MMBB 300 - Exam 2 - Fall 2002
KEY

1. (b) a-helix is an example of secondary structure
2. (c) 3.6 aa per turn. Each amino acid in the helix is hydrogen-bonded with at least one amino acid that is +/- 4 residues away
3. In a b-strand, the amino acids are stretched out; 3.5 Angstroms between amino acid centers. In the b-sheet, 2 or more strands are stabilized by hydrogen bonding between the carbonyl oxygen of one strand and the backbone nitrogen of the other strand. The side chains lie above and below the plane and play only a minor role in the b-sheet structure.
4. (b)
5. (c) urea is a chaotropic agent that helps proteins unfold, but it is not a reductant and can not reduce or break disulfide bonds.
6. (b) heme binds Fe^{3+} just fine; it is just that Fe^{3+} is not capable of binding O_2 very well.
7. (a) hydrophilic amino acids are found on the outside of proteins. The only hydrophilic amino acids found in the interior of myoglobin are the proximal and distal histidines.
8. (c) ferrimyoglobin is incapable of binding O_2 and is considered biologically inactive.
9. The distal histidine side chain plays a steric role by blocking the linear bonding of CO (carbon monoxide, NOT CO_2 which binds elsewhere). The distal His forces a bent geometry which is less stable for CO but does not affect O_2 binding which prefers a bent geometry (trigonal planar). CO still binds 200x stronger than O_2 but much less than the linear arrangement.
10. a. the amino acid sequences are mostly different; only 24 of 141 amino acids are identical.
b. the 3D structure of myoglobin and the alpha subunit of hemoglobin are nearly superimposable (i.e. same 3D structure).
11. (d) CO_2, H^+ & 2,3-BPG all bind different sites in hemoglobin and all three weaken the affinity for O_2; all 3 are allosteric effectors.
12. The myoglobin curve is hyberbolic & the hemoglobin curve is sigmoidal; both level out at a saturation of 1.0.
13. Lower pH shifts the sigmoidal curve to the right (i.e. weakens O_2 affinity). The Bohr effect.
14. There is a genetic point mutation where Glu_6 is converted to Val_6 in the b-subunit of HbS. This creates a hydrophobic patch near the outside of the protein which is exposed when O_2 is not bound. The exposed patch is capable of interacting with other HbS proteins to polymerize to the point where large filaments/aggregates fill the cell & force a sickle shape.
b. Malaria
15. (b) prions do not attack/exploit RNA or protein synthesis. They function by recruiting the cell's normal prion protein and converting them into the infectious form.
16 F
T
F
T
17. Enzymes/catalysts lower the energy of activation but not the Gibb's free energy of reaction.
18. (d)
19. In general, the substrate and enzyme active site are not a perfect prior to binding. Upon binding the substrate, the active site changes conformation to fit better; the substrate structure may also change upon binding.
This replaced the "Lock and Key" Model which stated that the substrate and enzyme active site are a perfect fit prior to binding.
20. (b)

21. a. $v = V_{max} \frac{[S]}{K_M + [S]}$

b. $K_M = \frac{k_2 + k_{-1}}{k_1}$

K_M = the substrate concentration at which $v = \frac{1}{2} V_{max}$

The K_M gives an indication of the affinity of the enzyme for the substrate. A low K_M indicates tight binding while a large K_M indicates weaker binding.

The K_M is also known as the Michaelis constant but this is only a name and is not much of a definition. This answer would get partial credit at best.

22. (c) $\frac{v}{V_{max}} = \frac{[S]}{K_M + [S]} = \frac{2}{1 + 2} = \frac{2}{3} = 67\ \%$

23. a. **Little or no product has formed**

b. **rate of ES formation = rate of ES breakdown; [ES] stays constant**

24. **competitive inhibitor only changes KM (makes it larger).**

$f_{ES} = \frac{[S]}{K_M + [S]} = \frac{2}{2 + 2} = \frac{2}{4} = \frac{1}{2}$ **½ the sites are filled.**