44. Purple is dominant.
45. Genotypic ratio: 1 *BB*: 2 *Bb*: 1 *bb*;
    Phenotypic ratio: 3 white: 1 black
46. A test cross with a black (*bb*) sheep
47. White is dominant.
48. a. 3/4 yellow: 1/4 green;
    b. 1/2 yellow: 1/2 green;
    c. all green
49. *Dd* x *Dd*
50. 9 *C-S-* mule-foot, belted pigs:
    3 *C-ss* mule-foot, solid pigs:
    3 *ccS-* cloven-foot, belted pigs:
    1 *ccss* cloven-foot, solid pig
51. There are 3 cloven-foot, belted pigs, but only one is homozygous (*ccSS*); the other two are heterozygous (*ccSs*).
52. 9 *F-T-* flat and toothed:
    3 *F-tt* flat and toothless:
    3 *ffT-* fuzzy and toothed:
    1 *fftt* fuzzy and toothless
53. black spotted male (*BBSS*) mated with brown solid female (*bbss*)
54. *BbSS* male mated with *bbss* female
55. 3 *P-RR* polled red:
    6 *P-Rr* polled roan:
    3 *P-rr* polled white:
    1 *ppRR* horned red:
    2 *ppRr* horned roan:
    1 *pprr* horned white
56. a. Incomplete dominance with cream being heterozygous
    b. 1 yellow: 2 cream: 1 white
57. F_1: *Rr* pink;
    F_2: 1/4 *rr* red: 2/4 *Rr* pink: 1/4 *rr* white
58. Half of the sons will inherit the defective gene from mom and get Y from dad. Half of the daughters will also inherit the defective gene from mom but they will be heterozygous since they also inherit a normal gene from dad.
59. None is expected to have hemophilia or inherit the gene.
60. a. *Cc* (normal but carrier woman) x *CY* (normal man) [X^CX^c x X^CY]
    b. No chance: she inherits a normal *C* gene (X^C) from dad as well as either a *C* gene (X^C) or *c* gene (X^c) from mom.
61. Black female, tortoiseshell female, black male, yellow male
62. F_2 for both sexes: 3/8 red long, 3/8 white long, 1/8 red vestigial, 1/8 white vestigial
63. a. all walnut
    b. 1/4 walnut: 1/4 rose: 1/4 pea: 1/4 single
    c. 1/4 walnut: 1/4 rose: 1/4 pea: 1/4 single
    d. 3/4 walnut: 1/4 pea
    e. 1/2 walnut: 1/2 pea
64. *DDee* x *ddEE* → *DdEe*
65. 9/16 *D-E-* normal: 3/16 *D-ee* deaf:
    3/16 *ddE-* deaf: 1/16 *ddee* deaf;
    so, 9/16 normal: 7/16 deaf
66. a. 3/4 restricted mallard: 1/4 mallard
    b. 1/2 restricted mallard: 1/2 mallard
    c. 1/2 mallard: 1/2 dusky mallard.
67. F_2: 1/16 white: 4/16 light: 6/16 medium: 4/16 dark: 1/16 black
68. a. black
    b. black
    c. medium
    d. black
    e. dark
    f. dark
    g. medium
    h. light
69. Like recessive deafness, there are several different genetic situations leading to albinism. In one type of albinism, the gene necessary for making an enzyme needed to convert a substrate into melanin pigment is missing (genotype *aa*), while in another type of albinism the gene necessary for transporting the substrate for melanin into the pigment-producing cells is lacking (genotype *bb*). So, both *aaBB* and *AAbb* people would have albinism. However, the children produced by individual *aaBB* and individual *AAbb* would be *AaBb* and would have normal pigmentation because the children would make the enzyme necessary to produce melanin and the enzyme necessary to transport the substrate for melanin into the pigment cells.

70. In the human blood types, a type A person has red blood cells (RBCs) with type A antigen proteins, and blood plasma with anti-B antibodies. A type B person has RBCs with type B antigen and plasma with anti-A antibodies. A type AB person has RBCs with type A and type B antigens and plasma lacking anti-A and anti-B antibodies. A type O person has RBCs lacking type A and type B antigens and plasma with anti-A and anti-B antibodies. Since type O red blood cells do not contain either the A or the B antigen proteins, they can be given to anyone in an emergency situation, since they will not be attacked by either anti-A or anti-B antibodies in the blood plasma of the recipient. Since type AB people do not have anti-A or anti-B antibodies in their blood plasma, they can receive any type of RBC in a transfusion, since they lack the antibodies to attack the donated cells.

71. All albino individuals are recessive for the albinism gene (*aa*). There are two possible genotypes for the normal parents, however. Some normal parents may be homozygotes (*AA*) and others may be heterozygotes (*Aa*). In those families with an albino parent and a heterozygous normal parent, we expect the following outcome: *aa* x *Aa*, 50% with *aa* (albinism) and 50% with *Aa* (normal). In those families with an albino parent and a homozygous normal parent, we expect the following outcome: *aa* x *AA*, all children with Aa (normal).

72. Let's say we are flipping a quarter and a dime. There is only one way that both coins land heads-up: a 1/2 (50%) chance that the quarter lands heads-up and a 1/2 (50%) chance that the dime lands heads-up. Since these outcomes are independent events, we multiply their results together: 1/2 x 1/2 = 1/4 (25%). If one coin lands heads-up and the other lands tails-up, there are two ways for that to happen: quarter-heads (1/2 chance) and dime-tails (1/2 chance) = 1/4 (25%) total chance, or quarter-tails (1/2 chance) and dime-heads (1/2 chance) = 1/4 (25%) total chance. Adding these two chances together, we get a total chance of 1/2 (50%) that one coin lands heads-up and the other coin lands tails-up.

73. There are several reasons why peas are better organisms than humans to study genetic principles. Peas have a short life cycle and produce many offspring per plant, so that meaningful statistical analysis can be performed from the data obtained. Peas are inexpensive to raise and take up little space. Peas have many easy to recognize characteristics, and it is easy to control the types of matings desired. In addition, there are no ethical problems associated with the genetic manipulation of matings using pea plants.

74. In dogs, narcolepsy is caused by defects in a recessive gene called *Hcrt-2*, which normally makes a protein receptor (that normally binds to hypocretin) present on the surfaces of certain brain cells. Because the receptor is defective in narcoleptic dogs, their brains cannot respond to the molecular signals delivered by the hypocretin molecules. In mice, narcolepsy is caused by a lack of the hypocretin molecules, caused by a defective recessive gene. Narcoleptic mice have normal *Hcrt-2* genes and make normal receptor molecules for hypocretin. Studies on humans with narcolepsy indicate that some patients lack hypocretin molecules but have normal receptors (like the mice) and others make hypocretin molecules but may have defective receptors (like the dogs).

75. According to the Huntington's Disease Society of America, Huntington's Disease (HD) is a devastating, degenerative brain disorder for which there is, at present, no effective treatment or cure. HD slowly diminishes the affected individual's ability to walk, think, talk and reason. Eventually, the person with HD becomes totally dependent upon others for his or her care. Huntington's Disease profoundly affects the lives of entire families: emotionally, socially and economically. More than a quarter of a million Americans have HD or are "at risk" of inheriting the disease from an affected parent. HD affects as many people as Hemophilia, Cystic Fibrosis or muscular dystrophy. Early symptoms of Huntington's Disease may affect cognitive ability or mobility and include depression, mood swings, forgetfulness, clumsiness, involuntary twitching and lack of coordination. As the disease progresses, concentration and short-term memory diminish and involuntary movements of the head, trunk and limbs increase. Walking, speaking and swallowing abilities deteriorate. Death eventually follows from complications such as choking, infection or heart failure.