Study Questions

Chapter 31

C-type Lectins

1. Many proteins that contain a C-type lectin domain do not bind glycans, and the ones that do are called C-type lectins. What is the difference in structure that distinguishes these two classes of proteins?
2. Why is it difficult to predict the type of glycan to which a C-type lectin will bind?
3. Some C-type lectins can form oligomers, which greatly increase the avidity of interactions with glycan ligands. Explain how oligomerization can also affect the specificity of the interaction.
4. Some C-type lectins, notably the selectins, bind with higher affinity to some glycoproteins than to others on the same cell, even though several glycoproteins may display similar glycan structures. Consider mechanisms that might confer such preferential binding.
5. Compare the interaction of P-selectin with PSGL-1 to the binding of a plant lectin to PSGL-1.

Chapter 32

I-type Lectins

1. There are now more than a dozen human Siglecs known. Why were these and other sialic-acid-binding proteins not discovered until very recently?
2. Compare the potential function of Siglecs with inhibitory motifs in their cytosolic tails with those that can recruit activatory motifs
3. Why are Siglec homologs found primarily in “higher” animals?
4. Explain the likely mechanism and driving forces for the rapid evolution of some Siglecs.
5. Why do plants and invertebrates that do not express sialic acids have sialic acid–binding proteins?

Chapter 43

Glycans in Acquired Human Diseases

1. What are the common underlying mechanisms for the roles of selectins in various diseases?
2. Although heparin is primarily used as an anticoagulant, its use has been proposed in connection with several other diseases. How can one drug have relevance to so many different mechanisms?
3. Give two examples where altered glycosylation has resulted in acquired blood cell diseases involving the hematopoietic stem cell. Why is it possible for somatic mutations to give rise to a phenotype?
4. Describe the common underlying molecular mechanism that causes changes in O-glycans in blood cell diseases, in IgA nephropathy, and in the altered glycosylation of cancer.