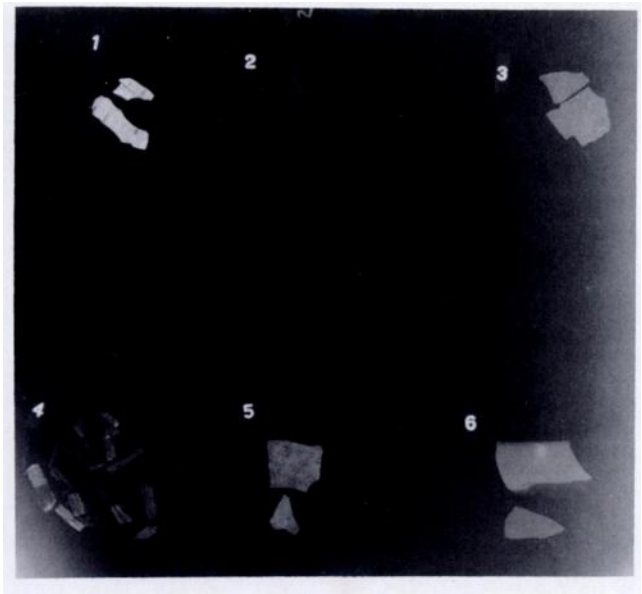

Fig 1. Radiographs of substances found on inspection of homes of children with high lead levels. 1, paint chips with 10 mg/cm^2 of lead; 2, wallpaper painted with non-lead paint; 3, unpainted plaster; 4, wood with paint with 1.4 to 1.8 mg/cm^2 of lead; 5, plaster painted with non-lead paint; 6, paint chips with 1.5 mg/cm^2 of lead.

In Reply.—

We agree with Dr O'Connor's assertion that radiopaque foreign bodies seen on abdominal radiographs of children with pica are not necessarily leaded paint chips. However, we consider it reasonable to infer that essentially all of the densities noted in the lead-poisoned children whose records we reviewed were, in fact, leaded paint chips.[1] For the great majority of lead-poisoned children in St. Louis, deteriorated leaded paint is found in their primary residence or in other residences the children frequent. During the period covered by our study (1989 through 1990), leaded paint hazards were found in 80% (2892 of 3595) of all such residences inspected. Although separate environmental data are not tabulated for children with blood lead levels greater than 55 μg/dL (for whom 12 of the 13 radiographs showed positive findings), almost all such children are found to have been exposed to leaded paint. In addition, abdominal radiographs of lead-poisoned children in St. Louis generally show densities that are almost completely radiopaque. For reasons stated in our paper, our data do not show precisely the proportion of lead-poisoning cases caused by paint chip ingestion, but they do support our essential conclusion: leaded paint chip ingestion remains an important cause of childhood lead poisoning in the United States.

After comparing the radiographic appearance of samples of paint chips, bare and painted plaster, painted wood chips, and painted wall paper with determinations of their lead content by an x-ray fluorescence analyzer, Dr O'Connor concludes that "all radiopaque substances are not lead chips." We have no doubt that her conclusion is correct in general, but for two reasons we are less sure that the unpainted plaster and the samples painted with "non-lead paint" could not contain potentially hazardous amounts of lead. First, portable x-ray fluorescence analyzers of the type used by most lead poisoning prevention programs are not accurate or precise enough at measuring low levels of lead to ensure, without confirmatory laboratory analysis, that paint testing negative by this method is essentially lead-free.[2] Although paint with high levels of lead, easily detectable by x-ray fluorescence, is more hazardous,[3] visible chips of paint with lower levels of added lead could amount to a dangerous dose if ingested by a child with pica. Second, lead residues can be found in porous substrates like plaster after leaded paint has been removed from them.

Finally, it is important to emphasize that an abdominal radiograph that indicates recent ingestion of leaded paint chips is not sufficient or even necessary to diagnose lead paint poisoning. As Dr O'Connor suggests, blood lead measurements and an investigation of a child's environment are required. When characteristic radiopaque densities are seen on an abdominal radiograph of a lead-poisoned child living in a residence with nonintact leaded paint, we believe it reasonable and prudent to assume that such densities are evidence of recent paint chip ingestion.

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α-Fetoprotein and Hypothyroidism in Infants

To the Editor.—

We recently have documented a relationship between serum α-fetoprotein (AFP) and congenital hypothyroidism (CH) in the newborn period. Previous studies in our laboratory and others had reported a relationship between elevated serum AFP and elevated thyroid-stimulating hormone (TSH)/low thyroxine T_4 in newborns and infants in the first few months of life.[1–4] The New York State Newborn Screening Program for CH routinely screens more than 300 000 specimens per year diagnosing 120 to 140 confirmed cases of CH annually. Recently, we assayed AFP in infants with thyroid dysfunction together with age-matched euthyroid controls from 1 to 3 months of age.[5] After the initial newborn dried blood screen and on physician's request, follow-up specimens from infants were assayed for serum AFP, TSH, and serum-bound T_4. Twelve of 12 infants with confirmed CH displayed elevated serum AFP levels (>2.0 SD) above our age-related reference ranges[6] and were consistent with high TSH and low T_4 levels. Following treatment with thyroxine in six infants that were tracked, all serum AFP levels declined to normal for age. Three of three infants with transient hypothyroidism (high TSH, normal T_4) also demonstrated elevated AFP levels. In 16 of 16 premature euthyroid infants (low T_4, normal TSH) elevated serum AFP levels were also demonstrable. Thus, we find elevated neonatal serum AFP levels consistent with situations displaying either elevated TSH levels, lowered T_4 levels, or both.

The physiological mechanism by which serum AFP is elevated both prenatally and postnatally in CH remains to be elucidated. Larsson et al[2] suggested either that the rate of AFP liver synthesis is increased or that the repression of this synthesis is delayed in the hypothyroid fetus/infant. However, Mengreli et al[4] argue that the half-life of AFP is extended in these infants (12 vs 5 days) and postulate that low thyroxine levels may be responsible for a slower catabolism of serum AFP in the liver. In a report predating these proposals, Belanger et al[7] demonstrated that both glucocorticoid- and thyroxine-treated newborn rodents displayed depressed AFP serum levels through a selective blockage of its hepatic synthesis and did not alter serum clearance of the fetal protein. In addition, thyroidectomy of these rodents did not modify the effects of administered glucocorticoid or thyroxine. In light of these prior studies and the data we presently submit, it would appear that