Name:_____ Dr. Reichler's Bio 325-uex Spring 2009 Quiz 3/5

1) What is the connection between fetuses who are exposed to poor nutrition and smoking?

2) What is different about the genes of a totipotent cell versus a pluripotent cell?

3) What evidence suggests that DNA packaging is different between animal and plant cells?

4) Are the A, B, and C proteins that determine flower parts the first proteins to function in determining flower development?

5) What is the function of many of the hox proteins?

6) If you were studying the SRY gene, coded on the Y chromosome. Could you tell when SRY begins to be expressed using a reporter gene? Would a reporter gene allow you to determine the stability of the SRY protein?

7) Would you expect the chimpanzee or human version of the huntingtin gene to be larger?

8) By looking at the DNA sequence, how would you identify a hox pseudogene?

9) What different information can be gleaned from comparing transposons between humans and chimps or different people?

10) Why would knowing the time since the last common human ancestor help you determine if you should be nervous about transposons disrupting one of your genes?

Answers:

1) Both may lead to the adaptation to thriftiness as adults due to poor fetal nutrition.

2) A pluripotent cell has already irreversibly packaged some of its DNA, none of the totipotent cell's DNA has been irreversibly packaged yet.

3) Most mature plant cells are totipotent while few mature animal cells are.

4) No, other genes must determine the four layers. Even when the A, B, or C genes are deleted, there are still 4 whorls.

5) They are transcription factors.

6) Reporter genes can tell when or where transcription of a gene is activated, but since the reporter gene protein is different from the SRY gene, no information about the SRY protein can be determined.

7) If we use the puffer fish-human comparison combined with the general sense that humans have more transposons then chimpanzees, we could surmise that the human huntingtin gene should have more transposons.

8) It will have some sequence similarity to active hox genes, but will lack some critical component, like a functional promoter, that keeps it from being expressed.

9) Between chimps and people we see how active transposons have been in the last 6 million years. Between different people, we can see how active transposons have been since the people shared a common ancestor.

10) The time of the last common human ancestor will allow an estimation of transposon movement per given time in humans.