



**Figure 4.11.** Diauxic growth curve for *E. coli* on glucose and lactose.

levels are still high, which results in higher levels of ATP and low levels of AMP and cAMP. Consequently, little cAMP–CAP complex is formed, and the interaction of the lac promoter with RNA polymerase is weak in the absence of cAMP–CAP. The rate of  $\beta$ -galactosidase formation would be slightly increased from the basal level—perhaps 5% of the maximal rate.

At 7 h the glucose has been fully consumed. The cell cannot generate energy, and the level of ATP decreases and cAMP increases. The cAMP–CAP complex level is high, which increases the efficiency of binding RNA polymerase to the lac promoter. This increased binding leads to increased transcription and translation. The rate of  $\beta$ -galactosidase formation is maximal and much higher than the basal rate or the 2 h rate. However, the cells have not yet accumulated sufficient intracellular concentrations of  $\beta$ -galactosidase and lac permease to allow efficient use of lactose and rapid growth.

At 10 h the intracellular content of proteins made from the lac operon is sufficiently high to allow maximal growth on lactose. However, this growth rate on lactose is slower than on glucose, since lactose utilization generates energy less efficiently. Consequently, the cAMP level remains higher than when the cell was growing on glucose. The level of production of  $\beta$ -galactosidase is thus higher than the basal or 2 h level.

Irrespective of whether an enzyme is made from a regulated or constitutive gene, its activity in the cell is regulated. Let us now consider control at the enzymatic level.