may also be facilitated. Research may need to cover a range of topics, the test being the relevance to solving nutrition problems. Finally, publications could include sections on public nutrition, which would fill a gap in the present system of communications.

This letter seeks to identify the conceptual area of nutrition in populations and to define its components and boundaries. It also proposes for discussion a new and specific terminology to facilitate communication in both academic and service contexts.

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1 The views expressed are those of the authors in their personal capacities, and not of their institutions.

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Effects of diets containing high or low amounts of stearic acid on plasma lipoprotein fractions and fecal fatty acid excretion of men

Dear Sir:

The recent paper by Dougherty et al (1) suggests that plasma lipids and lipoproteins may not be at steady state for up to 40 d after a dietary intervention consisting of whole foods containing high amounts of stearic acid. A potential requirement of ≥ 40 d per study arm has several negative implications for future studies of stearic acid metabolism. Feeding studies in which all foods are consumed in a central facility using standardized meal preparation techniques are notoriously expensive, and any prolongation of study duration multiplies these costs. To require ≥ 6 wk of participation per arm may also reduce subject compliance and increase the dropout rate, reducing the power of the study. The extensive literature supporting a lipid-neutral effect of dietary stearic acid has recently been reviewed in this journal (2).

An initial concern is the interpretation that a high-stearate diet contributed to an increase in plasma lipoproteins at 40 d compared with the 20-d concentration. The data clearly show
that the subjects consuming the high-stearate diet had concentrations of serum total and low-density-lipoprotein (LDL) cholesterol below those of both subjects consuming the baseline and the low-stearate diet. Thus, the diet high in stearic acid did not cause an increase in serum total or LDL cholesterol at any time, but rather showed some variability between 20 and 40 d. This leads to an examination of the study design itself as a possible explanation for this variability in which a high-stearate diet appeared to initially lower cholesterol concentrations only to revert toward the baseline value at 40 d.

First, the findings may reflect a rebound effect of dietary stearate. What is the mechanism for this rebound? In this regard, the data from serum and fecal analyses seem at odds, in that less 14:0, 16:0, and 18:0 fats were absorbed over time [see Figure 1 in (1)]. This should lead to a reduction in total and LDL cholesterol over time. The observation of the opposite trend suggests an effect outside the gut. Delayed effects of stearate on synthesis or catabolism of lipoproteins by the liver or other organs have not been described.

Is this phenomenon unique to sheanut oil or is it observed when other sources high in stearic acid are consumed? As a pilot investigation before conducting feeding studies of the metabolic effects of cocoa butter, we measured serum lipid, lipoprotein, and apolipoprotein concentrations in six healthy young male subjects at baseline and 1, 2, 3, 4, and 5 wk after a diet high in cocoa butter as the source of stearic acid. The dietary intervention and laboratory methods were described previously (3). Each week’s value was the average of two measurements per week (Table 1). Reductions in total, LDL, and very-low-density-lipoprotein cholesterol and triacylglycerols over weeks 1–3 appeared stable by 28 d. No increase from days 15–21 to days 28–35 was observed. Concentrations of apolipoprotein B and apolipoprotein A-I were also stable by week 3. Plots of individual subjects showed no evidence of an increase in lipid concentrations in the later phase of the study. We concluded that 27-d feeding durations would be conservative to ensure lipid concentrations at a steady state after a change in dietary stearic acid, with the change in lipid concentrations largely occurring in the first 14 d of feeding.

One design feature of the study by Dougherty et al (1) is the use of a single measurement of lipids and lipoproteins as the endpoint at 20 and 40 d. Considerable intrasubject variability in lipids and lipoproteins has been well recognized, and many studies control for this variance by using the average of several measurements of lipids and lipoproteins as the study endpoint. A figure showing individual changes in the single measurement of serum lipids between baseline, 20 d, and 40 d would provide evidence of systematic trends in lipid endpoints compared with a random variation in lipids. However, even a systematic trend may be due to causes other than diet. For example, laboratory drift could account for differences between either high- or low-stearate diets and baseline, unless all samples were measured in a single batch. This methodologic issue needs to be clarified.

In summary, the results of Dougherty et al are in variance with previous studies of diets high in stearic acid. Whether this is due to unique characteristics of sheanut oil or to study design features is difficult to ascertain. Our interpretation of the literature and our own laboratory data do not support the conclusion that serum lipid concentrations show an accommodation to dietary stearic acid concentrations over time. Serum lipid concentrations appear to be at a steady state after a maximum of 3 or 4 wk of diet.

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