

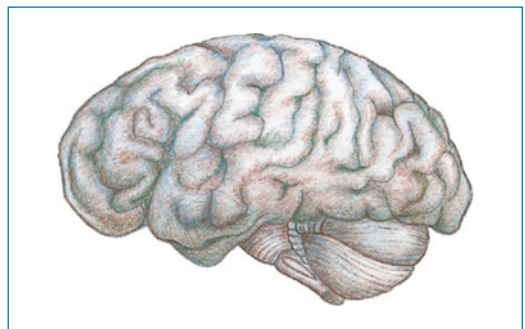
# 14 Control of energy metabolism – the reward system

Most people define nutrition in rather archaic terms, using the verbs to eat, drink and enjoy. Catch phrases like dining culture, culinary arts, delicacies, sumptuous spreads and feasting all give testimony to the high value our society places on people giving into their urge to eat. In fact, eating well contributes to life's enjoyment and when partaken of in moderation, can improve the state of our health merely by uplifting our psyche.

That said, our eating behavior is also strongly influenced by the people close to us. According to a long-term evaluation as part of the Framingham Heart Study, which repeatedly assessed a densely interconnected social network of 12,067 people from 1971 to 2003, there was a 57 % probability that a person would become overweight if their boyfriend or girlfriend had become overweight during the same period. The same probability was 40 % for siblings and 37 % for married couples. These effects did not transfer to other people in their immediate neighborhood. Genome analyses have also shown that very good friends exhibit hemophilic genotypes (Christakis et Fowler 2007, 2014).

Responsibility for the psychological effects of dietary intake has now been primarily attributed to the endogenous cannabinoids, discovered to modulate the feedback loops involved in hypothalamic appetite regulation via the specific endocannabinoid receptor CB1. The **endocannabinoids** are part of a reward system in the brain, which explains

their secretion after a well-tasting meal is consumed or food intake occurs after a period of fasting. Under normal conditions, this process is designed to maintain an energy balance. However, frequent excessive food intake leads to long-term overregulation of the endocannabinoid system, the consequence being an incessant craving for food and the consumption of increasingly large quantities thereof. This phenomenon is accompanied by a simultaneous further increase in endogenous cannabinoid levels (► Chapter 35). The administration of the anti-obesity drug rimonabant (tradename Acomplia) has been shown to break this vicious circle by medically blocking the CB1 receptor. However, the drug was taken off the European market in autumn of 2008 because of its strong psychological side effects.



■ Fig. 14.1