

Solutions

2.1

- Primary: peptide bonds; secondary: backbone H-bonds; tertiary: hydrophobic effect + H-bonds + disulfides; quaternary: same forces between subunits.

2.2

- Myoglobin is single-site (no partners to influence); haemoglobin's four sites communicate via T $\leftrightarrow$ R subunit shifts – binding helps binding.

2.3

- Conservative surface swap (both negative, similar size): minimal impact – outside the core and the active site.

3.1

- T is low-affinity, R high; each O₂ binding shifts equilibrium toward R, so later sites bind more easily (sigmoid, $n_H \approx 2.8$).
- $15/64\,500 \times 4 \times 22\,400 \approx 20.8$ mL/dL ✓.
- Anaemia halves it to ~ 10.4 mL/dL; compensation is raised cardiac output and erythropoietin-driven red-cell production.

3.2

- Val6 creates a hydrophobic patch on the β surface; in the deoxy T state it docks into a pocket on another Hb, polymerising fibres.
- Trigger: deoxy (T-state conformation exposes the patch).
- Capacity per molecule unchanged (haem intact); viscosity rises and cells deform/occlude.