TABLE 1
Proportion of unmarried women included in 1984 Ross Laboratories Survey of infant feeding practices (2)

	All infants		
	Weighted sample		Government data (3)
	%	n	%
Unmarried*	19.5	251	19.4†
Married	80.5	1029	80.6

* Single, widowed, divorced.
† Classified only unmarried.

by the National Center for Health Statistics (3) reflecting the proportion of unwed mothers in the United States.

It's difficult to determine the proportion of unwed women included in each of the Ross Laboratories surveys. However, I can say with assuredness that our sampling frame includes unwed mothers.

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Reply to letter by Martinez

Dear Sir:

We read with interest Mr Martinez's letter concerning the availability of data on infant-feeding practices among unwed mothers in the United States. These data were not presented prior to the 1985 article cited in his letter. Although Ross Laboratories surveys do include a percentage of unwed mothers similar to that of the US population, the data do not support the representativeness of the unwed mothers for computing national estimates of breast-feeding rates. First, the demographic mix of unwed mothers included in the Ross surveys may not be representative of the US population. Second, since the surveys are conducted only among mothers of older infants, mothers who have given up infants for adoption in the first few months of life may be missed. Finally, data collected from personal interviews (the survey method used in the 1985 Ross article cited in the letter) may produce appreciably different estimates of breast-feeding rates from estimates based on data collected from mailed questionnaires (the method used in the traditional Ross Infant Feeding and National Natality Surveys). Unless these potential differences can be taken into consideration, national estimates of breast-feeding among unwed mothers may not be accurately determined.

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Hypo- and hyperresponders to dietary cholesterol

Dear Sir:

In studies on the effect of dietary cholesterol on the level of serum cholesterol in humans, there generally is a considerable variability in individual response. In the literature the concept of hyper- and hyporesponders became widely accepted, and the recent study of Oh

and Miller (1) is another example. In the numerous cholesterol loading studies (in practice that is egg feeding studies) the response of serum cholesterol in a given subject was usually measured in one study only. When subjects were tested twice with a wash-out period in between, it was found that a subject hyperresponsive to egg-yolk consumption in one experiment, may appear hyporesponsive in the second experiment and vice versa (2, 3). Oh and Miller (1) had similar results when the challenge with three eggs/day was followed by a challenge with six eggs/day, and they introduced the terms of hypo-hyper-responders and hypo-hypo-responders.

These observations suggest that observed differences in response to dietary cholesterol in humans could be due to random fluctuations. Indeed, it is known that diet-independent, so-called "spontaneous" fluctuations in serum cholesterol concentrations in man can be as high as 20% (4). These fluctuations are of the same order of magnitude as the commonly observed response to dietary cholesterol loads. The study of Oh and Miller (1) nicely illustrates the impact of within-person fluctuations of serum cholesterol on the classification of subjects as hypo- or hyperresponsive on the basis of only one cholesterol determination on a low and high-cholesterol diet. Such a selection procedure will cause the inclusion in the hyperresponder group of some subjects whose serum cholesterol through chance fluctuations happened to be depressed at the beginning or elevated at the end of the high-cholesterol diet period. The reverse holds for the hyporesponder group. Indeed, Oh and Miller (1) found that the mean baseline cholesterol level was lower in the apparent hyperthan in the hyporesponders.

Thus observed differences in serum cholesterol responses between individuals are not necessarily due to stable differences in sensitivity to dietary cholesterol. We have carried out three controlled dietary trials with a large number of the same subjects in order to address the question whether individuals exist with a consistently high or low serum cholesterol response to dietary cholesterol (5). In each trial the healthy volunteers consumed a low-cholesterol diet (~120 mg of cholesterol/day) and a high-cholesterol diet (~650 mg/day in the first and second experiment, and ~1000 mg/day in the third trial), the cholesterol component of the diets being the only variable. The extra cholesterol was provided by egg yolk. Seventeen putative hyper- and 15 putative hyporesponding subjects with mean increases of 19 and 0%, respectively, were selected in the first trial and participated in the second and third experiment. Although the response in each subject was only partly reproducible, the selected hyperresponders showed significantly higher serum cholesterol responses in the second and third trial than did the putative hyporesponders (3). Standardized regression coefficients for individual responses (n = 32) in two experiments ranged from 0.34 to 0.53 (**Fig 1**).

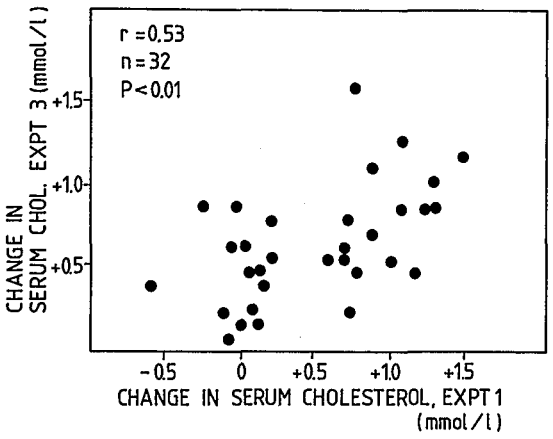

FIG 1. Relationship between the individual responses of serum cholesterol to dietary cholesterol observed in two different experiments. In experiment 1 the volunteers successively consumed 10 and 55 mg of cholesterol/MJ; in experiment 3 these values were 15 and 85 mg/MJ. Reproduced with permission from Beynen et al (6).

We have obtained similar results when the effect of cessation of egg consumption on serum cholesterol was studied twice in the same subjects (7). These subjects habitually consumed at least one egg/day. The linear correlation coefficient between the cholesterolemic responses in the two trials, which were 6 yr apart, was +0.32 ($n = 34$, $p < 0.05$). Thus it appears that at least part of the cholesterolemic response to dietary cholesterol in man is individually determined and stable. It is also clear that one will always find subjects who appear hyperresponsive in one experiment and hyporesponsive in another. This is caused by the diet-independent, within-person variability of serum cholesterol.

In our repeated, controlled trials with 32 subjects (5) we have determined the characteristics of hypo- and hyperresponders. Three response values (one from each trial) were averaged to give each subject's mean responsiveness. There was a positive relationship ($r = 0.41$, $p < 0.05$) between responsiveness and serum high-density lipoprotein$_2$ (HDL_2) cholesterol (8). A negative relation was found with habitual cholesterol consumption ($r = -0.62$, $p < 0.01$) and with body mass index ($r = -0.50$, $p < 0.01$). When Oh and Miller (1) divided their 21 subjects into 8 hyper- and 13 hyporesponders after 4 wk of cholesterol feeding, they found similar associations. Thus, a low habitual cholesterol intake, a high serum HDL_2 cholesterol level, or a low body weight do not make one less susceptible to dietary-cholesterol-induced hypercholesterolemia.

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Reply to letter by Beynen and Katan

Dear Sir:

Individual idiosyncracies in metabolizing cholesterol are well recognized as an overriding feature in the data of numerous cholesterol feeding studies. Variability in individual plasma cholesterol response to dietary cholesterol has been documented in humans (1, 2) and animals (3). Beynen and Katan's letter points out a valid concern regarding the variability in individual response of plasma cholesterol level to dietary cholesterol intake. Variations in plasma cholesterol level within individuals between occasions have been consistently observed.

Our recent study (4) focused not only on the changes in plasma cholesterol levels but also more on the ensuing changes in cholesterol levels of various lipoprotein fractions, especially high density lipoprotein (HDL) to assess the cardiovascular risk as the consequence of excess egg consumption. We observed the ubiquitous individual variation in plasma cholesterol response to changes in dietary cholesterol. Furthermore, the changes in total cholesterol levels of both hypo- and hyperresponders were characterized by the nearly parallel changes in both low density lipoprotein (LDL) and HDL cholesterol without grossly altering the ratio of cholesterol between the two lipoproteins. Citing previous observations (5, 6) on diet-independent random fluctuations in serum cholesterol concentrations in man, Beynen and Katan explicitly suggested that the hyperresponders classified in our study (4) could include some ostensible hyporesponders whose plasma cholesterol level through chance fluctuation happened to be depressed at the beginning of the experiment and subsequently elevated at the end of the high cholesterol diet. Though we at the present time, cannot rule out such possibility of inclusion in either group, it is the lipoprotein cholesterol data which undermine the likeli-