E. The Duffy antigens are located on the urea transport proteins of the kidney.

**Question 18:** What is the titer of anti-K in the serum examined in Table 5-4?

<table>
<thead>
<tr>
<th>Table 5-4.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dilution</td>
</tr>
<tr>
<td>Anti-K</td>
</tr>
</tbody>
</table>

m = microscopically positive

A. 8.
B. 1:8.
C. 16.
D. 1:16.
E. 1:32.

**Question 19:** Which of the following is true concerning the Sd^a antigen and the antibodies that recognize it?

A. The Sd^a antigen is expressed on 10% of red cell samples.
B. Sd^a is located on the C4 component of complement.
C. Anti-Sd^a is neutralized by human plasma.
D. The presence of anti-Sd^a is suggested by refractile orange agglutinates on microscopy.
E. Anti-Sd^a has been associated with HDFN.

**Question 20:** Which of the following statements is true of the antigens of the Lutheran blood group system and the antibodies that recognize them?
A. Lu\textsuperscript{a} is a high-incidence antigen.
B. Anti-Lu\textsuperscript{a} causes hemolytic transfusion reactions.
C. The Lu(a–b–) phenotype is common in individuals of African ethnicity.
D. The Lu(a–b–) phenotype results from an autosomal recessive gene only.
E. Antibodies to Lu\textsuperscript{a} produce a mixed-field pattern of reactivity with Lu\textsuperscript{a} positive cells.

**Question 21:** A 30-year-old female presents with a lacerated liver following an automobile accident. Four RBC units are ordered. The patient has no history of transfusion but has had five pregnancies. She types as group A, Rh positive. Her antibody screen is positive with the antibody identification panel given in Table 5-5. Additional testing is also performed, as shown in Table 5-6. Which of the following statements about this antibody and the antigen to which it is directed is true?

A. The antigen is named after its discoverers, Levine and Stetson.
B. The antibody was originally produced by immunizing guinea pigs with red cells from rhesus monkeys.
C. The antigen is destroyed by enzyme treatment.
D. The antibody is capable of fixing complement and causing intravascular hemolysis.
E. The antigen is poorly expressed on fetal red cells.

**Question 22:** Which is the most common red cell typing in people of African ethnicity?

A. Lu(a–b–).
B. Jk(a–b–).
C. Fy(a–b–).
D. O\textsubscript{h}.
E. Js(a–b–).
| Cell | D  | C  | c  | E  | f  | V  | C^a| K  | k  | Kp^a| Js^a| Js^b| Fy^a| Fy^b| Jk^a| Jk^b| Le^a| Le^b| P  | M  | N  | S  | s  | Lu^a| Lu^b| Xg^a| IS  | AHG | CC  |
|------|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| I    | +  | +  | 0  | 0  | 0  | 0  | +  | 0  | +  | 0  | +  | +  | +  | 0  | +  | 0  | +  | +  | +  | +  | 0  | +  | 0  | 0  | 1+ |
| II   | +  | 0  | +  | 0  | 0  | 0  | 0  | +  | 0  | +  | 0  | +  | 0  | +  | 0  | +  | +  | +  | 0  | +  | 0  | +  | 0  | 1+ |
| 1    | +  | +  | 0  | 0  | 0  | 0  | +  | 0  | +  | +  | +  | +  | 0  | +  | 0  | +  | +  | +  | +  | 0  | +  | 0  | 0  | 1+ |
| 2    | +  | +  | 0  | 0  | 0  | 0  | +  | 0  | +  | +  | +  | +  | 0  | +  | 0  | +  | +  | 0  | 0  | 0  | +  | 0  | 0  | 1+ |
| 3    | +  | 0  | +  | +  | 0  | 0  | 0  | +  | 0  | +  | 0  | +  | 0  | +  | 0  | +  | 0  | 0  | +  | 0  | +  | 0  | 0  | 1+ |
| 4    | +  | 0  | +  | +  | 0  | 0  | +  | 0  | +  | +  | 0  | +  | 0  | +  | 0  | +  | +  | 0  | 0  | 0  | +  | 0  | 0  | 1+ |
| 5    | 0  | +  | +  | 0  | +  | 0  | 0  | +  | 0  | +  | +  | +  | 0  | +  | 0  | +  | +  | +  | +  | 0  | +  | 0  | 0  | +  |
| 6    | 0  | 0  | +  | +  | +  | 0  | 0  | +  | 0  | +  | +  | +  | +  | 0  | +  | 0  | +  | 0  | 0  | +  | 0  | 0  | 0  | +  |
| 7    | 0  | 0  | +  | 0  | +  | 0  | 0  | +  | 0  | +  | +  | +  | +  | 0  | +  | 0  | 0  | 0  | +  | +  | 0  | 0  | 0  | +  |
| 8    | 0  | 0  | +  | 0  | +  | 0  | 0  | +  | 0  | +  | +  | +  | 0  | 0  | 0  | +  | 0  | +  | +  | 0  | 0  | 0  | +  |
| 9    | 0  | 0  | +  | +  | 0  | 0  | 0  | +  | 0  | +  | +  | +  | +  | 0  | 0  | +  | 0  | +  | 0  | 0  | +  | 0  | 0  | +  |
| 10   | 0  | 0  | +  | +  | 0  | 0  | 0  | +  | 0  | +  | +  | 0  | 0  | +  | 0  | +  | 0  | +  | 0  | +  | 0  | 0  | 0  | +  |

Patient's cells

0 0 +
D. The Bg antigens are sensitive to treatment with proteolytic enzymes and are also sensitive to the disulfide-bond-reducing agents AET and DTT.
E. Bg antigens can be stripped from red cells with chloroquine.

**ANSWERS**

**Question 1:** E

**Explanation:**

See Explanation for Question 2.

**Question 2:** C

**Explanation for Questions 1 and 2:**

The frequency of the Rh system antigens (D, C, E, c, and e) in blacks and whites is shown in Table 5-7.

<table>
<thead>
<tr>
<th></th>
<th>Blacks</th>
<th>Whites</th>
<th>Fisher-Race Terminology</th>
</tr>
</thead>
<tbody>
<tr>
<td>D</td>
<td>92</td>
<td>86</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>33</td>
<td>70</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>21</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>c</td>
<td>97</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>e</td>
<td>99</td>
<td>98</td>
<td></td>
</tr>
</tbody>
</table>
Question 3: A

Explanation:

- The observed frequency of Rh haplotypes in whites is $R^1$ (42%) > $r$ (37%) > $R^2$ (14%) > $R^o$ (4%).
- The observed frequency of Rh haplotypes in blacks is $R^o$ (44%) > $r$ (26%) > $R^1$ (17%) > $R^2$ (11%).
- The frequency of the $R^Z$, $r'$, $r''$, and $r'''$ haplotypes in people of African and European ethnicity is very rare.
- The principal Rh gene complexes and the antigens encoded are shown in Table 5-8.

<table>
<thead>
<tr>
<th>Haplotype</th>
<th>Genes Present</th>
<th>Antigens Present</th>
<th>Phenotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>$R^1$</td>
<td>$RHD, RHCe$</td>
<td>D, C, e</td>
<td>$R_1$</td>
</tr>
<tr>
<td>$R^2$</td>
<td>$RHD, RHcE$</td>
<td>D, c, E</td>
<td>$R_2$</td>
</tr>
<tr>
<td>$R^o$</td>
<td>$RHD, RHce$</td>
<td>D, c, e</td>
<td>$R_0$</td>
</tr>
<tr>
<td>$R^Z$</td>
<td>$RHD, RHCE$</td>
<td>D, C, E</td>
<td>$R_Z$</td>
</tr>
<tr>
<td>$r'$</td>
<td>$RHCe$</td>
<td>C, e</td>
<td>$r'$</td>
</tr>
<tr>
<td>$r''$</td>
<td>$RHcE$</td>
<td>c, E</td>
<td>$r''$</td>
</tr>
<tr>
<td>$r$</td>
<td>$RHce$</td>
<td>c, e</td>
<td>$r$</td>
</tr>
<tr>
<td>$r'''$</td>
<td>$RHCE$</td>
<td>C, E</td>
<td>$r'''$</td>
</tr>
</tbody>
</table>

Question 4: A

Explanation:

- Alloimmunization is a significant problem among patients with sickle cell disease, with up to 30% of sickle cell patients being alloimmunized depending on the population of patients being...
studied. This is particularly problematic among the chronically transfused.

- Although incompletely understood, the major cause for the high rate of alloimmunization among sickle cell patients appears to be the fact that sickle cell patients (predominantly of African ethnicity) are more likely to lack certain blood group antigens in comparison to a blood donor population that is predominantly of European ethnicity (eg, see Table 5-7). This means that they are more likely to be exposed to foreign blood group antigens.

- The most common blood group antibodies detected in sickle cell patients are anti-C, anti-E, and anti-K. This is because: 1) these are among the most immunogenic of all blood group antigens and 2) the corresponding antigens (ie, C, E, and K) are more frequently found in blood donors of European ethnicity.

- Although neither universally accepted nor applied, a means to avoid alloimmunization among sickle cell patients is to establish a baseline blood group antigen phenotype and then selectively match for the Rh antigens and K antigen. From the standpoint of the Rh antigens, this then means that blood lacking the C and E antigens (ie, c- and e-positive) would be provided.

- Among donors of European ethnicity, the r phenotype (ce) is most likely to lack the C and E antigens. Among donors of African ethnicity, the R0 phenotype (cDe) is most likely to lack the C and E antigens.

- See Table 5-9 for the frequencies of the Rh phenotypes found in donor groups of various ethnicities in the United States.

**Question 5: C**

**Explanation:**

- CGD is a primary immunodeficiency disorder in which phagocytes can ingest bacteria but are unable to kill them because of a defective oxidative burst. This results from abnormalities in nicotinamide adenine dinucleotide phosphate oxidase. This abnormality leads to the formation of granulomas and recurrent infections. CGD is often associated with a depression of Kell blood group system antigens, a component of the McLeod phenotype.