Reply to SP Bessman

Dear Sir:

Bessman's historical perspective in this issue (1) provides us with an interesting summary of one side of a controversy that occurred > 20 y ago concerning a possible role for tyrosine in the mental defect of phenylketonuria (PKU) and maternal PKU. Unfortunately, his editorial confuses the difference between early-treated and late-treated PKU. The study by Hsia et al (2) was conducted in institutionalized mentally retarded children and adults who had never received treatment for PKU. It was already known at that time, and we certainly know today, that dietary therapy in such individuals does not raise their intelligence quotient. The real benefit of dietary therapy is in preventing, not reversing, mental retardation by initiating the diet in early infancy. Hardly anyone today would dispute this.

Bessman's editorial contains misleading information about maternal PKU. The unreferenced statement, "Control of maternal phenylalanine concentrations with standard low-phenylalanine regimens did not prevent mental damage to the fetus...", is inaccurate. From the inception of dietary treatment for maternal PKU it was clear that mental retardation in the offspring could be prevented if the maternal blood phenylalanine concentration was well controlled through diet from either before conception or from early in pregnancy. Current data from the Maternal PKU Collaborative Study strongly support this concept (3). The experimental data from pregnant rats cited in Bessman's editorial is irrelevant to maternal PKU. The diet for maternal PKU (and for PKU) is phenylalanine deficient, not devoid of phenylalanine. A diet without phenylalanine, which is an essential amino acid, would produce severe depletion and a catabolic state and should never be given for the treatment of PKU. To my knowledge, such a diet is never prescribed. Finally, although tyrosine supplementation might be helpful in the treatment of PKU, treatment of PKU with tyrosine alone without a low-phenylalanine diet would be disastrous and would result in severe mental retardation. Bessman himself pointed this out more than a decade ago (4).

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Dietary cholesterol, serum cholesterol, and risks of cardiovascular and noncardiovascular diseases

Dear Sir:

The meta-analysis by Howell et al (1) confirms long-standing findings that consumption of egg yolks produces significant increases in blood cholesterol. Its quantitative results from " . . . the most tightly controlled, highest-quality experiments . . ." (1) —the only valid basis for a scientific assessment of the strength of the relation—are virtually identical to those in two other recent meta-analyses (2, 3): isoenergetic addition of two egg yolks per day (430 mg dietary cholesterol/d) to an adult's diet, without any countervailing decrease in cholesterol intake from other sources, results in an estimated average increase in blood total cholesterol of ≈0.31 mmol/L (12 mg/dL). This is an increase of ≈6% on the basis of a mean blood cholesterol concentration of ≈5.30 mmol/L (205 mg/dL) in the adult population in the 1990s. With 1% higher blood cholesterol resulting in an estimated 2% higher risk of coronary heart disease (CHD) for adults overall (4, 5), this increase in blood cholesterol means an estimated 12% greater CHD risk over 5–10 y. For young adult men, 1% higher blood cholesterol translates into an estimated 5% higher risk of CHD through middle age (5). This high long-term risk reflects the atherogenic effects of decades-long exposure to above-optimal serum cholesterol (6). Thus, for young adult men, a 0.31-mmol/L (12 mg/dL) higher blood cholesterol concentration resulting from consumption of two egg yolks per day means there is an estimated long-term increase in CHD risk of ≈30%. Howell et al (1) do not address this effect on CHD risk.

The main analyses in their paper (1) combined data unsoundly from high-quality trials with tight control and from inferior-quality trials with inadequate control. The inferior-quality trials yielded an ≈30% smaller estimated effect of dietary cholesterol on blood cholesterol than the high-quality trials. As a consequence of combining data from high-quality and inferior-quality trials, Howell et al arrived at an overall estimate that blood cholesterol increased significantly by 0.25 mmol/L (9.6 mg/dL, 4.7%) as a result of a 430-mg/d higher cholesterol intake. This value underestimates the risk of CHD by ≈25%.

Howell et al also did not address other important data on the

adverse effects of high dietary cholesterol:

1) As shown in thousands of animal experiments in mammalian and avian species, including nonhuman primates, addition of cholesterol to the usual diet is virtually a requirement for the production of atherosclerosis.

2) Addition of small amounts of cholesterol to the diet of rabbits, chickens, and monkeys— resulting in little or no increase in blood cholesterol—produces atherosclerosis in the long-term nonetheless (7, 8).

3) Prospective population studies of human cohorts have shown repeatedly that a higher cholesterol intake by individuals relates to a higher risk of CHD independent of (ie, over and above) the adverse influence of higher dietary cholesterol on blood cholesterol (5, 9).

4) Data are available indicating that a higher cholesterol intake adversely influences the composition and concentration of atherogenic lipoproteins (1–4), blood pressure (5), thrombogenic factor VII, and risk of breast, colon, lung, and prostate cancers (references available from authors on request).

5) Estimated *favorable* effects of *lower* dietary cholesterol on both blood cholesterol and, independently (additionally), on CHD risk are considerable. In the Western Electric Study, a dietary cholesterol intake lower by 430 mg/d (two egg yolks/d not consumed) was associated with an ≈43% lower long-term risk of CHD death, an ≈25% lower risk of death from all causes for middle-aged men, and a life expectancy greater by ≈3 y (5).

These briefly summated facts are the main scientific foundation for the conclusion that a habitually high cholesterol intake adversely affects human health and longevity, hence, the recommendation that Americans consume less dietary cholesterol, ie, < 300 mg/d on average, a recommendation made repeatedly by every responsible independent expert group (eg, the American Heart Association, the Inter-Society Commission for Heart Disease Resources, the National Academy of Sciences—National Research Council, the American Diabetes Association, and the National Cholesterol Education Program) from 1961 through the 1990s (5, 6). Despite its flaws, the data in the meta-analysis by Howell et al lend further support to this recommendation. Their data analysis does not support the following assertion in recent egg industry advertisements (*Newsweek*, July 7, 1997): "If you're healthy, go right ahead and enjoy your eggs: Your cholesterol will probably stay about the same."

It is a reasonable inference that the sizeable decline in per capita egg consumption in the United States in recent decades, and hence in per capita total cholesterol intake, has been one important component of the improved dietary patterns leading to a fall in mean serum cholesterol concentration in the adult population from ≈6.08 mmol/L (235 mg/dL) in the 1950s to ≈5.30 mmol/L (205 mg/dL) in the 1990s, and to the concomitant sustained marked reductions in mortality rates from CHD, all cardiovascular diseases, and all causes (5, 6).

Despite the considerable decline in per capita egg consumption in the United States in recent decades, the egg yolk is still on average a major source of dietary cholesterol. Therefore, Americans remain well advised to lower their egg yolk intake as part of their ongoing endeavors to achieve a daily dietary cholesterol concentration < 300 mg/d and generally improve their nutritional status, prevent major chronic diseases, and extend their life expectancy (10).

These efforts are poorly served by recent egg industry advertisements, which misinterpret results of recent meta-analyses, particularly those of Howell et al. About 20 y ago, on the basis of concerns from the American Heart Association and consumer groups, the Federal Trade Commission carried out successful legal action—upheld by the Supreme Court—to compel the egg industry to desist from false and misleading advertising that eggs had no harmful effects on health. In 1997, the scientific evidence is even more overwhelming that any such health claims for egg yolks are false and misleading. Current claims in egg industry advertisements appear to be essentially a rerun of old false and misleading claims.

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Reply to J Stamler et al

Dear Sir:

The letter from Stamler et al misrepresents the aims and findings of the paper, "Plasma lipid and lipoprotein responses to dietary fat and cholesterol: a meta-analysis"(1). Two specific points of clarification are necessary. First, the letter attributes our results as representing controlled experiments, when in reality we state specifically in our paper that previous meta-analyses focused on "the most tightly controlled, highest-quality experiments." Our intent, as stated, was to test whether the findings of these controlled investigations were generalizable to broad experimental settings and to the design of practical nutrition education interventions.

We accomplished this objective using rigorous meta-analytic techniques that allowed us to assess the effect of study quality (method of determination as defined in the paper) on the blood lipid response to dietary change. Specifically, interaction terms between study-quality rating and dietary-change variables were constructed and used as potential predictors of change in blood lipids. None of these study-quality interaction terms made a significant contribution to the prediction of blood lipid response to dietary change. We also compared our serum total cholesterol prediction model with those of other investigators and showed graphically the overlapping CIs among the "metabolic ward" and "free-living" studies of Hegsted (2) and our analyses of combined studies. We are unaware of the source of the data supporting the assertion of Stamler et al that "inferior-quality trials yielded an ≈30% smaller estimated effect of dietary cholesterol on blood cholesterol than the high-quality trials." Our paper did not report nor did our data support this differential effect. Furthermore, the estimated effect of dietary cholesterol on plasma cholesterol from our analysis of 224 studies is not significantly different from that estimated from 80 metabolic ward studies reported by Clarke et al (3).

The second point of clarification involves the quantitative interpretation of our results by Stamler et al. At the outset of their letter they indicate that according to our model, increasing dietary cholesterol by 430 mg/d would be expected to increase serum total cholesterol by 0.31 mmol/L (12 mg/dL). This calculation is incorrect. Rather, a 430-mg/d increase in dietary cholesterol would be expected to result in a 0.25-mmol/L (9.6-mg/dL) increase in serum total cholesterol. This correct calculation appears later in the same letter from Stamler et al. It is unclear why the two different computations are presented by them.

The question of any effect of dietary changes on coronary heart disease (CHD) risk was not evaluated in our report because this was outside the scope of the data being analyzed. On the basis of the Seven Countries Study (4), however, it was estimated that a 0.52-mmol/L (20 mg/dL) change in plasma cholesterol results in a 17% change in relative risk. Accordingly, a shift from the current diet to the National Cholesterol Education Program Step I diet, resulting in an average reduction of 0.26 mmol/L (10.2 mg/dL), would result in a 9% decrease in relative risk. The estimated effect of reducing dietary cholesterol from 450 to 300 mg/d would lower plasma cholesterol by 0.08 mmol/L (3 mg/dL) and reduce the risk of CHD by 2.5%.

The lack of a consistent, significant relation between dietary cholesterol intake and CHD mortality in several epidemiologic studies (*see* below) suggests that this may be more of a problem in hyperresponsive animal models of atherosclerosis than in humans. There is also the question of whether, in the absence of changes in total plasma cholesterol, there are any changes in the distribution of cholesterol between the lipoprotein particles or production of atherogenic lipoprotein particles in these hypersensitive animals. It is clear that more studies are needed to clarify these uncertainties.

Any association between high cholesterol intake and disease should be considered suspect because of the many other confounding variables. For example, in the Western Electric Study referred to by Stamler et al, the highest quintile of cholesterol intake (1079 mg/d) was the only group with increased cardiovascular disease incidence (5). At this level of intake it could be predicted that the subjects had a high intake of animal products, with a correspondingly low intake of fruit and vegetables. Similar findings were reported in the Ireland-Boston Diet-Heart Study (6) in which subjects had a low vegetable-foods score and a high animal-foods score. Under these conditions it would be expected that intakes of dietary fiber, antioxidants, folate, and B vitamins would all be low, resulting in increased CHD risk. The question is, does the increased cardiovascular disease incidence result from what is in the diet or from what is missing from the diet? Data reported by Ascherio et al (7) suggest just such a question because dietary cholesterol was unrelated to CHD incidence or mortality in multivariate analyses that included dietary fiber.

The authors also point out that the 300-mg/d recommendation has been "made repeatedly by every responsible independent expert group from 1961 through the 1990s," yet most countries do not restrict dietary cholesterol as part of their national dietary policy (8).

Our meta-analysis dealt specifically with changes in plasma lipids and lipoproteins in response to changes in dietary fat and cholesterol. Our conclusions remain the same: compliance with national dietary guidelines will reduce the average plasma cholesterol concentration in the population by ≈5%. What effects these dietary changes might have on CHD morbidity and mortality and by what mechanisms remain to be determined.

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Reply to J Stamler et al

Dear Sir:

It is curious that Stamler et al used the report by Howell et al (1) as a platform to critique advertisements developed by the American Egg Board. The meta-analysis by Howell et al (1) investigated effects of dietary cholesterol on plasma cholesterol, not the effect of eggs on coronary heart disease (CHD) risk nor the effect of the egg industry's advertising on egg-consumption patterns. That said, data from Howell et al (1) indicate that adding two eggs a day (430 mg cholesterol), every day, to the diet increases blood cholesterol by 0.25 mmol/L (9.6 mg/dL). Stamler et al postulate that this would increase the risk of CHD by 10%. The egg industry concurs with both points. We disagree, however, with the accusation that egg-industry advertisements stating "If you're healthy, go right ahead and enjoy your eggs: Your cholesterol will probably stay about the same" is "false and misleading." We also take exception to the suggestions that "egg yolk is still on average a major source of dietary cholesterol" and that a decline in egg consumption is "one important component" in the reduction in serum cholesterol concentrations in recent decades. These arguments are not based on the realities of per capita egg consumption.

Data from the third National Health and Nutrition Examination Survey (NHANES III) indicate that eggs contribute one-third of the cholesterol in the diet (2). In 1995, per capita egg consumption was 235 eggs/y (4.5 eggs/wk, 0.64 eggs/d), which added 138 mg cholesterol to the diet. Although eggs are a concentrated source of dietary cholesterol, other foods contribute two-thirds of the cholesterol in the diet.

The ≈0.78-mmol/L (30 mg/dL) fall in plasma cholesterol occurring between the 1950s and 1990s simply cannot be attributed to declining egg consumption. In 1945, at the peak of per capita egg consumption, intake was 405 eggs/y (almost 8 eggs/wk, 1.1 eggs/d). The decrease from 1.1 eggs/d in 1945 to 0.64 eggs/d in 1995 resulted in a 101-mg/d decrease in dietary cholesterol (0.47 eggs/d, 215 mg cholesterol/egg), which, based on the meta-analysis, would lower plasma cholesterol by ≈0.06 mmol/L (2.2 mg/dL). Thus, a 42% reduction in egg consumption lowered the average plasma cholesterol concentration by 1%.

Even at the peak of consumption, intakes were never as high as two eggs a day, every day. The egg industry advertisement states "Your cholesterol will probably stay about the same" because returning to peak egg consumption would result in an unmeasurable plasma cholesterol change in most individuals. We recognize that 15–20% of the population are hyperresponders to dietary cholesterol, and the industry is funding research to identify characteristics associated with hypersensitivity so that egg restrictions can be targeted to those who would benefit and restrictions relaxed for the 80–85% in whom there is no effect.

The four highest per capita egg-consuming countries (Japan, Mexico, Spain, and France) also happen to have the lowest rates of CHD. And although it can be argued that these countries have dietary patterns that differ from those of the United States, it is unlikely that the stated negative effects of eggs can be offset by lower intakes of energy from fat, soy protein, fiber from beans, olive oil, and red wine. As reported by Artaud-Wild et al (3), countries with similar cholesterol-saturated fat index values differ threefold in rates of CHD mortality, and a high intake of cholesterol and saturated fat combined with a higher consumption of fruit, vegetables, and vegetable oils results in lower rates of CHD. Verschuren et al (4) also reported that whereas some countries have the same relation between changes in relative risk of CHD and changes in plasma cholesterol, at the same plasma cholesterol concentrations absolute risk can vary fivefold.

It is only possible to provide limited comment on the hypothetical "cholesterol independent effect" on CHD risk because data supporting this thesis are rather limited. In 1984 Clarkson et al (5) reported that regression of existing atherosclerotic lesions in *Macaca mullata* was higher in animals who were consuming a high amount of dietary cholesterol to maintain a stable plasma cholesterol concentration (ie, nonresponders) than in animals with a greater sensitivity to dietary cholesterol (responders) (5). If dietary cholesterol is atherogenic, independent of effects on plasma cholesterol concentrations, then one would expect that the animals fed the higher amounts would exhibit less regression, not greater regression, than the animals fed the lower amounts of cholesterol.

The egg industry has supported research on the dietary cholesterol-plasma cholesterol relation for years and the reports by respected investigators, published in quality peer-reviewed journals, are labeled "industry funded" and disregarded. Rarely are these studies from the same research group and, as shown by the meta-analysis, the results are consistent with nonindustry- funded studies. The egg industry wants to address the hypothesis that there is an "independent effect of dietary cholesterol," yet investigators are reluctant because they fear accusations that their research was "industry funded" should their results not conform with this hypothesis. The fact is that many epidemiologic studies have not found a direct or indirect effect of dietary cholesterol on CHD incidence. Among these studies are the Lipid Research Clinics Prevalence Follow-Up Study in 4546 subjects (6); a study of 43 757 male health professionals (7); the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study in 21 930 men (8); and numerous other studies as reviewed by Ravnskov (9). Even the Twenty Countries Study data indicate that dietary cholesterol is not related to CHD mortality when multivariant analysis includes saturated fat, polyunsaturated fat, and alcohol intakes (10). The egg industry believes that the evidence is clear that reductions in plasma cholesterol require reductions in total and saturated fat, and that confusion about the dietary cholesterol–plasma cholesterol relation only complicates effective dietary interventions.

Finally, the American Egg Board, a commodity research and promotion board with US Department of Agriculture oversight, has never in its 21-y history been enjoined by the Federal Trade Commission to withdraw an advertisement. The actions of individual groups of egg producers should not be attributed to an industry-wide commodity promotion program, and such accusations are, in their own right, false and misleading.

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Standardization of nomenclature of body composition in weight loss

Dear Sir:

Roubenoff et al (1), writing from three major research centers, provide your readers with a valuable review of results from recent body-composition work. They propose standardized nomenclature as follows: *wasting* for involuntary weight loss, *cachexia* for involuntary loss of lean body mass with little weight loss, and *sarcopenia* for specific involuntary loss of muscle mass.

Roubenoff et al give good examples of conditions in which these three different types of depletion occur, and of course there are intermediate forms, as in marasmic kwashiorkor. It would certainly be useful for us to have agreed names for these conditions. But two of the proposed terms are already used with different meaning (cachexia) and broader meaning (wasting). The small nutrition establishment has little chance of changing existing usage by the much larger medical profession.

Sarcopenia is an almost new term, hardly taught as yet to medical students or listed in dictionaries. There are no alternative meanings to Rosenberg's definition. The concept of assessing it, for example, by measuring midthigh circumference is clinically important. With continued promotion this term should become established in medical vocabulary.

But *cachexia* goes back to Galen and came into English medical usage in 1541 (2). It is a somewhat vague term but describes gross generalized wasting (emaciation) and ill health, usually associated with chronic disease. *Harrison's* (3) and *Davidson's* (4) textbooks of medicine and five major medical dictionaries (2, 5–8) agree on this. The 16th edition of the World Health Organization international classification of diseases lists in its index 11 different varieties of cachexia (some with more than one name), such as cancerous cachexia, cardiac cachexia, and hypopituitary cachexia. The established meaning of cachexia is thus substantially different from the proposal of Roubenoff et al.

Wasting is used in medicine to mean a loss of bulk or substance of a part or the whole of the body. For example, there can be wasting of one leg after poliomyelitis. So wasting is not as clear as body weight loss, but a safer description than cachexia. Disproportionate wasting of lean body mass would be a safer description than cachexia. *Wasting* can equally be used to mean wasting of body fat with preservation of lean body mass.

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Reply to AS Truswell

Dear Sir:

We are grateful to Truswell for his instructive comments. We agree that *cachexia* and *wasting* have been used in various connotations and denotations for many years. In fact, that was the impetus for our small suggestion. However, we differ with Truswell in our understanding of the current usage of *wasting*. It is certainly true that this term has been used to describe atrophy of a part of the body, such as a limb after polio. However, more recently this usage has been overwhelmed by the sense of undesirable loss of weight that is implicit in the term AIDS wasting. In fact, the AIDS epidemic has largely been responsible for the replacement of this meaning of *cachexia* by *wasting*. We therefore sought to retain the elegant term *cachexia* (from the Greek for what is politely termed poor condition) in what is biologically its most important meaning: the loss of lean body mass (or body cell mass, depending on the measurement technique), which is thought to be directly responsible for the poor condition and reduced functional capacity that occurs with cachexia.

Although it is true that the nutrition community is but a small part of the medical research and practice establishment, we do not despair of slowly educating our colleagues. If we do not clarify our nomenclature among ourselves, we shall never educate our peers. We hope this effort at rationalizing nomenclature will help in this regard, as a common language is essential. Even if our suggestion is not accepted, we believe it is useful to stimulate the kind of debate that may lead to consensus and progress in the future.

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Moderate zinc deficiency

Dear Sir:

In the paper by Blostein-Fujii et al (1) it is suggested that moderate zinc deficiency occurs frequently in subjects with type 2 diabetes. The requirements for zinc in diabetic subjects are discussed further in an editorial by Sandstead and Egger (2), who emphasize the difficulties of diagnosing zinc deficiency and also draw attention to the importance of dietary zinc bioavailability. Blostein-Fujii et al, unfortunately, did not measure dietary zinc intake or bioavailability in their group of diabetic volunteers.

We recently studied zinc metabolism in a group of healthy men and women with type 2 diabetes and matched control subjects (3). Habitual zinc intakes of the diabetic patients, determined from a 5–7-d weighed food diary, ranged from 5.5 to 15.4 mg/d. The size and rate of turnover of exchangeable body pools of zinc were measured by administering an intravenous dose of a stable isotope of zinc and monitoring its rate of disappearance from the plasma, and the data were analyzed by using kinetic modeling. In the absence of good measures of zinc status, this method is believed to be a useful means of predicting body zinc concentrations. We found no differences in exchangeable zinc pool sizes between the diabetic patients and the control group. We also observed no significant differences in the efficiency of zinc absorption from a standard breakfast or in endogenous losses of zinc, apart from a higher urinary zinc excretion in the diabetic men.

The results of our study clearly indicate that healthy people with type 2 diabetes who are consuming a diet containing zinc in excess of the UK estimated average requirement (7.3 and 5.5 mg/d for men and women, respectively) (4) do not appear to have any changes in zinc metabolism that require an increased dietary intake of zinc.

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Reply to SJ Fairweather-Tait

Dear Sir:

Fairweather-Tait, in a recent paper from her laboratory (1), questioned whether moderate zinc deficiency occurs frequently in type 2 diabetic subjects. A paper from my laboratory had contended that moderate zinc deficiency may be common among type 2 diabetic women (2).

The study by Fairweather-Tait's group (1) deserves attention. Our recent paper did not provide this attention simply because their work

was published after our manuscript was submitted. Their work raises the possibility that some indexes of zinc metabolism may not be abnormal in all diabetic subjects. Their study with stable isotopes shows normal values for certain indexes in male or female diabetic subjects compared with control subjects of the same sex. However, for many reasons, I feel that this study cannot be used to conclude that zinc status is of no concern to diabetic individuals. One reason is that diabetic subjects are not a homogeneous population. The subjects examined by Fairweather-Tait's group represent one particular group of type 2 diabetic subjects whereas ours represent another. These groups likely differ in many ways. One definite difference is body mass. The subjects in the study by Fairweather-Tait's group (1) seem to have had good weight control but our study subjects did not (2).

Another difference was noted by the authors themselves in their paper (1). Their study had a very small subject number (five per group) and large relative SDs. The latter was particularly true for zinc exchangeable pool data. Thus, small differences may not have even been detectable. Small differences may be very important. Conceivably, some zinc functions could be greatly compromised despite small difference in total zinc in a relatively large pool. Furthermore, even if there are absolutely no differences for one type of zinc pool, this does not mean that other pools cannot be abnormal. I am not certain that the zinc pool assessed by Fairweather-Tait's group encompasses all body zinc pools. After all, the measurements taken only reflected fairly short-term fluxes (on 1 d). There have been other studies of zinc kinetics that have used considerably longer equilibration periods.

There are other reasons not to dismiss the idea that zinc status may be a concern for diabetic subjects. One is that our paper (2) shows very low activities for a zinc-dependent enzyme. Moreover, the activities are increased by zinc supplementation. This does not prove that there is zinc deficiency, but that is the simplest explanation. In any case, such data implies abnormal zinc metabolism. This idea is also supported by the swing in plasma zinc values from low readings before supplementation to high readings after supplementation (1). In a somewhat similar observation with type 1 diabetic subjects, plasma zinc showed an unusual time course after intravenous zinc injection (3). Initially, there is an abnormally high rise in plasma zinc, but then values fall at above-normal rates. Whether or not these results indicate unusually high zinc requirements remains to be seen, but this possibility merits consideration.

This contention is reinforced by considering the point made at the beginning of our recent paper (2). Namely, that several other laboratories have found a variety of signs consistent with moderate zinc deficiency in type 2 diabetic subjects.

Fairweather-Tait correctly points out that our study does not include certain types of measurements. However, the same can be said about the study by her group—they did not assess any functional-status indicators. Actually, all studies have limitations, especially when the studies are funded by small grants. It is hoped that future work will clarify whether zinc status is a major concern for many type 2 diabetic individuals. However, I feel that we have enough data right now to seriously consider this possibility.

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REFERENCES

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2. Blostein-Fujii A, DiSilvestro RA, Frid D, Katz C, Malarkey W. Short-term zinc supplementation in women with non-insulin-dependent diabetes mellitus: effects on plasma 5′-nucleotidase activities, insulin-like growth factor concentrations and lipoprotein oxidation rates in vitro. Am J Clin Nutr 1997;66:639–42.
3. Martin AM, Extremera BG, Soto MF, et al. Zinc levels after intravenous administration of zinc sulphate in insulin dependent diabetes mellitus patients. Klin Wochenschr 1991;69:640–4.

Erratum

Hilson JA, Rasmussen KM, Kjolhede CL. Maternal obesity and breast-feeding success in a rural population of white women. Am J Clin Nutr 1997;66:1371–8. On page 1373 in the paragraph in the right column beginning "In contrast," the proportions 95.7%, 91.1%, and 87.8% should be replaced with 4.3%, 8.9%, and 12.2%. The Production Office regrets the error.