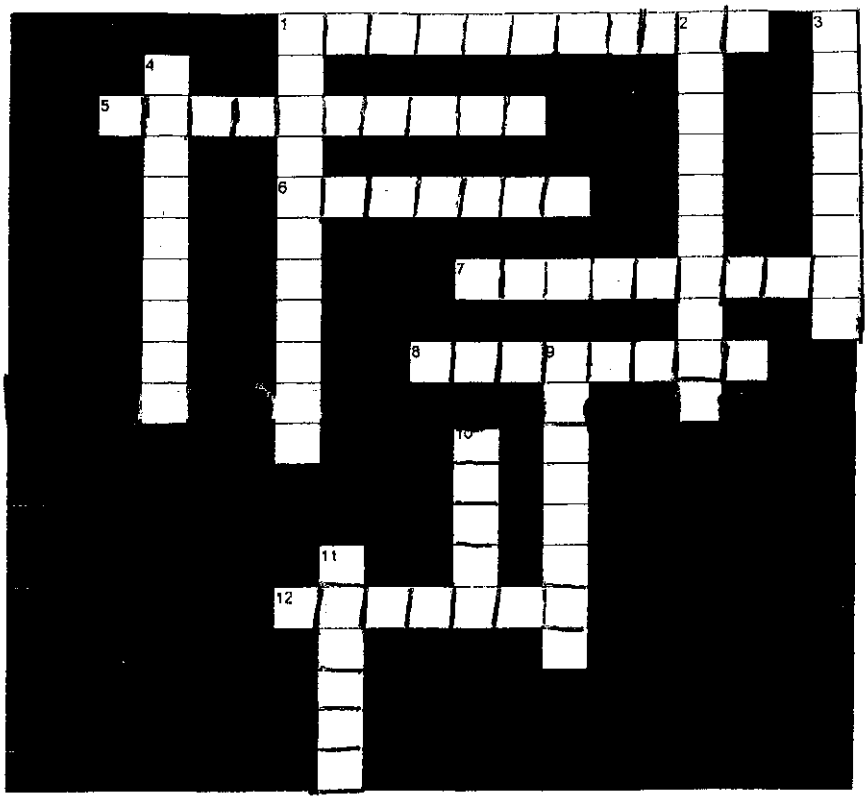
Mitosis



ACROSS

1. cytoplasm divides into two new cells
5. responsible for pulling cromatids apart in mitosis
6. cell division
7. chromosomes line up in the middle of the cell
8. chromatids are pulled apart
12. contains chromosomes

DOWN

1. contains the genes
2. the resting stage of a cell cycle
3. _____ cells result from mitosis
4. new nucleus begins to form around the chromosomes
9. the first phase of mitosis
10. cell occurs in plant mitosis
11. cleavage _____ occurs in animal mitosis

COMPARING MITOSIS AND MEIOSIS

Name _____

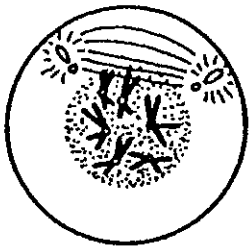
Determine whether the following characteristics apply to mitosis, meiosis or both by putting a check in the appropriate column(s).

	Mitosis	Meiosis
1. no pairing of homologs occurs		
2. two divisions		
3. four daughter cells produced		
4. associated with growth and asexual reproduction		
5. associated with sexual reproduction		
6. one division		
7. two daughter cells produced		
8. involves duplication of chromosomes		
9. chromosome number is maintained		
10. chromosome number is halved		
11. crossing over between homologous chromosomes may occur		
12. daughter cells are identical to parent cell		
13. daughter cells are not identical to parent cell		
14. produces gametes		
15. synapsis occurs in prophase		

STAGES OF MITOSIS

Name _____

Number the following six diagrams of the stages of mitosis in animal cells in the proper order. Label each stage with the proper name.



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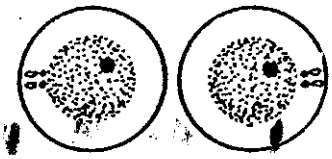

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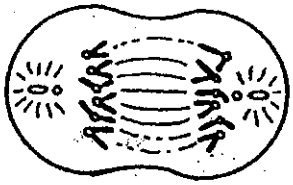

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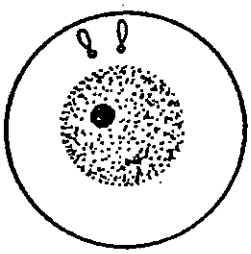

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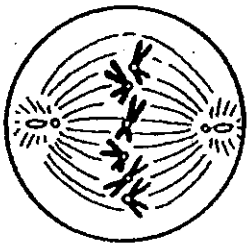

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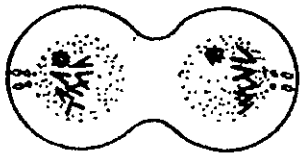
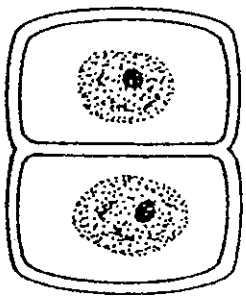


_____

Do the same for the following diagrams of mitosis in plant cells.



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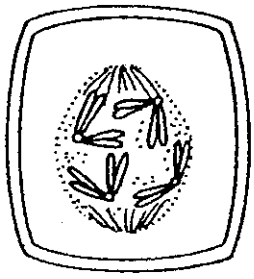

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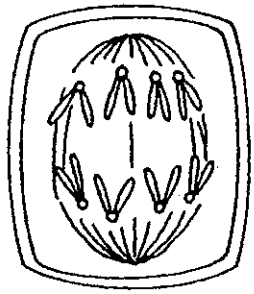

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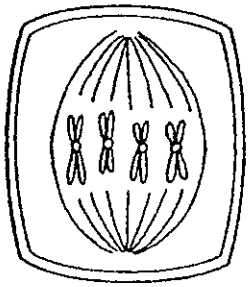

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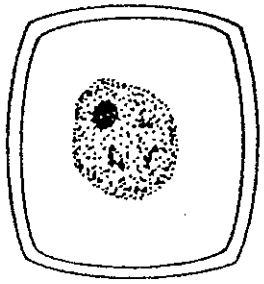

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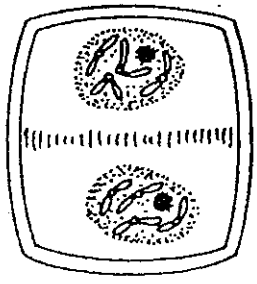

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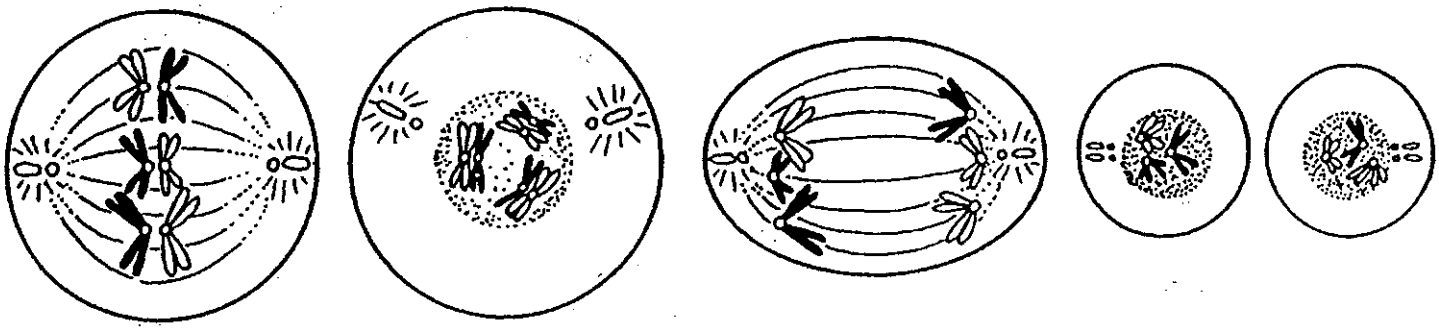
STAGES OF MEIOSIS

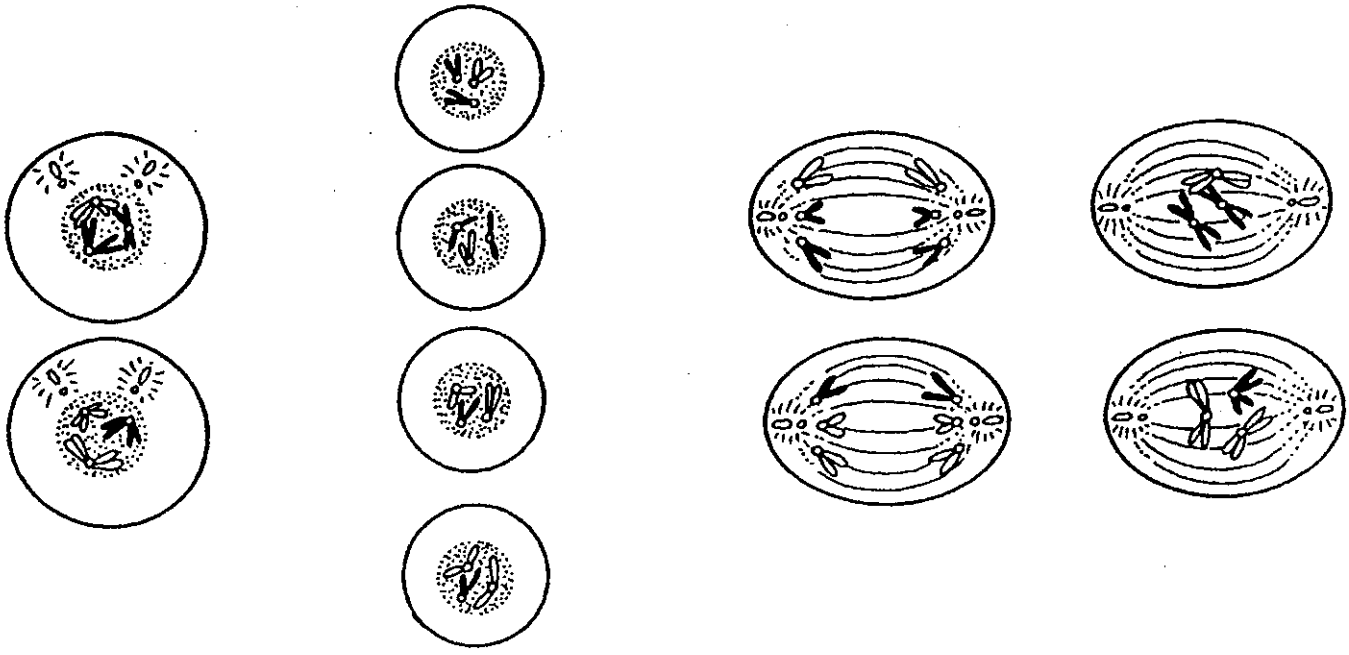
Name _____

Number the following diagrams of a first meiotic division in the proper order. Label each phase correctly as prophase I, metaphase I, anaphase I or telophase I.



_____ _____ _____ _____

Do the same for the diagrams of the second meiotic division. Label each phase correctly as prophase II, metaphase II, anaphase II, telophase II .



_____ _____ _____ _____

BIOLOGY CONCEPTS + CONNECTIONS Chapter 8

BOOK OF DAVID KROGH – CHAPTER 9, 10

CAMBELL / REECE CHAPTER 12 + 13

Audiolink pages 194–205 (Chapter 11)

Cell Division

Please read in your textbook, *Campbell's Biology*, Chapter 12- pages 215-224. This chapter offers a good overview of the processes of cell division. After reading this assignment, do the following activities:

Define the following terms in your notebook. You will need to learn these in order to understand the subject matter. You can find the definitions in the glossary of the Campbell text.

1. Identify the structural composition of each cell division structure .
2. Identify the location of each component of cell division.
3. Describe the function of each component of cell division.
4. Diagram the process of Mitosis and the process of Meiosis.
5. Explain cytokinesis.
6. Calculate chromosome number in each stage of Mitosis and do the same for Meiosis.
7. Contrast Mitosis in plants versus animals.
8. Contrast Meiosis and Mitosis.
9. Contrast Meiosis in females versus males.

Anaphase	Gamete	Mitosis
Cell Cycle	G1 Phase	M Phase
Centrioles	G2 Phase	Prophase
Centromere	Interphase	S Phase
Centrosomes	Kinetochore	Spindle
Chromatid	Meiosis	Somatic Cell
Chromosomes	Metaphase	Telophase
Cytokinesis	Metaphase Plate	

Be sure that you know and understand the following concepts:

A. Each stage of the cell cycle.

B. The different cell cycles for different types of cells.

C. The number of Chromosomes found in each stage of Mitosis and of Meiosis .

D. The purposes of mitosis and of Meiosis.

Audesirk: Pg 158+ Chapter 11

Please read in your textbook, *Campbell's Biology*, Chapter 12- pages 224-229. This chapter offers a good overview of the Cycles of Cell Division. After reading this assignment, do the following activities:

1. Define the following terms in your notebook. You will need to learn these in order to understand the subject matter. You can find the definitions in the glossary of the Campbell text.
2. Identify the different stages of the Cell Cycle .
3. Identify the Cell Cycle control Systems.
4. Describe the function of each Control Signal or Factor to direct the Cell Cycle .
5. Explain how benign tumors and cancer occur?
6. Explain cytokinesis.
7. Calculate chromosome number in each stage of the Mitosis Cell Cycle and do the same for the Meiosis Cell Cycle.

Anchorage Dependence	Density Dependent Inhibition	Meiosis
Benign Tumor	External Signals	Mitosis
Cancer	G0 Phase	Molecular Control Mechanisms
Cell Cycle	G1 Phase	M Phase
Cell Cycle Control System	G2 Phase	MPF

Checkpoints	Interphase	S Phase
Cyclin	Growth Factors	Somatic Cell
Cyclin Dependent Kinases Cdks	Internal Signals	Transformation
Cytoplasmic Chemical Signals	Malignant Tumor	
Cytokinesis	Metastasis	

Be sure that you know and understand the following concepts:

A. Each stage of the cell cycle.

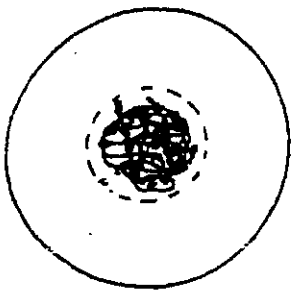
B. The different cell cycles for different types of cells

C. The purposes controlling mitosis and Meiosis.

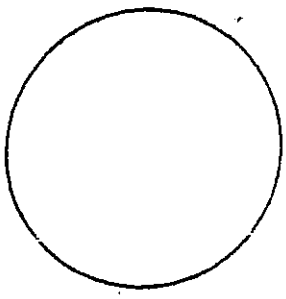
Name _____

Mitotic Division of a Diploid Cell

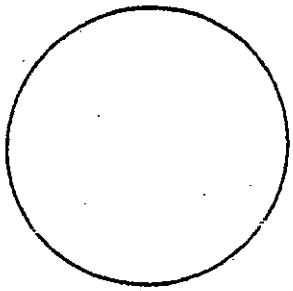
Diagram the stages of mitosis for a diploid Cell where 2n=6.



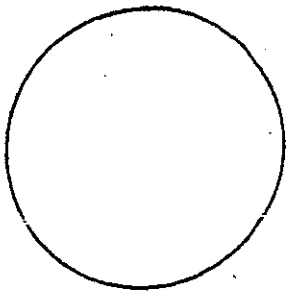
Interphase



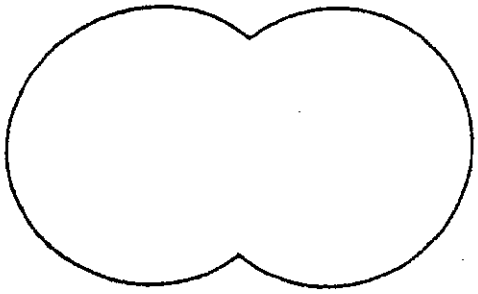
Prophase



Metaphase



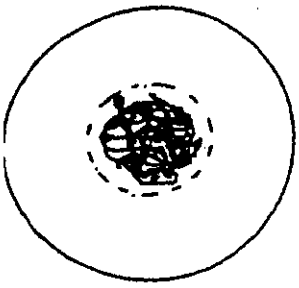
Anaphase



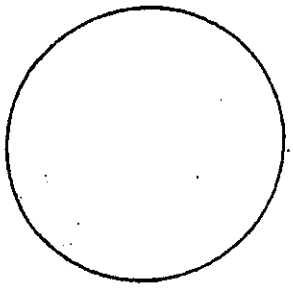
Telophase

Mitotic Division of a haploid Cell

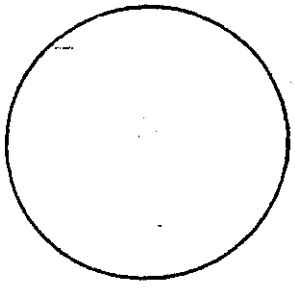
Diagram the stages of mitosis for a haploid Cell where n=4.



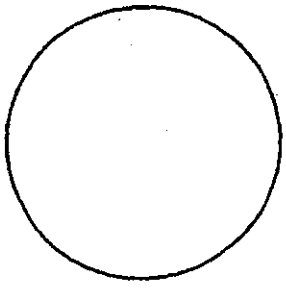
Interphase



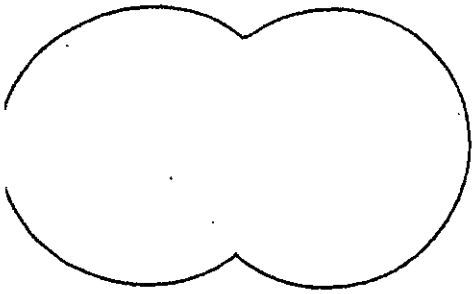
Prophase



Metaphase



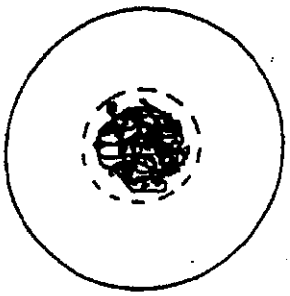
Anaphase



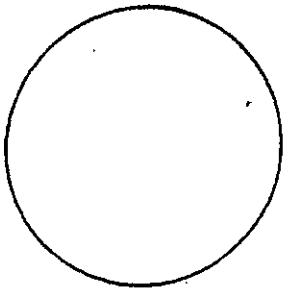
Telophase

Meiotic Division of a Diploid Cell

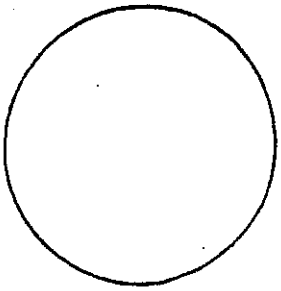
Diagram the stages of meiosis for a diploid Cell where 2n=6. Assume no independent assortment took place.



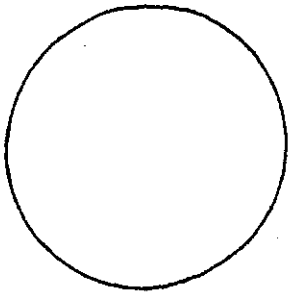
Interphase



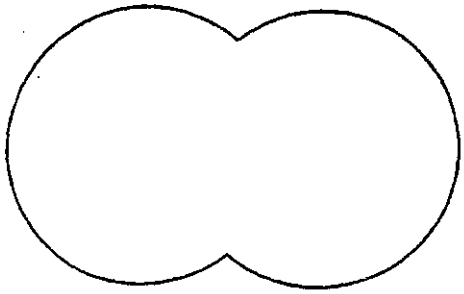
Prophase I



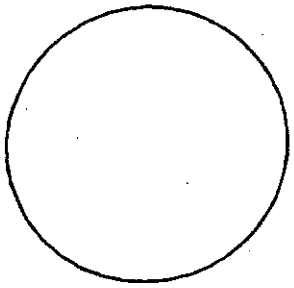
Metaphase I



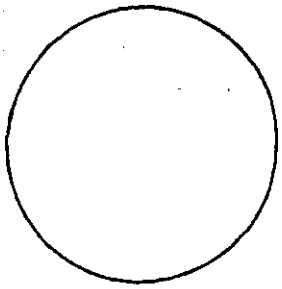
Anaphase I

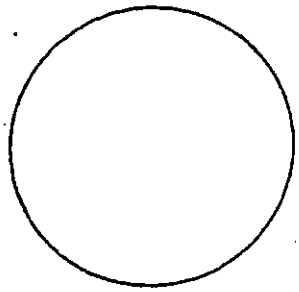


Telophase I - Prophase II

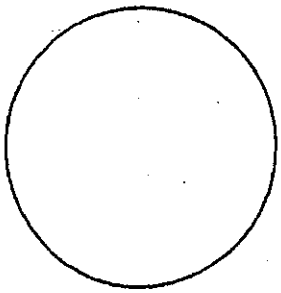


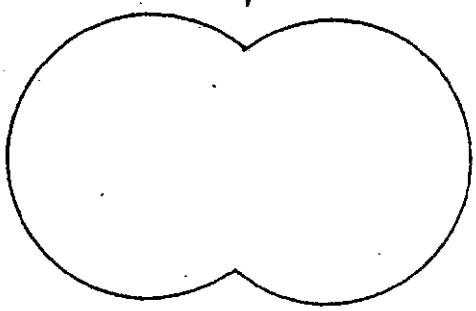
Metaphase II



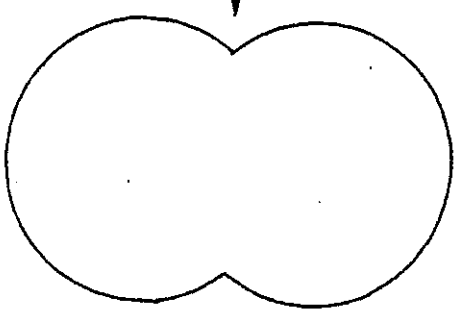


Anaphase II



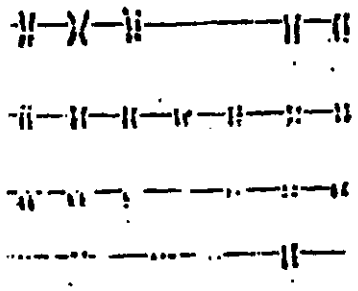


Telophase II



Chromosome Abnormality Syndromes

Down Syndrome (Trisomy 21)



Chromosome Complement:

- 94% of cases due to 21 trisomy
  47,XY, +21 or, 47,XX, +21
- 4% of cases due to translocation
  46,XY,t(14q 21q) or,
  46,XX,t(14q 21q)
  46,XY,t(21q 21q) or,
  46,XX,t(21q 21q)
  46,XY,t(21q 22q) or,
  46,XX,t(21q 22q)
- 2% of cases due to mosaicism
  46,XY/47,XY, +21 or,
  46,XX/47,XX, +21

Prevalence: 1:650 births

Key Clinical Features:

- Mental deficiency
- Hypotonia
- Upward slanting palpebral fissures epicanthal folds grayish spotting of the iris (Brushfield spots)
- Open mouth protruding tongue
- Short, wide head (brachycephaly) flat occiput
- Short, wide fingers (brachydactyly) curved little finger (clinodactyly) simian crease
- Cardiac abnormalities

Trisomy 18 Syndrome (Edwards Syndrome)

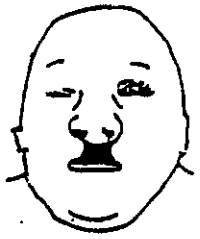
Chromosome Complement:
47,XX, +18 or, 47,XY, +18

Prevalence: 1:7000 births
3:1 female predilection

Key Clinical Features:

- Small for gestational age
- Polyhydramnios
- Single umbilical artery
- Elongated skull (dolichocephaly), prominent occiput
- Short palpebral fissures other eye anomalies
- Small lower jaw with recessed chin (micrognathia)
- Hypertonicity
- Overlapping fingers
- Rocker bottom feet
- Arches on six or more fingertips (low total ridge count)
- Mental deficiency
- Congenital heart defects

Trisomy 13 Syndrome (Patau Syndrome)



Chromosome Complement:
47,XX, +13 or, 47,XY, +13

Prevalence: 1:12000 births

Key Clinical Features:

- Microcephaly/sloping forehead impaired midline cleavage of the embryonic forebrain (holoprosencephaly)
- Microphthalmia/other eye anomalies
- Cleft lip/palate
- Postaxial polydactyly/flexion of fingers
- Cardiac abnormalities
- Mental deficiency

Turner Syndrome Monosomy X)

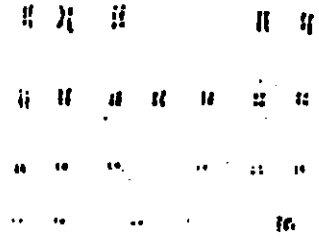
Chromosome Complement: 45,X

Prevalence: 1:2500 females
Many mosaics or structural aberrations of one X chromosome

Key Clinical Features:

- Small stature
- Transient congenital lymphedema with residual puffiness over the dorsum of the fingers and toes
- Low posterior hairline
- Shield chest widely spaced nipples
- Wide carrying angle of the forearm (cubitus valgus)
- Hyperconvex, deep-set nails
- Excessive pigmented nevi
- Streak ovaries
- Renal abnormalities
- Cardiac abnormalities (coarctation of aorta)

XXY Syndrome (Klinefelter Syndrome)



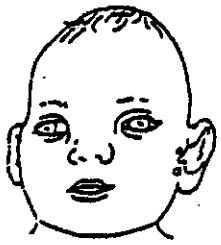
Chromosome Complement: 47.XXY

Prevalence: 1:500 males

Key Clinical Features:

- Hypogonadism
- Infertility
- Long limbs with low upper-to-lower segment ratio
- Sex chromatin positive

5p- Syndrome (Cri du Chat)



Chromosome Complement:

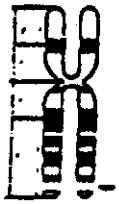
46.XX.del (5) (p ter → p12) or,
46.XY.del (5) (p ter → p12)

Prevalence: 1:50,000 births

Key Clinical Features:

- Cat-like cry (newborns only)
- Microcephaly
- Round face
- Hypertelorism
- Downward slanting of the palpebral fissures
- Epicanthal folds
- Mental deficiency
- Cardiac abnormalities

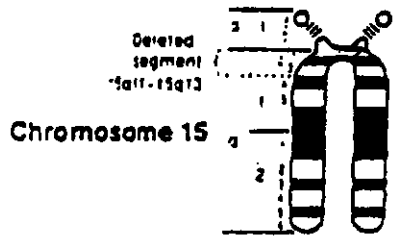
Syndrome of Macroorchidism and Sex-Linked Mental Deficiency (Fragile X, Martin-Bell Syndrome)



Key Clinical Features:

- Mental retardation
- Prominent forehead
- Large, simple ears
- Protrusion of the jaw (prognathism)
- Large testes after puberty

Prader-Willi Syndrome

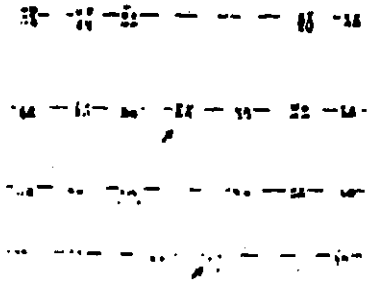


Basic Defect: Interstitial deletion of 15q11 to 15q13

Key Clinical Features:

- Small stature with especially small hands and feet
- Obesity
- Mental deficiency
- Hypotonia
- Hypogonadism
- Upslanting palpebral fissures, strabismus
- Narrow bifrontal diameter

Philadelphia Chromosome



Chromosome Complement:

46.XX, t (9q; 22q) or,
46.XY, t (9q; 22q)

The Philadelphia Chromosome abnormality (translocation of part of the long arm of a chromosome number 22 onto another chromosome, usually the long arm of a chromosome number 9) is present in 95% of patients with chronic myelogenous leukemia. These patients usually have a better prognosis than do those without this chromosome aberration.

Joan of Arc - Was It Really John of Arc?

Joan of Arc, the national heroine of France, was born during the Hundred Years War in 1412 in a village in northeastern France. At the age of 13 or 14 years she began to have visions that directed her to help fight the English at Orleans. Following victory she orchestrated the crowning of the new king, Charles VII. Joan was captured by the English during a siege of Paris, and in 1431 she was tried at Rouen for heresy. Although technically her trial was a religious one, conducted by the English-controlled church, it was clearly a political trial. Shortly after being sentenced to life imprisonment, she was declared a relapsed heretic, and on May 30, 1431 she was burned at the stake in the marketplace at Rouen.

In 1455 Pope Callistus III formed a commission to investigate the circumstances of her trial, and a new Trial of Rehabilitation took place over a period of 7 months in 1456. The second trial took testimony from over 100 persons who knew Joan personally.

Extensive documentation from the original trial and the Trial of Rehabilitation exists. The life and career of Joan of Arc has been told, retold, and interpreted in more than 100 plays and literally thousands of books. Although the story is well known, perhaps more remains to be told. For example, from an examination of the original evidence, R. B. Greenblatt has proposed that Joan had the array of physical symptoms associated with testicular feminization. By all accounts, Joan was a healthy female with well-developed breasts. Those living with her in close quarters testified that she never menstruated, and physical examination during her imprisonment indicated that she did not have pubic hair. While such circumstantial evidence is not enough for a diagnosis, it provides the basis for some interesting speculation about elevated testosterone levels and Joan's behavior. Undoubtedly it also provides a new stimulus to those medico/genetic detectives who prowl through history attempting to analyze the genetic makeup of the famous, the notorious and the obscure. □

HUMAN TRAITS EXERCISE

INTRODUCTION

Analyses of human traits offer genetic examples including complete dominance, incomplete dominance, codominance, and sex linkage. In this exercise you will examine the complexities and modes of inheritance of many human genetic traits.

Monogenetic characteristics

Analysis of human genetic characters is usually complex because many characters are influenced by multigene interactions and environmental factors. In this exercise we will concentrate on those characteristics determined by variations of a single gene without much environmental influence. You should be able to determine the phenotype and make good inferences (if not precise) about the genotypes controlling the characteristic. This will be particularly important when you analyze dominant phenotypes because you will not know by certain whether the second allele is also dominant or recessive for that particular characteristic under study.

Some of these characteristics are discussed below:

Ability to taste Phenylthiocarbamide (PTC)
Determined by a dominant allele T that seems to confer the ability to taste the chemical. Homozygous recessives for the allele lack the ability to taste PTC. In order to asses your tasting abilities, place a piece of control paper on your tongue and keep it there for at least 10 seconds. Then, place a piece of PTC paper in your mouth and keep it for 10 seconds. A distinct bitter taste will develop during this time if you are a taster. If you have to wonder if you taste or not, then you are a nontaster.

Ability to roll tongue
This characteristic is determined by a dominant gene R, that gives some people the ability to roll the tongue into a characteristic U shape.

Earlobes
Attached earlobes represent the homozygous recessive condition (gene f).

Interlocking fingers
Studies suggest that placing the left finger over the right when you cross your hand is due to a dominant gene L.

Bent little finger
Caused by a dominant gene B that causes the last joint of the little finger to bend inward toward the fourth finger. Lay your hands flat on the bench while relaxing the muscles to observe this phenotype.

Widow's Peak
Due to a dominant gene W, this shows in some people as a drop in the hairline downward forming a distinct point at the center of the forehead. Examine your front hairline to determine if you have this phenotype.

Hitchhiker's thumb (distal hyperextensibility of the thumb)
The expression of this trait is due to a recessive gene T with variable expressivity (only one thumb) with 5% reduction in penetrance.

Long palmar muscle
Homozygocity for the l gene determines the presence of long palmar muscle. This is detected by examining the tendons that run over the inside of the wrists. Close your fist tightly and flex your hand. If there are three tendons running into the wrist, you have the long palmar muscle. Otherwise, if you see two tendons with the large middle one will be missing, then you do not have the muscle.

Mid digital hair
Presence of hair on the second (middle) joint of one or more fingers is due to a dominant allele M.

The following chart shows the inheritance pattern of a number of known traits in human beings that are single locus traits. Underline your phenotype and possible genotype for each trait. Then circle the phenotype that is the most frequent in the population (your class).

	PHENOTYPE		
Trait	Dominant	Recessive	Possible Genotype
1. PTC tasting	Taster	Nontaster	TT Tt tt
2. Tongue rolling	Roller	Nonroller	RR Rr rr
3. Earlobes	Free	Attached	FF Ff ff
4. Sweat Glands (sex-linked recessive)	Present	Absent	SS Sa aa S_ a_
5. Rh Factor	Rh+	Rh-	Rh+Rh+ Rh+rh- rh-rh-
6. Blood Type	A, B, AB	O	AA, AO, BB, BO, AB, OO
7. Hair Color	Dark	Light	HH Hh hh
8. Hair Color	Nonred	Red	NN Nn nn
9. Dexterity	Right-handed	Left-handed	RR Rl ll
10. Interlocking Fingers	Left over Right	Right over Left	LL Lr rr
11. Bent Little Finger	Bent	Straight	BB Bb bb
12. Double Joints in Fingers	Present	Absent	DD Dd dd
13. Albinism	Normal	Albino	NN Na aa
14. Widow's Peak	Present	Absent	WW Ww ww
15. Curly Hair (incomplete dominance)	Curly, Wavy	Straight	CC CS SS
16. Dimples	Present	Absent	DD Dd dd
17. Nose Shape	Roman	Straight	EE Ee ee
18. Freckles	Present	Absent	FF Ff ff
19. Hitchhiker's thumb	Present	Absent	TT Tt tt
20. Color Blindness (sex-linked recessive)	Normal	Color Blind	CC Cc cc C_ c_

PHENOTYPE

Trait	Dominant	Recessive	Possible Genotype		
21. Long Palmar Muscle	Absent	Present	LL	Ll	ll
22. Mid Digital Hair	Present	Absent	MM	Mm	mm
23. White Forelock (sex-linked recessive)	Present	Absent	FF	Ff	ff
			F_		f_
24. Vision	Nearsighted	Normal	NN	Nn	nn
25. Vision	Farsighted	Normal	FF	Ff	ff
26. Vision	Astigmatism	Normal	AA	An	nn
27. Headaches	Migraine	No Migraine	MM	Mm	mm
28. Eyelashes	Long	Short	LL	Ls	ss
29. Fingers	Polydactylous	Normal	PP	Pn	nn
30. Fingers	Brachydactylous	Normal	BB	Bb	bb
31. Toes	Webbed	Normal	WW	Wn	nn
32. Inny-Outy Navel	Inny	Outty	II	Io	oo

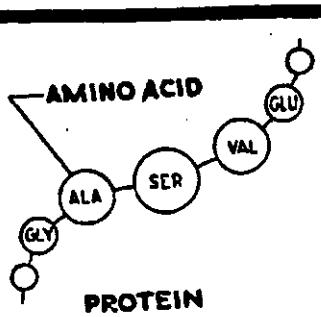
Now, construct a pedigree of your family that analyzes the inheritance of one of the traits shown in the previous table. Your pedigree should include your grandparents and their brothers and sisters in the first generation; your parents and their brothers and sisters in the second generation, and you and your brothers, sisters, and cousins in the third generation. Then, analyze your family pedigree and determine the mode of inheritance of the trait you selected.

Human Trait Analyzed _____

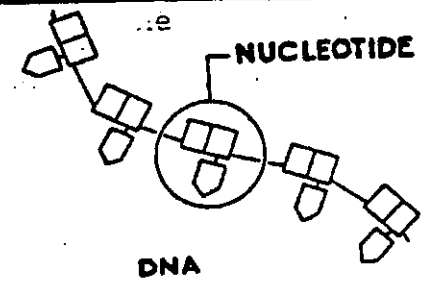
ORGANIC CHEMISTRY - WORKSHEET

TRANSCRIPTION OF DNA --> mRNA

1.



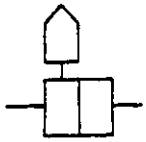
PROTEIN



DNA

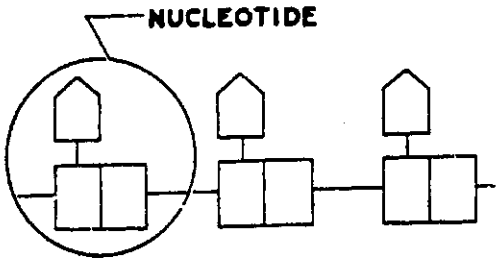
2. Just as proteins are chains of smaller molecules called _____ _____, so DNA molecules are chains of smaller molecules called _____.

AMINO ACID



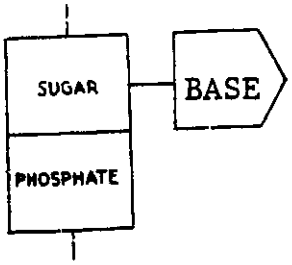
NUCLEOTIDE

3. Using the symbols above, draw a protein molecule and a DNA molecule-each consisting of three smaller molecules.



4. Since DNA is found in the nucleus, it is easy to remember that the smaller molecules hooked together to form DNA chains are called _____.

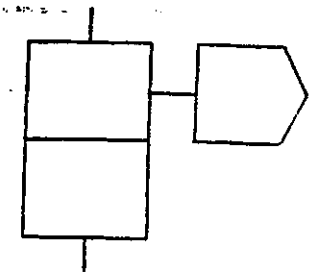
5. A simplified diagram of a DNA nucleotide is shown below:

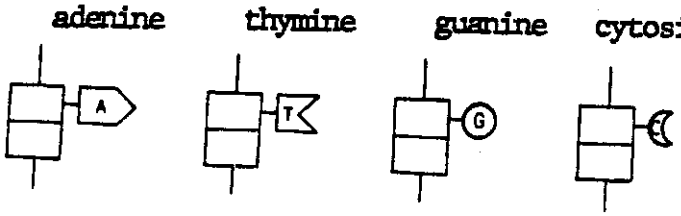


As shown, each nucleotide consists of three smaller chemical units:

(1)_____(2)_____(3)_____

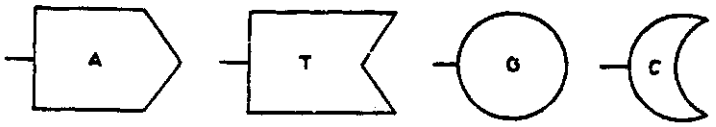
6. On the diagram at right, label the sugar, phosphate, and the base parts of the nucleotide.





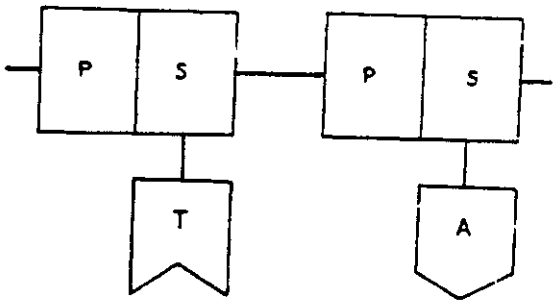
7. There are four main kinds of DNA nucleotides. All have the same _____ and _____ parts. Each has a different _____.

8. The chemical names for the (how many?) _____ different DNA bases are:
ADENINE, THYMINE, GUANINE, AND CYTOSINE.
We will use the first letter of these chemical names to stand for each base.



9. The symbols we will use for each of the four different bases found in DNA nucleotides are _____, _____, _____ and _____.

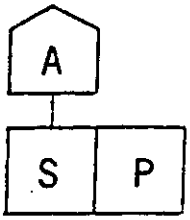
10. When two nucleotides hook together to start a DNA chain, the sugar part of one hooks to the phosphate part of another, thus:



Draw a horizontal nucleotide chain that includes all 4 different nucleotides, using the 4 different base symbols. Make sure the nucleotides are properly hooked together to form a chain.

11. The 4 different nucleotides in a DNA chain may be arranged in any order, and the chain may contain different numbers of nucleotides up to several thousand.

Complete the diagram of a DNA chain below showing the bases arranged horizontally in the order AGCTCT.



12. Just as different proteins are formed from different arrangements of about 20 _____, so different DNA molecules are formed from different arrangements of 4 _____.

DEOXY RIBO NUCLEIC ACID (DNA)
RIBO NUCLEIC ACID (RNA)

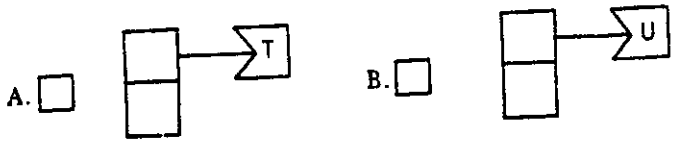
13. The names written above show that the names for RNA and DNA both end in _____ _____, and both contain the stem _____ referring to the sugar they contain.

14. The only difference between the names for RNA and DNA is that DNA's name begins with the 5-letter prefix _____.

15. "Deoxy" means "less oxygen," and so if we remove oxygen from ribonucleic acid (RNA) we have _____ ribonucleic acid, or _____.

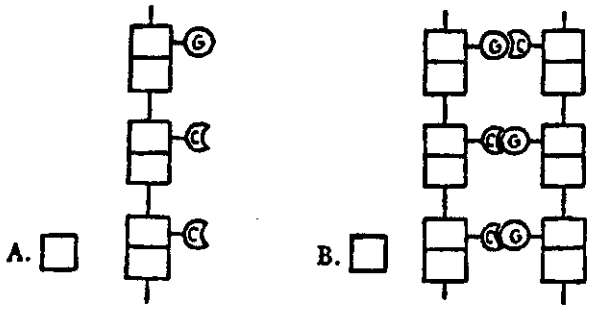
16. A second difference between RNA and DNA is that in RNA the base uracil is substituted for the DNA base thymine.

Check the nucleotide below that is RNA. Is it diagram A or B ?



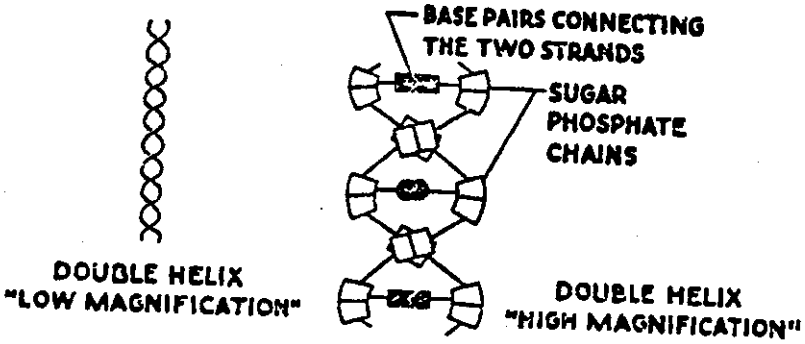
17. A difference between the structures of RNA and DNA is that RNA is found only as a SINGLE strand of nucleotides, while DNA in chromosomes occurs as two strands hooked together at their bases.

Check the drawing below (A or B) that represents a DNA molecule, and indicate why.



Why?_____

18. A helical shape of DNA with the base pairs forming a ladder between the double chains was hypothesized by J.D. Watson and F.H.C. Crick in 1953. In 1962 these brilliant scientists won a Nobel Prize for their work. It is due to their work that we think DNA molecules are twisted into the shape of a double_____.

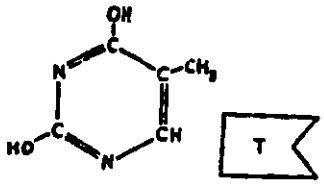


19. "Messenger RNA" is of intermediate molecular weight and is thought to be the "messenger" that carries DNA's code from the nucleus to the cytoplasm where protein is produced.

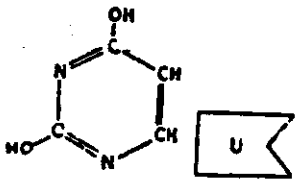
RNA of intermediate molecular weight is called _____ _____RNA. Its function is _____
_____

20. The messenger that carries DNA's base-coded information on protein production from the nucleus to the ribosome "assembly line" is a chemical similar to DNA. It is called _____.

21. We might now ask how messenger RNA carries DNA's "message." The series of amino acids found in each protein is determined by the series of _____ along the DNA molecule. A copy of that DNA series is carried from the nucleus to the ribosome by _____.

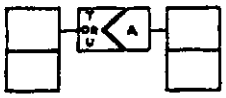


THYMINE



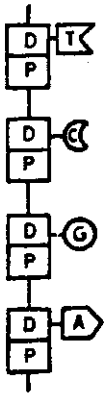
URACIL

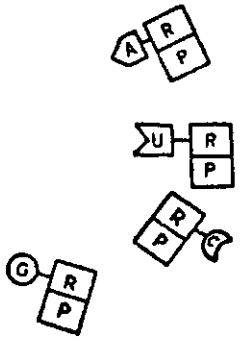
22. RNA contains the base uracil (U) instead of thymine (T). But the structure of U and T are so similar that a symbol of one shape is used for both.



Because of their different structures, the base A of DNA and the base U of RNA can pair up as shown at left.

Single nucleotides of RNA are always available in the nucleus. If the RNA bases fit, they may lock together with the DNA bases. Draw each RNA base below as it would lock into the four DNA bases in the chain at left.





23. When all RNA bases have locked into place against the DNA bases, the sugar and phosphates of the RNA join together. The new RNA molecule so formed separates from the DNA chain. This RNA molecule, which corresponds to the bases of DNA, is specifically called _____ RNA, and its function is to carry DNA's base code or message to the ribosome where _____ is produced.

24. Arrange the following items in order (1,2,3,4) to show how messenger RNA is made corresponding to DNA:

_____ a. Messenger RNA unlocks from the DNA and moves into the cytoplasm.

_____ b. RNA nucleotides lock into place as A and T or U pair and as G and C pair.

_____ c. The sugar and phosphates of the RNA nucleotides hook up to form a long messenger RNA molecule.

_____ d. DNA chain with bases exposed and single RNA nucleotides available in nucleus.

25. The messenger RNA, separated from the DNA chain and carrying DNA's information, moves from the nucleus into the cytoplasm. What does it do after that? _____

_____

26. Just as the boss in a big office building sends a foreman to supervise the building at a construction site, so the boss in the nucleus, _____, sends its foreman _____ to supervise the building of a protein at construction sites called _____.

27. When messenger RNA (carrying messages on protein production obtained from DNA's base code) has attached to a ribosome, the stage is finally set for the actual production of protein.

Provided by : Donald Maser 97-2

Genghis Khan a Prolific Lover, DNA Data Implies

Hillary Mayell
for National Geographic News
February 14, 2003

Genghis Khan, the fearsome Mongolian warrior of the 13th century, may have done more than rule the largest empire in the world; according to a recently published genetic study, he may have helped populate it too.

An international group of geneticists studying Y-chromosome data have found that nearly 8 percent of the men living in the region of the former Mongol empire carry y-chromosomes that are nearly identical. That translates to 0.5 percent of the male population in the world, or roughly 16 million descendants living today.

The spread of the chromosome could be the result of natural selection, in which an extremely fit individual manages to pass on some sort of biological advantage. The authors think this scenario is unlikely. They suggest that the unique set of circumstances surrounding the establishment of the Mongol empire led to the spread.

"This is a clear example that culture plays a very big role in patterns of genetic variation and diversity in human populations," said geneticist Spencer Wells, one of the 23 co-authors of the paper. "It's the first documented case when human culture has caused a single genetic lineage to increase to such an enormous extent in just a few hundred years."

Legacy of Genghis Khan

To have such a startling impact on a population required a special set of circumstances, all of which are met by Genghis Khan and his male relatives, the authors note in the study published in the *American Journal of Human Genetics.*

Khan's empire at the time of his death extended across Asia, from the Pacific Ocean to the Caspian Sea. His military conquests were frequently characterized by the wholesale slaughter of the vanquished. His descendants extended the empire and maintained power in the region for several hundred years, in civilizations in which

harems and concubines were the norm. And the males were markedly prolific.

Khan's eldest son, Tushi, is reported to have had 40 sons. Documents written during or just after Khan's reign say that after a conquest, looting, pillaging, and rape were the spoils of war for all soldiers, but that Khan got first pick of the beautiful women. His grandson, Kubilai Khan, who established the Yuan Dynasty in China, had 22 legitimate sons, and was reported to have added 30 virgins to his harem each year.

"The historically documented events accompanying the establishment of the Mongol empire would have contributed directly to the spread of this lineage," the authors conclude.

Tracking the Y-Chromosome

The study looked at blood samples collected over a period of ten years from more than 40 populations living in and around the former Mongol empire. Geneticists use the Y-chromosome in population studies such as this because it doesn't recombine as other parts of the genome do. When it comes to eye color, or height, or resistance or susceptibility to particular diseases, each parent contributes half of a child's DNA, which join together to form a new genetic combination.

The Y-chromosome is passed on as a chunk of DNA from father to son, basically unchanged through generations except for random mutations.

These random mutations, which happen naturally and are usually harmless, are called markers. Once the markers have been identified, geneticists can go back in time and trace them to the point at which they first occurred, defining a unique lineage of descent.

In this particular instance, the lineage originated 1,000 years ago. The authors aren't saying that the genetic mutations defining the lineage originated with Khan, who was born around 1162; they are more likely to have been passed on to him by a great great grandfather.

The lineage was found in only one population outside of the former Mongolian empire, in Pakistan.

"The Hazaras [of Pakistan] gave us our first clue to the connection with Genghis Khan," said Wells. "They have a long oral tradition that says they're his direct descendants."

Of course, the connection to Genghis Khan will never be a certainty unless his grave is found and his DNA could be extracted. Until then, geneticists will continue to seek out isolated populations in the hope of unraveling the mysteries of geographic origin and relatedness told by our genes.

The New York Times
nytimes.com

March 18, 2008

BASICS

In Most Species, Faithfulness Is a Fantasy

By NATALIE ANGIER

You can accuse the disgraced ex-governor Eliot Spitzer of many things in his decision to flout the law by soliciting the services of a pricey prostitute: hypocrisy, egomania, sophomoric impulsiveness and self-indulgence, delusional ineptitude and boneheadedness. But one trait decidedly not on display in Mr. Spitzer's splashy act of whole-life catabolism was originality.

It's all been done before, every snickering bit of it, and not just by powerful "risk-taking" alpha men who may or may not be enriched for the hormone testosterone. It's been done by many other creatures, tens of thousands of other species, by male and female representatives of every taxonomic twig on the great tree of life. Sexual promiscuity is rampant throughout nature, and true faithfulness a fond fantasy. Oh, there are plenty of animals in which males and females team up to raise young, as we do, that form "pair bonds" of impressive endurance and apparent mutual affection, spending hours reaffirming their partnership by snuggling together like prairie voles or singing hooty, doo-wop love songs like gibbons, or dancing goofily like blue-footed boobies.

Yet as biologists have discovered through the application of DNA paternity tests to the offspring of these bonded pairs, social monogamy is very rarely accompanied by sexual, or genetic, monogamy. Assay the kids in a given brood, whether of birds, voles, lesser apes, foxes or any other pair-bonding species, and anywhere from 10 to 70 percent will prove to have been sired by somebody other than the resident male.

As David P. Barash, a professor of psychology at the University of Washington in Seattle, put it with Cole Porter flair: Infants have their infancy; adults, adultery. Dr. Barash, who wrote "The Myth of Monogamy" with his psychiatrist-wife, Judith Eve Lipton, cited a scene from the movie "Heartburn" in which a Nora Ephronesque character complains to her father about her husband's philanderings and the father quips that if she'd wanted fidelity, she should have married a swan. Fat lot of good that would have done her, Dr. Barash said: we now know that swans can cheat, too. Instead, the heroine might have considered union with Diplozoon paradoxum, a flatworm that lives in gills of freshwater fish. "Males and females meet each other as adolescents, and their bodies literally fuse together, whereupon they remain faithful until death," Dr. Barash said. "That's the only species I know of in which there seems to be 100 percent monogamy." And where the only hearts burned belong to the unlucky host fish.

Even the "oldest profession" that figured so prominently in Mr. Spitzer's demise is old news. Nonhuman beings have been shown to pay for sex, too. Reporting in the journal Animal Behaviour, researchers from Adam Mickiewicz University and the University of South Bohemia described transactions among great grey shrikes, elegant raptorlike birds with silver capes, white bellies and black tails that, like 90 percent of bird species, form pair bonds to breed. A male shrike provisions his mate with so-called nuptial gifts: rodents, lizards, small birds or large insects that he

impales on sticks. But when the male shrike hankers after extracurricular sex, he will offer a would-be mistress an even bigger kebab than the ones he gives to his wife — for the richer the offering, the researchers found, the greater the chance that the female will agree to a fly-by-night fling.

In another recent report from the lubricious annals of Animal Behaviour entitled "Payment for sex in a macaque mating market," Michael D. Gumert of Hiram College described his two-year study of a group of longtailed macaques that live near the Rimba ecotourist lodge in the Tanjung Puting National Park of Indonesia. Dr. Gumert determined that male macaques pay for sex with that all-important, multipurpose primate currency, grooming. He saw that, whereas females groomed males and other females for social and political reasons — to affirm a friendship or make nice to a dominant — and mothers groomed their young to soothe and clean them, when an adult male spent time picking parasites from an adult female's hide, he expected compensation in the form of copulation, or at the very least a close genital inspection. About 89 percent of the male-grooming-female episodes observed, Dr. Gumert said in an interview from Singapore, where he is on the faculty of Nanyang Technological University, "were directed toward sexually active females" with whom the males had a chance of mating.

Significantly, males adjust their grooming behavior in a distinctly economic fashion, paying a higher or lower price depending on the availability and quality of the merchandise and competition from other buyers. "What led me to think of grooming as a form of payment was seeing how it changed across different market conditions," Dr. Gumert said. "When there were fewer females around, the male would groom longer, and when there were lots of females, the grooming times went down." Males also groomed females of high rank considerably longer than they did low-status females with nary a diamond to their page.

Commonplace though adultery may be, and as avidly as animals engage in it when given the opportunity, nobody seems to approve of it in others, and humans are hardly the only species that will rise up in outrage against wantonness real or perceived. Most female baboons have lost half an ear here, a swatch of pelt there, to the jealous fury of their much larger and toothier mates. Among scarab beetles, males and females generally pair up to start a family, jointly gathering dung and rolling and patting it into the rich brood balls in which the female deposits her fertilized eggs. The male may on occasion try to attract an extra female or two — but he does so at his peril. In one experiment with postmatrimonial scarabs, the female beetle was kept tethered in the vicinity of her mate, who quickly seized the opportunity to pheromonally broadcast for fresh faces. Upon being released from bondage, the female dashed over and knocked the male flat on his back. "She'd roll him right into the ball of dung," Dr. Barash said, "which seemed altogether appropriate."

In the case of the territorial red-backed salamander, males and females alike are inclined to zealous partner policing and will punish partners they believe to have strayed: with threat displays, mouth nips and throat bites, and most coldblooded of all, a withdrawal of affection, a refusal to engage. Be warned, you big lounge lizard: it could happen to you.

YAHOO! NEWS

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Indian DNA links to 6 'founding mothers'

AP Associated Press

By MALCOLM RITTER, AP Science Writer
Thu Mar 13, 10:33 AM ET

Nearly all of today's Native Americans in North, Central and South America can trace part of their ancestry to six women whose descendants immigrated around 20,000 years ago, a DNA study suggests.

Those women left a particular DNA legacy that persists to today in about about 95 percent of Native Americans, researchers said.

The finding does not mean that only these six women gave rise to the migrants who crossed into North America from Asia in the initial populating of the continent, said study co-author Ugo Perego.

The women lived between 18,000 and 21,000 years ago, though not necessarily at exactly the same time, he said.

The work was published this week by the journal PLoS One. Perego is from the Sorenson Molecular Genealogy Foundation in Salt Lake City and the University of Pavia in Italy.

The work confirms previous indications of the six maternal lineages, he said. But an expert unconnected with the study said the findings left some questions unanswered.

Perego and his colleagues traced the history of a particular kind of DNA that represents just a tiny fraction of the human genetic material, and reflects only a piece of a person's ancestry.

This DNA is found in the mitochondria, the power plants of cells. Unlike the DNA found in the nucleus, mitochondrial DNA is passed along only by the mother. So it follows a lineage that connects a person to his or her mother, then the mother's mother, and so on.

The researchers created a "family tree" that traces the different mitochondrial DNA lineages found in today's Native Americans. By noting mutations in each branch and applying a formula for how often such mutations arise, they calculated how old each branch was. That indicated when each branch arose in a single woman.

The six "founding mothers" apparently did not live in Asia because the DNA signatures they left behind aren't found there, Perego said. They probably lived in Beringia, the now-submerged land bridge that stetched to North America, he said.

Connie Mulligan of the University of Florida, an anthropolgist who studies the colonization of the Americas but didn't participate in the new work, said it's not surprising to trace the mitochondrial DNA to six women. "It's an OK number to start with right now," but further work may change it slightly, she said.

That finding doesn't answer the bigger questions of where those women lived, or of how many people left Beringia to colonize the Americas, she said Thursday.

The estimate for when the women lived is open to question because it's not clear whether the researchers properly accounted for differing mutation rates in mitochondrial DNA, she said. Further work could change the estimate, "possibly dramatically," she said.

On the Net:

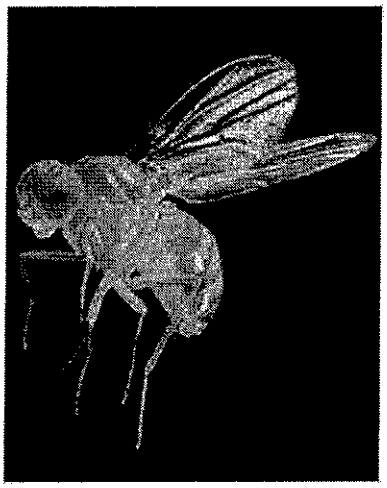
PLoS One: http://www.plosone.org

February 5, 2008, 9:14 pm

A Mutant Obsession

This week I'm introducing the first article in what will be an occasional series about mutation. And yes, I admit it: I'm obsessed with mutation (which is why I've already alluded to it in a couple of earlier articles). The reason is that mutations to DNA form the raw material for evolution. It's wondrous to think that mutations, accumulated over billions of years through the action of natural selection and the other forces of evolution, have produced such diverse life forms as vampire squid, coconut palms, death cap mushrooms, giant Gippsland earthworms, Etruscan pygmy shrews, *E. coli* — and us.

First things first: what are mutations? They are accidental changes to an organism's DNA; they typically happen when the cellular machinery makes a mistake as it copies DNA from one cell to the next. Once a mutation happens, it may or may not be preserved down the generations — whether it is depends on a variety of factors, not least natural selection.



A fruit fly. Photo Credit: Richard T. Nowitz/Photo Researchers Inc.

Early geneticists didn't know about DNA, so they thought of mutations in terms of how they changed an organism's appearance — a fruit fly with white eyes instead of red, or no bristles on the insides of its legs, peas that were wrinkly rather than smooth. But these days, mutations are typically classified by the effect they have on DNA. It turns out that there are many different types of mutation, with many different kinds of effects — and they contribute to the evolutionary process in surprisingly diverse ways.

Some are more common in plants; others, in animals. Some are more likely to occur in eggs; others, in sperm. Some have immediate effects; others don't — but instead have a substantial impact on long-term evolutionary potential. And in my view, it's only by appreciating the full diversity of mutations that the evolution of countless different life forms becomes comprehensible. The aim of the series, then, is to survey the mutational landscape.

The canonical mutation — the one everyone learns about in biology class — alters the part of a gene that contains the instruction to make a protein. In case you don't have an intuitive feeling for what proteins are, the easiest way to think about them is as small objects, each with a shape and a function — just as a teacup has a shape for holding tea. Proteins are built out of a kind of molecular Lego; the bits of Lego that are required are listed in the gene. So a mutation to the protein-coding part of a gene often has the effect of altering the protein's shape — like substituting a big Lego brick for a small one, or one with a hinge for one that's rigid. Such shape changes often affect how a protein does its job.

Mutations of this type tend to have an immediate effect on the organism, affecting its health, behavior, or looks. For instance, if you compare oldfield mice (*Peromyscus polionotus*) that live on the Florida mainland with their cousins that scamper around the sand dunes on the nearby Santa Rosa Island, the first thing you notice is that mainland mice have dark fur, and beach mice have pale, sandy-colored fur. A significant part of the difference in fur color has been traced to a single alteration in the DNA sequence of a gene called *Melanocortin-1 receptor*. The mutation alters the shape of the corresponding protein, and this interferes with the mouse's ability to produce a dark pigment. (To pick up last week's theme of evolution repeating itself at the genetic level, mutations to *Melanocortin-1 receptor* are also implicated in changes to hair and skin color in humans and (probably) Neanderthals, and to feather color in the lesser snow goose and the Arctic skua.)

Mutations that alter proteins have been linked to specific changes in a huge number of traits in organisms from bacteria to humans. Yet the proportion of a genome that contains the instructions to make proteins is tiny; in humans, it may be less than 2 percent. So there's a lot of other DNA that will experience mutations. The question is, what might such mutations do?

Here's one possibility. We know that some of that 98 percent is involved not in making proteins, but in regulating where and when the genes they are made from will get switched on. The biology of this gets pretty complicated — but what it amounts to is that most genes have an elaborate control region — a set of on/off switches officially known as cis-regulatory elements. When the right switches are on, the protein gets made; when they are off, it doesn't. So mutations to the switches can

alter how the protein is deployed. Then, the protein stays the same shape as it was before, but instead of being made in, say, just the liver, it starts being made somewhere else as well.

I touched on this in passing last week when I discussed the ghostly pelvis of certain freshwater sticklebacks. To recap briefly, most sticklebacks have an elaborate pelvic structure that includes a pair of spines. The structure grows as the fish develops; its growth is regulated by a gene called *Pitx1*. In some lakes, sticklebacks lack the pelvis: at the time the pelvis should form, *Pitx1* does not get expressed in the pelvic region, owing to mutations in the switches for the gene. A reader kindly pointed out a second example: the ability for human adults to digest milk. In most human populations, the ability to digest milk disappears in infancy, because the gene that enables milk digestion gets switched off. But in several different populations with a long history of herding cows, mutations to the switch allow the gene to remain on throughout a person's life. And here's one more: the fancy patterns of dark spots that some flies have on their wings. Again, having spots, or not, is not determined by a mutation to a protein that makes the pigment, but to one of its switches.

So how common are these mutations? We don't know yet. There are a large number of putative examples, but few have been conclusively proven. Tracking these mutations down is much more difficult than tracking down mutations that affect a protein's shape. One reason is that the regions of the genome that contain the instructions for making proteins are easy to spot — we know how to recognize them by looking at DNA sequences — so finding mutations in them is easy.

But we're still figuring out how to recognize switches by inspecting DNA sequences, so finding them is laborious. And the job is made more complicated by the fact that most genes have multiple switches, some of which may be far away from the gene itself. Worse still, whereas sequences that encode proteins often evolve rather slowly, so you can sometimes recognize the same gene in humans and jellyfish, switches appear to evolve fast, so the switches for a gene in one species may not look at all the same in the next.

Nonetheless, I'm betting that a substantial number of traits do evolve through this type of mutation. Altering the way a gene is turned on or off has the advantage of leaving the protein itself unaffected. Since many

changes to a protein are deleterious — they interfere with the protein's ability to do its job — it may sometimes be the case that tweaking how and where a protein is used results in less disruption to the organism. Moreover, with so little DNA given over to making proteins, and with long stretches of DNA given over to regulating them, it seems likely that mutations often happen in the regulatory regions, and that sometimes they will be naturally selected.

Another reason for my bet is that while the number of genes that different animals have does not vary nearly as widely as everyone expected, and does not seem to correlate closely with how complex organisms are, the complexity of gene regulation differs greatly from one group to the next. This allows the same basic set of proteins to be used in ever more elaborate ways.

Which leads me to wonder about the deep past. Millions of years ago, organisms began to evolve that, instead of having one cell, had many; and over time, those cells began to differentiate and take on separate roles. Perhaps regulatory mutations of the kind I've described here played an essential role in making this transition, allowing the evolution of organisms that are built from more than one cell. Allowing, in other words, the magnificent diversity of life.

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YAHOO! NEWS

Back to Story - Help

Gene Plays Key Role in Stopping Spread of Some Cancers

HealthDay

By Ed Edelson
HealthDay Reporter
Wed Jan 4, 5:04 PM ET

WEDNESDAY, Jan. 4 (HealthDay News) -- Researchers have found a genetic reason for the aggressiveness of some cancers, and perhaps a pathway toward taming their spread.

A gene that tells a cell to commit suicide if it wanders into the wrong part of the body gets silenced in some cancer cells, claims a report in the Jan. 5 issue of Nature. Those cells can grow in any tissue, however foreign, in a process called metastasis.

"It's not the growth of the primary tumor that kills," explained David Cheresh, associate director for translational research at the University of California at San Diego Moores Cancer Center. "Growth of metastatic tumors does kill."

The gene involved is called caspase 8. Cheresh and his colleagues previously have shown that it acts as a police officer, making sure that skin cells stay in the skin, liver cells in the liver, and so on. When a cell migrates to the wrong location, caspase 8 activates molecules called integrins that, in effect, tell the cell to commit suicide.

But experiments show that caspase 8 doesn't do its job in some cancer cells. The researchers found limited or no caspase 8 activity in metastases of a childhood cancer called neuroblastoma, and they have evidence that the same thing happens in other kinds of cancer.

Loss or suppression of caspase 8 is seen in about 70 percent of small cell lung cancers, 10 percent of colon cancers and 35 percent of medulloblastomas, the researchers said. It is seen in 70 percent of aggressive neuroblastomas in children.

The finding has some important medical implications, Cheresh said. Tests for caspase 8 function could be used to help guide treatment, he noted: "If it is missing, those are the most aggressive tumors, and [they] need to be treated early and aggressively."

Another clinical possibility is that caspase 8 activity could be manipulated to make cancer cells initiate apoptosis, the process of cellular suicide. "What we hope we can do is determine ways of initiating apoptosis in these invasive cells," Cheresh said.

The discovery casts light on a different way the body can attack cancer, said Marcus E. Peter, a professor of cancer biology at the University of Chicago Ben May Institute for Cancer Research. In 1995, his group first described the role of caspase 8 in programmed cell death.

"This has been viewed entirely as an immune system process," Peter said. "Tumor cells are recognized by the immune system, immune system cells move in, dock on the tumor and kill it. Here, integrins mediate death through caspase 8, with no role for the immune system."

The good news in the discovery is that "for the most part, tumors don't get rid of this gene entirely," Peter said. "They silence it through a process called methylation. There are a number of drugs that can block this methylation."

Whatever the ultimate impact of the finding on medical practice, "it gives us a molecular insight into the difference between cells that are metastatic and those that are not," Cheresh said.

More information

A constantly updated list of cancer-related genes is kept by the Sanger Institute.

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Articles

Main Reports Page

Are Cell Phones Causing Cancer?

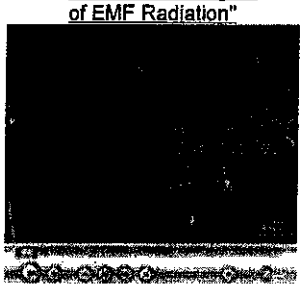
by Taraka Serrano

In 1993, a man filed a lawsuit against the cell phone industry, claiming that his wife died from a brain tumor caused by her repeated use of the cell phone. The tumor was on the same side of the head where she held her cell phone and was shaped like the cell phone antenna. The case got widespread media attention and was featured in CNN's Larry King show.

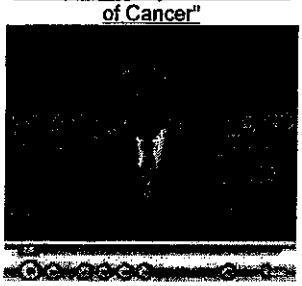
Although the claim was dismissed by the court due to lack of sufficient evidence, it was a public relations nightmare for the wireless industry. It also marked the beginning of the global search for a definitive answer to the question: are cell phones safe or not? Does it cause cancer and other degenerative diseases? **Brain cancer is up 25% since cell phones became popular. Every year, there are 183,000 more cases in the US alone.** Some health experts say there's a link with cell phone use, but is there proof?

In an effort to diffuse the negative publicity from the high-profile lawsuit, the cell phone industry itself funded a $25 million dollar research program to prove that cell phones are safe. After 6 years of intensive research, however, the results were not what they were looking for. Dr. George Carlo, the chief research scientist of the program, found evidence that cell phones pose some health risks, possibly even cancer.

Watch video report:
"The Invisible Dangers of EMF Radiation"



Watch Sydney TV report:
"Brain Tumors Cause More Deaths than Any Other Forms of Cancer"



The first evidence of cancer link that shook the cell phone industry came in 1997. Dr. Michael Repacholi and his colleagues from the Royal Adelaide Hospital in South Australia reported that long-term exposure to the type of radiation that comes from digital cell phones caused **an increase in the occurrence of lymphoma** in mice. The study received widespread international media attention because it was the first time that cancer has been linked to the cell phone in a well-conducted study.

THE RED FLAGS: SOLVING THE CANCER PUZZLE

In order to show a link between cell phone radiation and cancer, let's look at several studies Dr. Carlo investigated that made him blow the whistle, so to speak. These red-flag findings provide the pieces that fit together to form the cancer picture:

- DNA Damage in Human Blood Studies

- Breakdown in the Blood-Brain Barrier
- Studies of Tumors in People Who Use Cell Phones
- Studies of Cell Phone Radiation Dosage and Response

DNA DAMAGE IN HUMAN BLOOD
All tumors and all cancers are the result of genetic damage. Most often that damage includes the formation of **micronuclei**--fragments of chromosomes that form membranes around themselves and appear under a microscope as additional nuclei in blood cells (which normally have just a single nucleus). The relationship between micronuclei and cancer is so strong that doctors around the world test for their presence to identify patients likely to develop cancer. The presence of micronuclei indicates that the cells can no longer properly repair broken DNA. This deficiency is considered to be an indication of an increased risk of developing cancer.

- In December 1998, Drs. Ray Tice and Graham Hook of Integrated Laboratory Systems in North Carolina have shown that **blood cells exposed to cell phone radiation suffer genetic damage in the form of micronuclei.** In their studies, DNA and chromosome damage in human white blood cells occurred when exposed to signals from all types of phones--analog, digital, and PCS. Damage was shown even from signals occurring at a SAR level below the government's "safety" guideline.

- Using different methods, the above finding was confirmed by Dr. Joseph Roti Roti of Washington University in St. Louis in 2000. His research showed that human blood cells exposed to radiation at wireless phone frequencies did indeed develop genetic damage, in the form of micronuclei. This finding received a lot of notice because Dr. Roti Roti is a prominent scientist who does his work under funding by Motorola Inc.

This has a very serious implication. If cell phone radiation encourages the formation of micronuclei in blood cells, and **micronuclei are said to be "biological markers" for cancer**, then based on these studies alone cell phone use could be said to increase the risk of cancer.

BREAKDOWN IN THE BLOOD BRAIN BARRIER
The blood brain barrier is a special filter in the blood vessels of the brain that keeps dangerous chemicals from reaching sensitive brain tissue and causing DNA breaks and other damage.

- In 1994 and again, in 2002, Dr. Leif Salford from Lund University in Stockholm, Sweden found in his studies that **rats exposed to cell phone radiation showed a breakdown in the blood brain barrier, as well as areas of shrunken, damaged neurons.**

The micronuclei studies of Tice, Hook and Roti Roti and the blood-brain findings of Salford provide a two-step explanation for how cancer could be caused by cell phone radiation.

Step One: A leakage or breakdown in the blood brain barrier would provide a pathway for cancer-causing chemicals in the bloodstream (from tobacco, pesticides, air pollution, etc.) to leak into the brain and damage sensitive brain tissue that would otherwise be protected. These chemicals could break the DNA in the brain or cause other harm to reach those cells.

Step Two: While a number of studies showed that cell phone radiation by itself does not appear to break DNA, the micronuclei findings suggest that they do impair the DNA repair mechanisms in brain cells. Micronuclei result from a breakdown of the cell's ability to repair itself. **If the brain cells become unable to repair themselves, then carcinogenesis--the creation of tumors--induced by chemical toxins could begin.**

DNA carries the genetic material of an organism and its different cells. Any damage that

goes unrepaired affects the future generation of cells. The change has procreated and this mutation is seen as a possible cause of cancer.

TUMORS IN PEOPLE WHO USE CELL PHONES
Epidemiological studies, performed by different investigators using different methods, show some evidence of an increased risk of tumors among people who use cellular phones.

- In 1998, Dr. Ken Rothman of Eidemiology Resources, Inc. in Newton, Mass., did a study showing that users of handheld cell phones have more than twice the risk of dying from brain cancer than do car phone users--whose antennas are mounted on the body of the car, far removed from the users' heads.

- In 1998, Joshua Muscat, a research scientist from the American Health Foundation, showed in his study a **doubling of the risk of developing neuro-epithelial tumors on the outside of the brain among cell phone users**, particularly on the side of the skull where cell phone antennas are held during calls.

- Muscat also showed in another study that people who have used cell phones for six years or more have a **50-percent increase in risk of developing acoustic neuroma**, a benign tumor of the nerve that controls hearing and extends from the ear to the brain. Acoustic neuromas can cause hearing loss and can be life-threatening if untreated.

This was confirmed in a separate study in Stockholm, Sweden by Anders Ahlbom in 2004 and sponsored by the World Health Organization (WHO), which finds that people who have used cell phones, this time for at least 10 years, may have an increased risk of developing acoustic neuroma.

- In a study also requested by WHO, researchers headed by Dr. Lennart Hardell of the Orebro Medical Center in Sweden examined 1,617 patients aged between 20 and 80 who had been diagnosed with a brain tumour between 1997 and 2000. They were then compared to healthy people. **Those who used cell phones for less than 10 years faced a 20% higher risk of developing brain cancer. But for those who used them for more than a decade the risk was 80% higher.** The study also found that tumours were 2.5 times more likely to be on the same side of the head as the phone was held. The cancer of the auditory nerve, accoustic neuroma, showed a larger increase--3.5 times greater risk.

CELL PHONE RADIATION DOSAGE AND RESPONSE
All studies mentioned showed that an increase in cell phone radiation exposure also increases the likelihood of the adverse effect occurring.

In Repacholi's study of mice, the risk of lymphoma increased significantly the longer the mice were exposed to the radio waves.

In the research work done by Tice, Hook, and Roti Roti, the risks of genetic damage as measured by micronuclei formation increased as the amount of radiation increased.

In the three epidemiological studies--two by Muscat and one by Hardell--the risk of tumors was greater in the areas of the brain near where the cell phone was held.

In Salford's study, the higher the radiation exposure level the rats were exposed to, the more damage was apparent in the blood vessels in the brain and the neurons.

THE BIG CANCER PICTURE
The test tube studies by Tice and Hook; the mouse study by Repacholoi and Selford; and the epidemiological studies by Rothman, Muscat, and Hardell all agree in that they suggest an increased risk of cancer among cell phone users. They fit together to form the beginnings of a picture that everyone can see. They perhaps don't form the complete

picture yet, but there are enough already in place to see that there is cause fo genuine public health concern about cell phone safety.

According to Dr. Carlo, "The big picture is disturbingly clear. There is a definite risk that the radiation plume that emanates from a cell phone antenna can cause cancer and other health problems. It is a risk that affects hundreds of millions of people around the world. It is a risk that must be seen and understood by all who use cell phones so they can take all the appropriate and available steps to protect themselves--and especially to protect young children whose skulls are still growing and who are the most vulnerable to the risks of radiation." *(Cell Phones: Invisible Hazards of the Wireless Age)*

MORE PIECES COMING

- In 2000, a team of Sydney researchers published a scientific hypothesis about how mobile phone radiation causes cancer. The report claims that the radiation generated by cell phones causes ongoing stress to the body cells, causing them to give off **'heat shock proteins (HSP).'** The human cells sometimes release these proteins in response to injury or infection. Such a chronic activation of the heat shock response affects the normal regulation of cells, which could result in cancer.

- In 2002, cell biologist Fiorenzo Marinelli and his team at the National Research Council in Bologna, Italy, exposed leukemia cells to continuous radio waves similar to that of cell phones. The exposed cells had a higher rate of death than the controls initially, but after further exposure, a curious thing happened: instead of more cells dying, the exposed cells were replicating furiously compared to the controls. Genes that trigger cells to multiply were turned on in a high proportion of the cells. The cancer, although briefly beaten back, had become more aggressive. Marinelli suspects that the radiation may initially damage DNA, and that this interferes with the biochemical signals in a way that ultimately triggers the cells to multiply more rapidly.

- Dariusz Leszczynski at the Radiation and Nuclear Safety Authority in Helsinki found that one-hour exposure to mobile phone radiation caused cultured human cells to shrink. Leszczynski believes this happens when a cell is damaged. In a human being, such changes could destroy the blood-brain barrier. Radiation-induced changes in the cells could also interfere with normal cell death when the cell is damaged. If cells that are 'marked' to die do not, tumours can form.

SO WHY ARE CELL PHONES STILL AROUND?

Now with all the mounting evidence, the cell phone industry still maintains their position that cell phnes are safe and have even begun marketing towards children. The governments have been rather slow in stepping in to warn people of any danger from using cell phones. Fortunately, health officials and experts in several European countries are taking the first steps, having issued public warnings to parents urging caution about kids and cell phones,

If the previous environmental issues involving tobacco, asbestos, and lead are any indication, it takes years and even decades to accumulate the amount of evidence that would produce a definite ruling. **In the case of cigarette smoking, it took two decades of study and 100 years of consumer use to gather enough data to meet research standards** to demonstrate the need for the U.S. Surgeon General's warning label on cigarette packs. Some experts say that in the case of cell phones, it will not take that long as data are coming in at a faster pace. But at the present the authorities can only urge people to exercise caution.

Replication of research is another problem. A study that comes out with a new finding generally does not gain immediate acceptance in the scientific community or the wireless industry unless another research lab has been able to replicate the work and the findings. The industry has cleverly perpetuated their stance by creating an illusion of responsible follow up by always calling for more research.

When Dr. Salford published his study in 2003 showing that rat brain neurons were dying from exposure to cellphone radiation, he warned there might be similar effects in humans that over time could lead to degenerative diseases of the brain. His study was written off by the industry as a "novel" finding that needed to be replicated.

But achieving the scientific standard of replication can be complicated. Salford says if studies aren't absolutely replicated, providing an apples-to-apples comparison, there's wiggle room to dispute follow-up findings. Research studies also require funding, and the wireless industry, after Dr. Carlo's revelations, have been reluctant to put money into more comprehensive research. As for governments, again many European governments are taking the responsible course by funding research, but the U.S. and Canada are lagging poorly.

In 1999, CNN's Larry King once again featured a man who brought a multimillion dollar lawsuit against cell phone manufacturers. This time the man, a Maryland neurologist, was himself diagnosed with brain cancer--again located on the side of the head where he held his cell phone. The suit was yet again dismissed, however, and the man died not long afterwards.

According to WHO report, 0.1 billion people have died from tobacco use in the 20th century, and 10 times as many will die in the 21st century. No one is suggesting that cell phones could cause as much casualties, **but do we really want to wait and find out**?

AIMING FOR RESPONSIBLE TECHNOLOGY

Unlike tobacco, the cell phone has become as an indispensable part of our lives as television and computer. It has enabled us to make a gigantic leap in the way we communicate with one another and has been credited widely with saving people's lives in emergency situations. Cell phones are here to stay, and perhaps rightly so.

The question is not how to stop people from using this ubiquitous device but rather how to make it safer. The first step always is to admit there is a problem, hence the industry and the government have to acknowledge the health risks inherent with the present technology. This way we can all find the proper solutions that we may more enjoy the benefits of its use without sacrificing our health and wellbeing.

Taraka Serrano is a health advocate associated with BIOPRO Technology, a company that provides electromagnetic field (EMF) protection solutions. Watch the special video report: "The Invisible Dangers of EMF Radiation". For more information about the health dangers of EMF exposure and to find out how you can protect yourself and your family, visit: http://www.emf-health.com

Word count: 2,546

Heavy cell phone use tied to poor sperm quality

Men who talked more than 4 hours a day had lowest counts, study says

Reuters

updated 1:48 p.m. ET, Wed., Feb. 6, 2008

NEW YORK - Spending hours on a cell phone each day may affect the quality of a man's sperm, preliminary research suggests.

In a study of 361 men seen at their infertility clinic, researchers at the Cleveland Clinic found an association between the patients' cell phone use and their sperm quality.

On average, the more hours the men spent on their cell phones each day, the lower their sperm count and the greater their percentage of abnormal sperm.

The findings, published in the journal Fertility and Sterility, add to questions about the potential health effects of cell phones and other wireless devices. Some studies, for example, have linked long-term cell phone use to a higher risk of brain tumors, though many other studies have found no such connection.

The concern is that, over time, the electromagnetic energy emitted from mobile phones could theoretically harm body tissue — by damaging DNA, for example.

However, the new findings do not prove that cell phones somehow damage sperm, according to the researchers.

"Our results show a strong association of cell phone use with decreased semen quality. However, they do not prove a cause-and- effect relationship," lead researcher Dr. Ashok Agarwal told Reuters Health.

He and his colleagues based their findings on semen samples from 361 men who came to their infertility clinic over one year. All of the men were questioned about their cell phone habits.

In general, the researchers found, sperm count and sperm quality tended to decline as daily cell phone hours increased. Men who said they used their phones for more than four hours each day had the lowest average sperm count and the fewest normal, viable sperm.

"We infer from our results that heavy cell phone use ... is associated with a lower semen quality," Agarwal said. But whether cell phones somehow directly affect men's fertility is not clear.

Agarwal said he and his colleagues have two studies underway aiming to shed light on the issue. In one, they are exposing semen samples to electromagnetic radiation from cell phones to see what, if any, effects occur.

The second is a follow-up to the current study that is assessing a larger group of men. Agarwal said this study is more rigorously designed and will account for certain other factors like lifestyle habits and occupational exposures that might affect sperm quality.

URL: http://www.msnbc.msn.com/id/23033163/wid/11915773?gt1=10914

YAHOO! NEWS

Back to Story - Help

Frequent Cell Phone Use May Slow Brain Function

PCWORLD.COM

Matt Hamblen, Computerworld
Tue Sep 18, 9:00 PM ET

There have been worries about cell phones causing brain cancer. And certainly everyone worries about driving behind the guy who's holding the steering wheel with his knees while tapping in a message on a wireless e-mail device.

But now hear this: Mobile phone use may cause a slowing of brain activity.

Before anyone panics, the suggestion that frequent mobile phone use makes us behave a little unbalanced is, so far, based on a study of 300 people conducted by researchers in Australia, England and the Netherlands.

The study, published in the International Journal of Neuroscience this month, looked at the group of 300 people over 2.4 years, but researchers plan to expand the study over a longer period and with data involving 17,000 people.

According to the study, frequent mobile phone users demonstrated slowed brain function, but with the caveat that the slowed brain effects are still considered within normal brain functioning. A longer study with a larger sample group would consider whether the slowed brain activity should be considered an adverse health effect, according to a statement from Brainclinics Diagnostics in Nijmegen, the Netherlands, one of the groups involved in the study.

The noted slowed brain function could not be explained by differences in personality, according to researchers. "In Alzheimer's dementia you also find a severely slowing of brain activity," said Martijn Arns, the main investigator for Brainclinics Diagnostics, in a statement. "However, the slowing found in this study, with mobile phone users, can still be considered within 'normal' limits." Still, Arns predicted that a longer-term study would show more severe effects.

Of the 300 people in the study, only 100 were frequent mobile phone users, while 100 were non-mobile phones users and the third group of 100 were an intermediate user group. Differences in brain activity, as measured with quantitative electroencephalographic (EEG) studies, and neuropsychological functions such as attention, memory, executive function and personality, were assessed. Among the results, frequent users scored higher on ratings as extraverts and were found to be less open-minded.

The study also found that frequent users also showed improved focused attention, which was explained by a learning effect due to making more phone calls in busy places where users had to focus better on a phone call while filtering out background noise and other distractions.

Despite this improved focus and the findings about personality, the frequent users showed more instances of slowed activity as measured by delta and theta EEG power, as well as a slowdown in a measurement called alpha peak frequency.

The researchers cited several other studies going back to 1998 on the short-term effects of mobile phone use, some of which showed that frequent users improved their scores on cognitive tests. Those positive outcomes were linked to small increases in brain temperature, which led to faster metabolic activity and thus faster reaction times. However, the researchers in the current study said the previous studies are inconclusive.

In the recent study, Brainclinics was joined by researchers at Radbound University in Nijmegen, the Institute of Psychiatry in London and The Brain Resource Co. Ltd. in Sydney.

YAHOO! NEWS

Dinosaur Tumor Studied for Human Cancer Clues

Heather Whipps
Special to LiveScience
LiveScience.com
Tue Apr 4, 9:00 AM ET

Cancer in dinosaurs and illnesses in other animals are being studied in a groundbreaking new program that combines medical school with the study of natural history.

Educators hope the effort will produce doctors with a better understanding of why we get sick.

Despite being millions of years removed from our time and our own species, illnesses in animals like the dinosaurs can shed light on the evolution of human disease, says Christopher Beard, curator and specialist in vertebrate paleontology at Carnegie Museum of Natural History in Pittsburgh.

"Some diseases that afflict humans today, such as malaria, gout, and cancer, are truly ancient and were handed down to us from our distant ancestors," Beard told LiveScience. "By studying the distribution of these diseases in other living and fossil organisms, we can gain insights into the nature of these diseases."

Part of the course

Students will now be offered the chance to learn about the history of disease as part of their regular medical school training, the University of Pittsburgh Medical Center announced recently.

The university is welcoming four renowned curators from Carnegie Museum into its classrooms to teach seminars and use the museum collection, which is considered one of the world's premiere displays of natural history artifacts, for demonstrations. Included in the collection is a 150-million-year-old fossilized dinosaur bone complete with a tumor.

Finding a cancerous Jurassic lump doesn't surprise Michael Kennedy, a surgeon and professor at the University of Southern California. "Cancer is the most common cause of death in animals. It is not a uniquely human disease," he said in a recent telephone interview. The renewed focus on history in the teaching of medicine pleases Kennedy, also the author of "A Brief History of Disease, Science and Medicine" (Asklepiad Press, 2004), which describes the intricate historical links that connect diseases.

"History has traditionally been pushed aside in medical schools because it doesn't seem like a necessary part of the curriculum," he said. "But the history of disease has many practical applications today."

Backaches and the avian flu

Kennedy pointed to the current avian flu crisis as an example.

For several years now, health officials have been studying DNA extracted from frozen victims of the 1918 Spanish influenza pandemic buried in Alaska. According to Kennedy, they'll attempt to compare the deadly strain, which killed approximately 50 million people and was also thought to have jumped from birds to humans, to the contemporary flu to determine its potency.

The University of Pittsburgh's program will allow students to examine the birth of other familiar and modern health problems like back pain and hernias, which originated when humans began to walk upright about five million years ago.

"That evolutionary transformation radically altered the human skeleton, causing us to suffer many health problems that the typical

mammalian quadruped doesn't have to worry about," Beard explained. "For example, chronic lower back pain in humans results from our peculiarly S-shaped vertebral column, which places extraordinary pressures on our lumbar vertebrae."

Help from apes

While the museum does not have any early human fossils in its collection, its wide variety of prehistoric primate specimens should provide enough clues about the earlier phases of human evolution to keep students busy, Beard said.

"These fossils, along with skeletons of living primates, will allow us to trace the major changes that have occurred in the human skeleton as we diverged from our close primate relatives."

Students interested in the field of sports medicine might take particular interest in this section of the museum, according to Beard.

"Some changes in the human skeleton that we often regard as being unique to us—such as our remarkably mobile shoulder joints that allow us to pitch baseballs and play golf—are actually features that we hold in common with our closest primate relatives, the apes," he said.

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YAHOO! NEWS

Back to Story - Help

Gene Plays Key Role in Stopping Spread of Some Cancers

HealthDay

By Ed Edelson
HealthDay Reporter
Wed Jan 4, 5:04 PM ET

WEDNESDAY, Jan. 4 (HealthDay News) -- Researchers have found a genetic reason for the aggressiveness of some cancers, and perhaps a pathway toward taming their spread.

A gene that tells a cell to commit suicide if it wanders into the wrong part of the body gets silenced in some cancer cells, claims a report in the Jan. 5 issue of Nature. Those cells can grow in any tissue, however foreign, in a process called metastasis.

"It's not the growth of the primary tumor that kills," explained David Cheresh, associate director for translational research at the University of California at San Diego Moores Cancer Center. "Growth of metastatic tumors does kill."

The gene involved is called caspase 8. Cheresh and his colleagues previously have shown that it acts as a police officer, making sure that skin cells stay in the skin, liver cells in the liver, and so on. When a cell migrates to the wrong location, caspase 8 activates molecules called integrins that, in effect, tell the cell to commit suicide.

But experiments show that caspase 8 doesn't do its job in some cancer cells. The researchers found limited or no caspase 8 activity in metastases of a childhood cancer called neuroblastoma, and they have evidence that the same thing happens in other kinds of cancer.

Loss or suppression of caspase 8 is seen in about 70 percent of small cell lung cancers, 10 percent of colon cancers and 35 percent of medulloblastomas, the researchers said. It is seen in 70 percent of aggressive neuroblastomas in children.

The finding has some important medical implications, Cheresh said. Tests for caspase 8 function could be used to help guide treatment, he noted: "If it is missing, those are the most aggressive tumors, and [they] need to be treated early and aggressively."

Another clinical possibility is that caspase 8 activity could be manipulated to make cancer cells initiate apoptosis, the process of cellular suicide. "What we hope we can do is determine ways of initiating apoptosis in these invasive cells," Cheresh said.

The discovery casts light on a different way the body can attack cancer, said Marcus E. Peter, a professor of cancer biology at the University of Chicago Ben May Institute for Cancer Research. In 1995, his group first described the role of caspase 8 in programmed cell death.

"This has been viewed entirely as an immune system process," Peter said. "Tumor cells are recognized by the immune system, immune system cells move in, dock on the tumor and kill it. Here, integrins mediate death through caspase 8, with no role for the immune system."

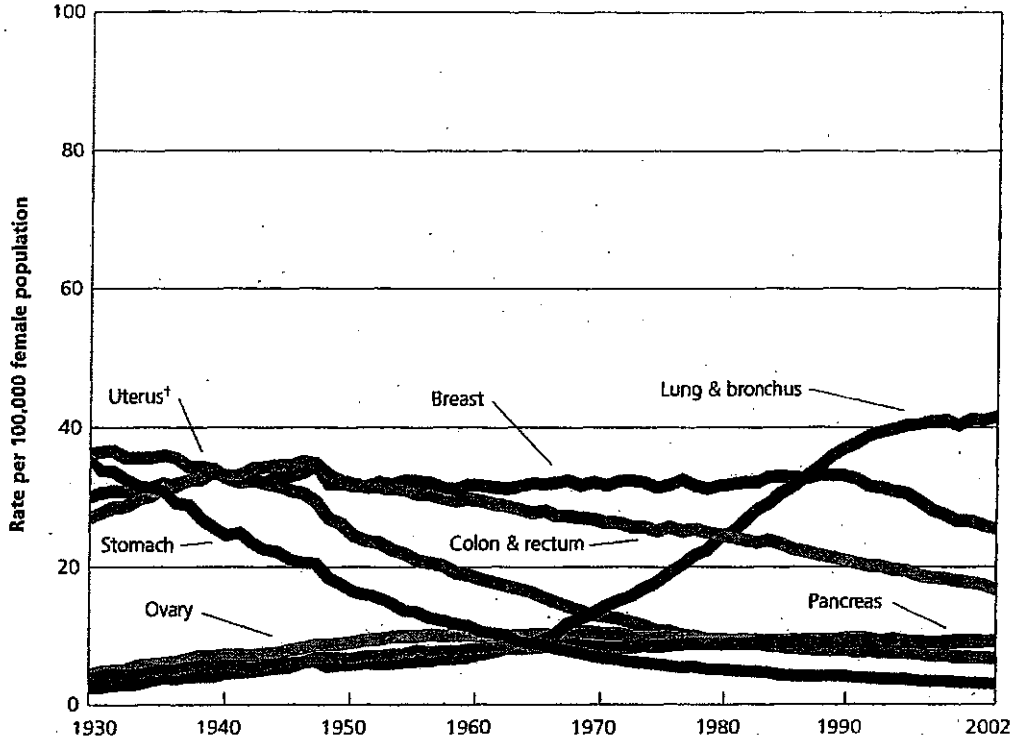
The good news in the discovery is that "for the most part, tumors don't get rid of this gene entirely," Peter said. "They silence it through a process called methylation. There are a number of drugs that can block this methylation."

Whatever the ultimate impact of the finding on medical practice, "it gives us a molecular insight into the difference between cells that are metastatic and those that are not," Cheresh said.

More information

A constantly updated list of cancer-related genes is kept by the Sanger Institute.

Age-Adjusted Cancer Death Rates,* Females by Site, US, 1930-2002



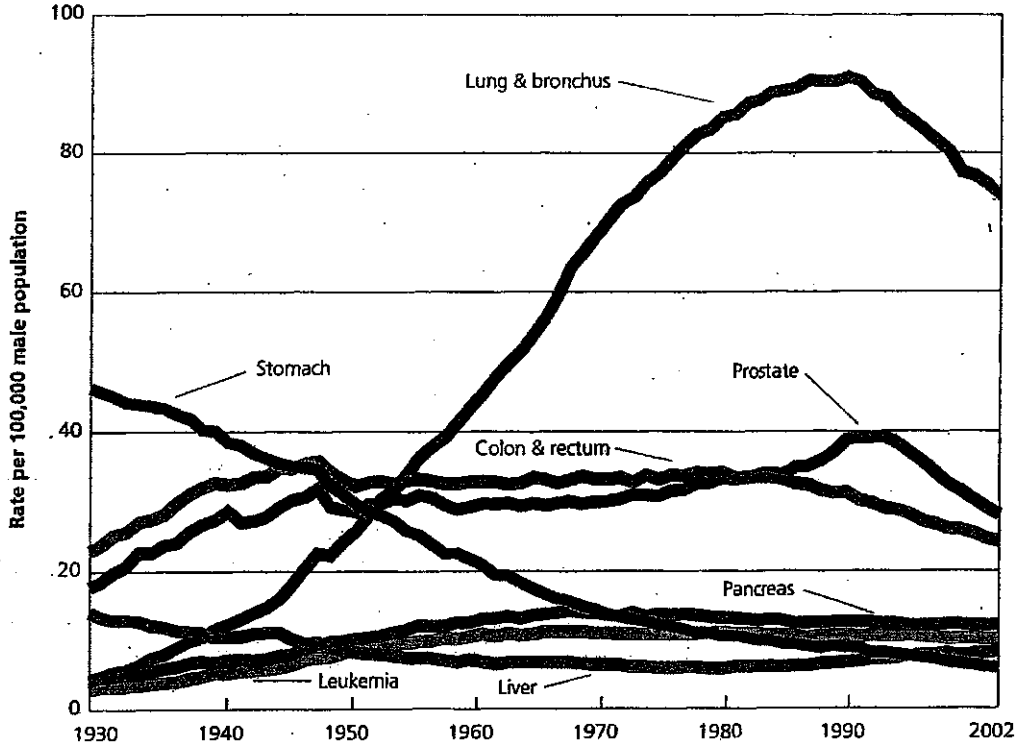
*Per 100,000, age-adjusted to the 2000 US standard population. †Uterus cancer death rates are for uterine cervix and uterine corpus combined.

Note: Due to changes in ICD coding, numerator information has changed over time. Rates for cancer of the lung and bronchus, colon and rectum, and ovary are affected by these coding changes.

Source: US Mortality Public Use Data Tapes 1960 to 2002, US Mortality Volumes 1930 to 1959, National Center for Health Statistics, Centers for Disease Control and Prevention, 2005.

American Cancer Society, Surveillance Research, 2006

Age-Adjusted Cancer Death Rates,* Males by Site, US, 1930-2002



*Per 100,000, age-adjusted to the 2000 US standard population.

Note: Due to changes in ICD coding, numerator information has changed over time. Rates for cancer of the liver, lung and bronchus, and colon and rectum are affected by these coding changes.

Source: US Mortality Public Use Data Tapes 1960 to 2002, US Mortality Volumes 1930 to 1959, National Center for Health Statistics, Centers for Disease Control and Prevention, 2005.

American Cancer Society, Surveillance Research, 2006

Incidence and Mortality Rates* by Site, Race, and Ethnicity, US, 1998-2002

Incidence	White	African American	Asian American and Pacific Islander	American Indian and Alaska Native	Hispanic/ Latino†
All sites					
Males	556.4	682.6	383.5	255.4	420.7
Females	429.3	398.5	303.6	220.5	310.9
Breast (female)	141.1	119.4	96.6	54.8	89.9
Colon & rectum					
Males	61.7	72.5	56.0	36.7	48.3
Females	45.3	56.0	39.7	32.2	32.3
Lung & bronchus					
Males	76.7	113.9	59.4	42.6	44.6
Females	51.1	55.2	28.3	23.6	23.3
Prostate	169.0	272.0	101.4	50.3	141.9
Stomach					
Males	10.7	17.7	21.0	15.9	17.2
Females	5.0	9.6	12.0	9.1	10.1
Liver & bile duct					
Males	7.4	12.1	21.4	8.7	14.1
Females	2.9	3.7	7.9	5.2	6.1
Uterine cervix	8.7	11.1	8.9	4.9	15.8

Mortality	White	African American	Asian American and Pacific Islander	American Indian and Alaska Native	Hispanic/ Latino†
All sites					
Males	242.5	339.4	148.0	159.7	171.4
Females	164.5	194.3	99.4	113.8	111.0
Breast (female)	25.9	34.7	12.7	13.8	16.7
Colon & rectum					
Males	24.3	34.0	15.8	16.2	17.7
Females	16.8	24.1	10.6	11.8	11.6
Lung & bronchus					
Males	75.2	101.3	39.4	47.0	38.7
Females	41.8	39.9	18.8	27.1	14.8
Prostate	27.7	68.1	12.1	18.3	23.0
Stomach					
Males	5.6	12.8	11.2	7.3	9.5
Females	2.8	6.3	6.8	4.1	5.3
Liver & bile duct					
Males	6.2	9.5	15.4	7.9	10.7
Females	2.7	3.8	6.5	4.3	5.1
Uterine cervix	2.5	5.3	2.7	2.6	3.5

*Per 100,000, age-adjusted to the 2000 US standard population. †Hispanic/Latinos are not mutually exclusive from whites, African Americans, Asian Americans and Pacific Islanders, and American Indians and Alaska Natives.

Source: Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Mariotto A, Feuer EJ, Edwards BK (eds). *SEER Cancer Statistics Review, 1975-2002*, National Cancer Institute, Bethesda, Maryland. http://seer.cancer.gov/csr/1975_2002/, 2005.

American Cancer Society, Surveillance Research, 2006

Probability of Developing Invasive Cancers Over Selected Age Intervals by Sex, US, 2000 to 2002*

		Birth to 39 (%)	40 to 59 (%)	60 to 69 (%)	60 to 69 (%)	Birth to Death (%)
All sites†	Male	1.43 (1 in 70)	8.57 (1 in 12)	16.46 (1 in 6)	39.61 (1 in 3)	45.67 (1 in 2)
	Female	1.99 (1 in 50)	9.06 (1 in 11)	10.54 (1 in 9)	26.72 (1 in 4)	38.09 (1 in 3)
Urinary bladder‡	Male	.02 (1 in 4375)	.40 (1 in 250)	.93 (1 in 108)	3.35 (1 in 30)	3.58 (1 in 28)
	Female	.01 (1 in 9513)	.12 (1 in 816)	.25 (1 in 402)	.96 (1 in 104)	1.14 (1 in 88)
Breast	Female	.48 (1 in 209)	4.11 (1 in 24)	3.82 (1 in 26)	7.13 (1 in 14)	13.22 (1 in 8)
Colon & rectum	Male	.07 (1 in 1399)	.90 (1 in 111)	1.66 (1 in 60)	4.94 (1 in 20)	5.84 (1 in 17)
	Female	.06 (1 in 1567)	.70 (1 in 143)	1.16 (1 in 86)	4.61 (1 in 22)	5.51 (1 in 18)
Leukemia	Male	.15 (1 in 650)	.22 (1 in 459)	.35 (1 in 284)	1.17 (1 in 85)	1.50 (1 in 67)
	Female	.13 (1 in 788)	.14 (1 in 721)	.19 (1 in 513)	.78 (1 in 129)	1.07 (1 in 93)
Lung & bronchus	Male	.03 (1 in 3244)	1.00 (1 in 100)	2.45 (1 in 41)	6.33 (1 in 16)	7.58 (1 in 13)
	Female	.03 (1 in 3103)	.80 (1 in 125)	1.68 (1 in 60)	4.17 (1 in 24)	5.72 (1 in 17)
Melanoma of skin	Male	.13 (1 in 800)	.51 (1 in 195)	.51 (1 in 195)	1.25 (1 in 80)	1.94 (1 in 52)
	Female	.21 (1 in 470)	.40 (1 in 248)	.26 (1 in 381)	.56 (1 in 178)	1.30 (1 in 77)
Non-Hodgkin lymphoma	Male	.14 (1 in 722)	.47 (1 in 215)	.56 (1 in 178)	1.57 (1 in 64)	2.18 (1 in 46)
	Female	.09 (1 in 1158)	.31 (1 in 320)	.42 (1 in 237)	1.29 (1 in 77)	1.82 (1 in 55)
Prostate	Male	.01 (1 in 10149)	2.66 (1 in 38)	7.19 (1 in 14)	14.51 (1 in 7)	17.93 (1 in 6)
Uterine cervix	Female	.15 (1 in 657)	.28 (1 in 353)	.15 (1 in 671)	.22 (1 in 464)	.74 (1 in 135)
Uterine corpus	Female	.06 (1 in 1641)	.72 (1 in 139)	.83 (1 in 120)	1.36 (1 in 74)	2.61 (1 in 38)

*For those free of cancer at beginning of age interval. Based on cancer cases diagnosed during 2000 to 2002.

†All sites exclude basal and squamous cell skin cancers and in situ cancers except urinary bladder.

‡Includes invasive and in situ cancer cases.

Source: DevCan: Probability of Developing or Dying of Cancer Software, Version 6.0. Statistical Research and Applications Branch, National Cancer Institute, 2005. http://srab.cancer.gov/devcan

American Cancer Society, Surveillance Research, 2006

From the staff of the Center for the Study of Technology and Society in Washington, D.C.

BIOTECHNOLOGY

Special Focus
Designer Babies

Latest News

Would Designer Babies Improve Our Species?
(London Guardian, through 6 Feb 01)

A British scientist has argued that our technology has allowed us to defeat evolution. "Gene mutations that would have been fatal to our ancestors" remain in the gene pool, so unless we engineer genetic improvements, we will become a "frail and sickly species."

- **RESPONSES:** In Search of Rogue Genes (London Guardian, 6 Feb 01)

How Does it Feel to Be a Designer Baby?
(various sources through 13 Feb 01)

Exactly 229 babies were born from sperm donated to the so-called "Genius Sperm Bank" -- a U.S. repository founded in the 1970s to collect sperm from Nobel prize winners and other scientists. Where are those children today? What are they like? Click here to read the story of one of those kids.

- The "Genius Babies" and How They Grew (Slate, 7 Feb 01)
- "Genius" Donors Come Forward (Slate, 13 Feb 01)

Other News

- Destiny Is In Our Grasp, Say Scientists (London Times, 9 Feb 01)
- Unease Over "Beauty Genes" (Financial Times, 25 Jan 01)
- Designer People: Are We Changing the Nature of Nature? (E-Magazine, Jan-Feb 01)
- **OLDER ARTICLES ARCHIVED BELOW**

The Problem

- Is it ethical for doctors and parents to "design" a baby by selecting or altering an embryo they wish to bring to term? Should it be legal? Should there be limits to the practice?

Background

- "Designer babies" is a term used by journalists and commentators - not by scientists - to describe several different reproductive technologies. These technologies have one thing in common: they give parents more control over what their offspring will be like.
- Designer babies are made possible by progress in three fields:
  1. **ADVANCED REPRODUCTIVE TECHNOLOGY.** In the two decades since the first "test tube baby" was born, reproductive medicine has helped countless women conceive and bear children. Today there are hundreds of thousands of humans who were conceived thanks to *in vitro fertilization* - the practice of mixing eggs and sperm outside the womb. Other advanced reproductive technologies include frozen embryos, egg and sperm donations, surrogate motherhood, pregnancies by older women, and the direct injection of a sperm cell into an egg.
  2. **CELL AND CHROMOSOME MANIPULATION.** The past decade has seen astonishing breakthroughs in our knowledge of cell structure. Our ability to transfer chromosomes (the long threads of DNA in each cell) has led to major developments in cloning. Our knowledge of stem cells (generic "starter" cells that eventually differentiate

into other types of cells) will make many new therapies possible. As we learn more about how reproduction works at the cellular level, we can have more control over the earliest stages of a baby's development.

3. **GENETICS AND GENOMICS.** With the mapping of the human genome in 2000, our understanding of how DNA affects human development is only just beginning. Someday, we might be able to switch bits of DNA on or off as we wish, or replace sections of DNA at will; research in that direction is already well underway.

Two Key Things to Remember

- Human reproduction is a tricky business. There are many factors involved in producing a baby: the genetic constitution of the parents, the condition of the parents' egg and sperm, and the health and behavior of the impregnated mother. When you also consider the enormous complexity of the human genome, with its billions of DNA pairs, it becomes clear that reproduction will always have an element of unpredictability.
- To a certain extent, we have always hoped to control our children's characteristics through our selection of mates. New technologies will give us more power to influence our children's "design" - but our control will still be far from total.

What are the Different Techniques for "Designing Babies"?

Unfortunately, since the term "designing babies" is so imprecise, it is difficult to untangle its various meanings in the public mind so as to make judgments about which techniques are acceptable. Below are several different procedures described by the term "designing babies"; please notice that some of these techniques do not yet exist.

- **SCREENING EMBRYOS FOR HIGH-RISK DISEASES.** Some parents have a high likelihood of passing on the genes for a disease. Many inherited diseases, such as cystic fibrosis and Huntington's disease, can be detected very early using a technique called *preimplantation genetic diagnosis* (PDG). This procedure, first used in 1990, helps doctors detect severe genetic disorders very early in a pregnancy. If it is used to screen test tube babies, an embryo with an inherited condition need never be implanted in the womb.
- **SCREENING EMBRYOS FOR UNKNOWN DISEASES.** The PDG technique could also be used to screen embryos even when there is no known risk of inheriting diseases. Also, doctors could screen for unpredictable disorders in the chromosomes - disorders which could result in miscarriage, birth defects, or diseases like Down's syndrome.
- **SELECTING THE SEX OF A BABY.** There are legitimate medical reasons to prefer a male or a female child; for instance, many diseases can only be passed on through the male line or the female line of descendants. While there has been some recent progress in selecting the sex before fertilization (by dividing the sperm likely to produce males from those likely to produce females), the sex of an embryo can more easily be determined after fertilization through PDG. It can also be determined several weeks into pregnancy - at which point an abortion is required if the baby is not of the desired sex.
- **PICKING AN EMBRYO FOR ITS SPECIFIC TRAITS.** This is not really possible today, but it is imaginable that as our understanding of the human genome improves, doctors might be able to develop a general genetic profile of several fertilized embryos. The parents could then choose an embryo based on its profile - although there would be no guarantees that the baby would grow to match its profile. This method has no therapeutic value.
- **GENETIC MANIPULATION FOR THERAPEUTIC REASONS.** As our knowledge of the human genome increases - and as our ability to modify it improves - we will be able to fix diseased or defective embryos at the genetic level. This technique is called *germ line therapy*, which refers to the fact that it would be performed on an egg, a sperm or a small fertilized embryo. Defective sections of DNA could be replaced with healthy DNA. Although, to date, there is no practical way to do this for humans, there have been several recent breakthroughs.
- **GENETIC MANIPULATION FOR COSMETIC REASONS.** It is conceivable that someday, the same technique used for genetic therapy could also be used for selecting other genetic characteristics. It is nowhere near possible today, but in the future, inherited characteristics like eye color, hair color or height could be selected with some rudimentary

amount of control. Other characteristics, however, such as intelligence, athleticism and beauty are so greatly influenced by environmental factors (such as parenting and nutrition) that genetic manipulation is never likely to have more than a slight effect.

Arguments *FOR* Designing Babies

Basic Arguments For Designing Babies
Using these techniques can help prevent certain genetic diseases, saving the children from debilitation and reducing the financial and emotional strain on the parents. If we want the best for our children, why shouldn't we use technology?
As of today, these techniques are only used by parents who need the help of fertility clinics to have children. Since they are investing so much time, energy and money in their effort to have a baby, shouldn't they have a healthy one?
A great many naturally-conceived embryos are rejected from the womb for defects; by screening embryos, we are doing what nature would normally do on our behalf.
"Imagine the reaction there would be if organ transplantation were prohibited because it is 'unnatural' -- though that is what some people called for when transplantation was a medical novelty. It is hard to see how the replacement of a defective gene is any less 'natural' than the replacement of a defective organ. Indeed, the major difference is the entirely beneficial one that medical intervention need occur only once around the time of conception, and the benefits would be inherited by the child and its descendants." - Dr. Roger Gosden[1]

Arguments *AGAINST* Designing Babies

Basic Arguments Against Designing Babies
"Even people who might welcome the growth of genetic knowledge and technology are worried about the power of geneticists, genetic engineers, and any governmental authority armed with genetic technology. Precisely because we have been taught by these very scientists that genes hold the secret of life, and that our genotype is our essence if not quite our destiny, we are made nervous by those whose expert knowledge and technique touches our very being." - Dr. Leon Kass
We could get carried away "correcting" perfectly healthy babies. Once we start down the slippery slope of eliminating embryos because they are diseased, what is to stop us from picking babies for their physical or their psychological traits?
There is always the looming shadow of *eugenics* - the practice of "improving" the human gene pool by eliminating undesirables. This was the motivation for some government policies in Europe and the United States in the first half of the twentieth century - including forced sterilizations, selective breeding and "racial hygiene." Eugenics is also practiced in China today, and the techniques for designing babies give us dangerous new powers to express our genetic stereotypes and preferences.
There are major social concerns. Will we breed a race of super-humans who look down on those without genetic enhancements? Will these new technologies only be available to the wealthy - resulting in a lower class which still suffers from inherited disabilities and diseases? Will discrimination against people already born with disabilities increase, if they are perceived as genetically inferior?
Economic pressures might come to play a role in making design choices regarding new babies. Insurance companies, for instance, may refuse to cover a newborn with a

condition that could have been corrected before birth, thus forcing parents to design their child. There is a connected concern about transferring the process of procreation from the home to the lab, and turning it into a manufacturing process.

Tampering with the human genetic structure might actually have unintended (and unpredictable) consequences that could damage the gene pool.

Many of these baby-designing techniques can be considered unethical, because they treat each embryo as a means for someone else's happiness, instead of as an end in itself.

Many of the procedures related to designing babies involve terminating an embryo, either inside or outside the womb; this is anathema to those who disapprove of aborting fetuses on moral or religious grounds.

What Happens Next?

Because the term "designing babies" describes so many different procedures, there are many different laws affecting the practice. Some countries, such as the U.S., have placed relatively light regulation on these procedures, only limiting the amount of government money that can be spent on related research. Other nations, such as the UK, seem headed toward greater regulation of "designing babies," at least in part because a number of high-profile cases have captured the public imagination and provoked heated debates. In the EU, many of the procedures related to designing babies are outlawed.

As our technical abilities progress, citizens will have to cope with the ethical implications of designing babies, and governments will have to define a regulatory course. We will have to answer some fundamental questions: How much power should parents and doctors have over the design of their children? How much power should government have over parents and doctors? As always, our judgments should be based on the facts and on our social beliefs.

Archive of Older Articles

American Couple Selected Embryo for Birth to Help Sick Daughter
(various sources through 5 Jan 01)
A U.S. couple with a daughter suffering from a rare form of anemia gave birth to a healthy son, conceived in a lab and selected when still an embryo because he did not have the disease. They decided to have the child at least partly because his tissues could help treat his sister. This case marks the first time treatment for another person been a consideration in embryo selection. Early reports indicate the therapy has been successful.

- U.S. Girl Saved by Brother's Stem Cells Goes Home (Reuters, 5 Jan 01)
- Designer Baby Ethics Fears (BBC, 4 Oct 00)

British Couple Wants to Select Sex of Baby
(various sources through 22 Oct 00)
A Scottish couple whose only daughter (they have four sons) was killed in a 1999 bonfire accident wishes use sex selection technology to have another daughter. Sex selection is banned in Britain, and the government body in charge of regulating reproductive technology has refused to make an exception. They are now suing under an EU human rights law recently passed in the UK.

- **DEBATE:** Should We Be Allowed to Choose the Sex of Our Children? (London Independent, 22 Oct 00)
- Bereaved Couple Demand Right to Baby Girl (London Guardian, 5 Oct 00)

Gene Screening Raises Ethics Questions Now
(London Guardian, 10 Jul 00)

We do not need a genome map to confront ethical dilemmas brought on by our knowledge of genetics. We can screen fetuses for the debilitating genetic disease cystic fibrosis, but because of the connection to abortion and eugenics, should we? Great Britain is facing precisely that question.

- Science Screens Out Defective Genes (BBC, 18 Nov 00)
- Test-Tube Screening Can Spot Chromosome Errors (New Scientist, 23 Oct 00)
- Screening Picks 'Best-Chance' Embryos (BBC, 23 Oct 00)
- Embryo Test Could Protect Couples from Fertility Woes (CNN, 23 Oct 00)
- **DISCUSSION:** Should Parents Be Able to Design Their Own Babies? (BBC, 11 Oct 00)
- Spanish Couple Picks Sex of Children to Protect Grandchildren (UniSci, 17 Oct 00)
- If You Can't Teach Your Baby, They Can Build You a Better One (London Times, 8 Oct 00)
- A Baby by Design (London Telegraph, 8 Oct 00)
- Anyone for Ethical Tennis? (London Guardian, 8 Oct 00)
- Not Quite Blondes to Order (London Guardian, 5 Oct 00)
- The Difficult Choices in Genetic Engineering are Only Just Beginning (London Independent, 5 Oct 00)
- **OPINION:** When a "Designing Baby" Can Be Right (London Independent, 5 Oct 00)
- Designer Babies? (Salon, 5 Oct 00)
- Report Frowns on Designer Babies (Associated Press, 19 Sep 00)
- Why I Want a Designer Baby (London Guardian, 29 Jun 00)
- Call for a Ban on Genetically Modified Humans (BBC, 29 May 00)
- Ban on "Designer Babies" May Be Lifted (Sydney Morning Herald, 16 Feb 00)
- Designing Babies: The Future of Genetics (BBC, 6 Jan 00)
- Babies by Design (CNN, 4 Jan 00)
- A Small Leap to Designer Babies (NY Times, 1 Jan 00 - free registration required)
- **DEBATE:** Could Embryo Screening Lead to Genetic Cleansing? (London Guardian, 20 Nov 99)
- Public Views on Embryo Genetic Testing Sought (London Guardian, 16 Nov 00)
- Major Advances in Germ-line Gene Therapy (New Scientist, 23 Oct 99)
- The Last Taboo: Human Genetic Engineering (New Scientist, 23 Oct 99)
- The Future of Medicine: Designer Babies (Time mag, 11 Jan 99)
- Tough Questions on Designer Babies (UPenn, 6 Jan 99)
- Designer Babies: Eugenics Revisited? (Policy.com, 30 Nov 98)
- Designer Babies (Newsweek, 9 Nov 98)

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NOTE: 1. Roger Gosden, Designing Babies: The Brave New World of Reproductive Technology, W.H. Freeman and Co., New York, 1999, p. 116.

Genetics Practice Problems

Work out these genetic problems. The answers are provided but the most important aspect is the practice of working out the problems.

1) The D gene controls pea plant height. The DD and dd genotypes confer tall and dwarf phenotypes, respectively. What is the relationship between D and d?
A) They are two different plant chromosomes.
B) They are alleles of the same gene.
C) They are two different genes on the same chromosome.
D) They are two possible homozygous genotypes.
E) They are two possible heterozygous genotypes.
Answer: B

2) In Mendel's basic experiment, he began with true-breeding parental (P) plants. What did he see when he cross-fertilized P plants that had different traits?
A) All F1 plants had the trait of one or the other P plant.
B) The F1 plants showed a combination of the two P traits, in a 3:1 ratio.
C) The F1 plants showed a combination of the two P traits in a 1:1 ratio.
D) The F1 plants had new traits that were a blend of P traits.
E) The F1 plants had an entirely new trait, not seen in either P plant.
Answer: A

3) Crossing red flowering snapdragons with white flowering snapdragons yields seed that grow into snapdragons with pink flowers.
A) These mating results support the blending hypothesis of inheritance.
B) These mating results support the hypothesis that there are unblended discrete units of inheritance.
Answer: A

4) Crossing two pink snapdragons yields some seed that produces red flowering plants, some seed that produces white flowering plants and some seed that produces pink flowering plants.
A) These mating results support the blending hypothesis of inheritance.
B) These mating results support the hypothesis that there are unblended discrete units of inheritance.
Answer: B

Use this information for the two questions below:
A and a are dominant and recessive alleles, respectively, of the same gene.

5) Which genotype(s) would result in an individual with the dominant phenotype?
A) AA and aa B) AA and Aa C) Aa and aa D) only AA E) only Aa
Answer: B
Type: MC

6) Which genotype(s) would result in an individual with the recessive phenotype?
A) aa only B) Aa only C) Aa or aa D) AA only E) AA or aa
Answer: A

Use this information for the four questions below.
Cystic fibrosis (CF) is caused by a recessive allele. A child has CF, even though neither of his parents has CF. Use this information for the questions below.

7) What can you conclude about the parents?
A) They are both homozygous dominant for the CF gene.
B) They are both homozygous recessive for the CF gene.
C) One is homozygous dominant for the CF gene, the other is heterozygous.
D) One is homozygous recessive for the CF gene, the other is heterozygous.
E) They are both heterozygous for the CF gene.
Answer: E

8) This couple has another child who does not have CF. What is the probability he or she is heterozygous?
A) 1/4 B) 2/4 C) 3/4 D) 1/3 E) 2/3
Answer: E

9) If this couple has another child, what is the probability he or she will NOT have CF?
A) 1/4 B) 2/4 C) 3/4 D) 1/3 E) 2/3
Answer: C

10) If this couple has another child, what is the probability it will be a boy, with CF?
A) 1/32 B) 1/20 C) 1/16 D) 1/10 E) 1/8
Answer: E

11) Widows peak hairline in humans is dominant to non-widows peak hairline. If a person has a widows peak hairline, what is his or her genotype?
A) It must be homozygous dominant.
B) It must be homozygous recessive.
C) It is either homozygous dominant or homozygous recessive.
D) It must be heterozygous.
E) It is are either heterozygous or homozygous dominant.
Answer: E

Use this information for the four questions below.
In humans, "unattached" earlobes are dominant over "attached" earlobes. "Widows peak" hairline is dominant over "non-widows peak" hairline. Use E and e for the earlobe phenotype alleles, and W and w for the hairline phenotype alleles.

12) A female and a male, both with unattached earlobes, have a child with attached earlobes. What is the probability their next child will have attached earlobes?
A) 4/4 B) 3/4 C) 2/4 D) 1/4 E) 0
Answer: D

13) A female and a male, both with genotype EeWw have a child. What is the probability it will have attached earlobes and a widows peak hairline?
A) 9/16 B) 3/16 C) 1/16 D) 1/3 E) 3/4
Answer: B

14) A female and a male, both with genotype EeWw have a child. What is the probability it will be a boy, and have attached earlobes and a widows peak hairline?
A) 1/6 B) 3/16 C) 1/16 D) 1/32 E) 3/32
Answer: E

15) A female with unattached earlobes and a widows peak hairline and a male with attached earlobes and a widows peak hairline have a child. The child has attached earlobes and a non-widows peak hairline. What are the genotypes of the parents?
A) EeWw and eeww
B) EeWw and eeWw
C) EEWW and eeww

D) EEWW and eeWw
E) EeWw and EeWW
Answer: B

16) What is the physical basis for the independent assortment observation that Mendel made?
A) Male and female gametes are produced in separate organs in separate individuals.
B) There are two chromosome divisions in meiosis.
C) Recombination (crossing over) occurs in meiosis.
D) Homologous chromosomes are randomly separated during meiosis I.
E) Sister chromatids do not separate until meiosis II.
Answer: D

17) The law of independent assortment states that
A) in fertilization, the combining of sperm and eggs is random.
B) in meiosis, crossing-over creates genetically diverse gametes.
C) in any dihybrid cross, it is possible to get any combination of phenotypes.
D) generation of male and female gametes must occur in separate organisms.
E) in gamete formation, gene pairs are transmitted independently of each other.
Answer: E

18) When Mendel crossed plants and followed two characters (a dihybrid cross), he saw a 9:3:3:1 ratio of characters in the offspring. What did he conclude?
A) The transmission of one character is unaffected by the other.
B) The two characters affect each other's transmission.
C) Dominant characters are always more common than recessive ones.
D) Characters are controlled by pairs of genes on homologous chromosomes.
E) In meiosis, one allele of each gene is passed to each gamete.
Answer: A

19) In a dihybrid cross, if heterozygotes are crossed, what fraction of the offspring are expected to have both the dominant phenotypes?
A) 1/3 B) 2/3 C) 1/16 D) 3/16 E) 9/16
Answer: E

20) In the individual with genotype AaBB, what percent of gametes will contain the A allele?
A) 100% B) 75% C) 50% D) 25% E) 10%
Answer: C

21) How many different types of gametes can be generated by an individual with genotype AaBB?
A) 1 B) 2 C) 3 D) 4 E) 8
Answer: B

22) What kind of phenotype ratio would you expect from a trihybrid cross?
A) 1:1
B) 3:1
C) 9:3:3:1
D) very complex
E) It cannot be determined.
Answer: D

23) A "trihybrid cross" would
A) follow a trait over three generations.
B) follow three traits in a cross.
C) be a cross involving gametes from three different individuals.

D) follow a gene for which there are three alleles.
E) follow a gene that affects three different traits.
Answer: B

Use this information for the seven questions below.
In Mendel's pea plants, yellow seeds are dominant to green seeds. Purple flowers are dominant to white flowers. Use Y and y for the seed color alleles and P and p for the flower color alleles. Flower color and seed color assort independently.

24) If a true breeding green seed-producing plant is crossed to a heterozygous yellow seed-producing plant, what percent of offspring produces green seeds?
A) 10 B) 25 C) 33 D) 50 E) 100
Answer: D

25) If a YyPp plant is crossed to a Yypp plant, what is the probability that the resulting plant will have the genotype Yypp? (Hint: Determine two separate probabilities and use the rule of multiplication.)
A) 1/2 B) 1/4 C) 1/8 D) 1/16 E) 1/32
Answer: B

26) A plant of unknown genotype with yellow seeds and purple flowers is crossed to a plant with green seeds and white flowers. The offspring all have yellow seeds, but some have purple flowers and some have white flowers. What is the genotype of the yellow-seeded, purple-flowered plant?
A) YyPp B) YyPP C) YYPP D) YYPp E) Yypp
Answer: D

27) A true-breeding plant with green seeds and white flowers is crossed to a plant that is heterozygous for the genes for both phenotypes. What is the probability that the cross will yield a plant with green seeds and white flowers?
A) 1/16 B) 3/16 C) 1/4 D) 1/32 E) 3/32
Answer: C

28) If a plant that is heterozygous for both flower color and seed color genes is self-fertilized, what proportion of the offspring will have one of the dominant phenotypes, either the seed color or flower color, but NOT both?
A) 9/16 B) 6/16 C) 9/32 D) 6/32 E) 6/64
Answer: B

29) When Mendel crossed heterozygotes for flower color and seed color, what proportion of the offspring had both dominant phenotypes?
A) 9/16 B) 3/16 C) 1/16 D) 1/3 E) 3/4
Answer: A

30) What is the relationship between the Y and P?
A) They are two different chromosomes in the pea plant.
B) They are incompletely dominant alleles of the same gene.
C) They are two different genes on the same chromosome.
D) They are the pleiotropic effects of a single gene.
E) They are two different genes on two different chromosomes.
Answer: E

Use this information for the three questions below.
A, B, and O blood type in humans is controlled by a single gene with three alleles: I to power of (A), I to power of (B) and i.

31) Imagine a fourth allele for blood type, I to power of (C). If it is also codominant with I to power of (A) and I to power of (B), and dominant to i, how many possible blood type phenotypes are there?
A) 3 B) 6 C) 7 D) 8 E) 9
Answer: C

32) Type O is the recessive trait. The i allele is recessive to both I to power of (A) and I to power of (B). Which of the following could be possible genotypes of the parents of a person with type O blood?
A) I to power of (A) I to power of (B) and ii
B) I to power of (A)i and I to power of (B)i
C) I to power of (A)i and I to power of (A) I to power of (A)
D) I to power of (A) I to power of (B) and I to power of (A) I to power of (B)
E) both parents must be ii
Answer: B

33) A person with the genotype I to power of (A) I to power of (B) has type AB blood. This is an example of
A) dihybridness.
B) the effect of the environment on phenotype.
C) codominance.
D) pleiotropy.
E) incomplete dominance.
Answer: C

34) If a female who is a carrier for the hemophilia gene has a child with a male who does not have hemophilia, which prediction is correct?
A) All of the sons and none of the daughters will have hemophilia.
B) All of the daughters and none of the sons will have hemophilia.
C) Half of the sons and half of the daughters will have hemophilia.
D) Half of the sons and none of the daughters will have hemophilia.
E) Half of the daughters and none of the sons will have hemophilia.
Answer: D

35) A female who does not carry the color blindness allele has children with a male who is color blind. What proportion of their children will be color blind?
A) all B) 1/4 C) 1/2 D) 3/4 E) none
Answer: E

36) A female is not color blind, but half her sons are. Her daughters are not color blind. Which conclusion is correct?
A) The father is color blind.
B) The father is not color blind, but is heterozygous for the color blindness gene.
C) The woman is heterozygous for the color blindness gene.
D) Color blindness is dominant.
E) Color blindness is autosomal.
Answer: C

Use this information for the question(s) below.
A man and a woman are both carriers of sickle cell anemia. The man is color blind.
The woman is not color blind, nor is she a carrier of color blindness.

37) How would the genotype of the man be written?
A) Hb to power of (S), XY B) Hb to power of (A) Hb to power of (A), Y to power of (C)Y C) Hb to power of (S) Hb to power of (A), Y to power of (C)Y D) Hb to power of (S) Hb to power of (S), X to power of (C)Y E) Hb to power of (S) Hb to power of (A), X to power of (C)Y

Answer: E

38) The proportion of all their children who will be carriers of color blindness who also have sickle cell anemia is
A) 1/2. B) 1/6. C) 1/8. D) 1/16. E) 1/32.
Answer: C

39) The percentage of all their children who will be color blind males with sickle cell anemia (meeting all three conditions) is
A) 0. B) 10. C) 25. D) 33. E) 50.
Answer: A

40) What proportion of their children will not have sickle cell anemia, yet will have malaria resistance?
A) 1/2 B) 1/4 C) 3/4 D) none E) all
Answer: A

41) A person who is heterozygous for the Huntington disease allele has offspring with someone who does not have HD. What proportion of their children will have HD?
A) 1/16 B) 1/10 C) 1/8 D) 1/4 E) 1/2
Answer: E

42) What is a karyotype?
A) a fetal cell
B) a technique that obtains fetal cells for testing
C) a test that determines if a cell is cancerous
D) a test for the activity of amino acid synthesizing enzymes in a cell
E) a picture of the set of chromosomes from a cell
Answer: E

43) Which condition is caused by a chromosomal deletion?
A) Down Syndrome
B) Huntington disease
C) Turner Syndrome
D) cri-du-chat
E) sickle cell anemia
Answer: D

44) Why might a chromosomal duplication be harmful?
A) It involves the loss of some genes.
B) It changes the orientation of a chromosomal segment.
C) It might result in production of too much of a protein.
D) It adds so many chromosomes that they might not fit in the cell.
E) It results in polyploidy, which is not tolerated by humans.
Answer: C

45) People with Down Syndrome have
A) a diploid set of chromosomes, plus one extra of number 21.
B) a haploid set of chromosomes, plus one extra of number 21.
C) a diploid set of autosomes, but only one sex chromosome.
D) three of all the chromosomes, including X and Y.
E) a diploid set of chromosomes, except only one of number 21.
Answer: A

46) A person with the genotype XO is mainly female, phenotypically. A person with the genotype XXY is mainly male. What can you conclude about the Y chromosome?

A) It has the same genes as X, just different alleles.
B) It has the same genes as X, in different orientation.
C) A Y chromosome confers maleness, regardless of the number of X chromosomes.
D) The only genes it carries are for male development.
E) A human cannot survive without a Y chromosome.
Answer: C

47) Gametes with too many or too few chromosomes can result from non-disjunction in
A) either mitosis I or mitosis II.
B) mitosis I only.
C) mitosis II only.
D) either meiosis I or meiosis II.
E) meiosis I only.
Answer: D

48) If a disease is autosomal recessive, it is caused by
A) failure to inherit one of the sex chromosomes.
B) failure to inherit one of the autosomes.
C) inheritance of an extra autosome.
D) a gene on a chromosome other than X or Y.
E) a gene on a chromosome other than an autosome.
Answer: D

49) What is the connection between sickle cell anemia and malaria?
A) Both are X-linked.
B) Both are autosomal recessive.
C) Both are dominant.
D) Heterozygotes for malaria have some resistance to sickle cell anemia.
E) Heterozygotes for sickle cell anemia have some resistance to malaria.
Answer: E

50) A person is heterozygous for an autosomal dominant condition. If they have children with someone who is homozygous recessive, which statement is correct?
A) All of their children will be carriers.
B) Half of their children will be carriers.
C) All their children will have the condition.
D) Half of their children will have the condition.
E) None of their children will have the condition.
Answer: D