Auxin and Protein Phosphorylation in Plants

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Auxin controls many physiological processes such as cell elongation, cell division, and differentiation in plants and elicits rapid responses in organelles such as plasma membrane, golgi apparatus, endoplasmic reticulum, and the nucleus as well as in the cell wall. Auxin functions through a signal transduction mechanism (Figure 1) which converts the external hormonal signals to various cellular responses. Protein phosphorylation is a post-translational reversible covalent modification which is recognized as a major regulatory mechanism by which various cellular metabolic activities are controlled (Budde and Chollet, 1988; Pierce Chemical, 1987). It is known that many key regulatory proteins are phosphorylated. This phosphorylation leads to alteration of biological properties in animal cells (Nishizuka, 1986; Pierce Chemical, 1987). Protein kinase plays an important role in signal transduction by amplifying the external signal (Nishizuka, 1986). External signals are transmitted to the inside of the cell by second messengers and these messengers in turn regulate the activity of protein kinases and phosphatases; thereby resulting in the expression of physiological responses. Messenger-regulated protein phosphorylation by these enzymes is required to maintain the physiological response because the increase in the level of second messengers is transient.

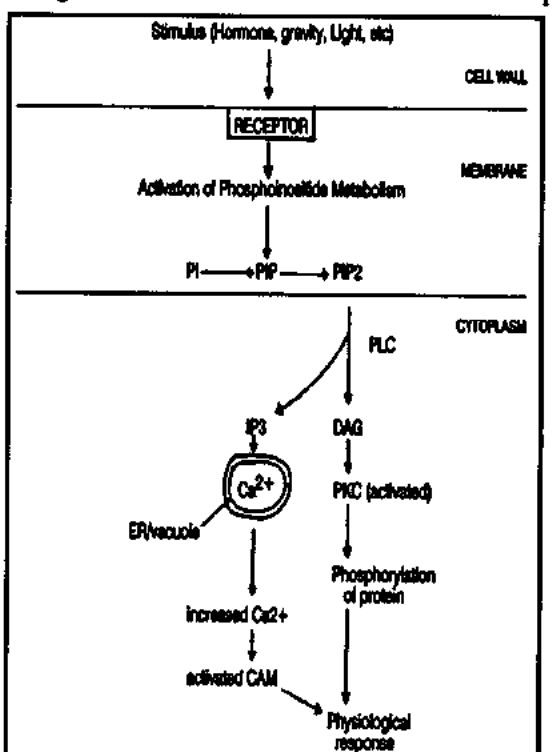

Figure 1. A Model Pathway of Signal Transduction in Plant Systems.
PI = phosphatidylinositol; PIP = phosphatidyl inositol-4-monophosphate; PIP2 = phosphatidyl inositol-4,5-bisphosphate; PLC = phospho-lipase C; IP3 = inositol 1,4,5-trisphosphate; DAG = diacylglycerol; PKC = protein kinase C like protein; ER = Endoplasmic reticulum; CAM = calmodulin.

The action of auxin may be linked to calcium which plays an essential role in the growth of plant cells. Auxin-promoted growth of isolated *Avena* (oats) coleoptile sections is inhibited by calcium chloride; the inhibitory action of calcium is expressed within 2 min (Cleland and Rayle, 1977). The movement of calcium is also correlated with auxin transport in roots exposed to gravistimulation (Lee *et al.*, 1983). Based on these results, the effects of auxin may be mediated by auxin-induced changes in internal calcium level. The calcium binding protein, calmodulin (CaM), is found in plants (Anderson and Cormier, 1978); calcium increases the in vitro phosphorylation of proteins in several plant species (Hetherington and Trewavas, 1984; Salimath and Marmé, 1983; Veluthambi and Poovaiah, 1986). Calmodulin may be involved in auxin-regulated activities in plants because of auxin mediation of calcium levels.

Auxin also alters the level of phosphorylation of pea epicotyl proteins (Reddy *et al.*, 1987). Furthermore, Zbell and Walter-Back (1988, Zbell *et al.*, 1989) have reported that auxin affects the phosphoinositide turnover response in cell membrane.

In this laboratory exercise, the effect of auxin on *in vitro* protein phosphorylation inthe cytoplasmic and membrane fractions of the primary roots of maize is examined.

GOALS OF THE EXPERIMENT

1. Examine the effect of auxin on *in vitro* protein phosphorylation in maize (*Zea Mays*) roots.
2. Comparison of the effect of auxin and calcium on *in vitro* protein phosphorylation in maize roots.

TIME REQUIREMENTS

0.25 hour (approximately) 3 days prior to experiment (soak grain)
3.00 hours (approximately) 1.5-2.0 days prior to experiment (plant grain)
2.00 hours (approximately) 1 days prior to experiment for gel preparation
4.00 hours to prepare cytoplasmic and membrane fractions
2.00 hours to prepare and load samples for electrophoresis
4.00 hours to run the gel
1.00 hour to separate the gel from glass plates
12.00 hours or over night to stain the gel
8.00 hours or more to destain the gel
2.50 hours to dry the gel
2.0-3.0 days to expose the gel to film
0.5 hours to develop the film

MATERIALS AND EQUIPMENT

Acetic acid
Acrylamide
Ammonium persulfate
ATP (gamma-32P, specific activity 185 MBq/ml)
bis-Acrylamide
Bromophenol blue
Centrifuge and Ultracentrifuge; tubes
Coomassie brilliant blue R-250
Dark room
Developer and fixer for film
Dithiothreitol (DTT)
EDTA (ethylenediamine tetraacetic acid)
EGTA (ethyleneglycol-bis[ß-amino ethyl ether]-N,N,N',N'-tetraacetic acid)
Electrophoresis kit
Ethanol
Film (Kodak X-OMAT AR, 13 x 18 cm)
Freezer (-70° C)
Glycerol
Glycine
Grain, corn
HCl
Indole 3-acetic acid (IAA)
Magnesium chloride
2-Mercaptoethanol
MES (2[N-morpholino]ethane sulfonic acid)
Methanol
Microcentrifuge; at least 11 tubes needed / group
Miracloth
Mortar and pestle
Paper towel
Plastic trays and tub
PMSF (Phenylmethylsulfonyl fluoride)
Protein assay kit (8)
SDS (Sodium lauryl sulfate)
Sea sand
Standard marker (Dalton Mark VI, Sigma, St. Louis)
Sucrose
N,N,N',N''-Tetramethylethylene diamine (TEMED)
Tris (Tris[hydroxymethyl] amino methane)
Triton X-100
Water bath

METHOD

Seedling Preparation. Corn caryopses (about 10 trays; approx. 300 grains per tray) are soaked overnight in running tap water to prevent anaerobiosis. Seeds germinate between wet paper towels on plastic trays in a vertical position. To obtain straight primary roots you should place the corn seeds in rows on a tray covered with 2-3 layers of paper towel. Cover the grains with 3 or 4 layers of paper towels; place another tray over final layers of towels to hold the paper towels and seeds in place. Position the trays vertically in a shallow tub containing 1-2 inches of water. Primary roots of approximately 1.5-2.0 cm should be used for the experiment. This should require 2-3 days of growth, depending upon the cultivar and temperature.

Isolation of Membrane and Cytoplasmic Fractions. One-cm root segments (at least 3 g of roots) which exclude the root tip (terminal 1 mm) are ground in ice with homogenization buffer containing 50 mM MES-NaOH, pH 7.0, 5 mM MgCl2, 0.5 mM DTT, 0.25 M Sucrose, 5 mM EDTA, and 0.5 mM PMSF using a mortar and pestle (1:2 w/v). All procedures are performed at 4°C. The homogenate is filtered through Miracloth and centrifuged for 10 min at 7,000 g to discard nucleus and cell wall. The supernatant is collected and centrifuged for 30 min at 100,000 g for 30 min. After centrifugation, the supernatant

is used for procedures requiring cytoplasmic fractions. The pellet is suspended in homogenization buffer at the same volume and centrifuged again at 100,000 g for 30 min. The final pellet is suspended in homogenization buffer (1 ml/5 g initial fresh weight of root), and is used for procedures requiring membrane fractions. The protein content of fractions is determined by BCA Protein Assay kit (8).

Protein Phosphorylation. *In vitro* protein phosphorylation assays are conducted at 30°C in a total reaction volume of 100 µl containing 50 mM MES-NaOH (pH 7.0), 0.5 mM DTT, 5 mM $MgCl_2$, 0.2 mM EGTA and 200 µg of membranous or cytoplasmic proteins. Ten µl of 1% Triton X100 is added 15 min before phosphorylation of the membrane only fractions. The reaction is initiated by adding 1 µCi [gamma-32P]-ATP. The reaction is terminated after 1 min by adding the same volume (100 µl) of electrophoresis sample buffer (Table 1, sample buffer) and by boiling the reaction mixture for 5 min. The reaction mixture of membrane protein is centrifuged at 10,000 g for 5 min in a microcentrifuge and the supernatant is used for analyses by SDS-PAGE. The reaction mixture of cytoplasmic protein is used without further centrifugation.

Gel Preparation. Linear gradient gel (12 to 18%) is prepared as shown in Table 2. Each mixture degasses for 10 min by vacuum, and add 70 µl 10% freshly made ammonium persulfate and 10 µl TEMED. Pour 5 ml of each gel solution from high % to low %; bottom is 18% and top is 12%. Layer 2 ml of distilled water on top of gel, and leave over night to polymerize. Stacking gels are prepared as shown in Table 3after the running gel is polymerized. Degass the mixture for 10 min, add 50 µl of 10% ammonium persulfate and 10 µl TEMED. Pour carefully with the electrophoresis gel comb in place, and leave it for 1.5-2.0 hours to polymerize. Remove the comb and carefully wash the wells produced by the comb with distilled water to remove the bubbles in the lanes.

Running the Electrophoresis Gel. Pour the electrode buffer into the bottom chamber, and place the prepared gel plates vertically in the electrophoresis apparatus. All the bubbles in the bottom of the gel plates should be removed by the pipette which has a curved tip or a syringe with a bent needle. Samples, terminated reaction mixture, are applied to each lane. The volume is 50 to 75 µl. Carefully pour electrode buffer in the upper chamber. Electrophoresis is started at 25 mA for 1 hr and then the current is changed to 30 mA for 3-4 hours. Initial voltage is 60-80 volts and rises to around 210 volts. When the dye reaches 2 cm from the bottom of the plate, turn off the

Table 1. *Stock Solutions for Electrophoresis.*

Acrylamide	30% and 50%; dissolve 30 and 50 g acrylamide in separate 100 ml aliquots of distilled water, and filter it. Store at 4°C in the dark
bis-Acrylamide (1%)	dissolve 1 g bis in 100 ml distilled water, and filter it. Store at 4°C in the dark.
Running Buffer	1 M Tris-HCl; pH 8.7; dissolve 60.5 g Tris in 250 ml distilled water, and adjust to pH 8.7 with HCl, and then make to 500 ml with distilled water.
Stacking Buffer	1 M Tris-HCl; pH 6.8; dissolve 12.1 g Tris in 70 ml distilled water, and adjust to pH 6.8 with HCl, and then make to 100 ml with distilled water.
Sample Buffer	50 ml; frozen as 5 ml aliquots; dissolve 0.0186 g EDTA (1 mM), a pinch of bromophenol blue and 15 g glycerol in 30 ml distilled water, then add 0.5 ml 10% SDS, 6.2 ml 1 M Tris-HCl pH 6.8, and 0.45 ml 2-mercapto-ethanol, and make to 50 ml with distilled water.
Electrode Buffer	pH 8.3; dissolve 5 g Tris and 38.8 g Glycine in 700 ml distilled water, then add 10 ml of 10% SDS, adjust pH to 8.3, and make to 1000 ml with distilled water.
Staining Solution	dissolve 0.35 g Coomassie blue brilliant R-250 in 454 ml of 95% ethanol. Add 454 ml distilled water and 92 ml acetic acid.
Destaining Solution	mix 75 ml of 95% ethanol, 75 ml of 95% methanol, and 50 ml of acetic acid, then add 800 ml of distilled water.

Table 2. Preparation of Running Gel (12-18% Acrylamide). Add the volumes in ml of each of the 5 solutions listed in the column under a % gel to obtain a 20 ml volume of a particular acrylamide solution.

% Gel / Solution	12%	14%	16%	18%
50% acrylamide	4.8	5.6	6.4	7.2
1% bis-acryl-amide	2.14	2.14	2.14	2.14
1 M Tris (pH 8.7)	7.74	7.74	7.74	7.74
20% SDS	0.1	0.1	0.1	0.1
distilled H2O	5.22	4.42	3.62	2.82

power supplier. Take the gel out carefully and stain overnight in staining solution (Table 1). The stained gel is destained in the destaining solution (Table 1) for 8 hours or more until bands can be observed and background is completely clear. The destained gel is placed on wetted filter paper and covered with wetted cellophane, remove all The bubbles inside the cellophane are all removed and dried. The completely dried gel is exposed to x-ray film in a cassette and keep at -70°C for 2-3 days.

EXPERIMENTAL PROCEDURES

1. One day prior to experiment, prepare the running gel.
2. Remove the tip of primary roots (1 mm) and make a 1 cm segments. All segments of roots should be kept in liquid nitrogen or ice.
3. Measure the fresh weight of segments (at least 3 g).
4. Grind the segments with homogenization buffer, and prepare cytoplasmic and membrane fractions (see METHOD). During this step, prepare the stacking gel and wait to polymerize.
5. Prepare the reaction mixture in microcentrifuge tubes and add each concentration of IAA (1 µM to 0.1 nM). This experiment need 11 tubes such as 1 for standard marker, 5 for membrane fraction, and 5 for cytoplasmic fraction with each concentration of IAA.
6. Start the reaction by adding 1 µCi [gamma-32P]-ATP and terminate the reaction by adding 100 µl sample buffer.
7. Load the sample (50 to 75 µl) in each lane, and start electrophoresis (see METHOD).
8. Place the gel stain in staining solution, and destain. Gel is then dried and exposed to film at -70°C for 2-3 days.
9. Develop the film and observe the pattern of bands which occur phosphorylation.

Table 3. Preparation of 5% Stacking Gel (5%). Add the volume of each of the 5 solutions listed to obtain 10 ml of 5% stacking gel..

Solution	Volume
30% acrylamide	2.7 ml
1% bis-acrylamide	2.1 ml
1 M Tris	2.0 ml
20% SDS	0.08 ml
distilled H2O	9.2 ml

OBSERVATIONS AND QUESTIONS

Compare the pattern of phosphorylation of membrane and cytoplasmic fractions. Are proteins phosphorylated in IAA treated samples which are not phosphorylated in the controls? Are any proteins phosphorylated in the controls which are not observed in the IAA treated samples?

Observe the effects of various IAA concentrations on protein phosphorylation in membrane and cytoplasmic fractions. What sizes of protein are phosphorylated in the various concentrations of IAA? Are different proteins phosphorylated at different IAA concentrations?

Is there a difference in the stained gel (total protein pattern) between the controls and the IAA treatments?

SUGGESTION FOR ADDITIONAL EXPERIMENTS

1. Add calcium chloride (1 mM to 1 µM) or EDTA/EGTA (1 mM to 80 mM) to the reaction mixture and observe the effect of calcium on *in vitro* protein phosphorylation.
2. Gravistimulate roots and separate upper and lower halves of the roots after 60-90 min. Compare the protein phosphorylation of the upper and lower halves of gravistimulated roots and control roots.
3. Compare the phosphorylation patterns which are observed with auxin treatment to the phosphorylation patterns observed in tissue treated with diacylglycerol, an activator of the second messenger system.

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Call for Nominations

This year, AMCBT members will elect four of our lot to serve in the following capacities:

President Secretary Two Members of the Steering Committee

Send recommendations to:

Professor Barabara Newman
Biomedical Sciences Department
Southwest Missouri State University
901 S. National Avenue
Springfield, MO 65804-0094

e-mail: SAR978@SMSVMA
Phone: (417)-836-5300
FAX: (417)-881-5208

Please send out recommendations of members whom you believe both would serve the organization well and would be eager to be elected.

Qualitative and Quantitative Pedigree Analysis: Graph Theory, Computer Software, and Case Studies

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Pedigrees, fate maps, and phylogenetic trees are three widely used graphical metaphors for representing historical relationships in genetics, developmental biology, and evolution. The word pedigree emanates from the French word for a stork's leg and three-toed foot (Bradie, pers. comm.). While widely employed in genealogy, ontogeny, and phylogeny, students struggle sometimes with comprehending the inferential power of these diagrams. Therefore, we have developed: a series of elementary mathematical tools for re-representing pedigrees; pedigree generators; pedigree-driven data base management systems; and, case studies for exploring genetic relationships.

One tool for examining a pedigree is to simplify it to a genetic graph. Inbreeding is most easily seen in this simplified representation of a pedigree. In 1736, Euler, the Swiss mathematician, published a solution to an entertaining puzzle on traversing seven bridges between the two banks of a river and two islands in Konigsberg without ever traversing the same bridge twice. He demonstrated that no such solution existed by establishing a logical system henceforth known as graph theory. We believe that the principles set forth here have considerable pedagogical promise as alternative ways of presenting genetics which will hopefully simplify genetics to and/or be appealing to you. Furthermore, these principles demonstrate that graph theory as an elementary subset of modern mathematics has considerable potential for application to biological problems.

Some of the fundamental definitions of graph theory are:

(1) A graph is a set of points (called vertices) and line segments (called edges) connecting pairs of vertices. We can assume for our purposes that a graph is a subset of real 2-or-3-dimensional space.

(2) A directed graph (digraph) is a graph with a direction, usually indicated by an arrow, assigned to every edge.

Thus, the simplest dioecious genetic graph is simply two parents and one offspring (Figure 1).

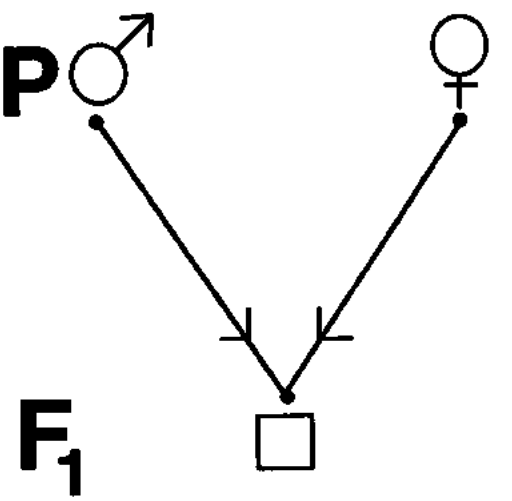


Figure 1. Simplest genetic graph.

Alternatively, the possible genotypes resulting from a mating could be represented by Figure 2.

(3) Notation: If G is a digraph, and A is a vertex of G, then the number of incoming edges to A is denoted by $p^*(A)$ and the number of outgoing edges from A is denoted by $p(A)$.

Every biology student can readily agree that each zygote must have two and only two biological parents, although one or both of them may be unknown. Also, any individual's parents consist of one female and one male. Finally, it is equally obvious that no one can be their own parent. Ore (1963) summarized these three biological axioms of sexual propagation in terms of graph theory:

"(a) $p^*(A) \leq 2$ for all A: no vertex has more than two incoming edges

(b) Every circular alternating path has a number of edges divisible by 4.

(c) The graph of a genetic experiment is acylic."

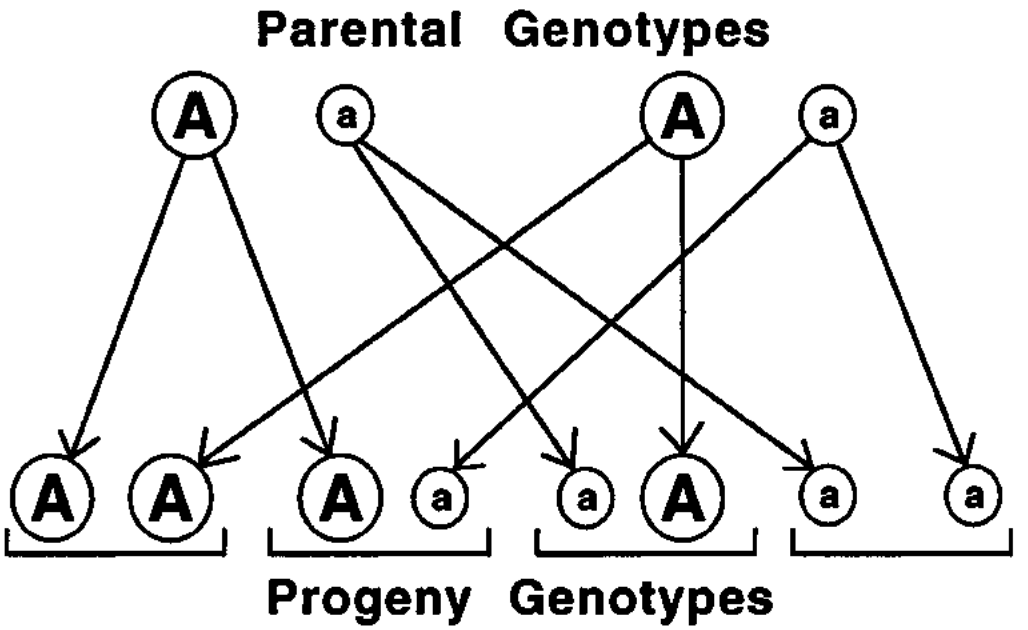


Figure 2. Genetic graph of a Punnett square. (After Mizrahi and Sullivan, 1976)

Secondly, we can illustrate Ore's (1963) point (b) by the following illustration of an impossible genetic graph (Figure 3). If parent P, is assigned as a female, then since P_1 and P_2 are the parents of child C_1, P_2 must be a male. Similarly P_1 and P_3 are the parents of child C_2; therefore, P_3 must also be male. How-ever, P_2 and $P_3$are the parents of child C_3; hence, both of them cannot be males. The reader or student is invited to try other circular alternating paths before generalizing about the divisible by 4 rule. Note exceptions in plants, protists, bacteria and hermaphroditic animals.

First, note that while the number of incoming edges, p* (A), (genetic material from parents) is maximally two, the number of outgoing edges from a vertex in a genetic graph, p(A), can vary considerably. For instance, human females release up to 400 eggs and human males release millions of sperm. While the maximum number of progeny borne by a single human female is roughly seventy, Attila the Hun supposedly fathered 6,000 children. Therefore, we can not easily place conditions on p(A).

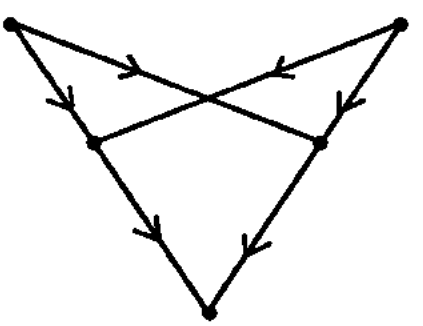


Figure 4. Brother-sister mating.

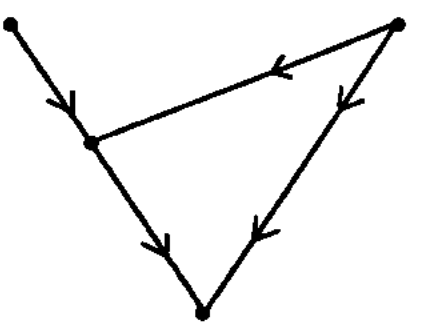


Figure 5. Child-parent mating. (An Oedipal graph?)

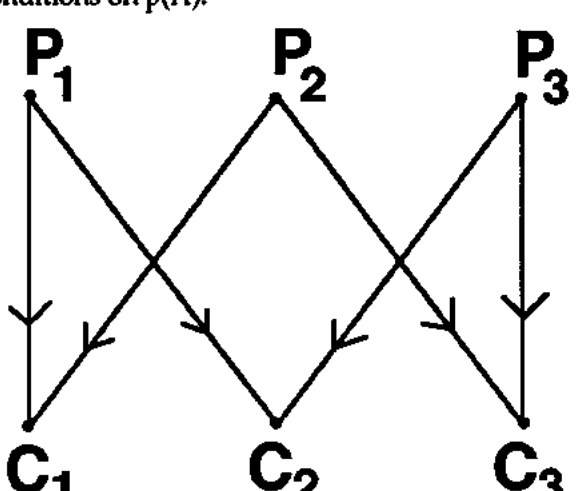


Figure 3. Unassignable parental genders in a circular alternating path.

Thirdly, while no individual is his own ancestor, we can take exception to Ore's (1963) stipulations: "For instance, since no individual may marry his own sister or brother there can be no configuration in our graph of the form given in Figure [4]. Since no individual may marry one of his parents there are no configurations of the type represented in Figure [5]." Unfortunately, Ore confuses both taboos and marriage with the realities of genetic relationship. Children can result both from incestuous and illegitimate matings; genetic graphs should reflect the actual biological heritage.

Crow (1976), following the work of Sewall Wright, has actually employed genetic graphs to

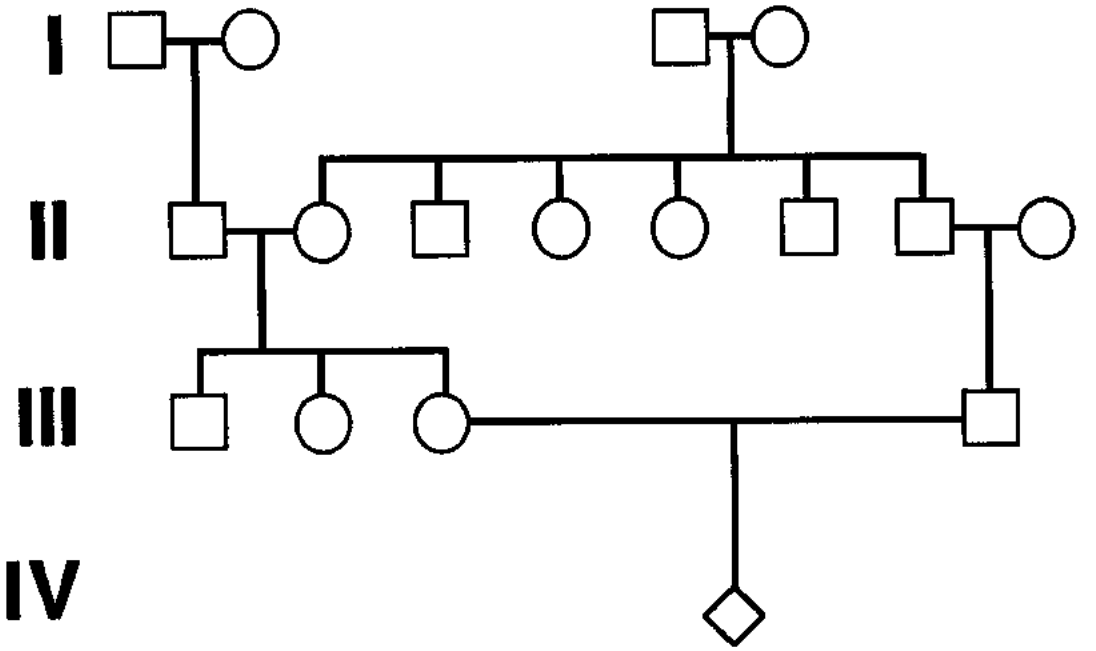


Figure 6. An inbred pedigree.

teach the analysis of inbreeding; however, he referred to his graphs of pedigrees as "arrow diagrams." Although his arrow diagrams implicitly follow Ore's (1963) three fundamental principles of genetic graphs, they were never explicitly stated. The pedigree in Figure 6 can be easily represented by the graph in Figure 7.

If we introduce an additional definition of graph theory, then the graph can be further simplified.

(4) A terminal edge is an edge between two vertices A and B such that there are either no other edges from A or no other edges from B.

Terminal edges cannot be part of a known inbreeding pathway. Thus, if a student is making a digraph of an inbred pedigree, then they should be entreated to:

(a) ensure $p^*(A) \leq 2$ for every vertex in the digraph;
(b) ensure all circular alternating digraphs are divisible by 4;

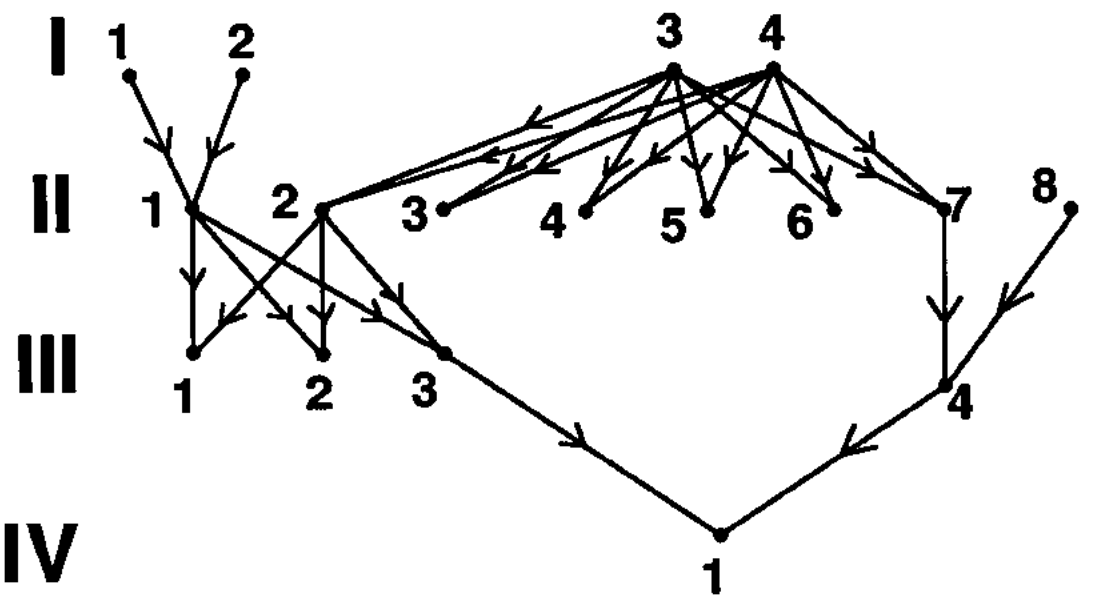


Figure 7. Genetic graph of the pedigree in Figure 6.

(c) ensure the digraph is acylic; and
(d) remove all terminal edges.

This policy would result in a genetic graph of the pedigree in Figure 6 which is illustrated in Figure 8.

The application of this policy reduces the arrow diagram or genetic digraph of an inbred pedigree, with sufficient self-checks, to the point where now subjective advice can be given for further simplifying the digraph with little room for confusion. At this point, the student should remove all edges which are not on circular paths; that is, if we want to calculate the inbreeding coefficient of individual IV-1, then we are only interested in retaining those directed edges for each parent of IV-1 which emanate from a common vertex (vertices I-3 and I-4). By applying this further restriction, the result is illustrated very simply by Figure 9 and Table 1.

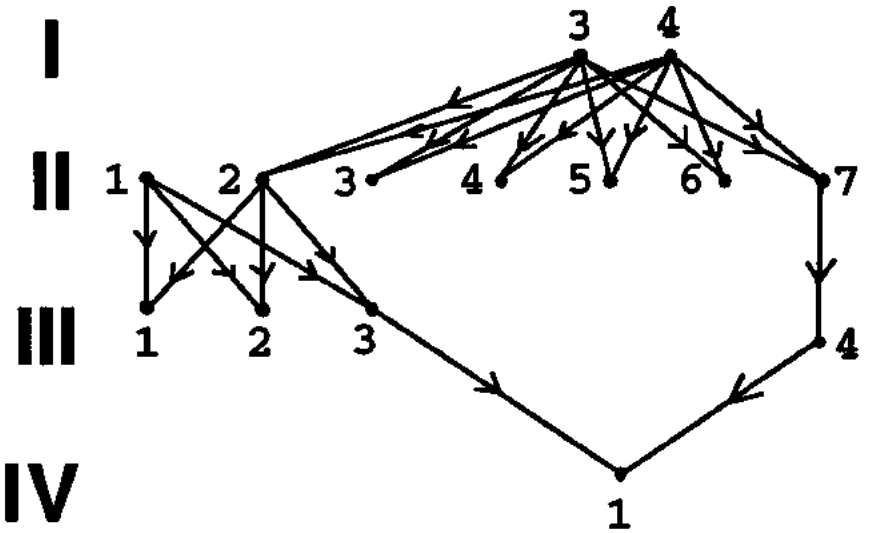


Figure 8. Genetic graph of the pedigree in Figure 4 without terminal edges.

We believe that the addition of these few principles of graph theory can help make the teaching of inbreeding analysis more explicitly logical and thus easier to learn. Additionally, we would like to reiterate the fact that you can check your arrow diagram of a pedigree to see if it fits the general rules of genetic digraphs.

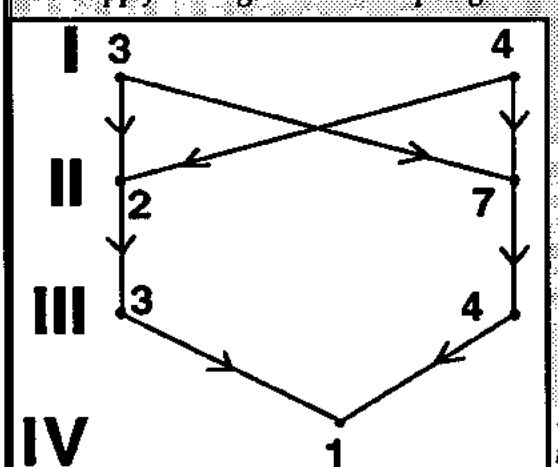
Another alternative for representing inbreeding is the "cyclogram" illustrated in Figure 10 which was developed by Nemat Hashem (1983), a genetic counselor in Cairo, Egypt.

Wright's formula for the inbreeding coefficient can now be readily applied:

$$F_I = \sum_{j=1}^{k} (1/2)^{n-1} (1 + F_A)$$

F_I = inbreeding coefficient of individual I; or, the probability that both alleles at a given locus in individual I came from a single ancestor
k = the number of independent circular paths
n = the number of directed edges in a given circular path
F_A = the inbreeding coefficient of a common ancestor; or, zero, if no additional information is known about the ancestry of the origin of a circular path

If we apply this algorithm to the pedigree in Fig. 9, then the following calculations will result:



$$F_{IV\text{-}1} = (1/2)^{6-1}(1 + F_{I\text{-}3}) + (1/2)^{6-1}(1 + F_{I\text{-}4})$$

$$F_{IV\text{-}1} = (1/2)^{5}(1 + 0) + (1/2)^{5}(1 + 0)$$

$$F_{IV\text{-}1} = (1/32) + (1/32)$$

$$F_{IV\text{-}1} = (1/16)$$

↑*Table 1. Inbreeding Coefficients*

←*Figure 9. Simplest genetic digraph of the inbreeding relationships of the pedigree shown in Figure 6.*

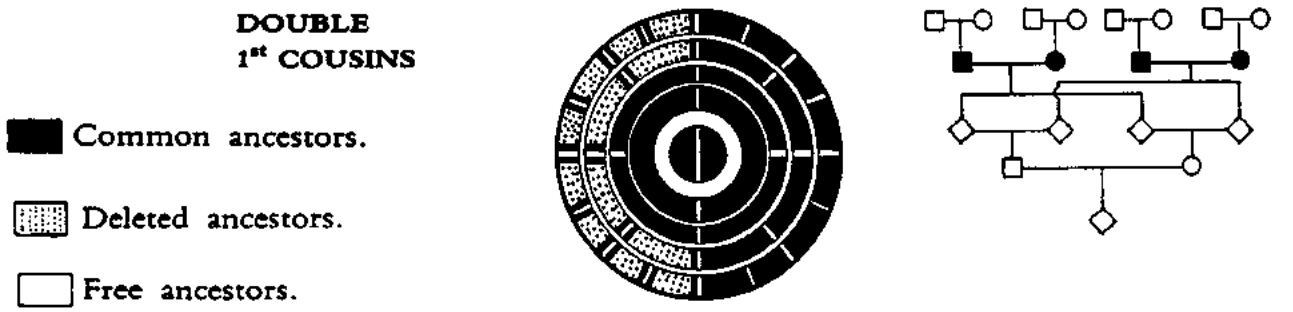


Figure 10. Cyclograms which project the inbred components of the different inbreeding patterns which prevail among Egyptians.

Computer Software

We have developed two different pieces of software to help students learn how to build and interpret pedigrees. Our older software, the Pedigree Construction Kit (Calley, Soderberg, and Jungck, 1990) allows the user to see multiple pedigrees generated for autosomal dominant and recessive and X-linked dominant and recessive traits (see Figure 11).

driven data base management system called Inherit that allows users to build pedigrees from scratch and to build a variety of templates for each individual. Since this is such a powerful tool and is easily available, we will only share two aspects as shown in Figures 13 and 14.

The palette of Inherit has a number of tools for creating, editing and displaying pedigrees. These are labeled in Figure 13 which shows the palette

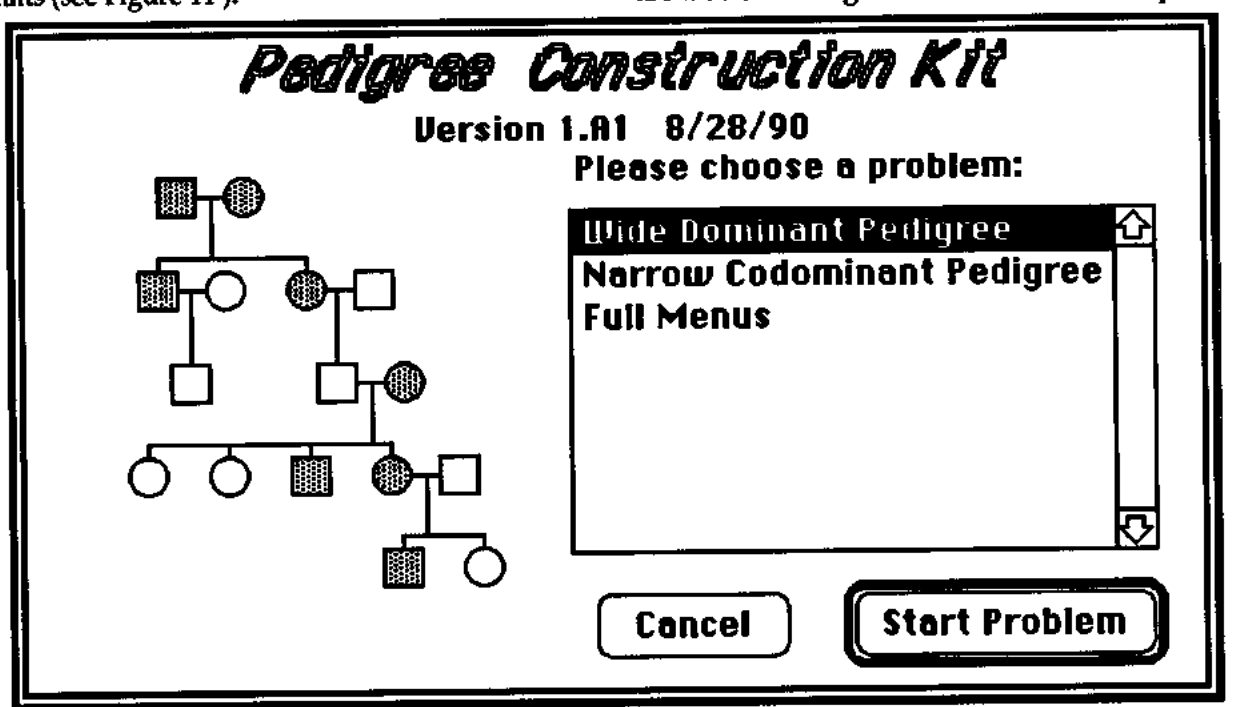


Figure 11.

After chosing a pedigree type from the generator, multiple examples (as many as your computer memory will allow) will be generated upon your command (Figure 12). The indiviudals in a pedigree can be moved laterally if that helps you clean up their appearance. Generally, we find that students print out several copies of each pedigree to carry out further analysis.

More recently, we (Jones, Calley, Soderberg, and Jungck, 1994) have developed a pedigree when it is in Show Phenotype mode; there is also available a Show Genotype mode.

Nichols, Sheffield, and Stone (1993) have devloped a Hypercard stack which is especially useful for linkage analysis. Also, Don Buckley at the University of Hartford is about ready to release a Supercard application on pedigree analysis that illustrates the transmission of alleles through a pedigree.

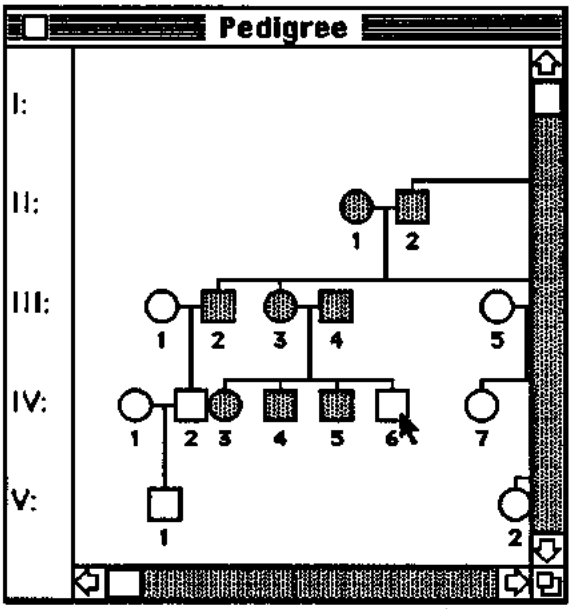


Figure 12. A PCK-generated pedigree

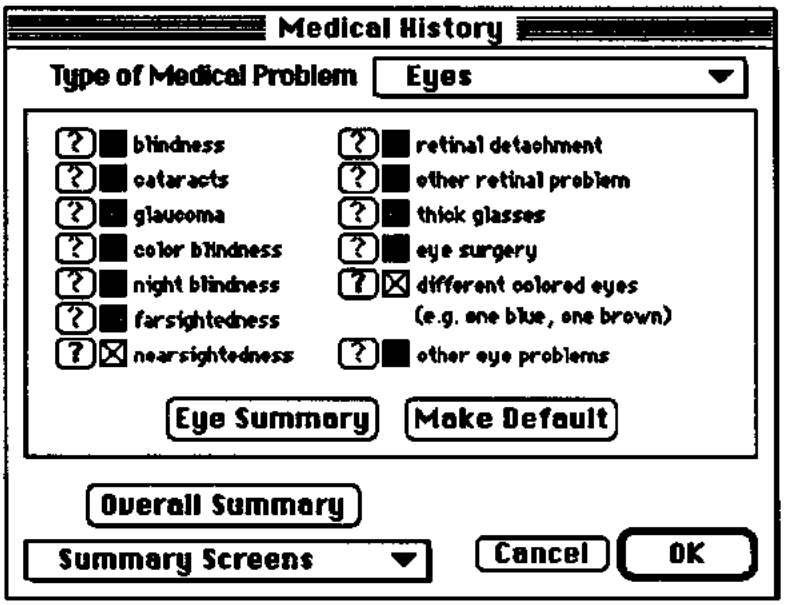


Figure 14. This is an example of one of dozens of templates that can be employed in Inherit to develop a clinical history for any individual in a pedigree that is being constructed.

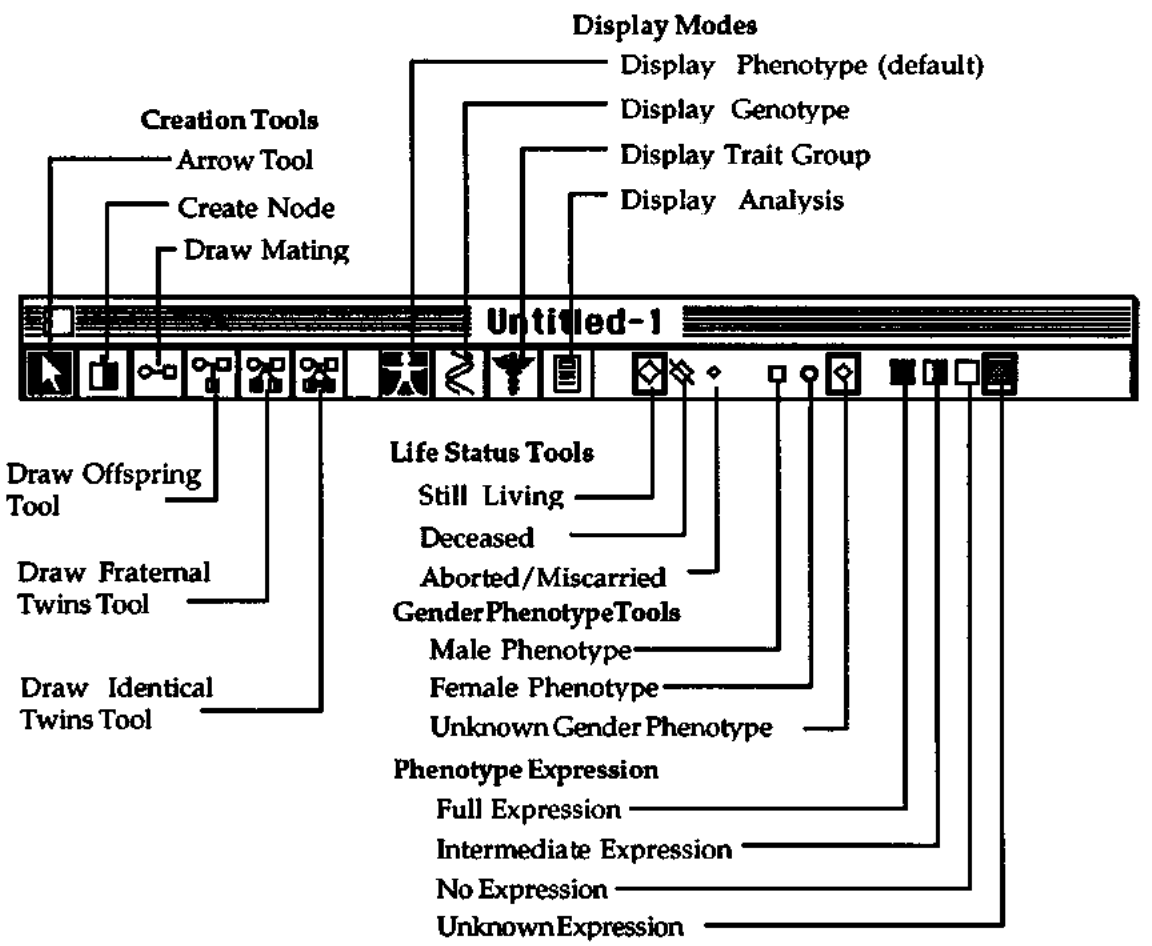


Figure 13. The Palette of Inherit in "Show Phenotype" mode.

confidentiality
disclosure/informed consent
prenatal screening
non-paternity
quality of life
individual rights (autonomy)
issue of individuality - when does life begin and end?
eugenics - "playing God"
euthanasia and the prolongation of life
right to decide (competency)
intervention
allocation of medical resources:
preventions vs. cures
acceptability of risk and cost-benefit analysis

Table 2. Bioethical Issues that can be Raised Using Case Studies

Using Pedigrees to Raise Social and Ethical Issues

Genetics is an ideal subject through which to explore controversial issues with students. Advances in genetic technology have raised dilemmas that have elicited strong emotional responses. The use of case studies and role playing scenarios can provide opportunities for students to acquire a better understanding of the complexities of controversial issues (see Table 2).

With PCK and/or Inherit, limitless combinations of situations can be created for classroom use. PCK facilitates flexibility in designing cases. A number of pedigrees can be generated and fit to a family history in numerous ways, or different disorders with the same mode of inheritance can be fit to the same pedigree. In either case, different issues will arise and different choices may need to be weighed. The involvement and creativity of the students make each case study unique. A case can be constructed around any genetic disorder. Pedigrees are generated by setting the mode of inheritance of a particular disorder along with preferences in regard to family size. A family history can then be written that describes the individuals represented in the pedigree. This will be illustrated with a case involving Marfan syndrome, an autosomal dominant disorder. Through this case, issues, choices and options can be examined, from a genetics counselor's position as well as from the point of view of each of the family members and their friends, colleagues and neighbors.

A Sample Case Study

In 1986 an article appeared in *Sports Illustrated* magazine concerning the death of volleyball star Flo Hyman. Autopsy revealed that she had had Marfan syndrome and had died from an aortic aneurysm. Individuals who have Marfan syndrome tend to be tall and lanky. Several basketball players who had been diagnosed as having Marfan syndrome or who had died from it were discussed in the article. Anne and her parents read this article and wondered if Anne had Marfan syndrome.

Proband Information

Anne is 16 years old, 5'11", plays varsity volleyball and basketball, wears contact lenses to correct nearsightedness, has slight scoliosis, a concave sternum, long fingers and toes, and wide spaced eyes. Her arm span exceeds her height by 5 inches, she wears orthodontic braces, and she is slightly knock-kneed.

The first decision that will have to be made is Anne's diagnosis. The proband information was hypothetically gathered from the first part of a visit to a clinical genetics center - the physical exam. Does she or does she not have Marfan syndrome? What information do you need to gather in order to make a diagnosis? Are there any confirmative tests for this particular disorder? What additional information will you need to know?

Most students will be unwilling to commit to a diagnosis on the basis of the information provided from the hypothetical physical exam. They are typically surprised and uncomfortable with the realization that there is no definitive test to base their answer on in this particular single gene disorder. Karyotyping can only reveal chromosomal aberrations, not single gene problems, and no DNA markers are available for analysis for Marfan syndrome. The idea that their diagnosis will depend on their best decision, given the information at hand, is disquieting. Most students are used to some source of confirmation of a "right" answer. A scenario such as this can force them to re-think what it means to label an answer as correct.

Siblings and Their Spouses

David - Age 25, married to Jessica (age 25), one daughter named Kristi (3 mos.), David wears glasses, is 6'3", has long fingers and toes, played basketball and ran track in high school, had some knee problems then.

Jessica - Age 25, 5'8", no glasses, no health complaints, had one miscarried pregnancy in her first trimester prior to the birth of daughter Kristi.

Cheryl - Age 14, 5'9", no glasses, slight case of scoliosis, born with club feet that responded well to treatment.

Parents

Mary - Age 47, 5'7", wears glasses, hay fever, has been diagnosed with carpal tunnel syndrom and mild diabetes, had two miscarriages in addition to her three children.

Peter - Age 49, 6'1", wears glasses, concave chest, high blood pressure, partial dislocation of lens in right eye, long fingers and toes.

Aunts and Uncles

Mary's siblings:

Dorothy - Age 46, wears glasses, 5'3", no major health problems, had an ovarian fibroid tumor removed at age 40, married with four children.

Ellen - Age 50, 5'5", high cholesterol, unmarried, no children.

Eric - Age 51, 6'0", wears reading glasses, back problems from car accident, suffers from exercised-induces asthma, is married and has two children from first marriage and three from his second.

Peter's siblings

Frank - Age 55, 6'4", wears glasses, slight hearing loss in one ear, was treated for alcoholism, is a heavy smoker and has developed chronic cough, divorced, the father of two children.

Alice - Age 56, 5'7", wears glasses, arthritis in one shoulder, married, has one daughter and a son who was born with cerebral palsy.

John - Deceased, heart attack at age 46, 6'2", had a dislocated lens in his right eye, had three children; the youngest girl is mildly mentally retarded and lives with his wife.

Larry - Age 58, 6'3", no glasses, high blood pressure, divorced twice, he now lives alone, had two children by his first marriage and one with his second wife, reported to be a heavy drinker.

Maternal Grandparents

Evelyn - Died at age 76 of a stroke, 5'4", arthritis in hands and feet, wore reading glasses, was said to have had as many as five miscarriages.

William - Age 81, no glasses, 5'10", no major health problems.

Paternal Grandparents

Martha - Age 86, 5'8", high blood pressure, some knee and ankle problems, wears glasses for distance and reading.

Charlie - Died at age 44 of a heart attack, severe vision problems, long fingers, described as tall and lanky, contracted polio at age 26 and had been wheelchair dependent since his polio treatment.

Table 3. Anne's Family History

When the students request information about Anne's family members to aid them in their diagnosis, they can be given a family history and pedigree (see Table 3). Each small group of students must come to a concensus regarding a diagnosis for each family member. Who has Marfan Syndrome and who does not? Their decisions will impact on the counseling issues they will explore next.

Despite the additional family information that has been given to the student groups, they are still often reluctant to commit to making decisions regarding diagnoses. However, faced with a case where they will have to come to concensus, the students will come to understand some of the complexitities of human genetics as they learn about specific disorders. The next section will provide the instructor with the information about syndromes and pleiotropy that can be taught from this case about Anne and her family.

About Syndromes, Pleiotropy, and the Diagnosis of Marfan Syndrome

A syndrome represents a disorder with a range of possible characteristics and severities. Not all individuals with a given syndrome will have the

same set of symptoms, yet each will have some sort of combination from the range of symptoms common to that particular disorder. In other words, not all individuals will fit the standard textbook list of symptoms for that disorder, yet there will be many similarities among individuals with the syndrome. This is true for Marfan syndrome (see Table 4).

Most syndromes are also examples of pleiotropy, multiple effects caused by a single gene. For example, in people with Marfan syndrome, you may see myopia; long limbs, fingers and toes; spinal curvatures; and aortic root dilatation with the consequence of an aortic aneurysm. All of these symptoms seem unrelated, yet are caused by a single gene (Marfan syndrome is an autosomal dominant disorder). However, in most cases of pleiotropy, if the underlying phenomenon can be identified, then the seemingly disparate symptoms can be understood in light of the causal mechanism. In Marfan syndrome, the genetic defect results in the production of sub-optimal connective tissue. Each one of the Marfan symptoms can be explained in terms of this defect. The eye lens is held in place by connective tissue. If the tissue is defective, the lens may move, causing myopia, or in severe cases, lens dislocations. Connective tissue is also the initial base material in long bone formation. This can result in the longer than average limbs and digits seen in most individuals with Marfan syndrome. The aortic root dilatation and aortic aneurysms can be explained by the defective connective tissue found in arteries. In Marfan syndrome, the aorta is the focal point of the defect-- it is under great force as the blood is forcefully ejected from the ventricle and enters the curvature of the aorta. This constant pounding of the blood against the wall of the aorta can lead to minute tears and stretching. Progressive damage to the aortic wall can ultimately result in rupture, otherwise referred to as an aortic aneurysm.

When diagnosing a genetic disorder, some of the most salient information comes from the family history. It is very important to sift carefully through the information obtained about each family member - what information is pertinent to the disorder in question, and what is unrelated health information? For example, an individual with Marfan syndrome is at increased risk for aortic dilatation and subsequent aneurysm. In taking a family history, you may discover that a family member has high blood pressure. It is unlikely that this is related to Marfan syndrome. However, you may also learn that several family members wear glasses. Again, vision problems may be a result of Marfan syndrome, or they may have other causes. Syndrome diagnosis is based on the presence of several symptoms occurring together, each of which can occur independently in the general population.

Creating a Family History for a Case Study

When creating a family history from a PCK-generated pedigree, choose the "extraneous" symptoms carefully. It is desirable to have some ambiguity so that students will debate the relevance of a health problem to the disorder in question. What information offers clues or insights into the identification of individuals at risk in the family? What information is incomplete? What additional information do you need to make a diagnosis? What family and individual issues and concerns should be addressed during the counseling session?

arm span greater than height (3-6 inches)
long fingers and toes
prominent shoulder blades
spinal curvature (scoliosis and thoracic lordosis)
dislocated eye lens
flat feet
highly arched palate with crowding of the teeth
myopia
mitral valve prolapse
mitral regurgitation (heart murmur)
lax joints

Table 4. Marfan symptom descriptors

This particular case study was created by choosing symptoms from a list of descriptions about Marfan syndrome and assigning them to individuals in the pedigree generated by PCK. Table contains a list of the spectrum of symptoms seen in individuals with Marfan syndrome. Such descriptors can be found in a variety of sources, including human genetics reference books and texts, the *On-line Mendelian Inheritance in Man (OMIM)*, and public education information from organizations such as the March of Dimes. Once the Marfan descriptors were assigned to various individuals, other health problems were chosen and assigned to the individuals.

Once the family history has been created for

the pedigree, the case study can be enriched by creating role play statements to give to the small groups to analyze in addition to the family history information. The role play statements are meant to help students recognize and understand the emotions of guilt, blame, anger, shame and denial that surround the birth and life of a person with a genetic disorder. Additionally, they create situations in which the students must put themselves in the roles of various members of the hypothetical family that is faced with reproductive decisions. What would they do if they were facing this situation, and more importantly, why did they make the choices they did? The students will have to assess their reproductive options (e.g., prenatal testing, artificial insemination by donor, adoption, no children, etc.) and the emotional implications of each. Role play statements generally fall into two categories: those that are generalizable to most genetic disorders, and those that are disorder-specific situations. Here are three examples of role statements that can be used with the sample case study.

Anne refuses to quit playing varsity level athletics despite the fact she is at risk for an aortic aneurysm. There is a good possibility that she will receive a full college scholarship upon graduation from high school.

Mary, Anne's mother, is terrified at the possibility that Peter may die at an early age. Mary has agoraphobia and experiences occasional panic attacks from anxieties about being in public places.

David and Jessica have planned on having two children.

Prenatal Tests

1. Ultrasound (major congenital malformations or deformations)
2. Serum AFP (neural tube defects)
3. Amniocentesis
   a. amniotic AFP
   b. Chromosomal abnormalities
4. Chorionic villi sampling (CVS)
   a. chromosome abnormalities
   b. DNA analysis (some single gene disorders)

Carrier testing

a. by enzyme assays
b. by DNA analysis

Table 5. Possible Tests for Genetic Disorder

Amniocentesis

- The results from your amniocentesis show a chromosome abnormality.
- The results from your amniocentesis do not show a chromosome abnormality.
- The cells taken from the amniocentesis did not grow. Would you like to have it performed again?

AFP Testing

- The results of your blood serum test indicate a higher than average AFP level. Would you like us to go ahead and test your amniotic fluid values?
- The results of your AFP testing was normal.
- The results of your amniocentesis show a higher than average AFP value.
- The results of your amniocentesis show a normal level of AFP.

Carrier Testing

- The results of your carrier test were positive.
- The results of your carrier test were negative.

Table 6. Some Examples of Outcomes Cards

Additional suggestions for role play statements that can be used when creating case studies can be found in the Appendices.

Ask the students to first place themselves in the role of the individual family members. What reactions, questions, concerns, and fears will each of the family members have? What ethical dilemmas or psycho-social implications are raised by the decisions that confront both the affected individuals and the individuals who do not have the disorder? In addition, what options and decisions must be made regarding prenatal and carrier status testing (see Table 5).

The role play scenarios can be extended to include outcomes of the decisions made by the students. First, what type of testing is appropriate for this case? Then, if students decide they would opt for prenatal or carrier testing, they can find out the results of their test by drawing from a stack of Outcome Cards, such as those listed in Table 6. In any scenario that involves testing, the students should consider the possibility of false negatives and false positives. Finally, what surprising additional inferences might be drawn about the inheritance of other characteristics of the individuals in the pedigree?

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News and Views

Time's Arrow, Time's Cycle: Myth and Metaphor in the Discovery of Geological Time,

by Stephen Jay Gould

Cambridge Massachusetts: Harvard University Press, 1987

Book Review

by

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Madison, Wisconsin 53706

Stephen Jay Gould is a Harvard University paleontologist who has written a number of popular books on various areas of natural history and evolution. Typically Gould's books are compilations of the monthly essays he writes for Natural History. This particular slim volume is different. The material contained in this book was written to work out and share some intellectual ideas that he has been grappling with for some time, namely, extending his insight into how past scientists used geological evidence to support arguments about the creation of the earth and the organisms that had lived here and live here now. In this work, originally delivered as part of the Jerusalem-Harvard lectures, Gould illustrates how early scientists began to portray time as a great abyss. He shares with us his story of how members of the theological/scientific community in the 17th, 18th and 19th centuries made the bottom of the chasm extend darker and deeper than ever before by the publication of their persuasive and compelling arguments.

As a teacher of geology and evolution, Gould struggles with how he can help his students understand and appreciate "deep time". Gould states, "An abstract, intellectual understanding of deep time comes easily enough - I know how many zeros to place after the 10 when I mean billions. Getting it into the gut is quite another matter. Deep time is so alien that we can really only comprehend it as a metaphor" (pg. 3). The metaphor that Gould chooses to use as a tool to more closely analyze the historical debate of the origin of earth and species is the dichotomy of time as arrow and time as cycle.

As scientists began acknowledging the concept of deep time from the period ranging from the mid-1600's through the early 1800's (Gould presents a quote from Rossi declaring that men in Hooke's time believed the earth to have a past history of 6,000 years while men of Kant's time were conscious of a past of millions of years), Gould chose to focus on the work of three famous scientists, each of whom worked during one of the three centuries in which deep time was accepted. Gould sets the stage by explaining, "... I have selected for intensive discussion only three cardinal actors on the British geological stage - the primary villain and the two standard heroes" (p. 4). As his argument unfolds, the reader learns that the basis of Gould's labels of villain and hero stem from his belief that the geology textbooks have grossly misrepresented the work of these three gentlemen by providing readers with a shallow and flimsy "cardboard history" depiction of the scientific community of this period by reducing the debate down to the religious catastrophists versus the atheistic uniformists. Gould cautions that Thomas Burnet, who wrote the *Sacred Theory of the Earth* in the 1680's, is portrayed by geology texts as a villain obscuring the way to scientific truth by popularizing the role of catastrophes while James Hutton, who published his *Theory of Earth* one hundred years later and Charles Lyell, who published the persuasive *Principles of Geology* in the 1830's are portrayed as the heroes of the scientific community, championing the reality of uniformity. Gould's work fabulously stands this cardboard representation on its head through his meticulous analysis of the arguments of the day by using his metaphoric tool of time as linear and time as circular.

Gould begins his analysis of Thomas Hutton's work by providing the reader with the frontispiece of Burnet's *Sacred Theory of the Earth,* which depicts Jesus standing on top of seven stages of the earth arranged in a circle beneath Jesus' feet. The engraving depicts the birth and the destruction of the earth as a distinct, never-

ending cycle. Thomas Burnet, we learn, was an Anglican clergyman, a practicing theoretical scientist at a time when the concept of science as a separate entity from religion was not yet discussed. Burnet was searching for rational, physical explanations for the great flood that wiped out all living things, save for Noah and the contents of his ark as depicted in the Bible. He attempted to speculate what the perfect earth was like before the flood, to look for geologically based explanations for enabling the flood to occur, and to describe the earth as it moves to destruction and renewal returning to the perfect earth.

If the Bible was to be taken literally, the earth was formed in six days. Burnet knew this was not true. He was not a biblical literalist, but was actually daring enough to publish work that could be viewed as blasphemous by his clerical colleagues. To work out his questions, Burnet corresponded regularly with Sir Isaac Newton. The letters provided the data Gould needed to track the evolution of Burnet's ideas. Gould describes how Burnet concludes one letter describing the great comet of 1680. Gould tells us that two years later that Halley would predict that another, smaller comet would return again in sixteen years. Halley's comet was in the sky as Gould wrote this book, and he offers this wondrous event as "... a primary signal of time's cycle (pg. 41)." Burnet was attempting to do for creationism what Newton did for matter and motion in the physical world - he was attempting to find the laws that govern the nature of creation and destruction of living things.

Burnet believed that the cyclical nature of birth and re-birth of the planet must also include the idea of progress over time. He visualized the original earth as consisting of perfectly smooth, concentric layers. The flood, he reasoned, arose from the water by emerging from one of the inner concentric layers and the craggy earth as it appears today occurred when the crust collapsed into the space that was previously occupied by the water. The land eventually erodes over time, falling back into the sea, until the time of conflagration occurs and the planet once again becomes the perfect, smooth round ball. This is time's cycle at its clearest. But Burnet, Gould proves, also believed in the notion of progress, time's arrow. He offers evidence in passages where it is clear that Burnet believed that there is directionality to time and that progress is inherent to directionality. For example, Burnet states that organisms are born, they grow, they die. Burnet, like his Danish colleague Steno, searched for vectors within the greater cycle of time.

Hutton's work, which followed in the next century, may have been lost, Gould points out, primarily due to its unreadability if it were not for Hutton's good friend John Playfair, who presented his friend's ideas in a much more accessible style of writing in his book *Illustrations of the Huttonian Theory of the Earth* published in 1802. Hutton , Gould claims, imposed "... the most pure and rigid concept of time's cycle ever presented in geology - so rigid, in fact, that it required Playfair's recasting to gain acceptability (pg. 63)." Hutton relied on large amounts of empirical evidence to support his argument that the land cycles through a series of destruction through erosion, consolidation through pressure and heat and subsequent renewal through upheavals. Gould uses Hutton's classic line " 'no vestige of a beginning, - no prospect of an end' (p. 63 & 65) to illustrate the author's clear belief in and dependence on the metaphor of time as a cycle.

Hutton differed from Burnet in that he believed that there is a mechanism to repair decay, the concept of uplift. So, when the mountains erode to provide soil and wash into the seas, strata build up in the seas where increasing pressure causes heat, which in turn causes the inner matter of earth to expand with force, causing uplift. These uplifts can be seen in unconformities, as depicted in an artist's portrayal of one of Hutton's first observations (provided at the beginning of Gould's section on Hutton).

Gould claims that Hutton is canonized in today's textbooks as the first empirical geologist, while Burnet has been castigated as the bad guy speculativist when the opposite is really true. Gould believes that although the frontispiece of Burnet's work depicts time as a cycle, his narrative portrays time as an arrow. Gould feels that this deeply held view of time as an arrow actually hindered Burnet from understanding the importance of using the view of time as a cycle as the later naturalists Hutton and Lyell successfully used to formulate explanatory laws for geological events.

Charles Lyell, like Hutton, believed in the cycle of time, and similarly did not argue for the concept of process. Lyell eloquently argues for the notion of time as arrow in his explanation of the empirical events noted in his work. Gould portrays Lyell as a superb rhetorician. He believes

that Lyell did far more than simply put down the "unscientific" catastrophists. Gould proves that the dichotomy of the war between catastrophists and uniformitarians is a false one, by providing us with evidence of Agassiz' support and praise that he found in the margins of Agassiz' copy of Lyell's *Principles of Geology*, a three volume work published between 1830 and 1833. Lyell believed that change occurred slowly and gradually over time and castigated catastrophism as being anti-empirical. He championed his own story of the triumph of uniformity through "perhaps the neatest trick of rhetoric," by claiming that if the methodological principles were true, then the substantive claims he derives from the methods must be true, too. Lyell managed to use theory to cover for imperfect data. Lyell criticized Hutton's catastrophic portrayal of upheavals. He smoothed his own argument for uniformity by believing that species that no longer exist, as evidenced by the fossil record, can one day exist again; Gould presents this belief at the beginning of the chapter on Lyell in a caricature by De la Beche portraying a future Lyell reincarnated as an ichthyosaurus which is teaching his long-billed students about the extinct humans from a pulpit on top of a human fossilized skull.

Gould shows us that Burnet was not the speculative armchair theorist that he is made out to be, but actually was the person who used empirical observations and careful logic to depict time more fruitfully as both an arrow and a cycle. On the other hand, Hutton and Lyell, typically presented as the objective scientists bringing enlightenment to the unscientific world of religion, actually held beliefs that are less accurate in the eyes of many present day evolutionary biologists and paleontologists.

So, what is the message to those of us who are scientists and science educators? The Chinese cookie take-home message is, beware of re-writes of other people's work in the guise of textbooks full of answers.

As Gould admonishes the skeptics among us, "But what does it matter? What harm is a bit of heroic folderol about an illusory past, especially if it makes us feel good about the progress of science? I would argue that we misrepresent history at our peril as practicing scientific researchers" (p. 114).

The power of the arrow/cycle metaphor becoming a key to unlocking greater insight into historical arguments came to Gould when he was on break from a "boring" meeting at the National Museum of Art in Washington, D.C. He saw at this gallery James Hampton's intricate throne on exhibit, carefully crafted out of bits of foil, wire, and other items he was able to gather at no or little expense. As Gould states at the end of his book, "This book congealed during the next ten minutes, one of those magic moments in any scholar's life"(p. 184). Gould shares the magic of his epiphany to a larger audience through the publication of this book.

In places Gould's argument is fascinating and illuminating, but it also can be frustrating for a reader who may be nominally knowledgeable in evolutionary biology, but uneducated in the geologic debate and players who underlie it. Gould's analysis of Hutton's work was wonderfully illuminating and captivating, but the analysis of Burnet and Lyell's work was difficult to grasp. He was obviously enthused and loving in his writing about these famous men, but I found myself reading and re-reading sections asking, what does he mean here, or what is he trying to do here? The intricacies of the use of the metaphor of time as a linear arrow and also as an endless cycle is obviously deep in Gould's belly, but as a recreational reader of history of science, I found it frustrating and irritating that he did not set the scene well enough for me to also be able to develop a gut-level appreciation of the debates at the time. I struggled with gaining a better appreciation of the complexities and subtleties of the times. Finally, after re-visiting section after section, I realized that despite being forewarned early in the introduction, I had been one of Gould's students who asked naively, which was better, time's arrow or time's cycle? So despite recognizing at one level the inappropriateness of the question, I was a student in his course frustrated by his response of "both and neither". Once I understood the wrongness of the question and held it as a talisman through my reading, I understood the beauty of his argument. He is convincing us that the idea of time as an arrow and as a cycle is deeply held in western Judeo-Christian thinking. His originally perplexing final chapter about James Hampton finally makes sense. Gould is describing how seeing a poor, black janitor's creation of an appropriate throne for Christ has incredibly managed to capture this metaphor so completely.

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*Seldin, 1991(Pace University)**

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Sr. Marion Johnson, Past President
Association of Midwestern College Biology Teachers

"By its very nature, teaching is evanescent. What does a faculty member have to show for all that effort at the end of a semester? A syllabus, a stack of corrected bluebooks, and perhaps some student evaluations. His or her lectures or seminars are rarely attended by colleagues or videotaped for posterity. The teacher is thus a bit like an actor whose performances theater critics never get to see. The theater program shows that he played Hamlet all last fall; but how do the critics know whether he was any good?"
*Wilkinson, 1991 (Harvard)**

* all quotes are from the OAH Council of Chairs Newsletter, June 1991.

BREAKING THROUGH TECHNOLOGICAL BARRIERS

AMCBT 1995 FALL MEETING

TENTATIVE SCHEDULE

Alverno College

Milwaukee, Wisconsin

SEPTEMBER 28-30, 1995

Thursday, September 28 — **LOCATION**

6:00-8:00 p.m. **REGISTRATION RECEPTION** — Nursing Building

8:00 p.m. **OPENING SESSION** — Nursing Building, Wehr Hall

Welcome for AMCBT
Donald "Buzz" Hoagland, Program Chair
Patricia Bowne, David Ferris, Leona Truchan, Local Arrangements

WELCOME to Alverno College
PRESIDENTIAL GREETING: Harold Wilkinson

OPENING ADDRESS:
Making Sense of New Medical Technologies
Suzanne Amador, Department of Physics, Haverford College, PA

9:00 p.m. **EXECUTIVE COMMITTEE MEETING**
(to immediately follow opening session)

6:00-8:00 p.m. & 9:00-12:00 midnight — **OPEN COMPUTER LAB**- software previews, electronic bulletin board, etc. — Nursing Building

Friday, September 29

7:00 a.m. **REGISTRATION** — Alumni Hall

7:00-8:10 a.m. **BUFFET BREAKFAST** — Alumni Hall
(price included in registration)
Interest Groups by Discipline

7:30-12:00 am **FIELD TRIPS I** ($5.00 each)
1. Wehr Nature Center/Mitchell Park (Birding)
2. Cedarburg Bog
3. Boerner Botanical Gardens/Mitchell Park

8:30-12:00 am **FIELD TRIPS II** ($5.00 each)
1. Museums/Historical Buildings Tour
2. Brewery Tour

12:00-1:00 p.m. **OPEN LUNCH**

1:00-2:00 p.m. **KEYNOTE ADDRESS** ALUMNI HALL
Title:
Speaker:

2:10-3:25 p.m. **CONCURRENT WORKSHOP SESSION I**
1. MACROMEDIA DIRECTOR AS AN AUTHORING TOOL
   Mark Bergland, University of Wisconsin, River Falls
2. GENBANK
3.
4.
5.

3:25-4:15 p.m. **COFFEE, POSTERS, EXHIBITS** Alumni Hall

4:15-5:00 p.m. **CONCURRENT PAPER SESSION I**
1. FRACTAL GEOMETRY IN BIOLOGY
   John R. Jungck, Beloit College, Beloit, WI
2. CONSTRUCTING ONE-PAGE TAXONOMIC KEYS
   Norman Waldow, Maryville University, St. Louis, MO
3. HISTORICAL LIFE SCIENCE TECHNOLOGIES IN THE CLASSROOM
   Philip Wilson, Northeast Missouri State University, Kirksville, MO
4. THE BIONIC TEACHER
5.

5:00-6:00 p.m. **BIOSCENE EDITORIAL BOARD MEETING**

6:00-7:00 p.m. **SOCIAL HOUR** Alumni Hall

7:00 p.m. **BANQUET** (price included in registration)
8:00 p.m. **BANQUET SPEAKER**
Title:
Speaker:

9:00-12:00 midnight **INTERNET WORKSHOP I** (World Wide Web; CDC, NMFS, THOMAS and other goverment Home Pages; AMCBT Home Page; etc.)
Tim Mulkey, Indiana State University, Terre Haute, IN; *Ethel Stanley,* Millikin University, Decatur, IL; *Karen Klyczek,* University of Wisconsin, River Falls, WI; *Buzz Hoagland,* Westfield State College, Westfield, MA

Saturday, September 30

7:00-8:15 a.m. **CONTINENTAL BREAKFEAST** Alumni Hall
Interest Groups by Discipline

8:30-10:30 a.m. **BALLOTING** Alumni Hall

8:30-9:15 a.m. **CONCURRENT PAPER SESSION II**

1. HOW THE USE OF MULTIMEDIA AFFECTS STUDENT RETENTION AND LEARNING IN AN INTRODUCTORY BIOLOGY COURSE
   Randy Moore, The University of Akron, Akron, OH
2.
3.
4.

8:30-10:10 a.m. **CONCURRENT WORKSHOP SESSION II**

1. INTERNET WORKSHOP II: CYBERSPACE REVISTED
   Tim Mulkey, Indiana State University, Terre Haute, IN; *Ethel Stanley,* Millikin University, Decatur, IL; *Karen Klyczek,* University of Wisconsin, River Falls, WI; *Buzz Hoagland,* Westfield State College, Westfield, MA
2. TEACHING HUMAN BIOLOGY: A WORKSHOP FORMAT
   Marion F. Fass and Marc M. Roy, Beloit College, Beloit, WI
3. AUTHORING PROGRAMS
   Dianne Bell, Avila College, Kansas City, MO
4.

9:25-10:10 a.m. **CONCURRENT PAPER SESSION III**

1.
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4.

10:10-10:30 a.m. **BREAK**

10:30-11:00 a.m. **CONCURRENT PAPER SESSION IV**

1.
2.
3.
4.

11:00-12:30 p.m. **LUNCHEON** (price included in registration fee)
BUSINESS MEETING

12:35-1:15 p.m. **EXECUTIVE COMMITTEE MEETING**
[N.B. Remember that newly elected officers must attend this very important planning meeting.]

1:30 p.m.-? **MILWAUKEE'S FINEST!** (Zoo, Art Center, Theater, etc.)
Why not join several of your colleagues in extending the meeting informally?

Why Come to Milwaukee September 28-30, 1995?

This year's AMCBT Meeting will be held at Alverno College, Milwaukee. Alverno is unique among U.S. colleges and universities because of their ability-based approach to education. This approach has gained them national recognition as a leader in making college education work. Alverno is minutes from downtown Milwaukee and only minutes from Mitchell International Airport. Pat Bowne, David Ferris, and Leona Truchan invite you to participate in a conference that promises to be as unique as Alverno. For example, opportunities to discover Milwaukee's natural and cultural history will not conflict with AMCBT presentations. Additionally, a greater number of hands-on workshops - two accessing the Internet - have been added to the program.

Registration Costs Are Anticipated To Be Under $50 (Field Trips Not Included)

What else might you discover? Did you know that early 20th century Milwaukee was a stronghold of Socialist thought and politics? Have you ever trekked into a forest clearing and felt the ground gently quake with each step? How would you like to watch a miniature train winding its way through 10,000 brightly colored blossoms, witness parallel evolution of euphorbs and cacti, and stroll among kapok, tamarind, and curate vines within a single superstructure? Where can you read The Water Street Journal while feasting upon charbroiled Usinger Bratwurst and Stutgarter Knackwurst along with a weizglas of Callan's English Red? The "gathering place by the waters" of course!

The Association of Midwestern College Biology Teachers (AMCBT) is dedicated to providing opportunities for you to share your teaching ideas in a public forum. At AMCBT's annual conference you can meet other Biologists who take their teaching seriously, and share ideas in formal presentations or workshops covering everything from laboratory problems to the latest ideas in teaching scholarship.

Application For Membership

ASSOCIATION OF MIDWESTERN COLLEGE BIOLOGY TEACHERS

NAME:_____ DATE:_____

TITLE:_____

DEPARTMENT:_____

INSTITUTION:_____

STREET ADDRESS:_____

CITY:_____STATE:_____ZIP CODE:_____

ADDRESS PREFERRED FOR MAILING:_____

CITY:_____STATE:_____ZIP CODE:_____

WORK PHONE:_____ FAX NUMBER:_____

HOME PHONE: _____ E-MAIL ADDRESS:_____

MAJOR INTERESTS:

( ) 1. Biology
( ) 2. Botany
( ) 3. Zoology
( ) 4. Microbiology
( ) 5. Pre-professional
( ) 6. Teacher Education
( ) 7. Other_____

SUB DISCIPLINES: (Mark as many as apply)

( ) A.	Ecology	( ) H.	Molecular
( ) B.	Evolution	( ) I.	Developmental
( ) C.	Physiology	( ) J.	Cellular
( ) D.	Anatomy	( ) K.	Genetics
( ) E.	History	( ) L.	Ethology
( ) F.	Philosophy	( ) M.	Neuroscience
( ) G.	Systematics	( ) N.	Other_____

RESOURCE AREAS:

_____

_____

_____

RESEARCH AREAS:

_____

_____

_____

How did you find out about AMCBT? _____

Have you been a member before?_____ If so, when?_____

PLEASE MAIL **MEMBERSHIP APPLICATION** FORM TO:

Edward S. Kos
Executive Secretary, AMCBT
AMCBT Central Office
Department of Biology
Rockhurst College
1100 Rockhurst Road
Kansas City, MO 64110-2561
Phone: 816-926-4049
FAX: 816-926-4666
Email: kos@vax1.rockhurst.edu

CURRENT DUES ARE **$25.00**