not expressed and excluded from analysis. In addition, because there is no scientific backup for determination of an expression threshold, criteria will always be arbitrary and usually based on former experience. We have chosen our criteria based on the fact that we are able to verify the expression of genes with a normalized signal >20 with quantitative polymerase chain reaction. Moreover, drastic filtering should be performed with care because it may negatively influence detection power when using statistical tests specifically designed for microarray analysis (7). Finally, signal intensity on a microarray is dependent on probe composition and thus does not per se relate to absolute mRNA abundances (8).

The third point that Dahlman raises is that, according to her experience, it is difficult to confirm changes in expression <20% and therefore those genes should be excluded from the analysis. However, in our Figure 4 we show the confirmation of our microarray data, including genes with mean fold changes <20%. In our opinion, caution should be taken with filtering based on fold changes in human intervention studies, because mean fold changes are the average of several individual responses and the average can be highly influenced by a few high responders, especially in small sample sizes. In our analysis, changes in expression of genes were only considered meaningful if genes are in one pathway or network, as is the case in our article.

An additional way to analyze the data that circumvents the issues mentioned by Dahlman is by the use of Gene Set Enrichment Analysis (GSEA). GSEA uses a ranked list as input and determines whether the genes within processes and pathways are either randomly distributed along the list or occur primarily at the top or bottom of the list (9, 10). In GSEA, no arbitrary preselection of significantly changed genes is applied, and therefore the GSEA method overcomes all of the issues raised by Dahlman regarding FDR correction, filtering, and fold-change thresholds. As mentioned in our article and shown in supplemental Table 1 under “Supplemental data” in the online issue, results of GSEA were highly consistent with the results obtained by using our selection criteria and confirmed the differential effect of an MUFA-rich diet on inflammatory gene expression.

On the basis of the above-mentioned arguments, we are convinced that our analysis method of microarray data as well as our conclusion that consumption of an SFA-rich diet resulted in a proinflammatory gene expression profile in adipose tissue was justified.

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a substantial increase in 24-h NaE over time in the US population, but this was not the case in Bernstein and Willett’s analysis. To try and shed more light on this, we recalculated the linear regression of 24-h NaE on time with and without adjustment for obesity prevalence, with the assumption that the prevalence of obesity in the study populations included in Bernstein and Willett’s meta-analysis was similar to the concurrent prevalence of obesity in the United States, by using data from the National Center for Health Statistics (Figure 2). The results of our analysis indicate that, whereas no association was apparent when using unadjusted 24-h NaE data, an inverse relation was detected when taking the rising prevalence of obesity into account, which suggests a trend toward a decrease in average salt intake over time of as much as 2.1 mmol/d per year. In other words, had the prevalence of obesity remained stable between 1970 and 2000, we might have seen a reduction of 63 mmol in sodium chloride excretion or 3.7 g in salt intake over 30 y.

The experience of several countries provides evidence in support of the possibility of reducing sodium chloride intake at the population level. To mention a few examples, in an intervention study in 2 villages in Portugal, salt intake was successfully reduced in one of the villages by the provision of processed foods reduced in salt, and blood pressure was reduced accordingly at 1 and 2 y of intervention (5). The Finnish national experience tells us of an impressive reduction in population average salt intake from as much as 14 g/d in 1972 to 8.5 g/d in 2002 and of a likewise striking decrease in mortality from stroke from 60% to 20% in men and from 45% to 10% in women over the same period (6). Notwithstanding McCarron et al’s analysis (2) of UK surveys conducted between 1984 and 2008, which apparently suggested no significant change in dietary sodium intake, the result of the most recent evaluation of 2 independent random samples of the British population led to an observed reduction in population salt intake from 9.5 g/d in 2000–2001 to 8.6 g/d in 2006 (7).

As noted by Bernstein and Willett, despite the relatively long scientific debate over the “salt hypothesis,” national strategies that aim at a population-wide reduction of sodium intake have been implemented only recently in most countries, including the United States, and are affecting public opinion only very recently. We need time before being able to detect a trend to reduced dietary salt intake in Western countries, and it may take even more time to detect a reduced prevalence of hypertension unless the steady increase in the prevalence of obesity is not reversed. The use of this time lag as evidence that governments’ efforts to deal with excess salt intake are doomed to failure is misleading and recalls the “benign neglect” with which blood pressures of up to 160/95 mm Hg were observed and left untreated in a vast proportion of the population a few decades ago, ignoring the substantial elevation in morbidity and mortality that they carry with them (8).

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Reply to P Strazzullo et al

Dear Sir:

Increasing energy intake in the US population over the past several decades has led to an increasing prevalence of overweight and obesity (1). With these changes, urinary sodium excretion would be expected to rise as well because greater energy intake would likely bring with it larger amounts of sodium. As pointed out by Strazzullo et al and as reported elsewhere (2), obesity is usually associated with greater sodium intake, which may be due to the higher energy