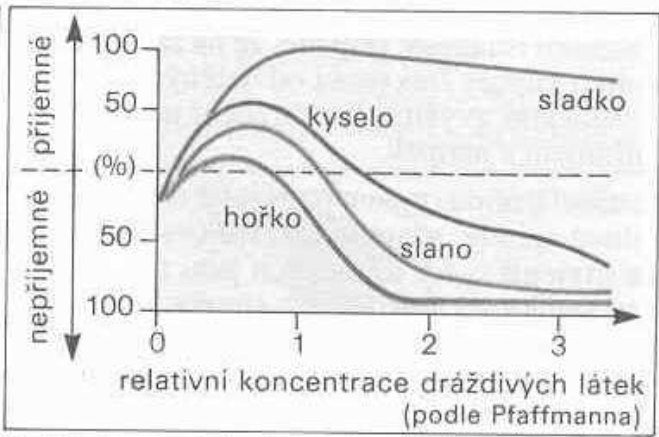


E. Chuťový pohárek (podle Andrese)



F. Hodnocení chuťových podnětů

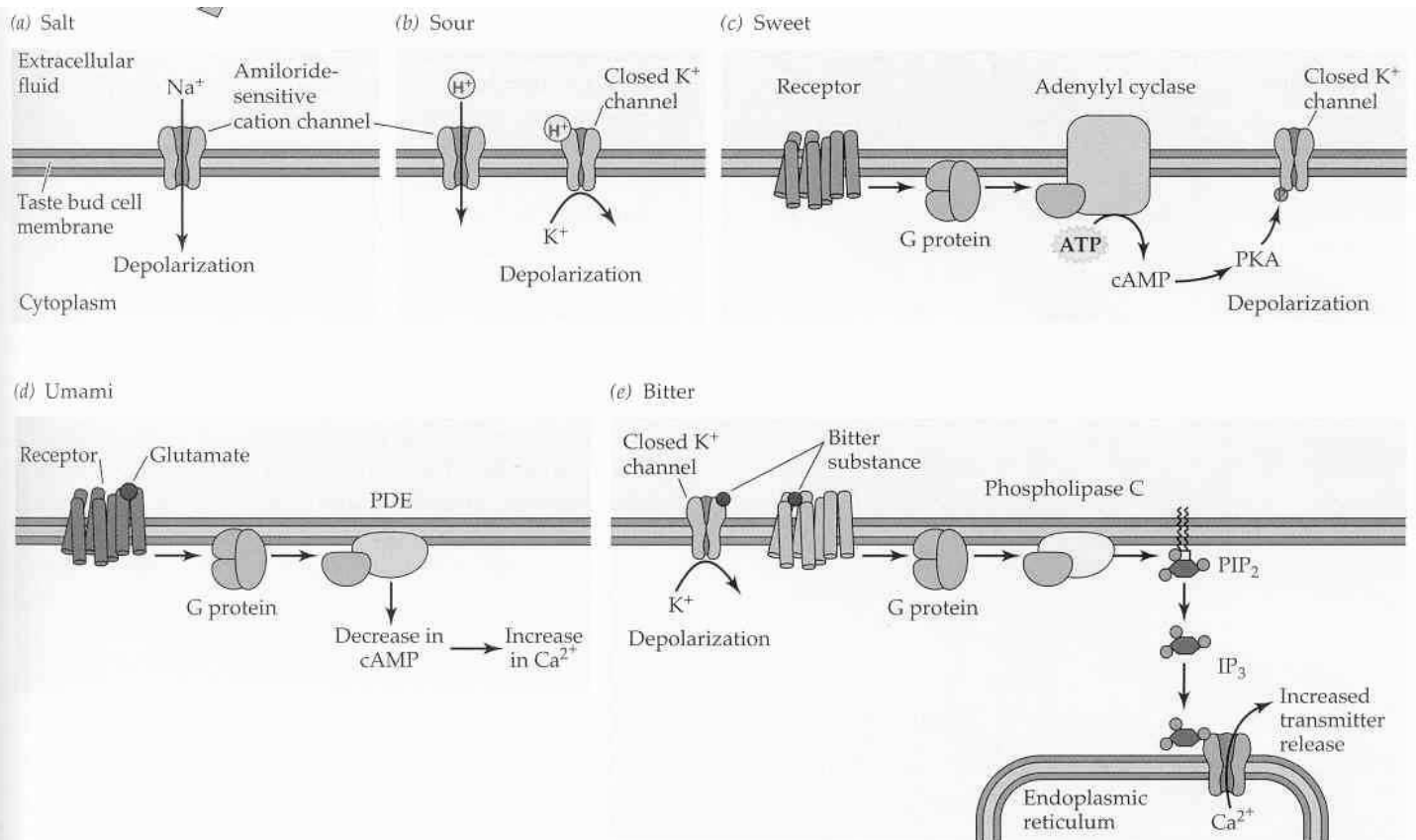
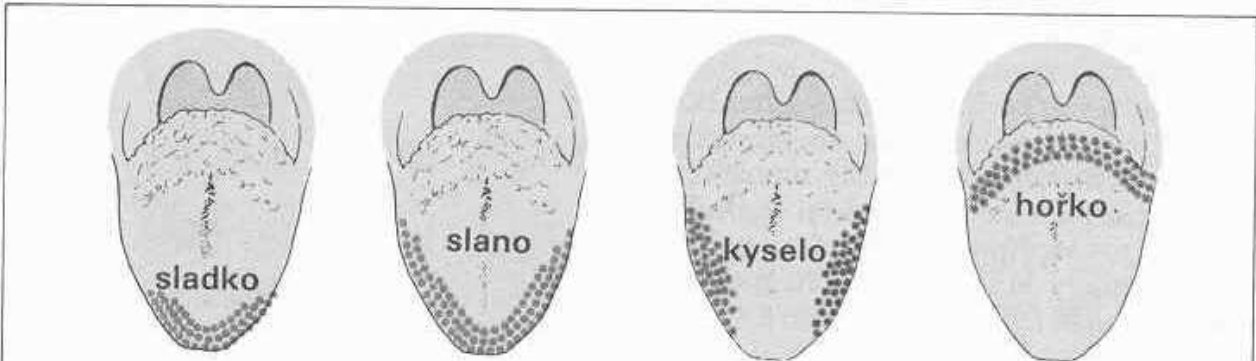


Figure 13.34 Taste-transduction mechanisms differ for different taste qualities. All transduction mechanisms except the  $\text{IP}_3$  action in (e) lead to *depolarization*, which spreads to the basal end of the cell and opens voltage-gated  $\text{Ca}^{2+}$  channels to allow  $\text{Ca}^{2+}$  entry and transmitter release. (a) For salt taste, sodium ions enter a taste bud cell through amiloride-sensitive cation channels, directly depolarizing the cell. (b) In sour taste, either  $\text{H}^+$  ions enter the cell through amiloride-sensitive cation channels, or they close  $\text{K}^+$  channels to produce depolarization. (c) Sweet taste is most commonly mediated by the binding of sugars to a G protein-coupled receptor, which acts via a G protein to activate adenylyl cyclase and produce cyclic AMP. Cyclic AMP then activates protein kinase A (PKA) to close a  $\text{K}^+$  channel (by phosphorylating

it), producing depolarization. (d) The amino acid glutamate (monosodium glutamate, MSG) stimulates the taste quality umami (a savory or meaty quality). Glutamate binds to a G protein-coupled receptor (related to synaptic metabotropic glutamate receptors) to activate a phosphodiesterase (PDE) and decrease the concentration of cAMP. The decrease in cAMP leads to an increase in intracellular  $\text{Ca}^{2+}$  concentration. (e) Bitter taste mechanisms can involve a G protein-coupled receptor for bitter substances that acts via a G protein and phospholipase C to produce  $\text{IP}_3$ .  $\text{IP}_3$  liberates  $\text{Ca}^{2+}$  ions from intracellular stores, eliciting transmitter release without requiring depolarization. Other bitter substances bind to  $\text{K}^+$  channels and close them to depolarize the cell.

