1) Describe the labeled line hypothesis of sensory perception.

Any particular neuron(s) responds maximally to a specific type of stimulus. This neuronal sensitivity to a particular type of stimuli is determined by the nature of the neurons that it is synaptically connected to, and ultimately to the sensory receptor neuron or other type of sensory cell that it is connected to. The labeled line hypothesis holds that cells in the CNS respond maximally to a particular type of stimulus originating in the outside environment because of the line of cells that it is synaptically connected to the receptors in the PNS.

2) Describe, in as much detail as possible, a G-protein-mediated receptor-second messenger cascade that results in a metabotropic response on a neuron’s ion channel.

Binding of a molecule to its receptor causes a conformational change in the receptor. This allows binding of the G-coupled protein to the receptor and phosphorylation of GDP to GTP. This causes the subunits of the G-coupled protein to dissociate from each other allowing the α-subunit to interact with adenylate cyclase. This activates the synthesis of cAMP, whereby an increase in cAMP causes the activation of a separate ion channel and thus a generator potential is produced.

3) You are experimenting on mice and you successfully knock-out all sweet receptors that exist in the mouth. You then genetically engineer the salty taste cells within these mice to have sweet taste receptors. What effect would glucose have on the mouse and why?

The mouse would perceive the sugar as salty. The glucose would activate the sweet receptor on the salty neuron which is part of a circuit that instructs the brain to perceive the tastant as salt.

4) Umami receptors are sensitive to which molecule?

Monosodium Glutamate
5) Describe three distinct mechanisms cells use to find their target cell. Consider how a cell can generally find its target, how a cell can specifically find its target, and how a cell can be ‘lazy’.

a) Pioneer cells have receptors on the growth cone which can sense chemicals in the environment. These chemicals can cause the axon to collapse, turn around, or grow towards the chemical signal, thus providing the signals for an axon to find its target cell.

b) Pioneer cells may have cell adhesion molecules at the tips of the growth cones which are necessary for the initial formation and maturation of synaptic contacts. Cell adhesion molecules interact with each other in a lock and key fashion, thus providing the specificity necessary for cells to find their exact target.

c) Secondary cells may use the axon of a pioneer cell as a tract for finding its way to its final destination.

6) The neurotrophic factor, Brain Derived Neurotrophic Factor (BDNF), is a chemical implicated in axon guidance. During development, growth cones will express the high-affinity receptor, tropomycin-related kinase B (TrkB), which will recognize BDNF in the environment. You are conducting an experiment in which you have knocked-out (removed) all TrkB receptors from Mus musculus, the common house mouse. You find that these mice have significantly fewer dendrites during early stages of development and have learning impairments in adulthood. What could be the reason for why loss of TrkB causes fewer dendrites? Why would this lead to learning impairments?

BDNF acts as a chemoattractant which allows dendrites expressing TrkB to grow towards their proper synaptic partners. Without TrkB, dendrites cannot recognize BDNF, and thus they cannot grow toward their proper synaptic contact and they are eliminated. With fewer dendrites, you also get fewer synapses. Since you are making less synaptic connections, you will not form the circuitry needed for proper learning and memory.