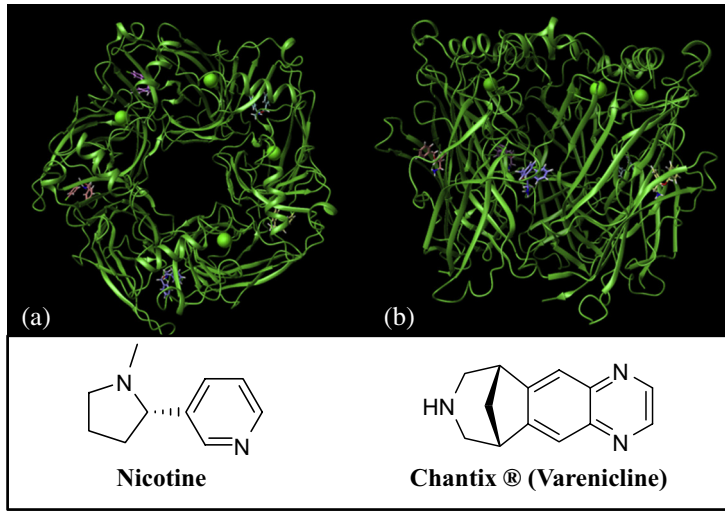

FIGURE 3.32 The acetylcholine-binding protein (AChBP) has been employed as a model system for the nicotinic acetylcholine receptor (nAChR). The crystal structure shows a ligand bound to the acetylcholine-binding sites. (a) Top view. (b) Side view. Nicotine and Chantix® (Varenicline) both bind to nAChR, but differential receptor responses provide an opportunity for therapeutic intervention in nicotine addiction. *RCSB 2XNT.*

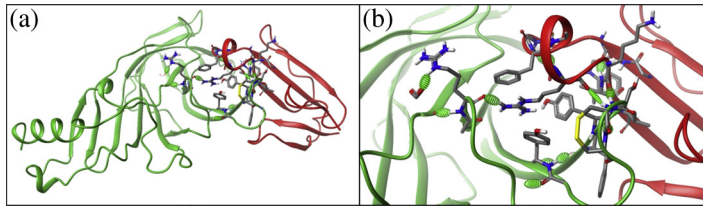

FIGURE 3.33 (a) Crystal structure of the extracellular domain of the nicotinic acetylcholine receptor 1 subunit (green) bound to α-bungarotoxin (red), from the venom of the snake Bungarus multicinctus, at 1.9 Å resolution. (b) Close-up of the binding-site interactions. *RCSB 2QC1.*

channel, but ion flow is prevented and the associated cellular response does not occur.

Allosteric activation of ligand-gated ion channels is also possible. The γ-aminobutyric acid type A receptor ($GABA_AR$), for example, is a ligand-gated chloride channel that plays a critical role in the central nervous system. Activation of $GABA_AR$ by the endogenous ligand γ-aminobutyric acid (GABA, Figure 3.34(a)), an inhibitory neurotransmitter, opens the chloride channel of $GABA_AR$, which leads to hyperpolarization of neurons, inhibiting neurotransmission.[70] The presence of benzodiazepines