used various techniques to help control for some of the more obvious flaws. There are relatively few areas in medicine where practice is guided by absolute proof.

We agree with Dr. DeFazio that for many obese persons who might not eat more than their thinner counterparts, to maintain a normal weight requires a lifelong commitment and struggle against food, a struggle not required of those who are of average weight. We recommend that physicians be honest with their fat patients about the unceasingly difficult nature of this struggle and tell them that strict discipline with regard to extensive eating restrictions and daily exercise is required without letup for the rest of their lives. As many of us know there are few who can maintain this level of commitment for a lifetime, but there are many persons, fat and thin alike, who show extraordinary levels of commitment and discipline in this area. It would certainly help to have the resources to hire personal trainers and cooks specializing in low-fat foods, as some celebrities such as Oprah Winfrey have done, to help us in this formidable struggle. Studies of the persons who are able to lose and keep weight off permanently would be useful and instructive, as suggested by Dr. DeFazio.

Dr. DeFazio rightly questions the evidence available that doing a "brief dietary and exercise history" is beneficial for the patient, given that physician intervention has not been proved to affect obesity. At least the history can give the physician specific information about that particular patient, which would enable the physician to make recommendations based on some knowledge of the patient's eating and exercise history, rather than some general, and we would argue dangerously unfounded, belief that all fat persons eat too much. It is hard to see how such an intervention can be harmful. Our admonition about the dangers of dieting is backed up by serious concerns, not only about weight cycling, which as Dr. DeFazio correctly points out is still being investigated, but also the dangers of triggering eating disorders and failure experiences, exposing patients to professionals who hold them in low regard, causing them to see themselves as deviant, flawed, and inadequate, and diverting their attention away from other problems or achievements.

Dr. Bett argues that she knows that "fat persons do indeed eat much more than lean persons" and notes that "in fact, most obese persons compulsively overeat in secret." She defines obesity as "compulsive seeking and continuing intake of certain foods despite increasing evidence of adverse effects." She then goes on to discuss the various techniques that compulsive overeaters use to conceal actual food intake and the psychological motivations and effects of such eating on the overeater.

We do not argue with Dr. Bett's description of compulsive overeating or that this disorder exists. We believe that this disorder could in fact be triggered by rigid dieting that the dieter is unable to maintain, which sets up the wild swings from diet-purge to binge and back again. Further, we do not believe that all, as Dr. Bett seems to imply, or even most fat persons are compulsive overeaters. In fact, as we discussed earlier, the preponderance of research on this subject suggests just the opposite. We are quite disturbed by her definition of obesity, which is characteristic of exactly the type of thinking about obesity we are challenging. We believe that genetics does play an important role in contributing to obesity as indicated by several references in our bibliography.

Dr. Bett cites her "extensive personal and professional experience" to back up her ensuing claims. In addition to our extensive review of the research literature, Dr. Robinson, the first author, has extensive clinical experience with the eating diaries of approximately 100 fat patients during the course of 12 years of work with this population. Her clinical experience confirms the research literature cited, i.e., most were eating average calorie amounts, i.e., approximately 2000 calories or less and maintaining above-average amounts of weight.

I certainly agree with Dr. Bett's contention that "any sedentary society such as our own that encourages the consumption of large amounts of low-cost, easily available, highly palatable, calorie-dense foods will inevitably have a large obese population." We are not arguing that food consumption has nothing to do with body weight, only that persons can eat similar amounts and types of food and have widely differing body weights. Certainly those whose bodies were very efficient with food would have survival value, because most of human history is one of food deprivation, not plentitude. These efficient metabolisms would be among the first to become obese in a society, like ours, that enjoys an abundance of food and freedom from starvation. In the past, this efficiency was adaptive; today it could be associated with health problems.

In sum, we want to stress the major message we were trying to get across to physicians working with their obese patients: be respectful of the emotional pain borne by many obese persons who live in a culture that stigmatizes them. Try to approach them respectfully and individually, and try to avoid some of the degrading techniques used in the past that just prevent fat patients from feeling comfortable and respected when they come for help and might prevent them from seeking the help they need.

Bean Robinson, PhD
Minneapolis, MN

Rapid Antigen Detection Testing

To the Editor: The article by Joslyn, et al.[1] provides useful information guiding the care of patients with suspected streptococcal pharyngitis. The authors make some misstatements, however, and draw some incorrect and incomplete conclusions in discussing their findings.

They state: "Results of these pilot studies indicate that an extremely low percentage (<1 percent) of subjects with GABHS [group A β-hemolytic streptococcus] escaped detection with our rapid screening test methods." In fact, based upon their published data, 4.55 percent (1 of 22) of patients in their sample who actually had GABHS tested negative by the screening

test. It is true that less than 1 percent of the patients who screened negative actually had GABHS. The confusion lies in that "false-negative rates" can refer to one of two distinct proportions. The number of patients who actually had GABHS and screened negative are known as the false negatives,[2] which can be expressed as the proportion of all of the individuals who had negative tests (the value reported by Joslyn, et al.) or as a proportion of all of the individuals who actually had the disease.

Also the authors should have reported confidence intervals around relevant results, such as sensitivity, specificity, positive predictive value, and negative predictive value of the screening test.[3] For example, using the binomial distribution[3] with a sample size of 22 and a probability of 0.95, the 95 percent confidence interval around the sensitivity would extend from 77 percent to 100 percent. Thus, these results could be completely consistent with a true sensitivity of 77 percent for their rapid screening test, a value similar to the lower sensitivities reported in previous studies noted by the authors.

Finally, the authors claim that "relatively low prevalence . . . would make case detection more difficult" and that because of low prevalence, estimates of sensitivity, specificity, positive predictive value, and negative predictive value "would be conservative estimates of actual values." Unless there is some characteristic of the disease that varies with prevalence and makes an individual more or less likely to have a positive test result, sensitivity (case detection) does not change. Positive and negative predictive values of a test do change with prevalence, but in opposite directions. Hence, given greater prevalence of GABHS in the population, positive predictive value of the test would be expected to rise, and negative predictive value to fall.[4]

In summary, Joslyn, et al. report useful information that does have clinical application but requires appropriate epidemiologic interpretation.

Sean Schafer, MD
Fresno, CA

References

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3. Dawson-Saunders B, Trapp RG. Basic and clinical biostatistics. Norwalk, CT: Appleton & Lange, 1994:73-5, 144-5.
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The above letter was referred to the authors of the article in question, who offer the following reply:

To the Editor: We acknowledge the comments by Dr. Schafer and appreciate the opportunity to reply.

Dr. Schafer's first comment concerns the expression of false-negative rates, which we described as the proportion of all patients screening negative who were actually positive for group A β-hemolytic streptococcal pharyngitis (GABHS). Dr. Schafer makes reference to Last,[1] who defines false negative as a "negative test result in a subject who possesses the attribute for which the test is conducted." This definition does not include recommendations for a denominator to express a false-negative rate. Inasmuch as we provided actual numbers used in our calculations, the reader can use either the total number screened or total with disease as the denominator to interpret the results. The false-negative value reported was not meant to be misleading but was a reflection of our opinion that a false-negative result has more serious consequences than a false-positive one. Because the predictive value negative (PV-) is the probability that a person with a negative screening test does not have the disease,[1] we believed that reporting the false-negative rate as (1 − [PV-]) (out of all patients testing negative, those who actually had disease) gave an accurate indication of how well the screening test performed.

Dr. Schafer gave recommendations for reporting confidence intervals around our reported values of sensitivity, specificity, predictive value positive, and predictive value negative. Confidence intervals are used to construct a range of values around a sample statistic, so that the range has a specific probability of including the true value of the variable.[1] Compared with using a sample, however, when an entire population is measured, confidence intervals are not necessary. The true values of the variables are known, not estimated.[2] In our study we tested all patients in the population of interest in a specific period, not a sample of the population. The reported values of sensitivity, specificity, and predictive values positive and negative were actual parameters of our population. This reporting convention is common in screening studies utilizing entire patient populations.

Finally, Dr. Schafer questions our claim of conservative estimates of sensitivity, specificity, and predictive values positive and negative during times of low GABHS prevalence. According to Hennekens and Buring,[3] "No matter how specific the [screening] test, if the population is at low risk of having the disease, results that are positive will mostly be false positive." If prevalence increases, proportions of true positive and true negative subjects will also increase relative to the number of false positives. This will result in improved values of sensitivity, specificity, and predictive values positive and negative, which would support our claim of conservative estimates during seasons of low prevalence of GABHS (spring through fall). We appreciate the opportunity to justify our methods and conclusions.

Sue A. Joslyn, PhD
University of Northern Iowa
Gregory L. Hoekstra, DO
John E. Sutherland, MD
University of Iowa College of Medicine
Waterloo, IA