HW11: Chapter 12 3, 10, 22, 23

(3) a) If adenyl cyclase is constantly activated the [cAMP] of the cells will increase dramatically

b) cAMP apparently functions as a [Na^+] regulator in intestinal cells., allowing the ions to leave the cell.

c) One treatment might be the blockage of the Na^+ transport proteins in the intestine

Another treatment might be to inhibit adenyl cyclase in the intestine somehow.

An easy treatment might be to replace the lost Na^+ and water in the body by drinking fluids

(10) Without Ras GTPase activity, cells would be constantly stimulated by resulting binding to the receptor. In other words, a single binding event would end up stimulating the cell for a long time

(22) a) If the TGFβ receptor has a mutation, it might result in constitutive activation of the pathway. This constant activation would result in unrestricted cell division

b) The virus has introduced the homologues to the CXCR4/CXCR2 receptors into cells that normally don't express them. This might lead to the cells that normally wouldn't respond to chemokines being able to bind them and activating cell growth/division.

i) pRb binds to E2F normally. Free E2F triggers the progression from G1 to S phase.

If the adenovirus injects E1A and E1A binds pRb, then that would free up E2F in the cells, which would lead to unrestricted cell growth

d) If a cell type doesn't express the protein then a mutation in that same protein's gene would not have an effect.

(23) Tumor suppressor genes encode proteins that stop cell division. As long as one of the two genes are normal, then the cell will not divide

Oncogenes encode proteins that triggers a cell to divide in response to some extracellular stimulus. If an oncogene is mutated, it triggers the cell to divide without the stimulus. A single mutated form is enough to trigger cell division