Cell biology

1. A. The protein takes the default pathway: through the ER, to the Golgi apparatus, to the cell surface. B. The protein fails to enter the ER and remains in the cytoplasm.

C. Same as A; the protein enters the default pathway through the ER, Golgi to the cell surface.

D. The protein is likely to end up as a single-pass transmembrane protein in the ER. If the hydrophobic core in the signal is preceded by more positively charged amino acids than following it on the carboxyl end, then the orientation of the transmembrane protein will be NCytosol/CER Lumen, otherwise the orientation will be NER Lumen/CCytosol.

2. The hybrid proteins in the experiment belong to two sets. In the first, the amino-terminal end of the thiolase is removed to various extents, and in the second, the carboxyl end of the thiolase is removed to various extents. In the first set, all hybrids with thiolase residue 100 intact have been imported into the peroxisome, and those lacking residue 100 or more remain in the cytosol. In the second set, all hybrids with residue 125 or greater have been imported into the peroxisome, and those lacking residue 125 or lesser remain in the cytosol.

Thus, the common sequence for peroxisome import from the given data lies between residues 100 through 125.

3. In the absence of hormones, protein B binds to protein A in the cytosol (immediately after translation of protein A in the cytosol). This binding prevents access to the nuclear transport machinery. This can happen in many ways, two of which are described here: protein B may mask the nuclear localisation signal (as in the case of the hsp90 - steroid hormone system), or protein B may anchor protein A in the cytosol. In each case, the

binding of the hormone to either protein causes dissociation of the two proteins, and protein A has access to the nuclear transport machinery. Thus, in normal cells, protein B is always found in the cytosolic extract, whereas the presence of hormone causes protein A to be found in the nucleus, rather than the cytosolic extract, where it is seen in the absence of hormone.

In cells lacking protein B, protein A has free access to the nuclear localisation machinery, and is therefore seen only in the nuclear extract.

4. A. The protein enters the ER and goes through the default pathway (ER-> Golgi-> Cell Surface). This is because ER import is co-translational, whereas nuclear import is post-translational. Thus, before the nuclear import signal can be recognized, the protein is already within the ER lumen.

B. The protein is imported into the mitochondria. This is because the nuclear export signal is different from the nuclear import signal, therefore the protein never enters the nucleus, and the mitchondrial import signal is recognized in the cytosol

C. The protein enters the ER and remains there. This is for the same reason as A, i. e. ER import is co-translational.

5. i) Membrane fusion requires the presence of Y and Z on the target and donor membrane respectively, or vice versa. Thus they are complementary.

ii) The chances of fusion are increased, whenever the same protein (either Y or Z) is present on both donor and target membranes. Thus, when none of the two are present on both, the chances of fusion is 50% of the maximal, when either Y or Z is present on both membranes, chances of fusion increases to 80%, and when both the proteins are present on both the membranes, the chances of fusion are 100% of the maximal.

iii) It is not important which of the two proteins is present on the donor and which on the target membrane for fusion to occur. However, whichever protein is present on the target membrane, the other one (its complement) should be present on the donor membrane

[Note: for the sake of clarity, I have denoted the membrane of one vesicle as target and the other as donor. This does not reflect asymmetry in function; the labels may just as well be reversed.]