

found 480 of 98,328 (0.49 percent) U.S. children living in high-risk areas to have blood lead concentrations in excess of 85 μg per 100 ml, indicating the possible magnitude of the present health hazard. This hazard may be complicated by a lack of overt symptoms of lead poisoning associated with these blood lead levels, rendering early diagnosis fortuitous in the absence of explicitly directed screening tests.

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References and Notes

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4. W. M. Grant, *Toxicology of the Eye* (Thomas, Springfield, Ill., ed. 2, 1974).
5. "Subclinical" lead poisoning has been defined as "the production of morbidity or mortality without the appearance of the classical signs or symptoms of clinical lead poisoning" by H. A. Waldron and D. Stöfen [*Subclinical Lead Poisoning* (Academic Press, London, 1974)]. Of necessity, assessment of subclinical poisoning requires examination and/or testing of the subject beyond gross clinical observation.
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7. Overt symptomatology we define as that set of clinical signs obvious upon gross observation of the subject, including, for lead poisoning, loss of appetite, tremor, seizures, and other neurological dysfunction. Conversely, covert signs include those not apparent without detailed assessment of an experimental subject's behavior or physiology, for example, retarded learning, impaired vision or balance, and hyperactivity of a quantitative degree.
8. H. F. Harlow and R. R. Zimmermann, *Science* **130**, 421 (1959); A. J. Blomquist and H. F. Harlow, *Proc. Anim. Care Panel* **11**, 57 (1961).
9. All blood lead samples were collected in lead-free, evacuated, rubber-stoppered tubes prepared by washing in 30 percent HNO_3 and rinsing in glass-distilled H_2O ; EDTA was used as an anticoagulant. Lead concentrations were determined for each blood sample by using a modified

Delves cup [R. D. Ediger and R. L. Coleman, *At. Absorpt. Newsl.* **11**, 33 (1972)] and a Perkin-Elmer model 306 atomic absorption spectrophotometer with a microsampling unit.

10. Unavoidable delays between blood sampling and blood lead assays occurred for these monkeys. Thus, their blood lead concentration exceeded the target levels of 85 μg per 100 ml before the assays detected the overshoot.
11. Weekly blood lead concentrations (in micrograms per 100 ml) in the three high-lead animals for the first 15 weeks of life were: for subject No. 1: 10, 30, 42, 53, 70, 137, 108, 120, 105, 123, 91, 114, 100, 65, and 83; for subject No. 2: 9, 23, 42, 59, 37, 69, 64, 42, 152, 72, 95, 124, 78, 140, and 84; and for subject No. 3: -, -, 9, 52, 300, 123, 136, 129, 142, 112, 85, 58, 60, 63, and 77. Peak values are italicized.
12. The U.S. Public Health Service classifies as "normal" any nonanemic child with a blood lead level less than 30 μg per 100 ml (Center for Disease Control statement: Lead Absorption and Lead Poisoning in Young Children, March 1975).
13. J. W. Davenport, A. S. Chamove, H. F. Harlow, *Behav. Res. Methods Instrum.* **2**, 135 (1970).
14. Stimulus luminances were measured with a Tektronix J16 option 2 Digital Photometer with luminance probe J-6503-2. The probe was corrected against the CIE standard observer, but was sensitive only to luminances greater than 1.0 nit (-0.50 log mlam). The function relating voltage to luminance on this meter was linear above this point, however, and the lowest luminance values were extrapolated from the linear regression equation computed on the voltages and luminances actually measured. The luminance values used were: +1.73, +1.42, +1.13, +0.82, +0.50, +0.04, -0.29, -0.81, -1.30, -1.55, -1.66, and -1.74 log mlam. The illuminations used to generate the luminance values were measured with a Gossen "Luna Pro" incident light meter. This instrument employs a CdS photocell which tends to overestimate brightness at the red end of the visible spectrum relative to the CIE standard observer. The readings taken with this instrument under low voltage incandescent light are thus slightly overestimated. The illumination values corresponding to the luminances listed above were: 2240, 1190, 700, 490, 166, 70.4 26.4, 6.60, 1.40, 0.525, 0.332, and 0.525 lux.
15. The analysis of variance involved one between-subjects variable (lead treatment, with 2 d.f.) and two within-subjects variables (light intensity, with 3 d.f., and stimulus pairs, with 4 d.f.); calculations were based on the model provided by J. L. Myers, *Fundamentals of Experimental Design* (Allyn & Bacon, Boston, 1971), pp. 202-203.
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18. J. L. Brown, in *ibid.*, p. 55.
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23. Publication No. 16-033 of the Wisconsin Regional Primate Research Center. This research was supported by a grant from the Food Research Institute, and grants RR-00167 and ES01062 from the National Institutes of Health. We thank P. Goldman, D. vanden Busch, I. Parrish, D. Goldhaber, B. Schneider, and D. Kehoe for assistance in data collection and R. Dodsworth for photometric assistance.

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The Capsian Escargotières: A Clarification

D. Grébénart of the Université de Provence, Aix-en-Provence, France, has asked us to point out that, in our article "The Capsian escargotières" (1), we neglected to properly cite his work. Grébénart's publications (2, 3) on the region provided us with the map coordinates and brief descriptions of most of the Capsian sites shown in our figure 1 (1, p. 911). His suggestion that the deposits exposed in Wadi Hamaja represented two distinct periods of occupation originally triggered our interest in the Ain Misteheyia escargotière.

To avoid confusion for those readers familiar with the literature, we also wish to point out that the Ain Misteheyia is identified as Ain Messaïa (site number 36) by Grébénart, who followed the no-

menclature used on the French topographic maps for the region. After extensive discussion in both 1973 and 1976 with the local inhabitants, who all insisted that the correct name was Ain Misteheyia, we elected not to use the apparently incorrect Ain Messaïa.

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