I. Match the vitamin or mineral with the corresponding amount and unit.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Amount</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Vitamin D</td>
<td>d,z</td>
<td>a) 20</td>
</tr>
<tr>
<td>2.</td>
<td>Vitamin B12</td>
<td>e,y</td>
<td>b) 0.3</td>
</tr>
<tr>
<td>3.</td>
<td>Biotin</td>
<td>b,x</td>
<td>c) 5000</td>
</tr>
<tr>
<td>4.</td>
<td>Vitamin A</td>
<td>c,z</td>
<td>d) 400</td>
</tr>
<tr>
<td>5.</td>
<td>Folacin</td>
<td>i,x</td>
<td>e) 6.0</td>
</tr>
<tr>
<td>6.</td>
<td>Vitamin E</td>
<td>h,z</td>
<td>f) 60</td>
</tr>
<tr>
<td>7.</td>
<td>Niacin</td>
<td>a,x</td>
<td>g) 10</td>
</tr>
<tr>
<td>8.</td>
<td>Pantothenic Acid</td>
<td>g,x</td>
<td>h) 30</td>
</tr>
<tr>
<td>9.</td>
<td>Vitamin C</td>
<td>f,x</td>
<td>i) 0.4</td>
</tr>
</tbody>
</table>

II. Water soluble vitamins in many cases can be taken in high doses without side effects.

   a. Explain why.
   Water soluble vitamins are readily metabolized and/or excreted due to their hydrophilicity.

   b. Give two examples where toxicity has been observed and list the symptoms of toxicity.
      B6 toxicity- > 200 mg/day leads to increased prolactin; >1-2 g/day leads to neuropathy.
      Niacin toxicity- peripheral vasodilation, GI upset, ulcers, diarrhea, liver damage.

III. For the following conditions:

   a. Name the vitamin that has been shown to be of some benefit.
   b. What enzyme/reaction is activated by high doses of the vitamin?

1. Homocystinuera- B6 activates cystathionine synthase
2. Wernicke-Korsakoff syndrome- B1 (thiamin) activates transketolase
3. Methylmalonic acidurea- B12 activates isomerase (methylmalonyl CoA to syccinyl CoA)
4. Xantheronic acidurea- B6 activates kyureninase
IV. Provide a metabolic explanation for the following (a scheme may be necessary for a
cogent answers-structures are not necessary).

a. A folate deficiency is observed during a B\textsubscript{12} deficiency.

\[
\begin{align*}
\text{N}_{5}\text{-methyl THFA} & \quad \xrightarrow{\text{B}_{12}} \quad \text{THFA} \\
\end{align*}
\]

B\textsubscript{12} is required to convert N\textsubscript{5}-methyl THFA to THFA. If B\textsubscript{12} is deficient, this conversion may
not occur, and a folate deficiency will occur.

b. Homocysteine levels may be elevated if B\textsubscript{6} intake is inadequate.

\[
\begin{align*}
\text{Methionine} & \quad \xrightarrow{\text{B}_{12}} \quad \text{Homocysteine} & \quad \text{B}_{6} & \quad \text{Cystathionine} & \quad \text{B}_{6} & \quad \alpha\text{-ketobutyrate} + \text{cysteine} \\
\end{align*}
\]

B\textsubscript{6} is involved in the conversion of homocysteine to cystathionine, and then alpha-
ketobutyrate and cysteine. When B\textsubscript{6} is inadequate, homocysteine levels will be increased.

c. Niacin may be deficient if B\textsubscript{6} intake is inadequate.

\[
\begin{align*}
\text{tryptophan oxygenase} & \quad \text{Tryptophan} & \quad \xrightarrow{\text{B}_{6}} & \quad \text{N-formylkynurenine} & \quad \xrightarrow{\text{kynureninase}} & \quad \text{3-hydroxykynurenine} \\
\text{3-hydroxy anthranilic acid} & \quad \xrightarrow{\text{B}_{6}} & \quad \text{quinolinic acid} & \quad \xrightarrow{\text{B}_{6}} & \quad \text{niacin} \\
\end{align*}
\]

If B\textsubscript{6} is inadequate, the conversion from tryptophan to niacin may be blocked, resulting in
niacin deficiency.

V. The following are signs of a vitamin deficiency. For each, list the most appropriate single
deficient vitamin.

\begin{align*}
a. \text{increased urinary xanthurenic acid} & \quad \text{B}_{6} \\
b. \text{increased plasma pyruvate} & \quad \text{B}_{1} \\
c. \text{increased plasma homocysteine} & \quad \text{B}_{6}, \text{folic acid}, \text{or B}_{12} \\
d. \text{decreased erythrocyte glutathione reductase activity} & \quad \text{B}_{2} \\
\end{align*}

VI. What vitamins are associated with the following functions?

\begin{align*}
a. \text{decarboxylation} & \quad \text{B}_{1} \text{ and B}_{6} \\
b. \text{transamination} & \quad \text{B}_{6} \\
c. \text{purine and pyrimidine synthesis} & \quad \text{folic acid} \\
d. \text{electron transport} & \quad \text{B}_{2} \text{ and B}_{3} \\
e. \text{niacin synthesis} & \quad \text{B}_{6} \\
f. \text{collagen synthesis} & \quad \text{C} \\
\end{align*}
VII. Why is it important that a woman who is pregnant or is planning on becoming pregnant should be receiving a supplemental folate?

Folic acid deficiency early in a pregnancy could lead to teratogenesis with neural tube defects. It is recommended that women who are pregnant or are thinking about becoming pregnant should be receiving the RDA value of 0.4 mg/day folic acid from fortified foods or from a supplement. This is in addition to that provided in the diet.

IX. Folic acid requires a prescription for high doses, even though it is essentially non-toxic. Why?

Folic acid deficiency (anemia) can be caused by two conditions: 1) lack of folic acid or 2) lack of B12 (involved in the recycling of folic acid). By giving a high folate dose, nucleic acids will be synthesized even though folic acid is not regenerated in the process. Therefore, pernicious anemia associated with B12 deficiency will be masked and nerve damage will progress irreversibly if not treated.

X. The following are vitamin antagonists.

a. Suggest the vitamin antagonized.
b. Suggest one symptom that you might expect with chronic administration of the antagonist.

\[
\begin{array}{c}
\text{NH}_2 \\
\text{N} \\
\text{N} \\
\text{S} \\
\text{OH}
\end{array}
\]

a. B6
b. Wernike-Korsakoff syndrome (confusion, memory loss, etc)

\[
\begin{array}{c}
\text{OH} \\
\text{HO} \\
\text{N}
\end{array}
\]

a. B1
b. Peripheral neuritis
a. Folic acid
b. Megaloblastic anemia