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Lead Toxicity in the 21st Century: Will We Still Be Treating It?

To the Editor.—

In the April 1992 issue of *Pediatrics*, Needleman and Jackson[1] claimed that asymptomatic blood lead levels—those ≥10 μg/dL—represent a serious health problem, affecting three to four million children in the United States, and referred to lead contamination in children as a "man-made epidemic" as well as "a blunter of children's cognition and silent thief of their futures."[1, p680] These authors recommended that all infants and young children be screened for blood lead, arguing that infants are screened routinely for conditions such as phenylketonuria which are much less common.

Needleman and Jackson neglected to mention that a successful program to eliminate lead from the environment has existed for >20 years, the result being an almost threefold decrease in mean blood lead levels of US children from 17 μg/dL to 6 μg/dL.[2] The epidemic the authors referred to apparently represents a self-fulfilling prophecy. Both authors were on the Centers for Disease Control Advisory Committee which in October 1991 lowered the blood lead level to be considered toxic from 25 μg/dL to 10 μg/dL,[3] thus—by a stroke of the pen—automatically increasing the number of asymptomatic "affected" children from about 300 000 nationwide to between three and four million and creating an "epidemic by edict."

The high prevalence of elevated blood lead levels described by Needleman and Jackson[1] is questionable. They stated that the free erythrocyte protoporphyrin test is not reliable at blood lead levels <25 μg/dL and that capillary blood lead tests are easily contaminated, yielding false-positive test results; however, we are not told whether these methods were used in determining the high-prevalence rates cited. One California study was a vivid example of how poor methodology and biased selection of subjects can lead to greatly exaggerated prevalence rates. In a 1987 to 1988 study of children living in a high-risk census tract in Oakland, the California Department of Health Services (CDHS) found that 67% of children tested had blood levels ≥10 μg/dL.[4] In an earlier reference to this study, submitted to the California legislature,[5] it was stated that the capillary method used by the CDHS was "extremely inaccurate even when precautions are taken"[5, p19] and resulted in a false-positive results rate of >50%; the recent MMWR report[4] did not include this qualifier—a serious omission. Subsequent studies using venous samples from San Francisco and Oakland children found that prevalence of levels ≥10 μg/dL was five to nine times lower (7.4% to 14.6%) in larger samples of children (6). Patricia Chase, MD, unpublished data, 1992; Raymond Davis, MD, unpublished data, 1992; (Children's Hospital, Oakland, California; 5461 Foothill Blvd; Oakland, CA), most of whom received public assistance program benefits and were at higher risk than the general population. In the Oakland inner-city private practice of Dr Raymond Davis, whose patients are mainly black children receiving welfare benefits, 136 children younger than 6 years of age were tested for lead by the venous-sample and graphite furnace method from February to May 1992; none of these children had lead levels ≥15 μg/dL, and 10 (7.4%) had levels ≥10 μg/dL. Similarly, in the state of Washington, Konig and Robertson[6] found that among 271 high-risk toddlers, only 6.3% had blood lead levels ≥10 μg/dL, indicating that data reported by the Environmental Defense Fund purporting a 40% prevalence at these levels were "severely flawed and grossly exaggerated."[6, p13] In contrast, the practitioner cited by Needleman and Jackson found that 22% of children had lead levels ≥15 μg/dL (the prevalence of values ≥15 μg/dL was < 3% in the above cited California studies), but the authors did not state how many patients were tested or what method was used. This information is needed before the study results can be accurately interpreted.

The authors state that, like blood lead contamination, metabolic disorders have a low prevalence and argue that because neonatal screening tests for metabolic disorders are nevertheless mandated in all states, pediatricians should support universal blood lead screening as well. In reply, however, lead screening does not meet the criteria used for mass neonatal testing. No simple and inexpensive yet accurate test for blood lead exists which is analogous to filter-paper blood testing of neonates. Mental retardation is severe and unequivocal in untreated neonatal disorders such as congenital hypothyroidism (CH) and phenylketonuria (PKU); the presumed psychocognitive defects caused by low blood lead levels, if any, are slight and are outweighed by confounding variables. Treatment of CH and PKU is safe and effective in preventing mental retardation; no convincing evidence exists to indicate that chelation treatment alleviates or reverses the claimed psychocognitive defects of low lead levels, and such therapy may cause major side effects.

Although the CDC report accepts that low blood lead levels (≥10 μg/dL) have clinically significant, detrimental, neurobehavioral affects, this contention is still debated. A detailed British analysis states that "it is not possible to conclude with any certainty that lead at low levels is affecting the performance or behavior of children."[7, p42] The data remain controversial and contradictory. Moreover, the validity and methodology of Dr Needleman's own work is being challenged.[8, 9]

Drs Needleman and Jackson propose that we spend billions of dollars on complex and controversial lead screening, treatment, and abatement programs. To do so in view of the many more serious, unaddressed health needs of US children is to misprioritize current health issues. Before major funding is allocated to universal lead screening programs, the issues of misleading information, flawed data, and biased statistical manipulation must be settled.

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TABLE 3. Women (15 to 44 Years of Age) with Acquired Immunodeficiency Syndrome (AIDS)* and Estimated Numbers of AIDS Orphans Born to Deceased Women, by Racial/Ethnic Group

	Racial/Ethnic Group			Total
	White	Black	Hispanic‡	
Women, N (%)	4094 (23)	9796 (55)	3868 (22)	17 758
Deaths				
N	2336	6040	2215	10 591
Median age, y	32	33	32	
AIDS orphans, N (estimated)§	2700	8400	3400	14 500

* Reported the Centers for Disease Control through December 31, 1991.
‡ 27.7% of Hispanic women are residents of Puerto Rico.
§ Calculated by applying the Current Population Survey cumulative age-specific fertility rates by racial/ethnic group to the number of women reported to Centers for Disease Control as deceased with AIDS by 5-year age intervals, then rounded to the nearest 100.

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Estimated Number of AIDS Orphans in the United States

To the Editor.—

The human immunodeficiency virus (HIV) epidemic in childbearing women impacts our society in a number of ways. One of these is the legacy of uninfected children left without their mothers when these women succumb to acquired immunodeficiency syndrome (AIDS). We have calculated a crude estimate of the current and future number of these AIDS orphans.

As of December 31, 1991, 21 225 women (≥13 years of age) with AIDS were reported to the Centers for Disease Control from the United States, Puerto Rico, and the Trusts and Territories; 17 910 (84%) were of reproductive age (15 to 44 years) at the time they were diagnosed with AIDS. Of these women, 55% were black, 23% were white, 22% were Hispanic, and <1% were of other race/ethnicity.

Because the published information on the impact of HIV infection on fertility is limited,[1,2] were made three simplifying assumptions in calculating our estimates of the numbers of AIDS orphans: (1) the fertility of HIV-infected women does not differ from that of the US population as a whole; (2) the fertility of women with AIDS who are residents of Puerto Rico is the same as Hispanic women who were surveyed by the Current Population Survey[3] of the fertility of American women in the 50 states and Washington, DC; (3) the period (1990) fertility rates will remain constant throughout the decade.

By December 31, 1991, 10 591 black, white, and Hispanic women with AIDS were reported to have died. To estimate the total number of children that were born to these women, we applied cumulative racial/ethnic- and age-specific fertility rates[3] to the number of women reported to have died (Table). (The age-specific fertility rate was adjusted downward to assume no fertility in the year the woman died with AIDS.) Of the estimated 19 300 children, we assumed that 25% are HIV-infected as a result of vertical transmission from mother to child.[4] Thus, there are approximately 14 500 uninfected children who will require long-term care by relatives, foster parents, and social service personnel. The remaining 25% (4800) of children are likely to be HIV-infected, and many may have developed AIDS already.

Using a similar method we estimated the future number of uninfected children who will be left without mothers (AIDS orphans). At present, the median incubation time from HIV infection to AIDS in adults is 10 years. The CDC estimates that there are currently at least 80 000 HIV-infected women of childbearing age.[6] We assumed that the distribution of these women by age and racial/ethnic group is the same as that for women reported with AIDS. Because the median age at death of women with AIDS is currently 33 years, we used age- and race/ethnic-specific cumulative fertility rates[3] by 5-year age groups through ages 30 to 34. We estimated that these women will leave approximately 125 000 to 150 000 children upon their deaths during the course of this decade. Thus, if the perinatal transmission rate remains approximately 25%, we estimate that 93 000 to 112 000 uninfected children will have been born to HIV-infected women during the next 10 years. Many of these will become orphans by the end of the decade.

We consider this estimate of the future number of uninfected children to be conservative because (1) we assumed all children born to HIV-infected women have a 25% risk of HIV infection, thus reducing the estimate of the number of uninfected children because some proportion of children will have been born prior to the mother's HIV infection, and (2) the estimate is based on prevalent HIV infection in childbearing women in 1990 and does not take into account current and future incident HIV infections in women of reproductive age. Barring significant breakthroughs in therapies that delay the progression of HIV to allow these women to survive and care for their children, we expect that caring for these AIDS orphans will require substantial economic and social resources in the coming decade.

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