March 29, 1993

TO: Bob Jeffery

FROM: Henry Blackburn

RE: MHHP Manuscript on Obesity

I was pleased to see this. It may be the first major publication of MHHP results to hit the press. Because of its import, I guess in addition to the people who had the ideas and did the analytical work for this study, that you should probably acknowledge the principal investigators by putting their names on the paper. I would be pleased to have my name on the paper perhaps in the tail-end position.

I have only these few running comments in perusing the article:

It is short, sweet and to the point. In the abstract I think you know my strong bias against our using the descriptive term "successful" in regard to our own interventions. It is not only better science but better diplomacy to let other people say that we are successful and to describe our results as results, not successes. In this instance, the use of the word "success" could be even more criticized because it was hardly a resounding success in any terms. I propose the word "influence" rather than "success".

In general, the abstract is complete and succinct.

We have a generic problem in that obesity is the metabolically-determined phenomenon but overweight is the measurement used. I really counsel strongly to use overweight throughout. Obesity is what you like to talk about in your specialty. On the other hand, I think it is more appropriate when we are measuring overweight that we call it that. If you are not happy with that, at least from time to time mention that we are not measuring obesity but overweight, or say "obesity measured as overweight", etc.

In the second paragraph where you discuss the absence of experimental studies, I think it would be highly appropriate to mention that "a specific condition sufficient to cause population obesity" <u>has</u> been well established by the ecologic correlations of the Seven Countries Study showing that "daily energy expenditure of 800 to 1200 activity calories is associated with the absence of obesity defined by skinfold thickness" (or your own interpretation of those correlations). I personally find this

correlation the most dramatic and convincing of all data that habitual population physical activity is a primary determinant of population obesity.

I don't know about you, but I usually use the diagrams on total calories and activity calories from the five year Seven Countries Monograph to illustrate this point, always qualifying that calorie expenditure is well characterized in a population by the precise measurements of calorie intake for whole populations.

As I have indicated on the text, I am not particularly comfortable with the reference to population exposure to "causal pathogens" rather than influences on risk factor levels. Pathogenicity usually means some specific disease mechanism. Because we are talking more about genetic susceptibility than about resistance, I prefer the term "susceptibility of host organisms". But I think we are primarily talking about population susceptibility rather than individual organisms. Your own broad public health concept of obesity clearly includes population susceptibility such as among blacks and Hispanics, which is only partly dependent on genetic susceptibility.
I am very uncomfortable with the concept of increasing host resistance as the aim of a public health intervention with respect to behaviors or even with respect to metabolic phenomena. It is, of course, appropriate in respect to immune phenomena, but I wonder if the model you are using throughout is the most appropriate for behavioral and cultural "resistance and susceptibility". You are quite right that mass immunization is a proto-typical example, but human resistance to obesity and hypercholesterolemia and hypertension, I would suggest, doesn't fit the prototype comfortably.

I guess I am generally uncomfortable with food as a pathogen and with speaking of technology as central to reduced energy expenditure when it is attributable to behavior as well as technology.

You might want to get a disinterested second opinion on my strong reaction against the whole "susceptibility-pathogen exposure concept in regard to culturally determined risk phenomena. I would think that John Potter's opinion would be useful. Moreover, I believe the term "host resistance" almost exclusively refers to individual phenomena not mass phenomena. I think herd resistance or herd susceptibility refers to mass phenomena.

I wonder why the so strong focus on such things as genetically determined individual differences in metabolic rate and efficiency, and individual behaviors, rather than the mass phenomena of overweight that perhaps should be our emphasis in a population-wide strategy to weight reduction and overweight prevention, where both the unit of intervention strategy and the unit of analysis is the community.

I see that you get around finally to "population interventions to influence mass obesity" at the top of page 4. I wonder if you may get us in considerable trouble by talking about "pathogenic foods". Our general policy in behavioral interventions, I thought, and certainly in public health policy statements, is not to call any foods bad, but to use descriptive terms for them. Do you not set us up to have our heads knocked off when

you talk about pathogenic foods? I don't understand the major focus on food supply when it is so much a pattern of food desires, purchases and consumption. Are we not talking about eating patterns?

In the next paragraph, I hope obesity was not "a primary outcome of the Karelia Study". The statement is confusing. It doesn't say whether there was any change in overweight in the population. I would also qualify strongly the statement "favorable changes were seen in diet, serum cholesterol, blood pressure and smoking behavior", based on our knowledge of the limited degrees of freedom in that particular comparison. The same applies to Stanford.

Under the intervention section, I hope that our intervention faculty, principally David Murray, will help guide a reorientation of this presentation which seems to emphasize a high risk strategy over the more general population-wide strategy that was in effect our goal and procedure.

I would have expected to see a more detailed recounting of exposures, at the very least, for the weight loss classes and home correspondence and work site programs, etc. Will not readers be primarily concerned to know how much intervention was delivered for this kind of result? Your descriptions are wonderfully brief, but do they really provide evidence of the individual or the community exposure that this article should provide?

Your paragraph description of the analysis is an excellent summary.

The discussion is superb. Does it ignore what I thought we had come to an understanding about, that habitual physical activity is a (the?) primary <u>population</u> cause of obesity.

Though you put great emphasis on foods and food marketing as a major influence, you say very little about the various cultural tendencies toward moving around less. You may find that average calorie consumption is probably not increasing, whereas body mass index is, so it cannot be that increased consumption of nutrient dense foods is the primary population cause of the phenomenon.

You finally come around to the environmental strategy which I am sure is the proper one. Bravo! I would be happy to end up with a strong social and public health approach. But I would <u>not</u> like to be painted unnecessarily with the brush of social activism and regulation. Might we reconsider some of the wording here so that we are talking about volunteerism among industry, based on good evidence, on persuasion, and redirection of marketing rather than primarily an issue of imposing laws.

I very much like your closing summary paragraph.

Bravo for this excellent effort. My main problem is the apparent, not real, I'm sure, confusion of individual models with population ones, etc.