

## Human chorionic gonadotropin in weight reduction: a reply

Dear Sir:

We appreciate Dr. Bradley's interest in our original communication (1). Since Dr. Bradley realizes "that trials to date do not substantiate our clinical impressions" of the value of human chorionic gonadotropin (HCG) in weight reduction, he should have no difficulty understanding the skepticism of practitioners and academics who have carefully reviewed the available literature on this subject.

Dr. Bradley has presented a teleological argument for HCG having a central role in weight control through regulation of the "adipostat." Evidence indicates that the hypothalamus contains the satiety center in the ventromedial nucleus and an additional center for integrating food-seeking behavior (2, 3). Theories based on laboratory evidence suggest that alpha adrenergic stimulation of neuroreceptors leads to hunger while beta adrenergic stimulation leads to satiety (2, 3). In contrast to Dr. Bradley's argument, we agree with Bray (3) . . . "Although the biological and physiological mechanism leading to the positive caloric balance are often unclear, the fact remains that for a patient to add fat to his body stores he must ingest more calories than he is expending in his daily activities."

Some of Dr. Bradley's references need clarification. His first reference by Edholm et al. (4) is more completely identified below. His second reference is improperly cited—the author and title do not fit the journal, date, and page listed. The cited authors, Sims et al. (5) do have a study comparing the physiological changes of experimental and spontaneous obesity with attention to hormonal changes. In the cited journal there is a study by Miller et al. (6) which examines thermogenesis in overeating. Dr. Bradley cited experimental evidence (7, 8) to support a metabolic role for HCG in weight reduction. The study of Melichar et al. (7) demonstrated effects of HCG on free fatty acids and glucose concentrations in newborns but failed to demonstrate any similar changes in the adults tested. Surmaczynska et al. (8) concluded that, "Existing information concerning the effects of

HCG on intermediary metabolism is insufficient to translate the HCG changes into metabolically meaningful terms."

Dr. Bradley has criticized five aspects of our study: "group spirit," analysis of hunger responses, analysis of differences in hip and waist measurements, lack of long-term follow-up, and the first 3 days of diet. These will be addressed respectively.

By randomly assigning volunteers to each treatment group, our experimental design neutralized factors that could influence differences in weight loss between the study groups. The effect of "group spirit" was reduced in part by this randomization and in part by the staggering of daily appointments. Since our clinical investigation was a double-blind study, both the placebo and HCG subgroups had opportunity for exposure to any overriding effects of peer pressure and reinforcement (if these were factors). We doubt that any leveling effect took place in view of the favorable comparison of our HCG group and the HCG treatment group reported in the Asher and Harper study (9). As we noted in our analysis of the two studies (1), we suspect that the apparent differences between the two placebo groups result from uncontrolled variables in the Asher and Harper study which lead to a "disadvantaged" placebo control.

We would agree with Dr. Bradley that there are certain difficulties in assessing food deprivation responses in obese patients. Using the basic design of Asher and Harper (9) we attempted to provide a better quantitative analysis by using qualifying terms for hunger (see *Parameters measured* (1)). We consider degrees of hunger a more useful measurement tool than simple recording of symptoms. The former is readily amenable to nonparametric statistical analysis.

Dr. Bradley says he has the impression that "patients who continuously break the diet and whose weights do not decrease by much that they lose inches off their waistline while receiving injections of HCG." He suggests that we should have compared measurements of patients in both treatment



groups who lost very little weight or many pounds. This challenge we accepted and have examined the weight-loss by separating the HCG and placebo patients into groups (low, < 13 pounds, n = 10; high, > 19 pounds, n = 11). The data were evaluated by *t* test and Mann-Whitney U test. All subgroups in both treatment categories lost inches from their waistline and hip measurements, but there was no statistically significant differences between comparable measurement changes in the HCG and placebo treatment groups.

The purpose of our study may be re-read by Dr. Bradley in our original communication (1). Long-term follow-up was not a part of the study (9) we were attempting to duplicate.

Finally, Dr. Bradley has criticized our study for ignoring one of Simeons' most crucial rules, the importance of overeating during the first 3 days of injections. Since Dr. Bradley has noted that Asher and Harper (9) observed this rule and all other rules, he must have misinterpreted our statement that, "The diet was identical to that used in the protocol of Asher and Harper" (1). This diet described in Asher and Harper's article (9) includes the provisions for overeating during the first 3 days of injections of HCG. We believe that we followed all of Simeons' rules as carefully as Asher and Harper had.

As physicians treating obese patients (M. S., R. J., and J. D.) we are not satisfied to accept "commercial success" and practitioners "impressions" as the criteria for efficacy of a product injected in patients! Therefore, we would suggest to Dr. Bradley that, in view of the equal success in weight reduction between our placebo and treatment groups and the complications noted in our study and in other reports of HCG use (10-15), he should either initiate his own double-blind study or use a placebo instead of HCG.

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## References

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