1. **Night blindness is one important deficiency state of which vitamin? Explain the relations.**

Night blindness is caused by a deficiency state of Vitamin A, an essential component of rhodopsin in the form of 11-cis-retinal. Trans-retinal is obtained from the diet and is converted to retinol by NADPH dehydrogenase. Retinol is then converted to 11-cis-retinol by isomerase. Finally, NAD converts cis-retinol to cis-retinal, which combines with opsin to give the light sensitive pigment rhodopsin. Hence, deficiency in Vitamin A results in night blindness.

![Chemical Structure of Vitamin A](image)

2. **What is this structure? How can it help with night blindness?**

This structure is beta-carotene. It is obtained from plants as a dietary source of Vitamin A. An enzyme, 15,15-dioxygenase, will metabolize beta-carotene to yield two equivalents of retinal. Thus, it can be used for treatment of Vitamin A deficiency, which may lead to night blindness.

3. **Briefly describe the function of Vitamin D in the body.**

Vitamin D is necessary for absorption of Ca through the intestinal mucosa, and it is necessary for a functioning calcium pump. 1,25-dihydroxy D₃ acts as a hormone in regulating synthesis of calcium binding protein at the gene expression level.

4. **A patient suffering from renal failure (diabetes) is also experiencing osteomalacia. Suggest a possible reason for the complication. Would Vitamin D be a suitable treatment for osteomalacia here? Why or why not?**

Osteomalacia is a condition of bone demineralization as a result of calcium depletion. Vitamin D helps in the uptake of calcium from the gut to circulation. The most active form of Vitamin D, 1,25-dihydroxycholecalciferol, is synthesized in the kidney through a metabolic pathway. In an event of kidney damage, the renal cells may not be able to form
this active Vitamin D metabolite. Thus, the effectiveness of the calcium uptake in the gut is reduced.

Vitamin D would not be suitable here because it would still require functional renal cells to do the metabolic conversion to the active Vitamin D. A better alternative will be to use Calcitriol.

5. Why is Vitamin E useful in patients that have hemolytic anemia associated with a glucose-6-phosphate dehydrogenase deficiency (G6PD)?

With the deficiency of the enzyme glucose-6-phosphate dehydrogenase, ROOH (cellular lipids) will not be converted to ROH there will also be an accumulation of peroxides (reactive oxygen species). These species will cause cellular damage leading to hemolytic anemia. Under normal physiology, the following pathway is functional.

Glucose-6-phosphate dehydrogenase provides NADPH to glutathione reductase in order to reduce GSSG to GSH. GSH then converts ROOH to ROH in the presence of glutathione peroxidase. So, when this pathway is non-functional, Vitamin E can be used to reduce the formation of peroxides via radical scavenging. In other words:

RH → R•
R• + O₂ → RO₂•
RO₂• + RH → ROOH + R•
R•, RO₂• + Vit E → RH, ROOH + oxidized Vit E

6. A patient has been on long term broad spectrum antibiotics for a year for Crohn's disease. Recently, there were signs of unexplained hemorrhage. What could be the reason for the bleeding?

Long term antibiotic therapy can lead to decreased intestinal bacteria concentration. Since 50% of Vitamin K comes from gut bacteria, the patient might be low in Vitamin K. Vitamin K is important for maintaining blood coagulation. A Vitamin K deficiency would cause an increased bleeding tendency.
7. Based on the Daily Value, complete the following table.

<table>
<thead>
<tr>
<th>Daily Value</th>
<th>Vitamin</th>
<th>Deficiency State</th>
<th>Toxicity</th>
<th>Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>400 IU</td>
<td>D</td>
<td>Rickets, osteomalacia</td>
<td>Calcification of soft tissues, esp. in infants</td>
<td>Rickets, osteomalacia, renal failure, hypoparathyroidism</td>
</tr>
<tr>
<td>80ug</td>
<td>K</td>
<td>Hypoprothrombinemia</td>
<td>None</td>
<td>Promote coagulation; Warfarin overdose</td>
</tr>
<tr>
<td>30 IU</td>
<td>E</td>
<td>Hemolytic anemia edema</td>
<td>None-possible bleeds at very high doses</td>
<td>Antioxidant (free radical scavenger); e.g. eye damage in premature infants</td>
</tr>
<tr>
<td>5000 IU</td>
<td>A</td>
<td>Xerophthalmia</td>
<td>Hypervitaminosis A</td>
<td>Xerophthalmia keratinization of epithelial tissues</td>
</tr>
</tbody>
</table>

8. Name two cases where multivitamin supplements are clearly worthwhile.

a. Inadequate intake: alcoholics, poor, elderly, dieters (<1500 calories/day), poor diet
b. Poor absorption: elderly, GI disorders, cystic fibrosis, diarrhea
c. Increased needs: pregnancy, lactation, infants, smokers, injury, trauma, surgery, infection
d. Iatrogenic vitamin deficiencies: oral contraceptives, long term antibiotic use, isoniazid, cholestyramine