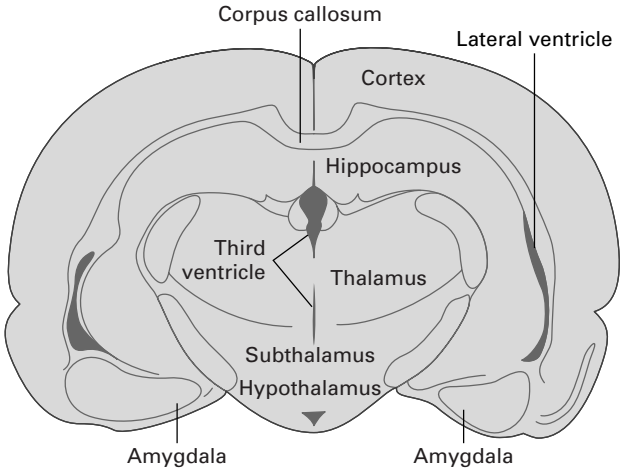


FIGURE 11.1 • Cross-sectional view of a rat brain. The hypothalamus is located near the bottom of the brain, in about the middle from front to back.

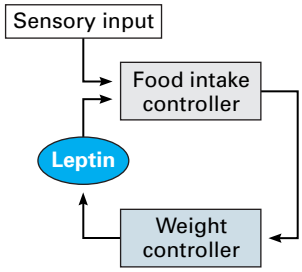


FIGURE 11.2 • Your initial model for weight control.
Sensory input includes both environmental and physical stimuli.

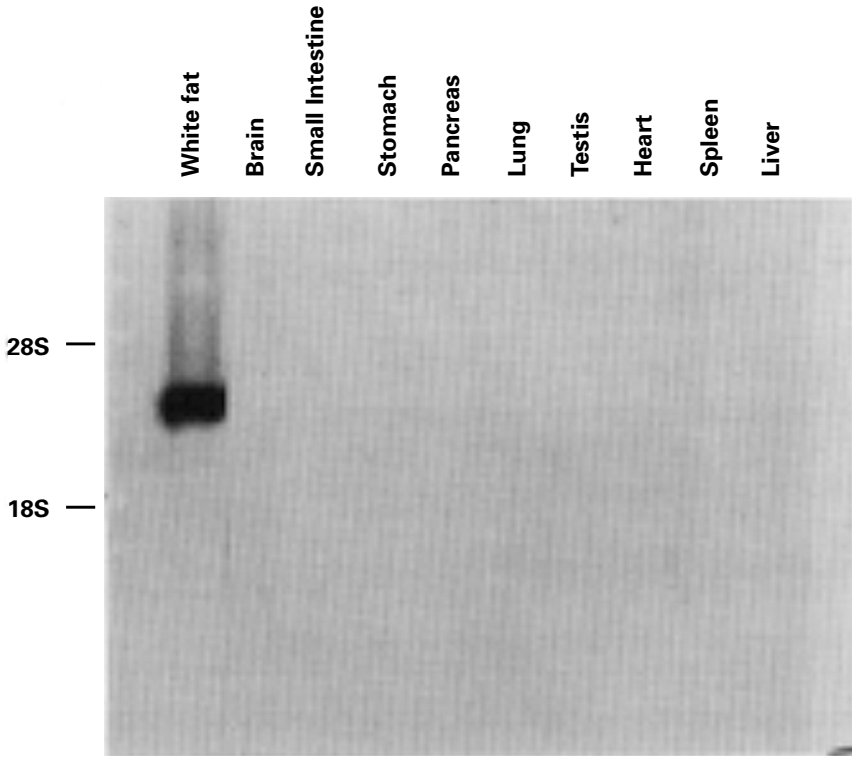


FIGURE 11.3 • Northern blot using 2G7 DNA as probe. RNA was isolated from *wt* mouse tissues as indicated. 28S and 18S are ribosomal RNA used as molecular weight markers.

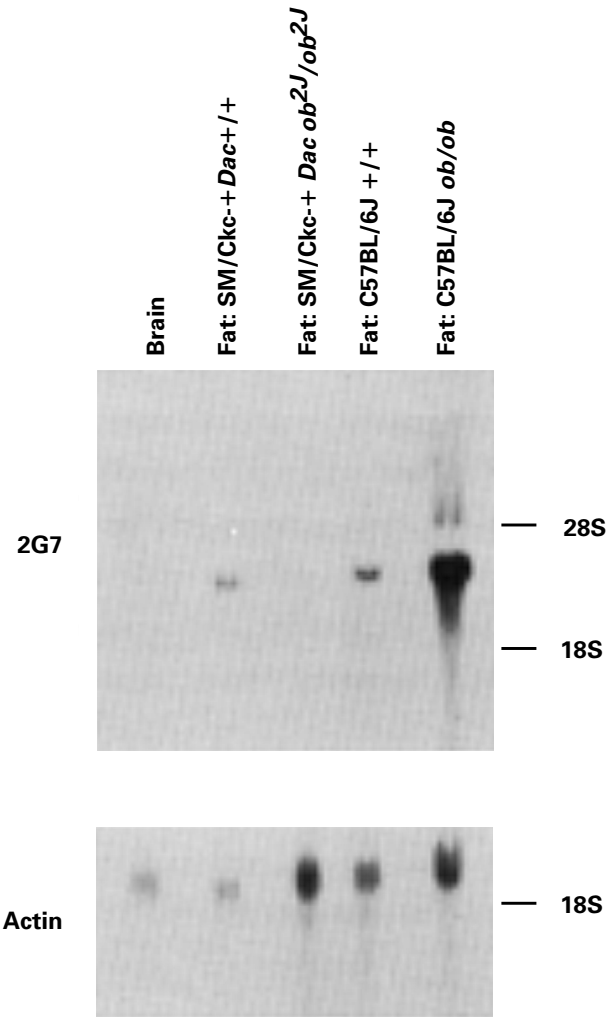


FIGURE 11.4 • Northern blot of fat RNA from various strains of mice. The probes were either 2G7 (see Figure 11.3) or actin cDNA. 28S and 18S are ribosomal RNA bands used as molecular weights. RNA was extracted from fat or brain as indicated. The source of the brain mRNA was a *wt* mouse.

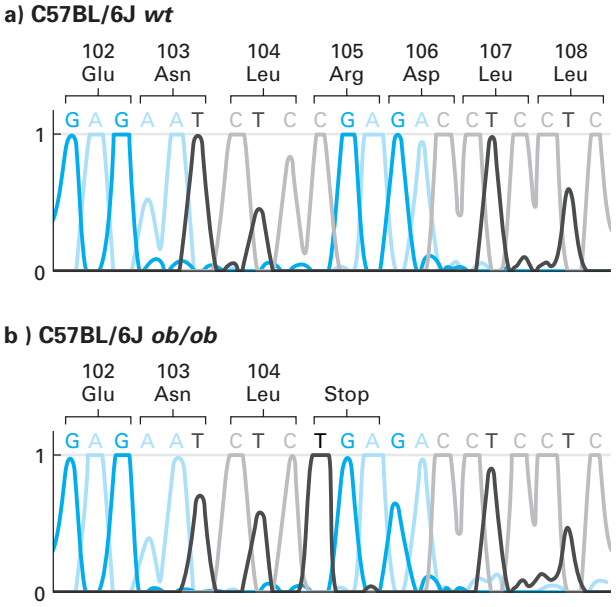


FIGURE 11.5 • Genetic cause for obese phenotype. Sequencing chromatograms of **a)** *wt* C57BL/6J and **b)** *ob/ob* C57BL/6J mouse leptin coding regions.

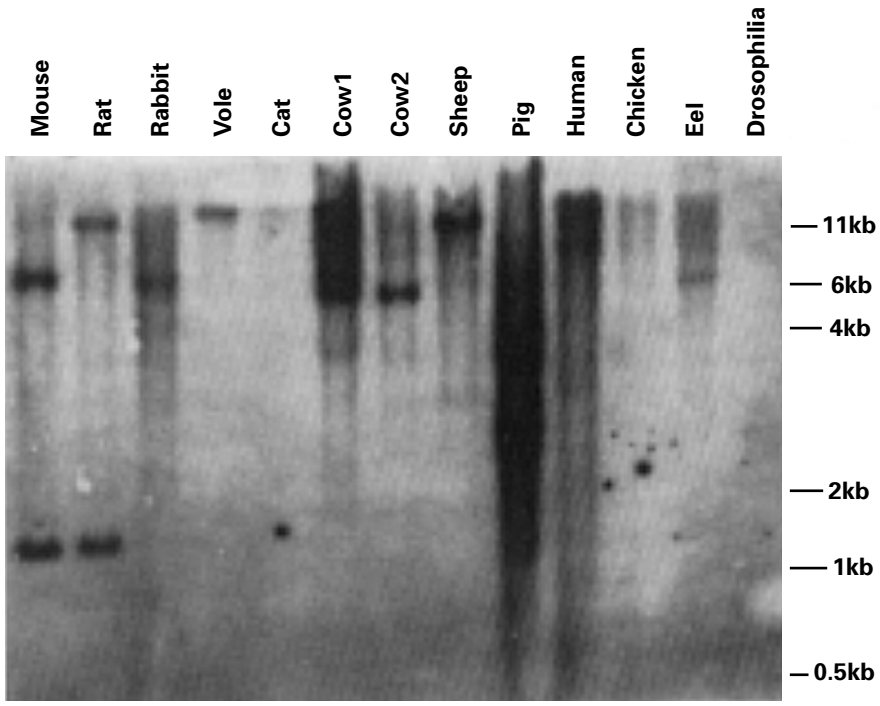


FIGURE 11.6 • Conservation of leptin gene in animals. Southern blot using genomic DNA from a wide range of species, as indicated. The probe was made from *ob* cDNA.

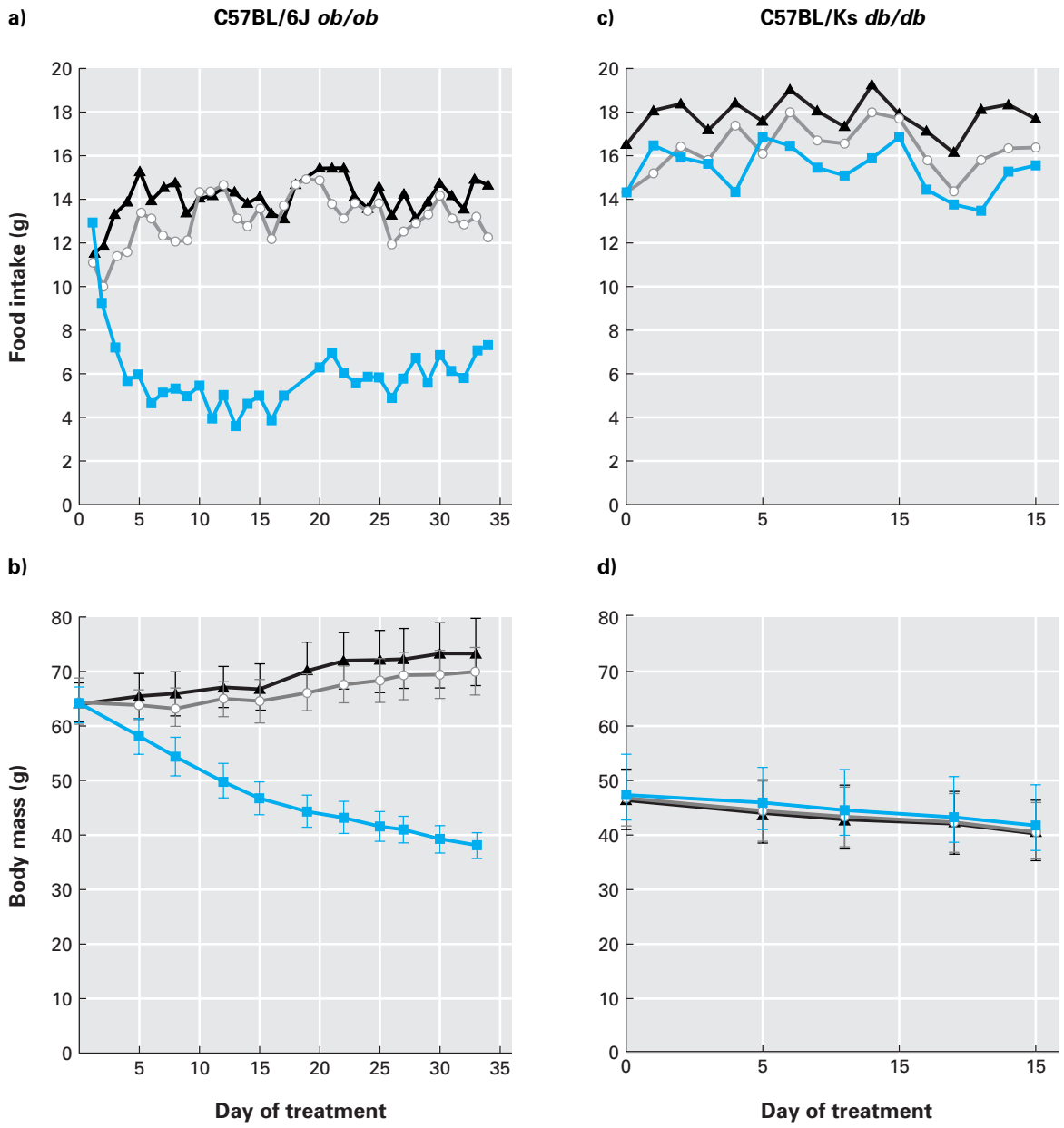


FIGURE 11.7 • Effect of leptin on *ob* and *db* mice. **a)** and **b)** Ten homozygous *ob* mice, and **c)** and **d)** 10 homozygous *db* mice received daily intraperitoneal injections of 2G7-encoded protein (filled squares) or saline (open circles), or no injections (filled triangles).

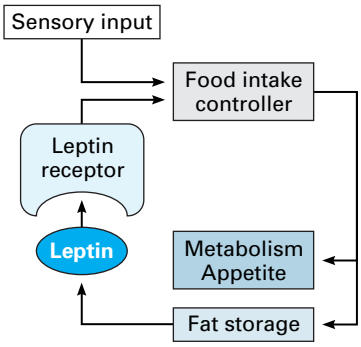


FIGURE 11.8 • Revised model for weight control. Notice that the leptin receptor has been added. In addition, the controller now produces two separate signals: one for metabolism/appetite and the other for weight gained through fat production.

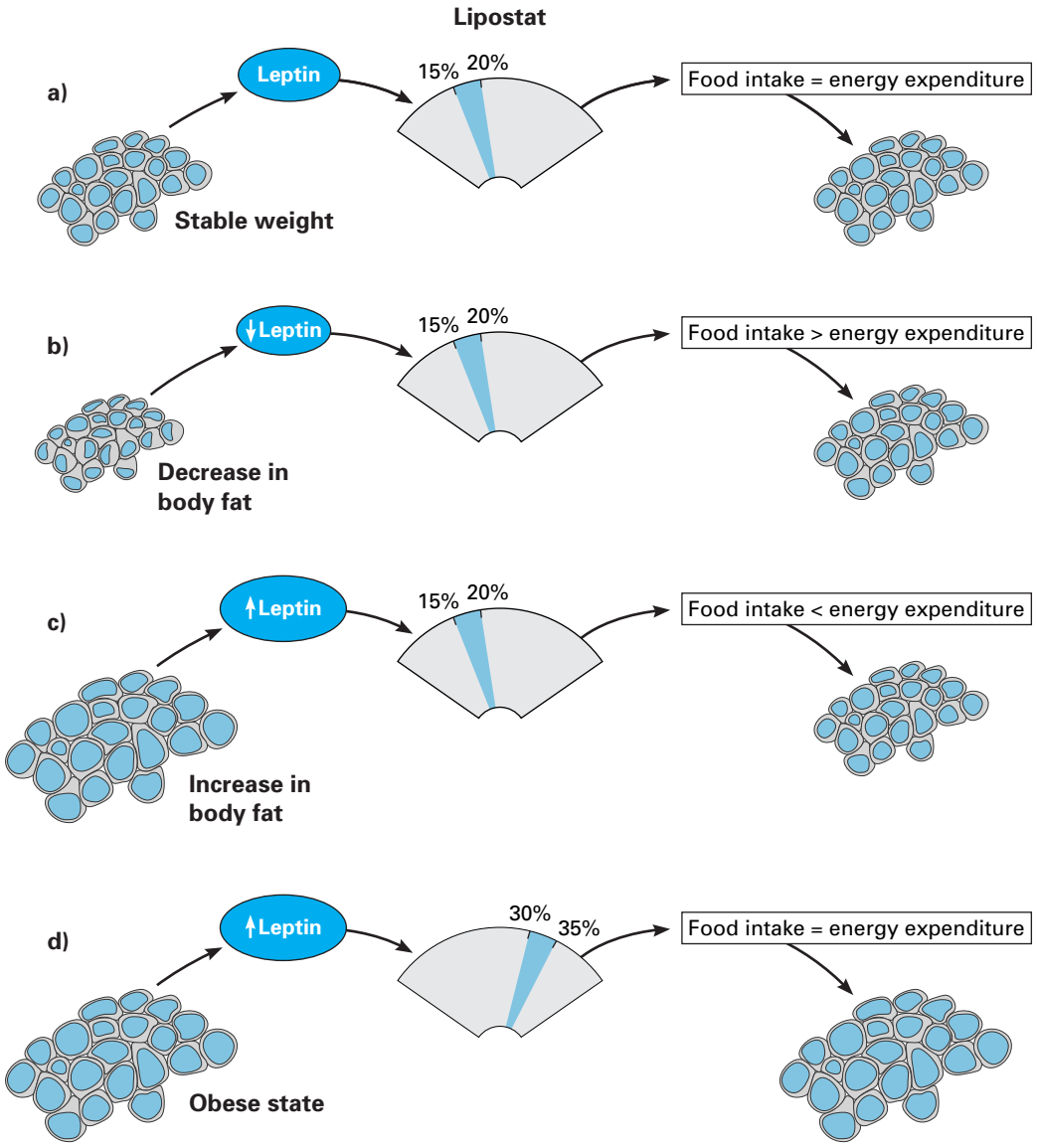


FIGURE 11.9 • Lipostat controls body fat content. **a)** Leptin affects the lipostat (scale) in a feedback loop regulating fat mass. At an individual's stable weight (shown as 15–20% fat for a nonobese subject), the amount of circulating leptin elicits a state in which food intake equals energy expenditure. Lipostats respond **b)** to a loss in body fat, and **c)** an increase of fat. **d)** A lipostat in an obese person has a higher set point for body fat (30–35% fat).

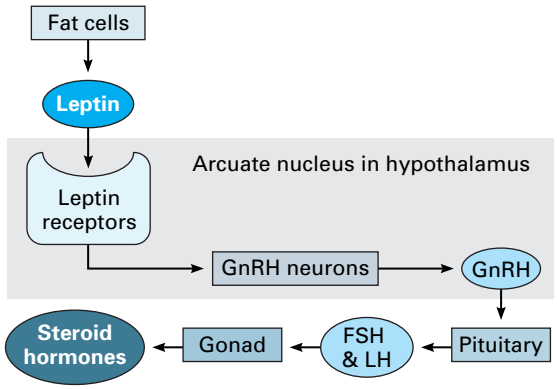
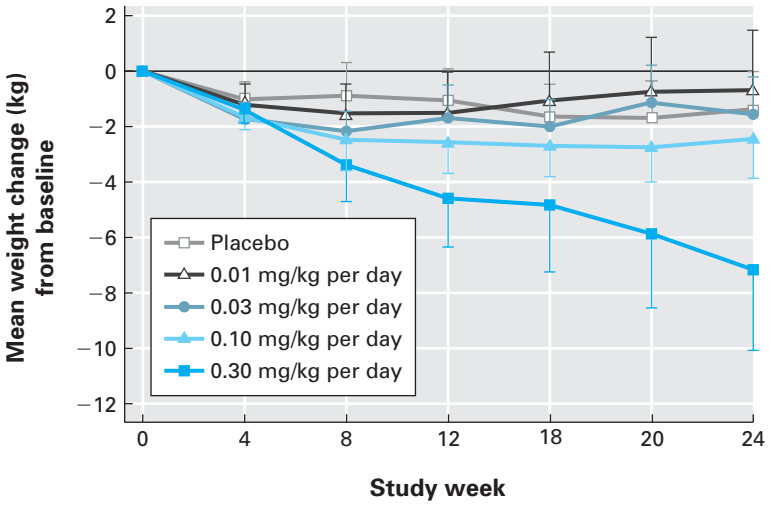


FIGURE 11.10 • Circuit diagram illustrating the role fat accumulation plays in sexual maturity. Abbreviations: GnRH is gonadtrophin-releasing hormone; FSH is follicle-stimulating hormone; and LH is leuteinizing hormone.

FIGURE 11.11 • Pattern of weight change over 24 weeks in obese people who received recombinant human leptin. The amount of leptin given to people depended on their weight as indicated in the figure's key. The number of people was not constant over the course of the study. Error bars indicate s.e.m.; thin horizontal line indicates no change.



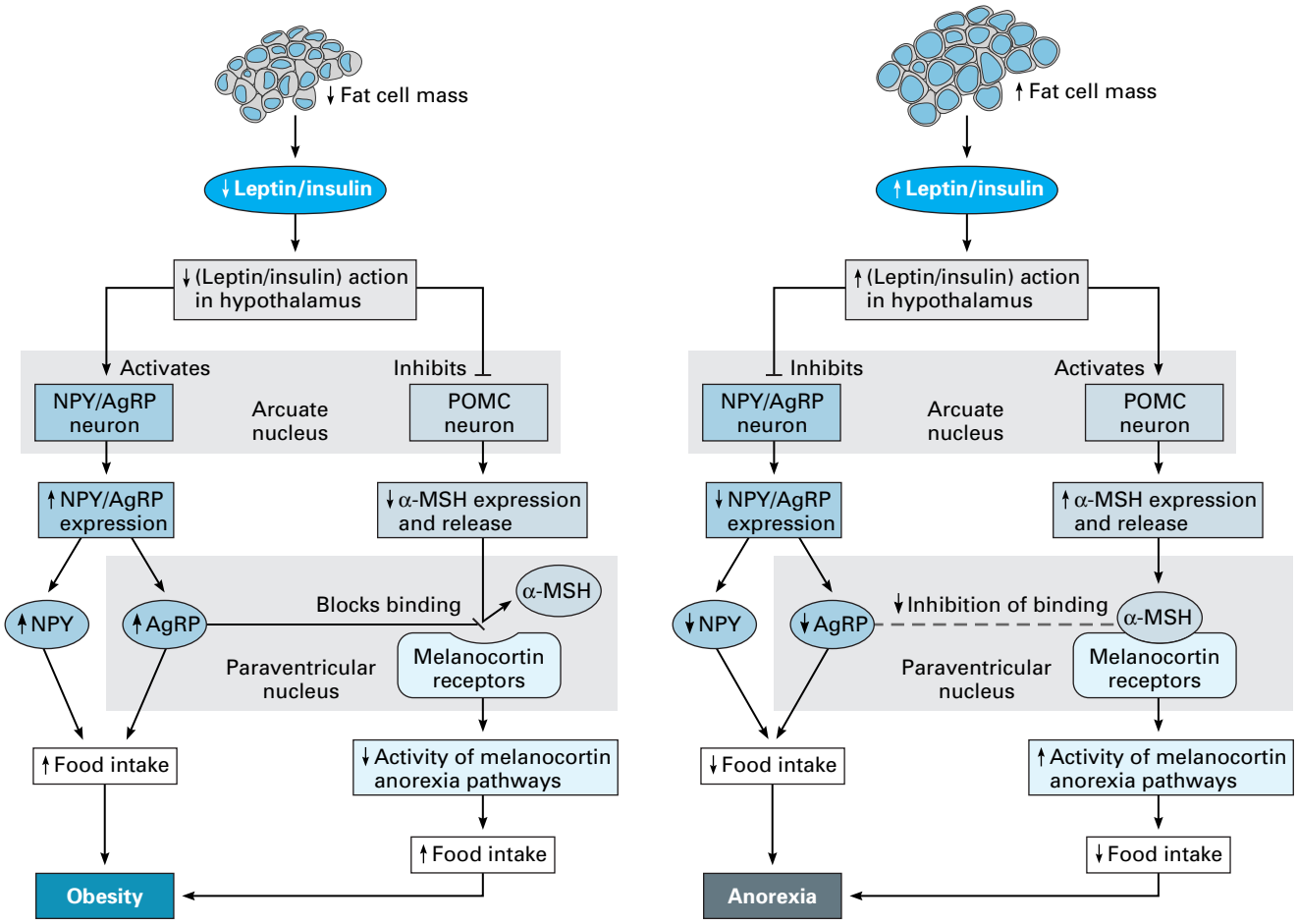


FIGURE 11.12 • Lipostat response to rapid changes in body fat. a) When fat is lost through surgery or "fad" diet, the body responds by producing less leptin. The outcome of reduced fat is increased food intake and fat storage. **b)** When you eat too much at a holiday meal, you accumulate body fat, which produces more leptin. The result of increased fat storage is reduced food intake, technically called "anorexia" but not to be confused with the pathological condition anorexia nervosa. Eventually, the body fat will be reduced to its preholiday level.