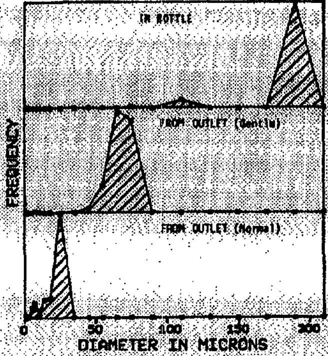

Figure 1. Size distributions for kaolinite flocs in the Niskin bottle and after removal from the outlet with gentle handling and with normal handling.

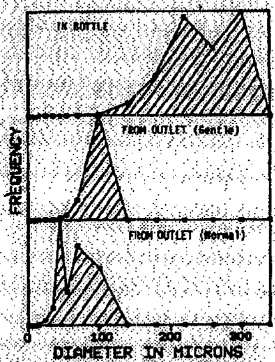

Figure 2. Size distributions for flocs of natural sample in Niskin bottle and after removal from the outlet with gentle handling and with normal handling.

water free falling 5 cm into the beaker from the outlet, the majority of the flocs were 45 μm with a small portion approximately 70 μm. This was only $^1/_5$ the size of the flocs inside the Niskin bottle.

The turbulence in the Niskin bottle created by four mixing propellers producing a G of 10 s^{-1} is comparable to that encountered in natural environments and, probably, most comparable to high-energy environments. If a slower stirring rate were used, resulting in a lower shear, the flocs would be larger and weaker, exhibiting even more dramatic breakage of flocs.

Conclusion

These controlled experiments demonstrate that significant breakage of flocs can occur when suspended material is withdrawn from a Niskin bottle in the usual manner, either allowing the water to fall freely from the discharge valve at the bottom of the bottle or allowing the water to run gently down the inside wall of a beaker through a tube connected to the outlet valve of the Niskin bottle. Alternative methods of obtaining samples from Niskin bottles for coagulation studies (such as using pipets through the top opening of the bottles) should be considered.

Registry No. Water, 7732-18-5.

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CORRESPONDENCE

Comment on "Development of an Air Quality Standard for Lead from Community Studies"

SIR: Snee recently proposed a methodology to develop an air quality standard for lead from community studies (*1*). The methodology can be summarized as follows: (a) specify the acceptable (distribution of) blood lead levels in the population (preferably the population at risk); (b) specify the air lead/blood lead relationship; (c) specify the (distribution of) blood lead (PbB) levels in the population at risk as it can be expected from nonair sources, using the air lead/blood lead relationship; (d) calculate the maximum amount of lead in the air that can be tolerated without violating the acceptable distribution of blood lead levels as specified under a.

In my opinion, the methodology can only be used when a set of assumptions are made that Snee does not make explicit, and I will show that some of these assumptions are not very realistic.

(a) Zielhuis has proposed an acceptable—not "desirable" as Snee mentions—distribution of PbB in 1974 (*2*). For young children, the population at the highest risk, this biological quality guide is not considered adequate anymore (*3*, *4*), and much debate is still going on concerning the health risks of low-level lead exposure (*5*). This situation is not atypical in the environmental health field and can be handled by applying safety factors or margins of safety. Snee, however, seems to think that this is not necessary, as his methodology tacitly assumes that it is wise to bring blood lead levels only down to (or all the way up to) the upper limit of what is considered acceptable at a certain moment in time. Essentially this is a policy proposal that can be (and should be) confronted with other established environmental policy principles like "best practicable means" etc.

(b) Snee has devoted much energy in specifying the air lead/blood lead relationship for both adults and children (*6, 7*). As his analyses appeared in other journals and are only referred to briefly in his recent contribution (*1*), I will only comment on them in a qualitative sense. There is one requirement that an air lead/blood lead relationship has to fulfill if one wants to use it in Snee's methodology, and that is that one be absolutely sure that air lead varies independently of lead in other environmental pathways to men or that the air lead/blood lead relationship takes other environmental pathways into account when independent variation is not possible or likely.

For children, pathways like soil and dust have been shown to be as important or maybe even more important than air (*8, 9*). Lead is brought into the air by automobiles and other sources, and its concentration in air depends, among others, on source strength. Independent variation of lead in air is unlikely: where lead in the air is elevated, lead in crops, soil, dust, etc., are or become elevated too.

Thus, it is necessary to analyze the air lead concentration as an *indicator* of the combined environmental pathways. Snee does not mention this fundamental requirement, and it is easy to show that his deduction of the air lead/blood lead slope for children from the Silver Valley lead study (*7*) through the use of multiple regression models fails to meet this requirement. The indicator approach would yield a slope close to 3 rather than 1.0, and Snee's analysis of the data in fact simply confirmed once again that for children, air is not the most important environmental pathway.

It is necessary to stress that the relationship between environmental lead and children's blood lead is a very complicated issue because of the many pathways and intervening variables that are involved. Quantitative relationships between the input of lead in the environment (e.g., through gasoline) and the resulting blood lead levels in children do not exist to date, and it will be difficult to derive them from community studies. An approach that seems to offer more perspective is the use of stable isotopes or stable isotope ratios. Unfortunately, few studies have followed this approach, and even fewer results are available (*10, 11*). It is also unfortunate that Snee has failed to mention these studies with their inherent advantages over the traditional community studies.

(c) The assumptions that have to be made here are largely the same as those concerning b. It is interesting to note that Snee uses rather old PbB data to derive his base line; these are only valid today when it is assumed that PbB values remain constant over time—which they obviously do not as the general decline in both adult and child PbB values in different developed countries shows (*12–14*). Even more important than variations in time are probably variations between population groups of different regional or cultural background (*15, 16*). The methodology assumes that something like a nationwide distribution of blood lead levels, related to nonair sources, can be established.

The reality, of course, is that regional and cultural differences will persist and that the environmental lead problem, e.g., the lead exposure problem, requires different forms of management for populations with different regional or cultural backgrounds.

Thus, specifying a base line is a matter of choice—a choice as to what extent the high-risk subpopulations within the population at risk are to be disregarded in the process of setting a national air quality standard for lead.

Registry No. Lead, 7439-92-1.

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SIR: The quantitative determination of an air quality standard for lead is an important and complicated issue that has not been given adequate attention. Brunekreef accurately summarizes my proposed approach (*1*) and raises some important issues. I will discuss his concerns in the order in which he raised them. It will be apparent that I do not agree with some of his views.

We must keep in mind at the outset that there are two key elements under discussion: the *statistical methodology* used to develop the air quality standard and the *data base* used to derive the estimated distribution of blood lead values and the blood lead–air lead relationship used in the procedure. Changes in the latter require only a recomputation while disagreements over the former require a reformulation of the statistical model used. The proper formulation is critical because it can result only after we have an understanding of the system we are trying to regulate.

(a) As I pointed out (*1*), the methodology I proposed will work for *any* "acceptable" (i.e., safe) level of blood lead. I chose the biological guideline proposed by Zielhuis (*2*), which calls for 50%, 90%, and 98% of the population at risk to have blood lead levels less than 20, 30, and 35 μg/dL, respectively. As I noted, the United States Environmental Protection Agency (EPA) guideline for children of 99.5% of the population to have blood lead levels less than 30 μg/dL (*3*) can also be used. Other guidelines may be appropriate. If the guideline distribution of blood lead levels changes in the future, then the

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air quality standard will need to be reevaluated.

The question of safety factors is, for the most part, a societal (policy) judgment outside the methodology used to calculate the standard. Margins of safety can be incorporated. It seems reasonable to assume, however, that any "acceptable" blood lead level contains a margin of safety. As discussed later, the procedure described in ref 1 is conservative, thereby providing an additional margin of safety, because the distribution of true blood lead values is broadened by blood lead measurement error, plus any variation due to the inability to accurately measure an individual's air lead exposure.

We must be careful in developing air quality standards to keep the scientific considerations separate from the societal (i.e., political) judgments. The calculations should be based on average values of the various inputs. Policy makers can promulgate a standard that is lower than the value calculated from the best available data and scientific knowledge if they feel that such action is necessary. If done in this manner, this action is clearly recognized as a societal judgment made outside of scientific considerations. Incorporating societal judgments within the scientific analysis (e.g., the use of upper confidence limits rather than average values of model inputs) creates confusion and reduces the effectiveness of the standard setting process because it is not clear to all parties which decisions are based on scientific knowledge and which are based on societal judgments.

The approach proposed in ref 1 is conservative because the distribution of observed blood lead values used contains measurement error that was not taken into account by the modeling procedure. It is a fundamental principal that an observed blood lead value for a given individual is equal to the *true* blood lead level plus measurement error due to sampling variation and analytical error: observed blood lead = true blood lead + measurement error. Blood lead measurement error, therefore, broadens the distribution of observed blood lead values compared to the distribution of true values (*4*). This produces a lower air quality standard than would occur if the standard were computed on a true value basis by taking the measurement error into account. Lucas (*4*) found that 50% or more of the total variation in blood lead measurements can be due to measurement error. Statistical procedures to properly account for measurement error are discussed by Lucas (*4*) and Hahn (*5*). These techniques can be used to further refine the methodology described in ref 1.

(b) Brunekreef properly points out that it is necessary to take all sources of lead exposure into account when setting an air quality standard and notes that soil and dust are important sources of lead for children. Lead exposure from sources other than the air is an integral part of my proposed procedure (*1*) and that used by the EPA (*3*). Improvements are needed in this regard and should be incorporated as new data on the other pathways become available.

Lead in dust and dirt comes from many sources including house paint, soil lead, previous air lead levels, and current air lead levels. The linear correlation coefficient between air lead and soil and dust lead is not large, however, being in general less than 0.5 (*6, 7*) and sometimes close to zero (*8*). Quantitative modeling of these relationships is an important area for further research, but the data available do not suggest a correlation strong enough to justify the use of air lead as an indicator variable as suggested by Brunekreef.

In my procedure (*1*) lead exposure from nonair lead sources is accounted for by the distribution of observed blood lead values used to derive the standard. The distribution used is that for a population at a fixed air lead level. The spread in this distribution represents person-to-person variation due to lead exposure from other sources as well as measurement error. In the Azar study (*9*), the inability to model the contribution to blood lead from pathways other than air shows up as variation in the blood lead measurements around the fitted curve. In the Tepper–Levin study (*10*), lead exposure from other pathways also resulted in a larger within-group variation. In both of these instances, lead exposure from other pathways increases the variation in observed blood lead levels, thereby broadening the distribution. As noted earlier, a broader distribution produces lower values for the resulting air quality standard.

Lead exposure from soil and dust was incorporated in the air quality standard developed by the EPA (*3*). They assumed that at a zero air lead level, a child's blood lead level would average 12 $\mu g/dL$ due to all sources of lead exposure except from the air. They then determined that an air lead level of 1.5 $\mu g/m^3$ would result in a geometric mean blood lead of 15 $\mu g/dL$, which is consistent with the blood lead guideline of 30 $\mu g/dL$ for the 99.5 percentile of the distribution. The EPA assumed that blood lead values follow a log-normal distribution with a geometric standard deviation of 1.3.

In any study the inability to unbiasedly sample each individual's air lead exposure also broadens the distribution of blood lead values at a fixed air lead level. This is particularly true in stationary sampler studies such as that conducted by Tepper and Levin (*10*), in which a person's lead exposure is approximated by a single air sampler situated in the area in which the person lives or works.

An extreme case is population sampling studies such as the NHANES II study (*11*), in which air lead measurements are not made. In these studies, the distribution of observed blood lead values contains variation due to different air lead exposures as well as lead exposure from other pathways. The variation in the NHANES II study, due to the 64 sampling locations, as well as any time trends that occurred during the 4-year study (1976–1980), further broadens the observed blood lead distributions. The use of distributions such as those developed in the NHANES II study will result in a conservative air lead standard due to the broader distribution of blood lead levels.

Brunekreef states, without documentation or reference to other papers, that the blood lead–air lead slope is greater than 1 and may be as large as 3. The basis for this conjecture is unclear, for an exhaustive study of the literature shows slopes ranging from approximately 1 to 2 with an overall average value between 1.0 and 1.4 (*12*). The determination of the appropriate slope is critical to the determination of the standard. It is, however, an input to my proposed methodology and is independent of the methodology. The standard could be derived by using a variety of slopes if appropriate.

(c) I do not share Brunekreef's concern about the age of the data base used in my calculations. The studies of Azar et al. and Tepper and Levin (*9, 10*) are, today, generally considered to be the best available *prospective* epidemiologic studies of the relationship between blood lead and air lead. The critical items are the distribution of observed blood lead levels, as estimated by the Tepper and Levin (*10*) study, and the blood lead–air lead relationship, as estimated by the Azar et al. study (*9, 13*) and by a linear model with a slope of 1. These inputs to the air quality standard calculations apply equally well to today's population. Blood lead levels in the general population are

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lower now than they were when these two studies were conducted for several reasons, including the decrease in the lead content of our food and water supply, the reduction in the number of houses with leaded paint, and the declining use of lead in gasoline. These changes should have no effect on the relationship between blood lead and air lead and can only lessen the spread of the within-population distribution. It is certainly appropriate to reevaluate the standard in the future and redo the calculations as new data become available, suggesting that such calculations are appropriate.

Brunekreef also raises concerns about the effects of variations in population groups. Contrary to his statement, the methodology assumes a distribution for the population at highest risk and not a nationwide distribution. It should also be recognized that differences present in the population due to culture, sex, race, etc., will broaden the distribution of observed blood lead levels. Lead exposure problems may require different forms of management for different populations; however, there is only one air quality standard. The base line will be determined by the regulatory agencies and is an input to the methodology described in ref 1.

Registry No. Lead, 7439-92-1.

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