suggests that the same relationships exist in these settings. Findings from the Global School–Based Student Health Survey for a pooled sample of 22,256 respondents from five African countries demonstrate significant dose–response relationships between adversities such as forced sex and bullying and risk behaviors such as smoking, alcohol abuse, unsafe sex, and attempted suicide.[51] Similarly, findings from community-based surveys in mainland China, Hong Kong SAR, Taiwan, Singapore, and Malaysia also show that adolescents in these cultures experience a substantial health burden from exposure to various adversities.[52]

Building a framework for global surveillance of the prevalence and broad public health impact of ACEs requires moving beyond the dream of finding the "perfect" measures of these common exposures to implementing

methods that are epidemiologically credible and feasible within the U.S. and the often resource-poor environments of more global interest.

The practice of public health surveillance of ACEs and their wide array of health and social consequences is just beginning at WHO and CDC. These practices will evolve rapidly as the U.S. state-level data from the BRFSS are reviewed and critiqued and as public health practitioners in the U.S. and countries involved in the WHO/CDC collaborative effort define and track the global burden of exposure to ACEs and their public health sequelae.

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the CDC or the authors' affiliated institutions.

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References

1. WHO. Addressing adverse childhood experiences to improve public health. Expert consultation, 4–5 May 2009 [Meeting report]. Geneva, Switzerland: WHO, 2009.
2. Andrews G, Corry J, Slade T, Issakidis C, Swanston H. Child sexual abuse. In: Ezzati M, Lopez AD, Rodgers A, Murray C, eds. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. Vol. 2. Geneva, Switzerland: WHO, 2004.
3. Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities. Building a new framework for health promotion and disease prevention. JAMA 2009;301(21):2252–9.
4. Gilbert R, Spatz-Widom C, Browne K, Fergusson D, Webb E, Janson S. Burden and consequences of child maltreatment in high-income countries. Lancet 2009;373(9657):68–81.
5. Felitti VJ, Anda RF, Nordenberg D, et al. The relationship of adult health status to childhood abuse and household dysfunction. Am J Prev Med 1998;14:245–58.
6. Anda RF, Brown DW, Felitti VJ, Bremner JD, Dube SR, Giles WH. Adverse childhood experiences and prescribed psychotropic medications in adults. Am J Prev Med 2007;32(5):389–94.
7. Anda RF, Brown DW, Dube SR, Bremner JD, Felitti VJ, Giles WH. Adverse childhood experiences and chronic obstructive pulmonary disease in adults. Am J Prev Med 2008;34(5):396–403.
8. Brown DW, Anda RF, Tiemeier H, et al. Adverse childhood experiences and the risk of premature mortality. Am J Prev Med 2009; 37(5):389–96.
9. CDC. Adverse Childhood Experiences study. www.cdc.gov/nccdphp/ace.
10. CDC. Behavioral risk factor surveillance system. www.cdc.gov/brfss.
11. McGinnis JM, Foege WH. Actual causes of death in the U.S. JAMA 1993;270:2207–12.
12. U.S. Department of Health, Education, and Welfare. Smoking and health: report of the Advisory Committee to the Surgeon General of the Public Health Service. Washington DC: U.S. Department of Health, Education, and Welfare, Public Health Service, CDC, 1964. PHS Publication No.: 1103.
13. CDC. State-specific prevalence and trends in adult cigarette smoking—U.S., 1998–2007. MMWR Morb Mortal Wkly Rep 2009;58(9):221–6.
14. WHO. WHO report on the global tobacco epidemic, 2008: the MPOWER package. Geneva, Switzerland: WHO, 2008.
15. Anda RF, Williamson DF, Escobedo LG, Mast EE, Giovino GA, Remington PL. Depression and the dynamics of smoking. A national perspective. JAMA 1990;264:1541–5.
16. Glassman AH, Helzer JE, Covey LS, et al. Smoking, smoking cessation, and major depression. JAMA 1990;264:1546–9.
17. Pomerlau OF, Pomerlau CS. Neuroregulators and the reinforcement of smoking: towards a biobehavioral explanation. Neurosci Biobehav Rev 1984;8(4):503–13.
18. Anda RF, Brown DW. Root causes and organic budgeting: funding health from conception to the grave. Ped Health 2007;1(2):141–3.
19. Anda RF, Felitti VJ, Bremner JD, et al. The enduring effects of abuse and related adverse experiences in childhood: a convergence of evidence from neurobiology and epidemiology. Eur Arch Psychiatry Clin Neurosci 2006;256(3):174–86.
20. Chan KL. Sexual violence against women and children in Chinese societies. Trauma Violence Abuse 2009;10(1):69–85.
21. Jewkes R, Dunkle K, Koss MP, et al. Rape perpetration by young, rural South African men: prevalence, patterns and risk factors. Soc Sci Med 2006;63(11):2949–61.
22. Martin EK, Taft CT, Resick PA. A review of marital rape. Aggress Violent Behav 2007;12:329–47.
23. Söchting I, Fairbrother N, Koch WJ. 2004. Sexual assault of women: prevention efforts and risk factors. Violence Against Women 2004;10:73–93.
24. Widom CS, Raphael KG, DuMont KA. The case for prospective longitudinal studies in child maltreatment research: commentary on Dube, Williamson, Thompson, Felitti, and Anda. Child Abuse Negl 2004;28(7):715–22.
25. Kendall-Tackett K, Becker-Blease K. The importance of retrospective findings in child maltreatment research. Child Abuse Negl 2004; 28(7):723–27.
26. MacMillan HL, Fleming JE, Trocme N, et al. Prevalence of child physical and sexual abuse in the community: results from the Ontario Health Supplement. JAMA 1997;278(2):131–5.
27. Brown DW, Riley L, Butchart A, Meddings DR, Kann L, Harvey AP. Exposure to physical and sexual violence and adverse health behaviors in African children: results from the Global School-Based Student Health Survey. Bull WHO 2009;87(6):447–55.
28. Mercy JA, Saul J. Creating a healthier future through early interventions for children. JAMA 2009;301(21):2262–4.
29. Dong M, Anda RF, Felitti, VJ, et al. The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. Child Abuse Negl 2004;28(7):771–84.
30. Teicher MH, Andersen SL, Polcari A, Anderson CM, Navalta CP. Developmental neurobiology of childhood stress and trauma. Psychiatr Clin North Am 2002;25(2):397–426, vii–viii.
31. Heim C, Nemeroff CB. The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. Biol Psychiatry 2001;49(12):1023–39.
32. Repetti RL, Taylor SE, Seeman TE. Risky families: family social environments and the mental and physical health of offspring. Psychol Bull 2002;128(2):330–66.
33. Gutman D, Nemeroff CB. Neurobiology of early life stress: rodent studies. Semin Clin Neuropsychiatr 2002;7(2):89–95.
34. Gorman JM, Mathew S, Coplan J. Neurobiology of early life stress: nonhuman primate models. Clin Neuropsychiatr 2002;7(2):96–103.
35. De Bellis M, Thomas L. Biologic findings of post-traumatic stress disorder and child maltreatment. Curr Psychiatry Rep 2003;5(2): 108–17.

36. Bremner JD, Vermetten E. Stress and development: behavioral and biological consequences. Dev Psychopathol 2001;13(3):473–89.
37. McEwen BS, Angulo J, Cameron H, et al. Paradoxical effects of adrenal steroids on the brain: protection versus degeneration. Biol Psychiatry 1992;31(2):177–99.
38. Sapolsky RM. Why stress is bad for your brain. Science 1996; 273(5276):749–50.
39. Sapolsky RM, Uno H, Rebert CS, Finch CE. Hippocampal damage associated with prolonged glucocorticoid exposure in primates. J Neurosci 1990;10(9):2897–902.
40. Perry BD, Pollard R. Homeostasis, stress, trauma, and adaptation: a neurodevelopmental view of childhood trauma. Child Adolesc Psychiatr Clin N Am 1998;7(1):33–51,viii.
41. Perry BD. Neurobiological sequelae of childhood trauma: post-traumatic stress disorders in children. In: Murburg M, ed. Catecholamine function in post-traumatic stress disorder: emerging concepts. Washington DC: American Psychiatric Press, 1994:253–76.
42. FW Putnam. Developmental pathways in sexually abused girls. Presented at Psychological Trauma: Maturational Processes and Psychotherapeutic Interventions. Harvard Medical School, Boston MA. March 20, 1998.
43. DiLalla L, Gottesman II. Biological and genetic contributors to violence—Widom's untold tale. Psychol Bull 1991;109(1):125–9.
44. Caspi A, McCray J, Moffitt TE, et al. Role of genotype in the cycle of violence in maltreated children. Science 2002;297(5582):851–4.
45. Champagne FA, Curley JP. Epigenetic mechanisms mediating the long-term effects of maternal care on development. Neurosci Biobehav Rev 2009;33(4):593–600.
46. Danese A, Moffitt TE, Pariante CM, Ambler A, Poulton R, Caspi A. Elevated inflammation levels in depressed adults with a history of childhood maltreatment. Arch Gen Psychiatry 2008;65(4):409–15.
47. Rutter M, Moffit TE, Caspi A. Gene–environment interplay and psychopathology: multiple varieties but real effects. J Child Psychol Psychiatry 2006:47(3–4):226–61.
48. Kendler KS, Kuhn JW, Vittum J, Prescott CA, Riley B. The interaction of stressful life events and a serotonin transporter polymorphism in the prediction of episodes of major depression: a replication. Arch Gen Psychiatry 2005;62(5):529–35.
49. Kendler KS. "A gene for. . .": The nature of gene action in psychiatric disorders. Am J Psychiatry 2005;162:1243–52.
50. Rusk TN, Rusk N. Not by genes alone. Bull Menninger Clin 2007;71:1–21.
51. Dunne MP, Chen JQ, Wan Yuen Choo. The evolving evidence base for child protection in Chinese societies. Asia Pac J Public Health 2008;20(4):267–76.
52. Butchart A, Kahane T, Harvey AP, Mian M, Furniss T. Preventing child maltreatment: a guide to taking action and generating evidence. WHO and International Society for the Prevention of Child Abuse and Neglect, Geneva, Switzerland, 2006.