



Two Mice with a Defect in a Gene Called Obese (*ob*).

The defect usually results in a marked increase in the amount of body fat. Administration of leptin, the protein encoded by the *ob* gene, has reduced the body weight of *ob* mice. After 4½ weeks, the mouse on the left, which did not receive leptin, weighed 67 g; the mouse on the right, which received daily injections of leptin, weighed 35 g.

but must be applied to our understanding of the physiology of energy homeostasis and the biology of obesity, and then to the treatment of obesity and its coexisting conditions.

Obesity reviews the history, evolution, and future directions of obesity research. The editors have divided the book into 11 sections, but the chapters can also be divided into three basic categories. The first category includes established fields, such as the classic family and twin studies that clearly demonstrated the existence of a genetic component to obesity. These chapters contain excellent discussions of the reported data as well as some new summaries. The chapters on the monogenic causes of obesity in mice and humans provide comprehensive discussions of our current understanding of the biology of obesity. The material covered by these chapters is unlikely to change rapidly, making these sections a useful resource for years to come.

The chapters in the second category summarize linkage and candidate-gene studies and provide a good overview of current knowledge in these areas. The summaries are timely and valuable, but the information is likely to be modified in the near future, particularly as replication studies in independent cohorts are emphasized. The findings from candidate-gene studies will also need to be integrated with results from genomewide association studies.

The chapters in the third category concern topics that have not yet been definitively tied to

obesity in a mechanistic way (e.g., epigenetics). The inclusion of these chapters is understandable given the potential importance of these areas. However, with the current absence of data on how these topics apply to obesity, recent journal reviews may provide the most up-to-date reading.

One minor issue with the book is its subtitle. At this time, the term “postgenomics” means different things to different researchers, and one might look to current journals for the latest approach to the integration of systems biology into obesity genetics and genomics. Overall, this is a timely publication that will serve as a useful reference in the broad and growing field of obesity research.

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CORRECTION

Gestational Diabetes — Setting Limits, Exploring Treatments (May 8, 2008;358:2061-3). In the paragraph that begins “Also in this issue of the Journal...” (page 2062), the final sentence should have read, “Rates of cesarean delivery and birth weights were similar in the two groups.”

NOTICES

Notices submitted for publication should contain a mailing address and telephone number of a contact person or department. We regret that we are unable to publish all notices received. Notices also appear on the Journal's Web site (www.nejm.org/meetings). The listings can be viewed in their entirety or searched by location, month, or key word.

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