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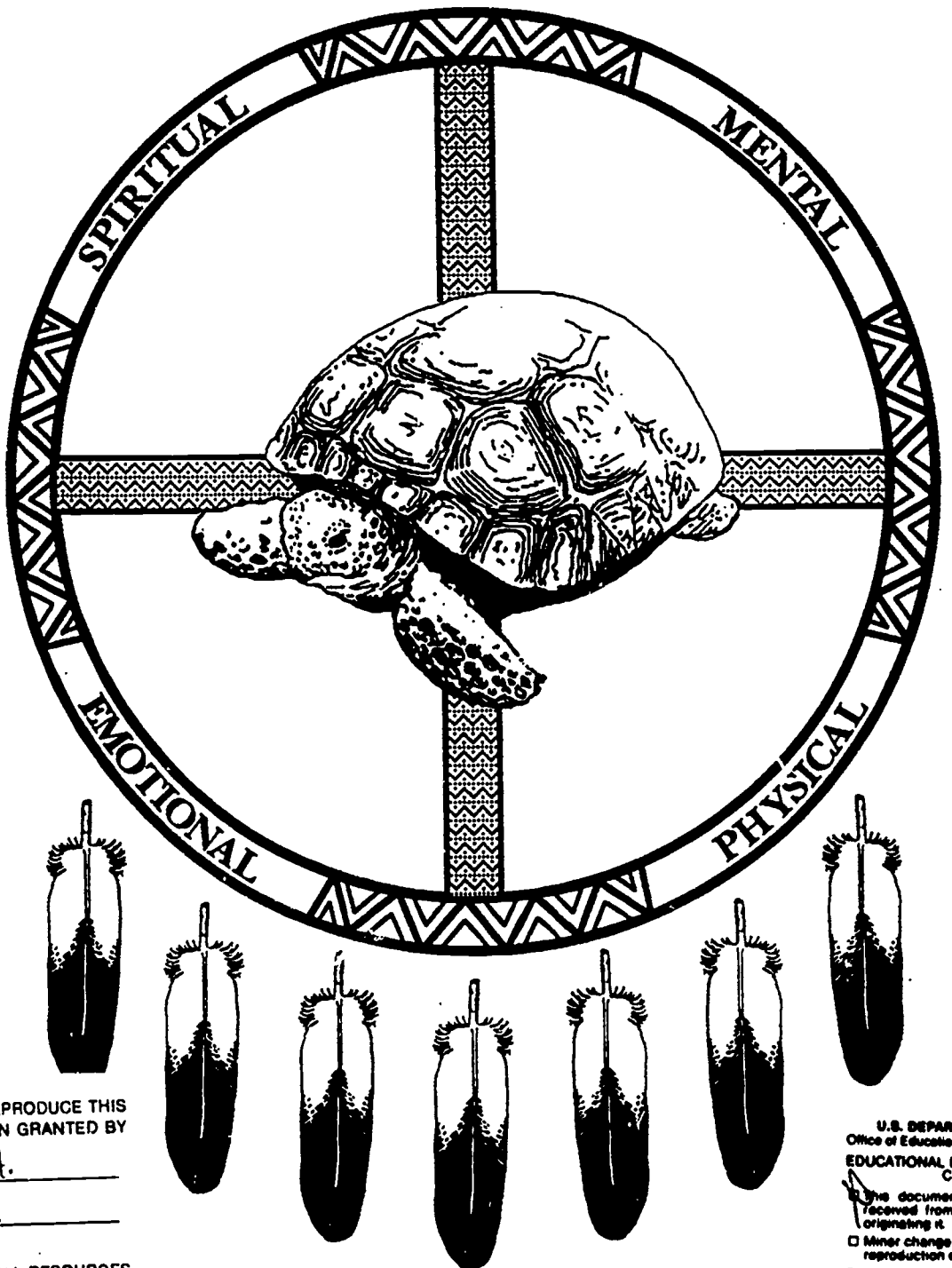
ABSTRACT

This genetics curriculum was conceived to fill the void in the general public's awareness of the importance of heredity and environment in health. The module contains a teacher's manual, student manual, and appendix. Topics are presented in a manner that is intended to be culturally sensitive and meaningful, and to reflect the concerns of the Native American population in the South Dakota region. In the context of traditional Native American beliefs and culture, students learn about the genetics of diabetes, heart disease and alcoholism, the causes of some birth defects, and how lifestyle choices have an impact on individual and community health. Seven lessons present a series of classroom discussions, hands-on experiences, group research projects, and interactions with individuals from the community. The module is designed to promote student participation in the learning process. Direct teaching methods are utilized only when necessary. The teacher's manual presents lessons with the following components: introduction, goal, objectives, materials and advance preparation, directions for conducting the activity, background information, resources, and optional activities. The student manual is teacher reproducible. An appendix contains material highlighting health care issues in the Native American community. (LZ)

ED 381 378

HEALTH IN THE YEAR 2100:
WHAT'S HEREDITY GOT TO DO WITH IT?



GENETICS CURRICULUM FOR HIGH SCHOOL BIOLOGY STUDENTS

About the Cover

The Medicine Wheel represents all of the directions and the meaning in life. It portrays the sacred relatedness of all that is in the universe. The seven Eagle feathers acknowledge the Seven Sacred Ceremonies. The Eagle is a messenger of and from the Winged people. The Turtle teaches by example.

The Turtle, from the Four Legged and the Water people, represents endurance, fortitude and solitude. These and other Turtle attributes are especially admired by Native peoples. The symbol of the Turtle encourages learners and reminds them to study genetics in a cultural context.

The Medicine Wheel appropriately represents the genetics curriculum, because the curriculum focuses on issues of health and on making healthy life choices. The Medicine Wheel symbolizes balance and harmony for each individual and for all of creation.

All of the symbols on the cover encourage learners to persist in their quest for knowledge and wisdom.

- Wayne H. Evans, EdD

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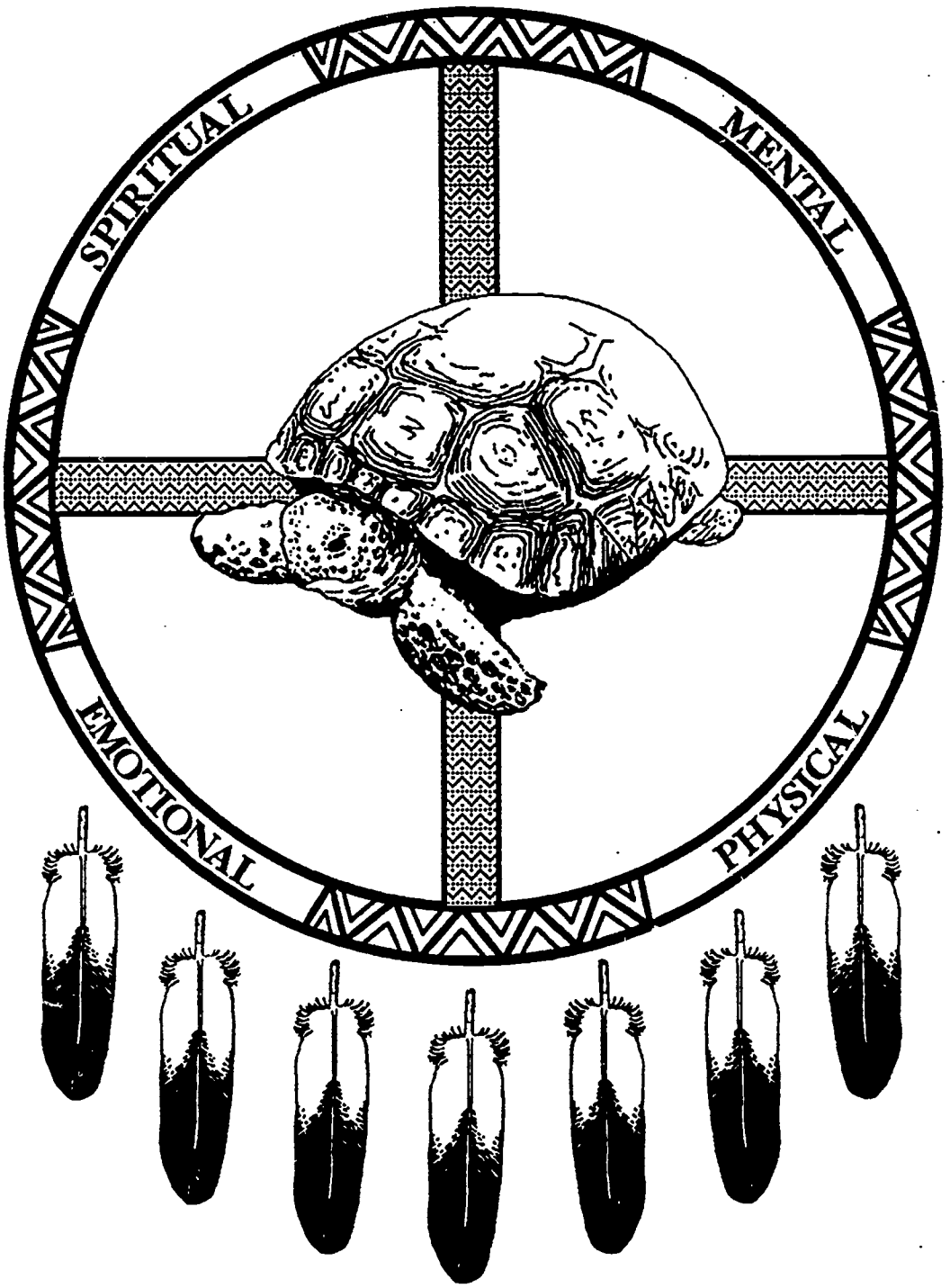

GENETICS CURRICULUM FOR HIGH SCHOOL BIOLOGY STUDENTS

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TABLE OF CONTENTS

FOREWORD

Health in the Year 2100 was conceived to fill the void in the general public's awareness of the importance of heredity and environment in health.

The effects of genetic disorders are ubiquitous: 36% of spontaneous abortions are due to chromosomal errors, 80% of mental retardation has a genetic component, 40% of infant mortality is caused by genetic factors, 33% of pediatric hospitalizations are due to genetic problems, 3-5% of newborns have genetic birth defects[1]. Among common diseases in adults, genetic factors play a substantial role in 30% of cancer, 40% of cardiovascular diseases, and 20% of neurodegenerative disorders[2]. This curriculum stresses the role of heredity in these common human ailments.

Environmental components leading to ill health--infectious disease, malnutrition, poor sanitation--have been brought under control. However, the last couple of decades have witnessed disturbing changes in the social and moral fabric of society. Nationwide, teenage pregnancies continue to increase, 80% of teen pregnancies are unintended, and more than 2/3 of those are to unwed mothers[3]. Added to these are lifestyles deleterious to general health or to fetal well-being. This curriculum stresses the role of environmental factors against the background of genetic susceptibility.

Finally, in an attempt to "make a difference," topics were chosen to reflect concerns of the Native American population. These topics are presented in a manner that is intended to be culturally sensitive and meaningful. This module was designed to meet the needs of high school students and adult learners. Hopefully, lifestyle choices made now will be beneficial to individual health by the year 2100.

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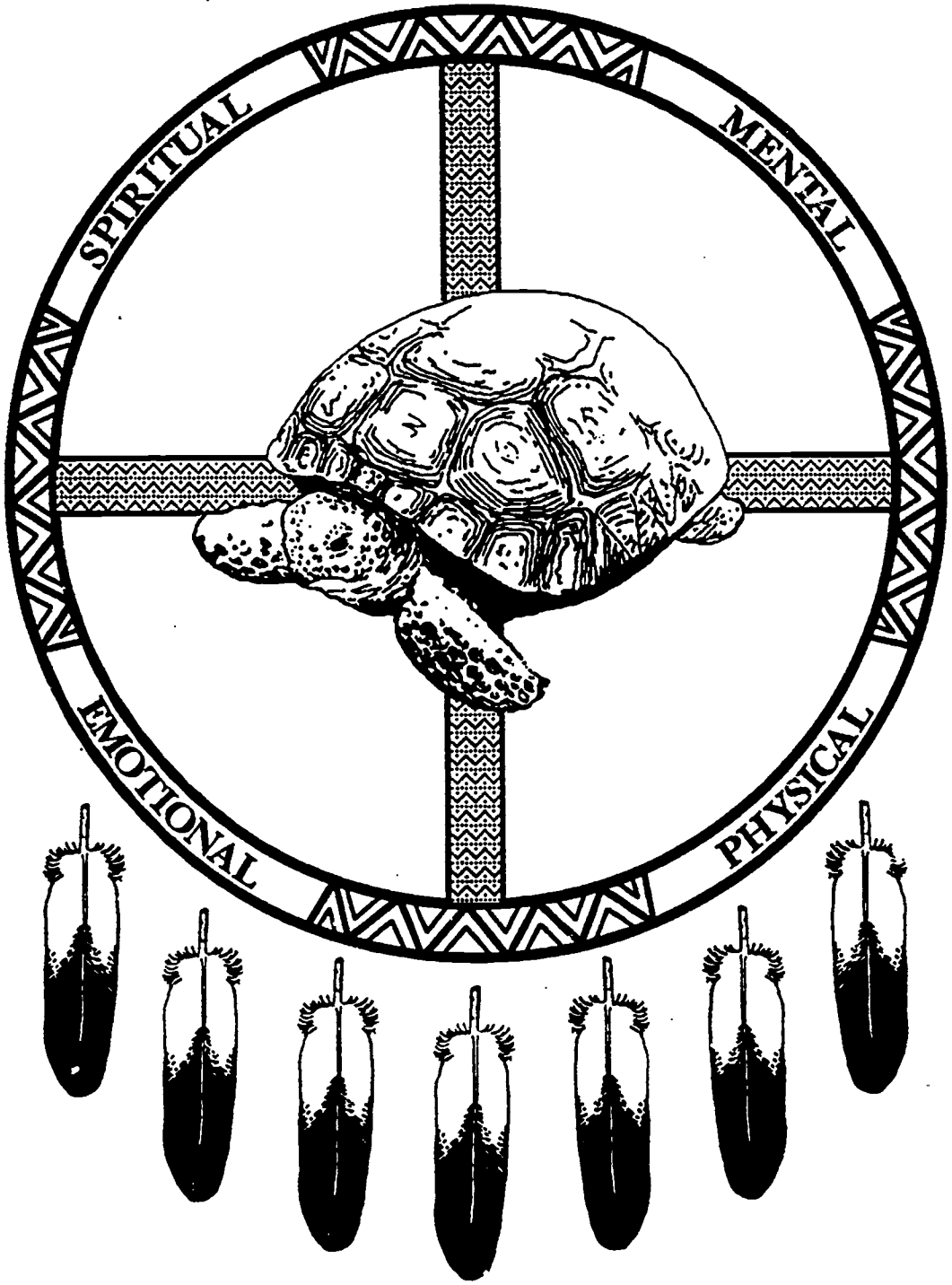

TEACHER'S MANUAL

TABLE OF CONTENTS

HEALTH IN THE YEAR 2100

INTRODUCTION

INTRODUCTION AND BACKGROUND RATIONALE

In the book *Teaching the Native American*, Clarence Wesly, Chairman of the San Carlos Apache Tribe, is quoted as saying of the majority society education: "The school curriculum is geared to a whole set of concepts and literary background too often totally unfamiliar to an Indian child"[1]. In response to this assertion, and to other comments made about existing curricula, the authors of *Teaching the Native American* encourage readers to develop instructional materials related to native students' experiences. They contend that native culture should be an integral part of basic instruction. Native heritage, values and contributions to thought and knowledge should be included in classroom discussions. Assignments should be related to students' backgrounds, and students should be encouraged to see how their course work relates to life in their communities.

The challenge put forth by the authors of *Teaching the Native American*, and the rapid advances occurring in the field of genetics, led to the development of this module. It is clear that the need to create culturally sensitive materials for school-aged children has never been greater. There is also increasing support for the development of educational materials that provide information about the role genetics plays in human health and development[2,3,4,5,6].

Health in the Year 2100 attempts to provide Native American biology students in South Dakota with information about heritable traits that may affect their health, the health of their children, and the health of their communities. The lessons, however, are appropriate for any student wanting to learn more about the role genetics plays in common diseases and birth defects.

MODULE DEVELOPMENT

To create a human genetics curriculum that accurately reflects the life experiences and cultures of the Native Americans in this region, teachers, superintendents, tribal health officials and community members were invited to attend one of four planning meetings conducted at various sites throughout South Dakota. Meeting participants were asked to consider what they thought students should know about the effects of heritable traits on their health, and the health of their communities.

Meeting participants defined the content of the curriculum, and their comments were used to create an outline. The proposed outline was then reviewed and revised by the planning meeting participants and a group of independent reviewers. The revised outline served as a guide for the writing team members.

The first version of the curriculum was field-tested in the fall of 1993. High school biology teachers in Lower Brule, Marty and Wanblee used this curriculum and critiqued each lesson. Comments were also solicited from each student who used these materials and independent reviewers with expertise in the areas of education and genetics. The comments made by the field-test participants and members of the review team were used to guide the revision

process. The writing team met for a second time in the spring of 1994 to draft the final version of the curriculum.

COURSE GOAL

In the context of traditional Native American beliefs and culture, students will learn about the genetics of diabetes, heart disease and alcoholism, the causes of some birth defects, and how lifestyle choices impact individual and community health.

OBJECTIVES

Through a series of classroom discussions, hands-on experiences and interaction with individuals from the community, students who use this module will:

1. Gain an appreciation of how the lifestyles of Native American people have changed in the past two hundred years, and the effects these changes have had on the health of their communities.

2. Learn that the risk of developing a particular health problem, such as diabetes, heart disease or alcoholism, may be increased because of lifestyle choices, the environment and genetic factors.

3. Learn the basic concepts of inheritance and normal fetal development.

4. Learn that some birth defects, like fetal alcohol syndrome, are preventable, and that the majority of birth defects are not preventable.

5. Learn about the kinship belief system that governed their ancestors' relationships and the importance of these guidelines in their communities today.

6. Learn about the role that traditional beliefs and customs played in the decisions made by their ancestors, and the role that these beliefs might still play in their lives.

ORGANIZATION OF THE MODULE

Health in the Year 2100 can be used in an introductory high school biology course or health class. No prerequisites are required to use this module. However, an understanding of basic genetic concepts and normal human development is helpful. Experience in classroom discussion and library research skills are also helpful, given that student participation in the learning process is emphasized.

This module presents some basic genetic concepts and provides students with opportunities to personalize this information. A conscious decision was made to exclude a large number of technical terms. Only those terms necessary to convey basic genetic concepts are included. This text is designed to provide students with information that will be of value to them throughout their lives. For those students wishing to learn more about molecular genetics additional course work is recommended.

INTRODUCTION

This module is divided into three sections: the "Teacher's Manual," the "Student Manual" and the "Appendix." The curriculum includes seven lessons that can be taught separately or as a three week unit.

LESSON SUMMARIES

Lesson 1 In this lesson, life as it was for the Plains Indians 200 years ago is compared to life in the 1990's. This lesson encourages students to look at both the similarities and differences between their lives and the lives of their ancestors. Specifically, students will focus on the differences in lifestyles, foods and health.

Students will create a class vision for health in the year 2100. Throughout the remainder of this unit you will challenge students to think of creative ways to make their vision become a reality.

Lesson 2 In Lesson 2, students will explore the role that genetics and lifestyle choices play in the development of heart disease, diabetes and alcoholism. After selecting one of these diseases to study, students will research the disease and present their findings to the class. Information will also be provided by a member of the community who has personal and/or professional experience with the condition.

Lesson 3 The concepts of genotype and phenotype are presented in this lesson. Students will also learn about multifactorial inheritance and the influence of environmental factors and lifestyle choices on gene expression.

Lesson 4 Students will cut out and compare the chromosomes present in the egg to the chromosomes present in the sperm. They will organize the chromosomes by shape, size and banding patterns to create a karyotype. This lesson reinforces the idea that each parent contributes a total of 23 chromosomes to each child, and that a child's sex is determined by his or her father's genetic contribution.

Lesson 5 The video *Fetal Development: A Nine Month Journey* presents the stages of normal fetal development. Following this introduction, each student group will research a specific birth defect and make a brief presentation to the class. The "Teacher's Manual" includes information about fetal alcohol syndrome along with a set of questions to help stimulate classroom discussion.

Lesson 6 An elder or a native scholar will present information about the traditional rules governing relationships and the selection of marriage partners. Students will consider why people chose to abide by these rules, keeping in mind the traditional community structure and what they have learned about recessive single gene disorders.

Lesson 7 An elder will share information about the traditional beliefs, customs and values that influenced the decisions made by their ancestors. Students will then consider their own decision-making strategies, and the long-term effects of their life choices on their health and the health of their children. In conclusion,

students will identify those things they can do to make sure their vision of health in the year 2100 becomes a reality.

TEACHER'S MANUAL

In the "Teacher's Manual," each lesson is organized in the following manner:

INTRODUCTION: Major ideas covered in the lesson.

GOAL: A statement of what the students will learn.

OBJECTIVES: A list of concepts the students should master in order to achieve the lesson goal.

MATERIALS AND ADVANCE PREPARATION: An outline of the activities that must be completed prior to beginning a lesson.

DIRECTIONS FOR CONDUCTING THE ACTIVITY: Strategies to facilitate discussions, organize small group activities, and coordinate student experiments.

BACKGROUND INFORMATION: Information that should be mastered prior to beginning each lesson.

RESOURCES: Reference materials that will provide additional information on the lesson topics.

OPTIONAL ACTIVITY: Instructions for additional activities that reinforce the ideas presented in the lesson.

STUDENT MANUAL

The "Student Manual" is located on pages immediately following the "Teacher's Manual." The specified pages should be photocopied and distributed to your students with each lesson.

APPENDIX

Material highlighting health care issues in the Native American community is included in the "Appendix."

ADVANCED PREPARATION

Preparation is required to use this module. In many schools, it is necessary to consult with both the building and district administrators prior to implementing a new curriculum. You may wish to check with your building administrator if you are unfamiliar with the policies at your school.

Prior to beginning this unit, provide each student with a copy of the handouts, "Health in the Year 2100" and "Health 200 Years Ago," found on pages S-1 and S-2 of the "Student Manual." Instruct your students to talk to the elder members of their families, or elders in the community, about the health of their ancestors.

INTRODUCTION

You will need to collect materials and order a videotape prior to beginning this module. In Lesson 1, for instance, you will need to collect information about the most prevalent health concerns in your community. You will also need to identify health care providers, and people knowledgeable about the traditional ways of healing who are willing to visit with your students. In Lesson 2, students will need to have access to materials on diabetes, heart disease, and alcoholism. If this material is not available in your school library, begin collecting resource materials to have on hand in your classroom. A set of pop beads and two containers per student group must be on hand for Lesson 3. In Lesson 4, each student must have a pair of scissors and scotch tape. You should order the videotape, *Fetal Development: A Nine Month Journey*, and collect materials on the more common chromosome abnormalities, single and multiple gene defects, and teratogens prior to beginning Lesson 5. Copies of the videotape are available through the South Dakota State Library System. Invitations should also be extended to the people who will be speaking to your class during Lessons 2, 6 and 7.

TEACHING STRATEGIES

This module is designed to be taught using both direct and indirect teaching methods. Direct teaching methods include giving facts and information about the lesson content and procedures. You will have to use direct teaching methods to introduce the module and to assist students with small group discussions and projects. If you choose to invite traditional people to speak to your class they may also use more direct teaching methods.

If you select an elder from the community to speak to your class, instruct your students to sit in a circle and listen to the speaker without interruption. When the speaker has finished speaking, the students should get up, shake the hand of the speaker and thank him for coming. Only if the speaker gives permission should the students be encouraged to ask questions.

Given that this module is designed to promote student participation in the learning process, direct teaching methods should only be used when absolutely necessary. Except in the few instances cited above, this module should be taught using indirect teaching methods. Through a series of open-ended questions, classroom discussions and group research projects, encourage your students to "discover" basic genetic principles, explore the causes of birth defects, and consider their lifestyle choices in the context of traditional belief systems.

CLASSROOM DISCUSSIONS

Many lessons involve class or small group discussions. If this format is unfamiliar to your students, spend time during the initial class period creating group norms for discussion. Consider having students generate a list of behaviors they think will ensure that people feel comfortable sharing their thoughts, feelings, and ideas. This list could include such things as respecting each person's right to express an opinion; not making fun of a person's comments when they are presented in a serious manner; encouraging each student to participate in the discussion; and not sharing comments made by class members with people outside the classroom. When the list is complete, post it in the classroom and refer to it throughout the remainder of this unit.

INTRODUCTION

Consider the physical circumstances of the classroom prior to beginning class discussions. Arrange students so that they are comfortable interacting with one another. Also, consider your position in the classroom. To foster the idea that this is a student-directed learning experience, consider sitting with your students.

If group discussion is not the norm in your classroom, begin by asking open-ended questions. Allow students time to formulate their responses, and use questions to redirect the discussion if it is getting too far from the specified topic.

When facilitating group discussions, it is important to model behaviors that encourage uncensored dialogue and class participation. Praise and encouragement are essential. Nodding your head, or saying "um hm" or "go on," are ways to encourage student participation. When offering praise, only praise the student's actions or behavior. When students are talking about their feelings or beliefs, it is essential that you remain neutral. If the class perceives that you support one argument over another, this may seriously affect the open exchange of ideas and feelings.

Acceptance of students' feelings and ideas is another important behavior to model. Remember, that feelings may be difficult to articulate, and some students will need help putting their feelings into words. Help students clarify their feelings when appropriate, or ask another student to build on the ideas and feelings that have been expressed. Asking other students to build on an idea is a great way to affirm the person who first suggested the idea, and to encourage continued class participation.

When facilitating classroom discussions, you should not provide students with information they can obtain from resources available in your room, or from other students. It is important not to allow a few people to dominate the discussion. You should also guard against rejecting comments presented in a serious manner, or responding in a negative way to ideas that go against your own beliefs.

IDENTIFYING RESOURCES

Many of the lessons in this module are designed to build on your students' own interests and curiosity. Broad topics are presented and students are asked what they would like to learn about these topics. They are then instructed to find the answers to their questions.

To be successful in their search for information, students will need to have access to a collection of resources. Prior to starting this unit, you will need to evaluate the resources available in your classroom, your school and within your community. If your school has access to Internet or other on-line computer databases, you may also want to familiarize yourself with the resources available on these systems. On Internet, for instance, there is a copy of the text *Mendelian Inheritance in Man* which provides up-to-date information about thousands of known single gene disorders.

You will also need to determine what your students know about identifying and using the resources in their community. If your students have never had to research health-related

questions, ask them to generate a list of places they would go to find the desired information. An initial list might include a classroom text, the library, or possibly the counseling office at school. If your students have not listed community resources, national organizations such as the American Heart Association, or the on-line computer services available at your school, make sure they know about these resources and how to use them before you begin Lesson 2.

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HEALTH IN THE YEAR 2100

LESSON 1

PAST, PRESENT AND FUTURE

INTRODUCTION

In Lesson 1, students will explore what they know about the role heredity plays in their lives. They will also be involved in determining the direction of future lessons. Through a series of open-ended questions, you will find out what your students know about genetics, and the relationship between genes, the environment, lifestyle choices and health. You will identify areas of confusion and misconceptions. You will also determine which topics your students are most interested in studying. This information is essential, as you will build on your students' interests in future lessons.

GOAL

To involve students in discussions that explore what they know about the relationship between genetics, the environment, lifestyle choices and health.

OBJECTIVES

Students who participate in this lesson will:

1. Identify some of the similarities and differences between themselves and their ancestors, such as differences in lifestyles, traditions, and health;
2. Identify some of the health problems in their community;
3. Discuss what they know about the impact of genetics on health;
4. Create a vision statement which defines the changes in health they hope will occur by the year 2100.

MATERIALS AND ADVANCE PREPARATION

Prepare an introductory packet for each student to take home prior to beginning Lesson 1. This packet should include the handouts, "Health in the Year 2100," and "Health 200 Years Ago." These handouts are found on pages S-1 and S-2 of the "Student Manual." Instruct your students to read the introductory materials, and encourage them to talk to people in the community about what life was like for Native Americans in this area 200 years ago.

If you are not familiar with the history of the indigenous people who live in your region, refer to one of the texts listed in the "Resources" section on page 1-5, or talk with an elder in your community. Familiarize yourself with the lifestyles, social structure, and health concerns of Native Americans who lived in the plains area 200 years ago.

LESSON 1

Read the material included in the "Appendix," and obtain community health statistics, if possible. Find out if there is a person who practices Native American medicine and ceremonies in your community, and familiarize yourself with the work he or she does. Determine whether or not this person is willing to talk with your students, and include this information in your files. If you are unable to identify a person who has expertise in Native American medicine, visit with the members of the community who work at your school or your building principal.

Familiarize yourself with the services available at the hospital or clinics in your area. Specifically, gather information about preventive health care programs and prenatal health care services. Collect brochures and handouts that describe the available services. To facilitate the interaction between the health care providers in your community and your students, find an individual who is willing to respond to questions your students might have about local health care services, and include this information in your class files.

DIRECTIONS FOR CONDUCTING THE ACTIVITY

DAY ONE

Begin with a brief introduction to the module. Explain to your students that they will be spending the next three weeks learning about factors that can influence health. To help focus their discussion, explain that a person's health is determined by the interaction between the environment, inherited traits, and lifestyle choices.

When it is clear that your students understand the goals of the module, establish group norms for discussion as described in the "Introduction" on page 0-5. Then, ask students to share what they know about the health and lifestyles of the native people who lived in the region 200 years ago. If they are reluctant to talk, refer to the handout, "Health 200 Years Ago," and ask students to share their answers to these questions.

To expand on this discussion, inquire about the types of dwellings that were commonly used 200 years ago. Have your students talk about the day to day activities of their ancestors. Were their ancestors active people? What was the common mode of transportation? What did their ancestors eat? Were there periods of time when food was scarce? What were the common causes of death? What type of medical care was available? What types of drugs or medications were available?

Ask your students to compare this information with their own experiences. What types of dwellings do your students live in? How do they travel from one location to another? What do they eat? What are the common diseases in their community? What types of medical care are available to them, and what types of care do they choose? Do they use traditional Native American healing practices? Do they receive hospital or clinical services?

When comparing life 200 years ago to life today your students will probably point out the differences between daily activities, the dwellings, modes of transportation and food. If they do not mention the difference in the types of health problems or causes of death, encourage

your students to explore these two areas. They should begin to appreciate that prior to the discovery of antibiotics and vaccinations, more people died from infectious diseases. Today, chronic conditions such as heart disease and diabetes claim more lives each year.

Ask your students to consider the causes of some of the chronic diseases they have listed. Are there environmental factors that influence who develops a particular disease? What about lifestyle choices; does a person's lifestyle affect his or her health? Are some diseases more likely to occur in members of the same family? If your students answer "yes," ask them why they think this might occur.

During this lesson, your students should be encouraged to talk about aspects of health that interest them. This discussion is intended to help you learn what your students know about genetics, health, and the past, and what they anticipate will happen in the future.

The exercise must be student-centered. It will require active listening on your part. You should pay close attention to those topics that are of interest to your students and the types of questions and misconceptions that they have. Do not try to answer their questions or correct misconceptions during this discussion. Simply keep a list of questions and misconceptions and be sure to address each later in this module.

ASSIGNMENT

Ask each student to write a paragraph about what he or she thinks the health concerns will be in their community in the year 2100 if nothing changes. Then have each student write a second paragraph outlining what his or her vision is for health in the year 2100.

It is possible that your students will paint a very bleak picture of health in the year 2100 if no changes occur. Their vision statements, however, should be more positive. They may envision a world where no one is born with birth defects, or suffers from chronic illnesses. Their vision statements may also include a cure for AIDS, the absence of alcoholism, and "Star Trek"-like medical technology.

If your students have not had experience creating vision statements, have them do some brainstorming before the end of the first class period. Begin by asking them to consider some of the following questions: Will there will be disease in the year 2100? Will people develop chronic health problems? What will be the leading cause of death? If people do not die, how will the planet accommodate everyone? What type of medical care will be available? Will people even need medical care? Will babies be born with birth defects? If babies are born with birth defects, will it be possible to correct these problems? Will it be possible to correct learning problems or mental retardation?

Encourage your students to be creative and expand on the ideas that have been presented. Use your discretion if you find that your students need a lot of coaching. Remember, too much guidance may stifle their creativity.

LESSON 1

DAY TWO

On Day Two, your students will work to create a class vision for health in the year 2100. If there are students in your class who would like to lead this discussion, encourage them in their efforts to develop consensus among their classmates.

A strategy for creating a class vision is outlined below. You, or your student leaders, may want to use another strategy to reach the stated goal.

Begin the process of creating a class vision by asking students to share their vision for health in the year 2100. If they are reluctant to speak, ask a few students to read their vision statements. Then, have students identify a few of the common themes. For example: Do most students comment on the presence or absence of disease? Do they talk about the availability of health care? Do they mention the health of children, or birth defects? Do they make a distinction between the health of Native Americans living on the reservations and those living in urban areas?

Once your students have generated a list of common themes, ask them to list their specific visions. For example, a number of students may have included comments in their personal vision statements about the health of children in the year 2100. Some students may envision a world where children are not born with birth defects. Other students may envision that medical technology will exist to correct all birth defects and health problems.

If your students' visions are similar, they should be summarized and included in the class vision statement. If their visions are not similar, have each student share why his or her vision should be incorporated into the class vision. Then, have the class reach a consensus on what visions to include before moving on to the next theme.

Repeat this process until your class has created a vision for health in the year 2100 that all of your students can support. Have one of your students write this vision on a transparency, or a piece of newsprint, and post it in your room each class period.

At the end of each of the following lessons refer back to the class vision. Have your students consider what they can do, in light of what they have just learned, to make their vision a reality.

LESSON 1

RESOURCES

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Rhoades ER, Hammond J, Welty TK, Handler AO, Amler RW, The Indian burden of illness and future health interventions. Journal of the US Public Health Service 1987;102(4):361-368.

Welty TK. Health of the Oglala Sioux People: a turning point. Report prepared for the Oglala Lakota Sioux Tribe by the Aberdeen Area IHS, Epidemiology Program, Rapid City, South Dakota; November 22, 1992.

HEALTH IN THE YEAR 2100

OPTIONAL ACTIVITY

FOOD AND YOU

INTRODUCTION

This activity is designed to be used in conjunction with Lessons 1 and 7. It requires that students examine the food choices they make, and challenges them to think about how these choices may affect their health in the future.

GOAL

Students will know where to find nutrition information and how to plan a healthy diet.

OBJECTIVES

At the conclusion of this activity students will:

1. Be able to locate and read the "Nutrition Facts" label on any food item they wish to purchase;

2. Know their personal nutritional needs;

3. Be familiar with the "Food Guide Pyramid" and how to use the pyramid to plan meals for a day.

MATERIALS AND ADVANCE PREPARATION

Make one copy of the "Food and You" handout, and four copies of the "Grocery Store Worksheet" for each student in your class. These handouts are located on pages S-3 and S-4 in the "Student Manual."

Each student will need a copy of the brochures "Are You Ready for New Food Labels?," "Daily Values and You," and the "Food Guide Pyramid." The day you introduce this activity, you will also need to bring in four or five food items that have a "Nutrition Facts" label. Make sure that at least one of the food items you choose has a claim on the label such as "fat free," "sodium free," or "lite." You will also need at least one food item with a health claim, such as "high in calcium" or "low in sodium," on the label.

DIRECTIONS FOR CONDUCTING THE ACTIVITY

This activity is designed to complement the classroom discussions in Lesson 1 and Lesson 7. In Lesson 1 your students will have the opportunity to explore how their diet differs from the diet of their ancestors. If you choose to use this activity, you will want to expand this discussion.

OPTIONAL ACTIVITY

Begin by asking your students what they know about the foods they eat. Do they know how many calories they consume each day? Do they know what percent of their calories come from fat? Do they know how much sugar and salt is in the food they eat? Do they know how much cholesterol a person should eat each day? Do they know where to find this information?

If none of your students mention that the above information is available on food labels, distribute the food items that you brought in and ask your students to locate this information. When each student has had an opportunity to look at the "Nutrition Facts" label on at least one of the food items, spend a few minutes going over the various components of the "Nutrition Facts" label. Make sure that your students understand the significance of the "% Daily Value" before proceeding.

Next, ask your students if they noticed any other nutrition-related information on the labels. Those students who examined the food item you brought in with a label claim, such as "fat free," should respond affirmatively. Then ask your students to list other label claims they have seen and record these claims on the board. Distribute copies of the brochure, "Are You Ready for New Food Labels?" and ask your students to compare their list with the list of possible label claims. Then, ask your students if they noticed any health claims on the labels they examined, and repeat this process.

When you are confident that your students understand how to locate and read label claims and the "Nutrition Facts" panel, ask them where they can obtain information about food items that have no labels such as fresh fruits and vegetables. If they do not know the answer to this question, ask them to generate a list of people they think might have nutrition information about fruits and vegetables. This list may include a local nutritionist or registered dietitian, the school cook, a restaurant owner, health professionals in the community, or the organizations listed on the brochure "Are You Ready for New Food Labels?"

If your students do not mention the personnel at the local grocery store as a potential resource, mention the voluntary point-of-purchase nutritional information program. In this program, retailers are asked to provide consumers with information about the 20 most frequently eaten raw foods and the 45 major cuts of meats and poultry.

At the conclusion of this discussion, give each student a copy of the "Food and You" handout and four copies of the "Grocery Store Worksheet." Instruct them to complete these forms and have them available for discussion during Lesson 7.

At the conclusion of Lesson 7 students are encouraged to discuss how their life choices will affect themselves, their families and their vision of health in the year 2100. As part of this discussion, each student who does the optional activity will determine his or her own nutritional needs and create a meal plan for one day using the "Food Guide Pyramid." Instructions for completing this activity are found in the "Directions for Conducting the Activity" section of Lesson 7 on page 7-3.

OPTIONAL ACTIVITY

BACKGROUND INFORMATION

Nutrition labeling was first introduced in the 1970's. Since then, interest in changing the labeling laws has grown. In 1993 the collective work of government agencies, Congress, the food industry and other groups resulted in extensive changes in food labeling laws and the development of a new food label.

Proponents of food labeling changes wanted labels to reflect the emerging scientific research about the overall effect of diet on a person's health, and the role diet may play in reducing the risk for certain diseases. To achieve this goal, the new labels include a list of key nutrients and information to help consumers compare foods and make informed food choices.

The serving sizes on the new labels are now more comparable for similar food products. Percent daily values are calculated for a 2000 calorie diet, and can be used by consumers to quickly compare and choose products that will meet their specific dietary needs.

Under the new law, label claims such as "free," "low," "reduced," and "light" have uniform definitions. Labels can also contain health claims that describe the relationship between a food or food component and a disease or health-related condition. To make a health claim, however, a food must meet certain nutrient requirements.

The new food labeling laws went into effect on May 8, 1994. Some foods, however, are exempt from the nutrition labeling rules. Exempt foods include things like coffee and spices that have no nutritional significance, restaurant foods, foods prepared on-site in a grocery store or hospital kitchen, foods produced by small businesses, and medical foods. Foods sold in very small packages are not required to have a "Nutrition Facts" label; however, the FDA does require that a telephone number or address be placed on small products so that people can request nutrition information.

Under the new guidelines, nutrition labeling is mandatory for most processed meat and poultry foods. Single-ingredient, raw meat and poultry products, such as ground beef, chicken breast, and whole turkey, are subject to a voluntary nutrition labeling program.

Raw fruits, vegetables and fish are also subject to a voluntary nutrition labeling program. The law states that by May of 1993, at least 60 percent of all supermarkets must voluntarily label at least 90 percent of these products, or make nutrition information available at the point of purchase. If not enough stores participate in this voluntary program, labeling these foods will become mandatory.

OPTIONAL ACTIVITY

RESOURCES

If you would like more information about the Nutrition Labeling and Education Act, or other educational materials, contact the Food and Drug Administration, or the State Nutritionist.

Food and Drug Administration
Office of Public Affairs
5600 Fishers Lane, HFE-88
Rockerville, MD 20857
301-443-3220

SD State Nutritionist
SD Department of Health
Health and Medical Services
300 S. Courtland, Suite 109
Chamberlain, SD 57325
605-734-5486

HEALTH IN THE YEAR 2100

LESSON 2

GENETICS AND CHRONIC DISEASE: IS THERE A CONNECTION?

INTRODUCTION

This lesson focuses on three serious diseases that are especially common among members of the regional native communities: diabetes mellitus (or sugar diabetes), heart disease and alcoholism. Because there is not sufficient time to study all three diseases during class, students will select only one disease to study in detail. They will generate a list of things they want to learn about this condition and then search for answers to their questions. Some questions your students may choose to research include: How common is the disease among Native Americans? What are the signs and symptoms of the disease? How is the disease inherited?

Perhaps the most important message to convey is that not everyone who is genetically predisposed to a particular disease will develop that disease. By making thoughtful lifestyle choices, individuals can reduce their risk of developing many chronic, long-term diseases.

This lesson will require at least three days to complete. On Day One, introduce the topic and have students select one of the three diseases to study in detail. Divide the class into small groups and have each group research a specific topic of interest to them. When their research is complete, each group of students will present their results to the entire class. This lesson will end with a presentation by an invited speaker who will talk to your class about a disease they have studied and respond to any questions your students might have.

GOAL

Students will learn that genes, the environment and lifestyle choices interact to determine who will develop diabetes, heart disease or alcoholism. They will also learn that if an individual alters his or her environment or lifestyle, he or she can increase or decrease the risk of disease.

OBJECTIVES

By the end of this lesson, students will be able to:

1. Research literature dealing with medical topics of general interest, such as causes, symptoms, and treatment of diseases;

2. Describe the following aspects of the disease they studied (either diabetes mellitus, heart disease or alcoholism):

   a. The significance of the disease to Native American communities;

   b. The general features of the disease;

   c. The signs and symptoms of the disease;

d. The genetic and non-genetic factors that influence who will develop the disease, and;

e. How people who are predisposed to the disease can lower their risk of becoming ill through lifestyle choices.

MATERIALS AND ADVANCE PREPARATION

Prepare a file on each of the three diseases for class use. The files should contain information about the causes, symptoms and treatment of each disease. This information could include photocopied material from textbooks and other library resources, brochures and pamphlets from national associations, newspaper and magazine clippings, etc. If it is feasible, students should also have access to videotapes that discuss these diseases. Refer to the "Resources" section at the end of this lesson for information about the available resources.

Make enough photocopies of the handouts, "Diabetes Mellitus," "Heart Disease," and "Alcoholism" for each student in your class. These handouts are on pages S-5 through S-16 of the "Student Manual." Even though your students will only research one disease in class, the authors of this text recommend that you distribute all three handouts, and encourage your students to share them with their families.

Contact a person who is knowledgeable about one of the diseases your class chooses to study and arrange to have him or her speak to your class on Day Four. This person should be able to talk about the common signs of the disease, treatment, and the effects of the disease on the individual, the family and the community. Possible speakers include a community member who has the disease, a school nurse or counselor, Community Health Representatives, the Tribal Health Officer, or medical personnel from a local clinic or hospital.

DIRECTIONS FOR CONDUCTING THE ACTIVITY

DAY ONE

1. Divide the students into small groups and ask each group to write down five common diseases that they are familiar with, or that someone they know has.

2. Using an overhead projector or chalkboard, begin listing the diseases that each group of students has mentioned. As you write down each disease, mention whether the disease tends to run in families (and therefore has a genetic component), or if it is an infectious disease. Most diseases the students will mention should fall into one of these two categories.

   Examples of infectious diseases familiar to your students may include:

   a. Respiratory infections (colds, flu, tuberculosis, pneumonia, whooping cough, etc.);

   b. Digestive tract infections (diarrheal diseases, food poisoning, parasitic infections, etc.);

c. Sexually transmitted diseases (AIDS, gonorrhea, syphilis, etc.).

3. As you go through the lists your students have generated, draw your students' attention to diabetes, heart disease and alcoholism. These are diseases that possess a genetic component, and are prevalent in Native American communities.

4. Ask your students to decide among themselves which one of these three diseases (diabetes, heart disease or alcoholism) they are most interested in studying. You may have to reorganize your student groups at this time based on each student's interest. After each student group has selected one disease to study, ask each group to make a list of all the things it wants to learn about the disease.

5. Student groups should categorize their list of questions by major themes, such as: Who gets the disease? Does the disease run in certain families? How can you avoid getting the disease? What are the symptoms of the disease? What are the treatments?

6. Each student should then select a question to research that is of interest to him or her. This research will occupy the students until the end of the first period and will comprise their homework assignment. Be sure your students understand that they will present their findings to the rest of the class during the next class period.

7. As soon as possible after class, confirm with the community expert that he or she will talk with your students in two days.

DAY TWO

Some students may need a second day to research their question. If this is the case, make arrangements for class presentations on Day Three and for your quest speaker to appear on Day Four. Some classes may be ready to move into Day Three activities on Day Two of this lesson. Use your discretion.

DAY THREE

1. Each group of students will present its findings to the class. Allow some time for questions after each presentation. If a student group fails to mention a point you think is important, ask those students what their thoughts are on that particular topic. If the group members are unable to respond, ask the other students in your class. If no one knows the answer to the question, have the group research the question and share the answer with the class on Day Four.

2. Take notes identifying specific questions, misunderstandings or misconceptions about the disease, and share this information with your guest speaker. He or she needs to be aware of your students' interests and level of understanding.

LESSON 2

DAY FOUR:

1. On Day Four your guest speaker will share what he or she knows about the signs, symptoms, causes and treatment of one of the diseases your students chose to study. Allow him or her to determine the format, and set the tone and pace of the presentation.

2. After the guest presentation, be sure to pass out the student handouts on all three diseases. It is important for students and their families to have access to printed information about each of these diseases, regardless of which one the students chose to study in class.

BACKGROUND INFORMATION

This lesson addresses a complicated question: Is a person's health determined by the genes he or she inherits? There is no single answer to this question. For example, researchers have identified single genes that cause blood-clotting diseases, certain types of muscular dystrophy and inherited forms of high blood cholesterol, called "familial hyperlipoproteinemia." The causes of many other diseases, however, are extremely complex and often involve the interaction between genes, the environment and lifestyles.

This module focuses on three diseases which are more common among the native people of this region than among the United States population as a whole. They are diabetes mellitus, heart disease, and alcoholism. Most research suggests that susceptibility to these diseases is determined by both genetic and non-genetic factors.

Information about each of these diseases follows. You will notice that some of this text is included in the student handouts.

DIABETES MELLITUS

WHAT IS DIABETES?

The term "diabetes mellitus," or "sugar diabetes," refers to diseases in which the body cannot process glucose (a sugar) properly. The word "diabetes" comes from the Greek word meaning "to pass through." This word is used because people with diabetes urinate more often than do non-diabetics. "Mellitus" comes from the Latin word for "honey," and is used because the urine of diabetic patients contains sugar. Specialists recognize several categories of diabetes, but two of the most familiar forms are called Type I and Type II diabetes mellitus.

A. TYPE I DIABETES MELLITUS

Type I diabetes mellitus usually occurs in children, teenagers or young adults, and is sometimes referred to as juvenile-onset diabetes. The pancreas of an affected individual does not produce insulin, the hormone necessary for proper sugar (glucose) processing by muscles, liver and other tissue cells. As a result, people with Type I, or juvenile-onset diabetes, must take insulin for the remainder of their lives. For this reason, some doctors refer to this form of the disease as insulin-dependent diabetes mellitus (IDDM).

Approximately 10 to 15% of all Americans with diabetes have Type I diabetes. The rate is much lower among Native Americans. Less than 2% of American Indians in the Aberdeen area with diabetes have Type I diabetes[1].

B. TYPE II DIABETES MELLITUS

Type II diabetes mellitus usually affects people later in life, and is sometimes called maturity-onset, or adult-onset diabetes mellitus. Most people with Type II diabetes are overweight and physically inactive when they are first diagnosed with this disease. Many doctors, therefore, treat this form of diabetes by prescribing low-calorie diets and vigorous daily exercise, not insulin injections. Consequently, some people refer to Type II diabetes as non-insulin-dependent diabetes mellitus (NIDDM). This can be confusing, however, because some people with Type II diabetes also take insulin in addition to their dietary and exercise therapies. In the Aberdeen Area, 98 to 99% of Native Americans with diabetes have the Type II form of the disease[1].

The pancreas of a person with Type II diabetes usually produces insulin, but the insulin is ineffective, either because the pancreas produces too little of it, or because the body's cells have become resistant to the insulin. Being overweight is a major factor in increasing the cells' resistance to insulin.

There is no "typical" Type II diabetic. Many people with Type II diabetes produce normal levels of insulin, while others show decreased levels, and some even exhibit increased insulin levels. The most consistent features associated with Type II diabetes seem to be obesity and a resistance to whatever insulin the body produces.

INSULIN, BLOOD SUGAR AND DIABETES

Your students might wonder about the relationship between insulin, blood sugar and diabetes. A brief discussion could include the following points:

1. After a person eats, glucose (sugar) and other nutrients are absorbed by blood vessels in the small intestine.

2. Increases in blood glucose levels stimulate the pancreas (a small gland located below and behind the stomach) to release the hormone insulin into the bloodstream.

3. Normally, tissue cells are sensitive to circulating insulin and, with the help of insulin, absorb glucose from the bloodstream. The cells in Type II diabetes, however, become insensitive to insulin. These cells do not respond to circulating insulin and do not absorb glucose from the bloodstream.

4. As a consequence, body tissues of a person with Type II diabetes absorb less and less sugar and their cells enter a state of starvation.

5. Meanwhile, abnormally high levels of blood glucose can eventually overwork or stress the insulin-producing cells of the pancreas, and may cause them to produce less insulin.

6. The liver compensates for "starving" tissue cells by breaking down stored carbohydrates and releasing more sugar into the bloodstream.

7. Thus, a circulatory system already saturated with sugars becomes even more overloaded, and becomes less and less efficient at delivering oxygen to body tissues.

8. This cycle of ever-increasing blood sugar levels will continue as long as the diabetes remains untreated. Blood sugars that are not absorbed into tissues will be excreted in the urine (again, the basis for calling this disease sugar diabetes).

9. If a person with Type II diabetes conscientiously follows a daily weight reduction and exercise program, the exhausted pancreatic cells may, over time, recover enough to begin secreting insulin again. The tissue may also become sensitive to this insulin, allowing cells to absorb and utilize glucose normally.

GESTATIONAL DIABETES

Some of your students may be familiar with gestational diabetes, which occurs during pregnancy. This form of the disease is temporary, usually appearing during mid-pregnancy and disappearing upon the birth of the child. Nationwide, this form of diabetes occurs in 3 to 5% of pregnant women. Among Native Americans, however, the incidence varies from 5 to 10% of all pregnancies[1].

If some of your students choose to research gestational diabetes, make sure they include the following points in their presentation:

1. During pregnancy, the placenta and umbilical cord serve as the lifeline between the fetus and the mother.

2. Nutrients and fluids flow from the mother's circulatory system through the placenta into the fetal circulation. Wastes produced by the fetus flow back through the placenta to the mother to be excreted by her kidneys.

3. The placenta also produces the hormones needed to maintain the pregnancy. Some of these hormones interfere with the action of the mother's insulin.

4. The pancreas of most pregnant women will compensate for this interference by increasing the production of insulin.

5. Sometimes the amount of insulin produced by the mother's pancreas is not enough to overcome the blocking action of the placental hormones. In these cases, the pregnant woman develops a temporary form of diabetes known as gestational diabetes.

6. Gestational diabetes usually develops during mid-pregnancy (about the 24th week) and disappears after the birth of the child.

7. Gestational diabetes usually does not cause birth defects. Most birth defects occur during the first 12 weeks of pregnancy, long before gestational diabetes develops;

8. Gestational diabetes may, however, result in the birth of a larger than normal baby. The mother's blood is high in glucose, which triggers the fetal pancreas to produce more insulin. This combination results in the fetus removing sugars from its bloodstream and converting them into fatty deposits;

9. Who is at risk to develop gestational diabetes?

   a. Women with a family history of diabetes mellitus;

   b. Women who are overweight, because they may be placing stress on their pancreas;

   c. Women who have given birth to a very large baby, because they may have had undiagnosed gestational diabetes during the previous pregnancy;

   d. Older mothers, who are at greater risk than younger mothers because an older pancreas may become more easily stressed than a younger pancreas.

10. Is a woman who has gestational diabetes likely to develop Type II diabetes in the future?

    In one study, 50% of the women who were diagnosed with gestational diabetes developed Type II diabetes within 15 years after pregnancy[2]. Any woman diagnosed with gestational diabetes should be tested for diabetes regularly for the rest of her life.

11. Are children of women with gestational diabetes likely to develop Type II diabetes in the future?

    In one study of Pima Indians, over 45% of children born to women who experienced gestational diabetes during their pregnancies developed diabetes themselves before they reached the age of 24. These children are at high risk for diabetes; therefore, it is extremely important to monitor the weight and health of children of mothers who develop gestational diabetes when they were pregnant.

LESSON 2

COMPARISON OF DIFFERENT TYPES OF DIABETES MELLITUS

	TYPE I DIABETES	TYPE II DIABETES	GESTATIONAL DIABETES
When do the symptoms of diabetes usually appear?	During childhood or early adulthood	In adults older than 35 years of age	About midway through pregnancy
How common is this form of diabetes in the US?	10%-15% of all Americans with diabetes	85%-90% of all Americans with diabetes	3%-5% of all pregnancies in the US
How common is this form among Native Americans in the Aberdeen Area?	1%-2% of Native Americans with diabetes	98%-99% of Native Americans with diabetes	5%-10% of all Native American pregnancies
Is normal insulin present in affected individuals?	No, either not enough insulin is produced, or the insulin is abnormal in structure and cannot function properly.	Yes, normal insulin usually is produced, but the body's cells have become resistant to its action.	Yes, normal insulin usually is produced, but its action is blocked by placental hormones.
Is this form of diabetes associated with excess weight?	No, many children with Type I diabetes are normal or underweight.	Yes, more than 80% of adults diagnosed are overweight by 20% or more.	Most pregnant Native American women who are diagnosed are overweight, although a few may not be.
Are daily insulin injections required for treatment?	Yes, along with dietary and weight control.	Sometimes, but most doctors concentrate on weight reduction through diet and exercise, or on oral hypoglycemics (sugar reducing pills).	Sometimes, but diet and weight control are always important.

HOW COMMON IS DIABETES AMONG NATIVE AMERICANS?

Diabetes mellitus affects an estimated one in 20 Americans (10 to 12 million people), about half of whom are unaware that they have diabetes. One in 10,400 will die of this disease each year[3]. The death rate among Native Americans, however, is dramatically higher. In 1989, an

estimated one in 1,700 people on the Pine Ridge reservation died of Type II diabetes mellitus[4]. This is more than six times higher than the national average.

Historically, diabetes was not a major health problem for Native Americans. Indeed, diabetes was rare in all tribes before the 1940's. In 1900, for example, the census reported only two Native American deaths from diabetes in a population of about 266,000. In 1955, the proportion of American Indians dying from diabetes was equal to the proportion of diabetes deaths in the Caucasian population[3]. Now, the proportion of Native American deaths is much higher, and increasing. This rapid increase in the incidence of diabetes makes it one of the most common chronic diseases among Native Americans.

WHAT ARE THE SIGNS AND SYMPTOMS OF DIABETES MELLITUS?

The most common characteristics of diabetes mellitus are:

1. Sugar in the urine. Doctors call this condition "glucosuria." It indicates that the blood sugar is not consumed by tissue cells, but rather is "spilling over" into the urine.

2. Excessive urination. The elimination of sugar in the urine requires water as the carrier solvent. This results in more urine being produced by the kidneys, and in more frequent urination.

3. Excessive thirst. Because the body is losing more water through the urine than usual, diabetics are constantly thirsty and drink large amounts of water.

4. Increased appetite. The major source of fuel for the body is sugar. Given that individuals with untreated diabetes are not able to process sugar, they tend to eat more food in an effort to compensate for this lack of fuel.

5. Long-term effects of diabetes mellitus. Many long-term effects of diabetes can be extremely harmful, even life-threatening.

LONG-TERM EFFECTS OF DIABETES MELLITUS

If a group of students chooses to study the long-term effects of diabetes mellitus, the group should bring up the following points in its presentation to the class.

In uncontrolled diabetes, the disease process affects blood vessels. Damage occurs to the capillaries, the small blood vessels through which molecules pass into tissue cells. Such damage reduces the amount of oxygen and nutrients reaching the tissues, which then gradually become diseased. Many people refer to this decreased flow as "poor circulation."

Many long-term complications of diabetes can result in disease processes that are related to reduced amounts of oxygen reaching body tissues. Examples include:

1. Poor circulation to the eyes can lead to blurred vision and other vision problems, and sometimes even blindness.

2. Reduced circulation to the kidneys can damage kidney tissues which, in turn, can lead to kidney failure.

3. Reduced circulation to the skin can result in dryness, itching, infections, skin ulcers and poor healing.

4. Reduced circulation to the arms and legs can cause tissues in the hands and feet to die. This results in gangrene, a term indicating death of surrounding tissues generally due to lack of sufficient blood supply. Without early treatment, amputation may be necessary.

It is important for you to stress that these conditions are not inevitable consequences of diabetes. By faithfully following their doctors' instructions to control their blood sugar levels, people can limit the severity of problems caused by poor circulation.

IS DIABETES INHERITED?

Diabetes mellitus runs in families. Nationwide, about 17% of diabetics have a diabetic mother, and another 8% have a diabetic father. If a person develops diabetes, the chances are greater that he or she will have children who become diabetic as they grow older. If both a parent and a child develop diabetes, the chance that other children will develop the disease increases dramatically.

Some geneticists have suggested that "diabetes genes" probably exist among certain groups of Native Americans, Mexican Americans, Australian aborigines, migrant Asian Indians, urbanized Pacific Islanders and some other non-Western populations[5]. Such genes are hypothesized to have been essential to the survival of these peoples.

HOW COULD GENES THAT CAUSE DIABETES BE BENEFICIAL?

Geneticists hypothesize that "diabetes genes" make tissue cells more efficient at converting dietary glucose (sugar) into body fat[6]. According to this hypothesis, individuals who inherit these genes store more fat when food is consumed. When food is scarce, this fat serves as an extra source of energy, to tide these individuals over until more food becomes available. Individuals without these genes (and lacking the extra fat) are more apt to starve when food is not available for long periods of time.

This hypothesis suggests that the indigenous people who had these genes were more likely to survive periods of drought, or harsh winters, when food sources were scarce. Those that survived were, in turn, more likely to have children and pass on this particular genetic trait.

During the past century, the diets and lifestyles of Native Americans have changed. For example, meals today are lower in fiber and higher in fats and sugars. In addition, most people rarely exercise on a daily basis. Nevertheless, people who have inherited these genes continue to store fat. As droughts and harsh winters no longer affect the availability of food, this weight is not lost. Rather, people with these genes risk becoming overweight and, eventually, diabetic.

LESSON 2

REDUCING THE RISK OF DIABETES

A person's genes do not condemn him or her to becoming diabetic. Adopting healthy lifestyles and behavior patterns may prevent this disease. The behaviors most often recommended to reduce your risk of becoming diabetic include[6]:

1. Keeping your weight appropriate for your age and height;
2. Reducing fat intake by choosing lean meats (lean beef and chicken), and low fat dairy products and by decreasing the use of butter and shortening;
3. Reducing calorie intake by avoiding foods that provide little or no "nutrition," such as candy, sugar, chocolate and ice cream;
4. Increasing the amount of fiber and complex carbohydrates by eating more vegetables, whole grain cereals, grains, beans, peas and other legumes;
5. Exercising regularly by walking, swimming, running or doing other aerobic activities.

In addition to excess weight and a lack of exercise, advanced age is a risk factor for diabetes mellitus. Only 1 in 900 people less than 20 years of age have diabetes. The rate increases to 1 in 200 between the ages of 41 and 50. Over the age of 61, the risk of diabetes increases to 1 in 50.

A person's chances of developing Type II diabetes mellitus are greatest when he or she possesses two or more of the above risk characteristics. For example, a person who has a diabetic relative and is overweight is far more likely to develop diabetes than a 20-year-old with no family history of this disease.

HEART DISEASE

WHAT IS HEART DISEASE?

Heart disease, is a general term for several different conditions that can lead to heart failure. The American Heart Association describes five major forms of cardiovascular disease ("cardio" referring to heart and "vascular" to blood vessels)[7].

A. CORONARY HEART DISEASE OR CORONARY ARTERY DISEASE

The major arteries which nourish the heart muscles are called coronary arteries. Damage to these arteries can decrease the blood flow to the heart. Decreasing the blood flow reduces the amount of nutrients and oxygen reaching the heart muscle and causes heart attacks, or myocardial infarctions (MI).

The primary cause of coronary artery damage is the long-term build up of fatty deposits on the artery walls. Doctors call this condition atherosclerosis, and the fatty deposits, plaque. This build up results from high blood levels of cholesterol (a complex fatty molecule) and lipoproteins (fatty molecules linked to a protein).

People who have heart attacks often have a diet that is high in fat and cholesterol. This increases the chance that fatty deposits, or plaque, will build up on the walls of the coronary arteries. The build up of fatty deposits decreases the diameter of the arteries and the amount of oxygen and nutrients that reach the heart muscle. This, in turn, results in an increased risk of a heart attack.

B. CONGESTIVE HEART FAILURE

Congestive heart failure refers to the mechanical failure of the heart to pump blood efficiently. This condition may or may not be life-threatening, depending on which factors are causing the heart to pump less blood.

Some students might wonder how "congestion," which refers to an accumulation of fluids in body tissues, is related to heart failure. A generalized sequence of events follows: As the heart fails mechanically, it pumps out less blood. Blood begins to accumulate in the veins. This accumulation increases pressure within the veins, which eventually forces fluids out of the veins into surrounding tissues. The tissues gradually become swollen with fluids, or "congested."

A few causes of congestive heart failure include:

1. Damage to one or more heart valves, so that they do not close properly during a contraction. Normally, the valves allow blood to flow in only one direction, forward. Damaged heart valves may allow blood to flow backwards into the heart chambers.

2. Abnormally shallow and irregular heartbeats, called arrhythmia. Such contractions are not strong enough to propel the normal volume of blood forward through the major arterial system.

3. Myocardial infarction, discussed in the section above on coronary heart disease. A heart attack destroys heart muscle cells and decreases the force of the heartbeat.

4. Disturbances in the balance between water and salt concentrations in the blood. Heart muscle, as well as skeletal muscle, requires a certain level of calcium ions in order to contract properly.

5. High blood pressure, or hypertension, which increases the workload of the heart, and decreases the efficiency of each heartbeat.

C. CONGENITAL HEART DISEASE

Some babies are born with heart defects. In most instances there is a defect in the structure of the heart, such as a hole in the wall that separates the right and left chambers, or an abnormal heart valve. Heart anomalies that are present at birth are referred to as congenital heart defects.

D. RHEUMATIC HEART DISEASE

Rheumatic fever usually occurs in school-aged children following an infection of the throat with group A streptococci. In response to the infection, white blood cells produce antibodies directed against the invading bacteria. In some children, however, these antibodies also attack normal tissue, causing inflammation and pain. The most common tissues attacked are the joints. Less commonly, heart tissue is involved. Inflammation of the joints rarely causes permanent damage, but inflammation of heart tissue can injure the heart valves so that they no longer function properly.

Antibiotics can prevent the long-term consequences of rheumatic heart disease. Now rheumatic fever rarely causes actual heart failure, and most children who have had this disease can lead normal or near-normal lives.

E. HYPERTENSION

The term "blood pressure" refers to the pressure of the blood within the arteries. It is defined by two numbers. The upper number indicates the amount of pressure exerted by the blood against the artery walls as the heart contracts. The lower number represents the pressure exerted when the heart is relaxed, between beats. Technically, the upper number is called systolic blood pressure, and the lower number diastolic blood pressure.

High blood pressure, or hypertension, occurs when either systolic or diastolic values rise above normal. A prolonged increase in blood pressure can damage the blood vessels and lead to heart failure, stroke, kidney failure and other life-threatening conditions.

Hypertension is not a simple disorder and requires more space than is available in this module for a complete discussion; however, should your students want to discuss high blood pressure, a few comments are in order.

CAUSES OF HIGH BLOOD PRESSURE

Many people have high blood pressure for no apparent reason. This form of high blood pressure is called essential hypertension, and tends to occur more frequently in certain families.

In many cases, people with essential hypertension can bring their blood pressures down to normal or near-normal levels simply by modifying their diets and lifestyles. Recommended changes include losing weight, exercising on a regular basis, avoiding fatty foods, avoiding salty foods and learning to use salt-free seasonings on foods.

When the cause of high blood pressure is known, it is called secondary hypertension. Some causes of secondary hypertension include:

1. Kidney disease. Damage to the kidneys can prevent the removal of wastes and poisons from the blood. As these waste products accumulate in the blood, they interfere with the excretion of fluids, which then accumulate in the body as edema. A decrease in blood supply to the kidneys can also increase blood pressure. When the blood supply to the kidneys is decreased, the kidneys secrete renin, an enzyme that produces angiotensin. Angiotensin constricts blood vessels, which increases blood pressure.

2. Certain hormonal disorders. Certain glands produce hormones that influence blood pressure by regulating kidney function. The pituitary gland, for example, produces an antidiuretic hormone (ADH). This hormone regulates the reabsorption of water by the kidney. Also, the adrenal glands secrete aldosterone, a hormone that regulates salt reabsorption by the kidneys. Since "water follows salt," increased retention of salt leads to increased retention of water, which in turn leads to increased blood pressure.

3. Pregnancy. Some women are diagnosed with high blood pressure during pregnancy. In some cases, this is a result of conditions existing before pregnancy that have gone undiagnosed and untreated. In other cases, hypertension is a result of the pregnancy itself.

4. Contraceptive pills. About 5% of women taking oral contraceptives also develop high blood pressure. Most "pills" contain the female hormones estrogen and progesterone, which apparently stimulate the adrenal glands to produce aldosterone. As described above, increased production of aldosterone increases blood pressure.

SYMPTOMS OF HIGH BLOOD PRESSURE

Hypertension is almost always a disease without symptoms, at least in its earliest stages. In the later stages of the disease, symptoms of hypertension include; headaches that are present when waking in the morning and decrease as the day passes, episodes of dizziness and weakness, nosebleeds and blurred vision. Such symptoms are <u>not</u> the result of increased pressure itself, but of damage to the blood vessels and other tissues caused by high blood pressure over a long period of time.

LONG-TERM EFFECTS OF HYPERTENSION

People with untreated high blood pressure die prematurely. People who follow their doctors' orders carefully can lead normal or near-normal lives.

The most common causes of death resulting from untreated hypertension are heart failure, stroke and kidney disease.

1. Heart failure. Increased blood pressure in the arteries creates a strain on the heart. It forces the heart to pump harder in an effort to deliver an adequate amount of blood to the body's tissues and organs. Over time, this strain on the heart and damage to the inner

lining of the coronary arteries can lead either to coronary heart disease or to congestive heart failure (see the discussion in Sections A and B).

2. Stroke. Technically called a cerebrovascular accident, or CVA, a stroke occurs when part of the brain is deprived of blood. High blood pressure can cause the blood vessels in the brain to burst. Blood seeps out of the vessels into the surrounding brain tissue. The nature and severity of damage depends on the region of the brain affected and the amount of tissue destroyed. The most common, and frightening, symptoms of a stroke are loss of consciousness, mental confusion and paralysis. Paralysis occurs as a result of damage to cells that receive sensory stimuli or initiate muscular movements.

3. Kidney disease. High blood pressure can destroy delicate kidney capillaries that filter wastes out of the blood. If the kidneys cannot filter properly, these waste products accumulate in the blood and eventually poison the body. These capillaries may also burst from the increased pressure and spill blood into the urine. If untreated, the diseased kidneys will fail to function altogether, and without dialysis or a transplant, the patient will die.

These long-term consequences of uncontrolled high blood pressure <u>are</u> preventable. Because there are no symptoms associated with the early stages of hypertension, everyone should have their blood pressure checked at least once a year. If diagnosed early, people with high blood pressure can significantly reduce their risk of heart failure, stroke and kidney disease by following their doctors' orders, watching their weight, watching their diet and taking their medications as prescribed.

HOW COMMON IS HEART DISEASE AMONG NATIVE AMERICANS?

Heart disease accounts for nearly one-third of all deaths among Europeans and European-Americans. This means that one out of 571 Americans nationwide dies of heart disease each year, most often as the result of coronary heart disease or high blood pressure. The death rate among Lakota people living on the Pine Ridge reservation is one in 305, nearly double the national rate[4].

WHAT ARE THE SIGNS AND SYMPTOMS OF HEART DISEASE?

Symptoms of coronary heart disease and high blood pressure do not usually appear until after a great deal of damage has already occurred. The first noticeable sign of a heart attack is often chest pain that spreads to the arms, back, throat or jaw. During the attack, chest pain may vary from a tight feeling to a bursting sensation. The pain may be continuous or it may come and go. Other symptoms include dizziness, shortness of breath, sweating, nausea and fainting.

IS HEART DISEASE INHERITED?

Like diabetes, heart disease runs in families. The more relatives a person has with heart disease, the greater his or her risk is for developing heart disease as he or she grows older. One study showed that if two members of a person's immediate family (parents, grandparents,

siblings) had a heart attack before the age of 55, that person's risk of developing heart disease was five times the risk of someone who had no family history of heart disease[7].

As people grow older, their chance of having a heart attack also increases. Historically, men were at a greater risk for coronary heart disease than women. This trend, however, seems to be changing, at least in the industrialized countries.

None of these factors, family history, age or sex, are under our control. A person can, however, control many other factors that increase his or her chances of heart disease, such as:

1. Smoking cigarettes. Nicotine not only increases the heart rate but also intensifies the effects of high blood pressure. Heavy smokers who suffer heart attacks are much more likely to die than nonsmokers who suffer heart attacks.

2. Being overweight, primarily because the heart must work harder in order to pump blood to all the tissues in an obese person.

3. Eating foods containing high amounts of saturated fats, such as fatty meats, butter, bacon, cream and whole milk cheeses, because these foods contribute to atherosclerosis, (see Section A on coronary heart disease).

4. Eating sugar-rich foods such as pies, cakes, cookies, ice cream, candy, soft drinks, fruit drinks, fruit packed in syrup, jams, jelly, doughnuts and sweet rolls, because these foods contribute significantly to obesity.

5. Eating foods high in salt, such as potato chips, pretzels, salted nuts and popcorn, soy sauce, steak sauce, cheese, pickled foods and cured meats, because salty foods contribute to high blood pressure, which increases the risk of heart attacks.

6. Not exercising regularly.

Is heart disease inherited? The answer is yes, heredity does contribute to heart disease, but it is not the only factor that contributes to the risk of heart disease. In most cases, a person's risk of heart disease is determined by the interaction between several pairs of genes, the environment and lifestyle choices. By eating a balanced diet, exercising regularly and eliminating cigarettes and alcohol, even a person with a family history of heart disease can significantly reduce his or her risk of heart disease.

FAMILIAL HYPERLIPOPROTEINEMIA

Some of your students may have heard of "good" and "bad" cholesterol. The liver produces lipids which are joined to certain protein molecules and released into the blood. These complex compounds are known as lipoproteins. Low density lipoproteins (LDL) are molecules that deliver cholesterol to tissue cells. If LDL levels are abnormally high cholesterol accumulates in cells which contributes to heart disease. High density lipoproteins (HDL) remove cholesterol from tissue cells and deliver it to the liver for processing. Some people

oversimplify these types of lipoproteins by calling LDL "bad cholesterol" and HDL "good cholesterol."

There are five known genetic forms of high blood cholesterol, or familial hyperlipoproteinemia. Two of these are due to recessive genes, and three to dominant genes. The symptoms of these hereditary diseases can vary widely from relatively mild symptoms such as, deposits of fat on the skin called xanthomas, to severe abdominal pain and even heart attacks in people in their late 20's or early 30's. The frequency of these hyperlipoproteinemia genes among Lakota, Dakota and Nakota people is not known.

ALCOHOL AND ALCOHOLISM

WHAT IS ALCOHOL?

Alcohol is a small two-carbon molecule that acts as a depressant and interferes with the activity of the central nervous system (the brain and spinal cord). It is a mind- and mood-altering drug. If consumed in small amounts, alcohol usually has little or no apparent effect on the drinker. In moderate amounts, alcohol can produce an exaggerated sense of happiness and well-being. In large amounts, alcohol acts as a sedative.

Not only does alcohol affect the mind, it also affects the body. Alcohol can damage the stomach, pancreas, liver, cardiovascular system (heart and blood vessels) and the nerves throughout the body. There is also a high correlation between alcohol abuse and sugar diabetes among Native Americans.

WHAT IS ALCOHOLISM?

Although alcohol affects people differently, and everyone has their own definition of alcoholism, most definitions include the following factors:

1. An inability to control one's drinking habits, resulting in excessive alcohol consumption.
2. Drinking at inappropriate times.
3. A preoccupation with ways of obtaining and drinking alcohol.
4. An increased tolerance to alcohol.
5. Chronic, or repeated consumption of alcohol, leading to impairment of health and interference with normal functioning.
6. A physical dependency on alcohol, resulting in withdrawal symptoms when alcohol is no longer consumed.

WITHDRAWAL SYMPTOMS

Withdrawal symptoms vary from person to person. In most cases, the signs of withdrawal are relatively mild. They may include tremors of the hands, feelings of anxiety or panic attacks, insomnia and occasionally bad dreams. These symptoms often begin within 5 to 10 hours after an alcoholic person has had his or her last drink. These symptoms may disappear within a week, or they may last for months.

Approximately five percent of alcoholic individuals will have severe withdrawal symptoms, or delirium tremens (DTs). Symptoms can include a state of mental confusion and disorientation, restlessness, irritation and hallucinations. They may hear voices, see non-existent things, or feel as if something is touching them or crawling over their bodies.

HOW COMMON IS ALCOHOLISM AMONG NATIVE AMERICANS?

Native Americans exhibit a higher rate of problems associated with alcohol abuse than any other ethnic group in the US[8]. Death rates from chronic liver disease and alcoholism are 6 to 10 time higher than the national rate of one per 15,600[4]. Deaths due to injuries (18%), suicide (3%), and homicide (3%) resulting from alcohol abuse are among the top ten causes of deaths reported by the Indian Health Services[8].

HOW DOES ALCOHOL AFFECT THE BODY?

THE LIVER

Alcohol is quickly absorbed from the stomach and small intestine into the bloodstream and carried to the liver. Liver enzymes convert ethanol (CH_3CH_2OH) into acetaldehyde (CH_3CHO), and acetaldehyde into acetic acid (CH_3COOH). This process not only consumes needed oxygen, but also causes an abnormal build up of various chemicals in the liver.

Acetaldehyde is responsible for hangover symptoms and "the shakes" associated with heavy drinking. Acetaldehyde is also similar in molecular composition to the stimulant amphetamine.

With repeated drinking, fatty deposits build up in the liver. These deposits crowd out normal liver cells and interfere with their functions. Eventually the liver becomes inflamed (a condition called hepatitis) and scarred (cirrhosis).

THE HEART AND BLOOD VESSELS

Alcohol causes blood vessels to dilate, or increase in diameter. This creates both a mild drop in blood pressure and an increased heart rate. Higher doses will increase blood pressure, although how this happens is not clear. Continued drinking may eventually damage the heart muscle itself, resulting in heart enlargement and an abnormal heart rate. This damage can contribute to heart failure. Strokes (the rupture of blood vessels in the brain) can also follow episodes of heavy drinking.

LESSON 2

THE CENTRAL NERVOUS SYSTEM

Perhaps the most familiar effects of alcohol abuse on the central nervous system are altered behavioral patterns. People who drink may have an artificial sense of well-being or euphoria, followed by depression and sometimes hostility. They may have lowered inhibitions and impaired judgment. Alcohol use may cause staggering and slurred speech. Excessive drinking may also lead to episodes of unconsciousness or "blackouts."

Continued drinking can interfere with normal nutrition, causing malnutrition and vitamin deficiencies (especially of thiamin, a B-vitamin). In some people this leads to double vision, a lack of muscular coordination and decreased mental function. Individuals addicted to alcohol may also experience delirium tremens when they stop drinking. The symptoms include tremors, hallucinations and delusions.

IS ALCOHOLISM INHERITED?

Over the past several years, researchers have attempted to identify the gene, or genes, that cause alcohol abuse. Their results, however, are not conclusive. One group of researchers found that some alcoholics carry the A1 gene, a gene that has been associated with several psychiatric disorders. This gene, however, is absent in the majority of alcoholics, which leads to the conclusion that the A1 gene by itself cannot cause alcohol abuse.

Other researchers feel that alcoholism is far too complex a disease to be caused by a single gene. Most geneticists believe that alcoholism itself is not genetic, but that people can be genetically predisposed to develop alcoholism. That is the potential, or predisposition, to become an alcoholic is inherited.

The tendency to become alcohol-dependent is most likely determined by the interaction between several genes and a number of non-genetic factors. Non-genetics factors that influence a person's risk of becoming an alcoholic include[9]:

1. A family history of alcoholism. If relatives are heavy drinkers, the chances are increased that other members of the family will become alcoholic.

2. A family history of depression. Families with a high incidence of depression, especially among female members, tend to have more alcoholic members than other families. It seems that many people who are chronically depressed think they will find temporary relief by getting drunk.

3. A family history of total abstinence. Ironically, families that forbid drinking alcohol and that enforce strict moral controls are more likely to have alcoholic children. Children who feel the need to rebel against regulations at home (or school) may deliberately set out to engage in behaviors they know will upset their parents or other adults in the community.

4. A family history of divorce and parental discord. Children raised by single parents are more likely to become alcoholic than children raised by two parents.

5. Being a member of certain ethnic groups. The rate of alcoholism is higher among some ethnic groups than among others. Those that are most vulnerable include Native Americans, Northern Europeans (including Irish and Scandinavians), the French, Mediterranean peoples (Italian, Greek and Jewish), and the Chinese.

6. Being a heavy smoker. Persons who are heavy smokers are also at a higher risk for drinking alcohol to excess. Apparently, many people who find nicotine and tobacco calming also tend to drink alcohol.

RESOURCES

Begin your search for information by talking to the counselors in your school or community. They may have both written information and a list of local resource people. Health clinics on the reservation are also a source of materials (including guest speakers and visual aids) on diseases of concern to local residents.

DIABETES

Information is available from state and national organizations. Consider writing or phoning for information about diabetes from the following organizations:

Diabetes Control Program
South Dakota Department of Health
445 East Capitol
Pierre, SD 57501-2080
Phone: 605-773-3737

American Diabetes Association
National Service Center
1660 Duke Street
Alexandria, VA 22314
Phone: 1-800-232-3472

Juvenile Diabetes Foundation International
60 Madison Ave., 4th Floor
New York, NY 10010
Phone: 1-800-223-1138

National Diabetes Information
Clearinghouse
Box NDIC
9000 Rockerville Pike
Bethesda, MD 20892
Phone: 301-468-2162

HEART DISEASE

Perhaps the most familiar national organization dealing with heart disease is the American Heart Association. In addition, the National Heart, Lung and Blood Institute distributes good consumer information upon request.

American Heart Association
1005 12th Ave. SE
PO Box 1287
Jamestown, ND 58402
Phone: 1-800-437-9710

NHLBI Information Center
PO Box 30105
Bethesda, MD 20824-0105
Phone: 301-951-3260

LESSON 2

ALCOHOLISM

There has been a great proliferation of organizations set up to advise, counsel and treat persons suffering from alcoholism and substance abuse. Perhaps the most familiar national organization is Alcoholics Anonymous (AA). For information from a local resource, contact Anpetu Luta Otipi in Kyle, on the Pine Ridge Reservation.

Alcoholics Anonymous National Office
General Service Board
Box 459
Grand Central Station
New York, NY 10163
Phone: 212-870-3400

Anpetu Luta Otipi
PO Box 275
Kyle, SD 57752
Phone: 605-455-2331

Other organizations include:

Alateen World Service Headquarters
PO Box 862
Midtown Station
New York, NY 10018-0862
Phone: 212-302-7240

Division of Alcohol and Drug Abuse
500 East Capitol
Pierre, SD 57501
Phone: 605-773-3123

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HEALTH IN THE YEAR 2100

LESSON 3

WHAT ARE GENES?

INTRODUCTION

This lesson on genes is too long to cover in a single class period so it is divided into two sections. The first section, "Multiple-Gene Traits," expands on the concept introduced in the previous lesson; that more than one pair of genes can cause certain diseases to develop later in life. The concept of multiple-gene traits will be developed in this section through an exercise using pop-beads drawn randomly from two separate containers.

You will cover the second section, "Non-Genetic Factors," during the following class period. The proposed activity is designed to help students understand that non-genetic factors, such as the environment and lifestyle choices, can modify the expression of genes. They will learn that inheriting a particular combination of genes does not automatically mean that a person will inevitably develop the disease. Whether or not a person develops a particular "multiple-gene" disease depends on both the genes that person inherits and the lifestyle he or she chooses.

SECTION I: MULTIPLE-GENE TRAITS

GOAL

The students will learn that some traits are determined by more than one pair of genes and that various non-genetic factors can influence gene expression.

OBJECTIVES

By the end of this lesson, students will understand:

1. That people do not inherit physical characteristics or traits; they inherit instructions, or genes, for making those traits;
2. That one-half of the genes come from their mother and one-half from their father;
3. That these instructions are provided by chemical units called "genes;"
4. The difference between genotype and phenotype;
5. That the expression of some traits is governed by a single pair of genes, and that other traits are coded for by two, three or even more pairs of genes.

LESSON 3

MATERIALS AND ADVANCE PREPARATION

This lesson requires a significant amount of preparation. Before class, prepare two containers of pop-beads for each group of students. Each pop-bead represents a single gene, and each container represents gametes, either the sperm or the egg, produced by the parents.

Label one container "Father's Genes" and the other container "Mother's Genes." Each container should have equal numbers of red and white pop-beads (or any other contrasting colors available). Keep a separate supply of beads at your disposal to replenish the beads after each draw.

Make one copy of the worksheet, "Genotype/Phenotype," for each student. This worksheet should assist your students in understanding the significance of these experiments, and will serve as the basis for class discussion.

Make a transparency of the "Genotype/Phenotype Key," or recreate this key on the blackboard or on newsprint. This key will be completed during the course of this activity. Both the worksheet and the key are located on pages S-16 and S-17 in the "Student Manual."

DIRECTIONS FOR CONDUCTING THE ACTIVITY

DAY ONE

INTRODUCTORY DISCUSSION

1. Briefly review the disease discussed last period. All references in this lesson to "disease" will refer to the specific disease (diabetes, heart disease or alcoholism), your class discussed in Lesson 2. Make sure that your students have read the material handed out at the end of the previous lesson. They should be prepared to discuss the genetics of the disease they chose to study.

   In many cases, Lesson 2 will have ended on a Friday. With the passing of a weekend, some students will probably need to review the significant points covered in Lesson 2 before proceeding further with Lesson 3.

2. Ask your class to consider the following question; "How do people inherit physical traits such as eye color or the tendency to develop certain diseases?" If your students have a difficult time concluding that children inherit physical characteristics from both their mothers and fathers, ask them to consider what physical characteristics they share in common with their mothers. Then, ask them if they have any traits that are different from their mothers', and how they inherited these different traits.

   When your students are comfortable with the idea that physical traits are inherited from each parent, ask them to consider how these traits are passed on. If your students are unfamiliar with the concept of genes, define this word in very basic terms. Remember, it is not the goal of this module to introduce a large number of technical terms or molecular genetic concepts. To complete this lesson, all your students need to know is that genes

are small units of inherited material that code for physical traits, and that gene expression is affected by the environment, lifestyle choices and other genes.

Emphasize that genes are not "good" or "bad." Genes simply influence the production of traits. It is the traits that are beneficial or detrimental to a person's health. Also, point out that genes are inherited in pairs. A child inherits one gene coding for each trait from her mother, and another gene coding for the same trait from her father.

When you are convinced that your students understand the concept of genes, and the fact that a child inherits two genes coding for each physical trait, (one from the mother, and one from the father), introduce the first exercise.

FIRST EXERCISE: Inheritance of a Single Pair of Genes

EXAMPLE ONE

To begin this exercise, make sure your students understand that the beads represent individual genes, or sets of instructions. Students should also understand that the container labeled "Mother's Genes" represents the instructions a child inherits from his or her mother in the egg. The container labeled "Father's Genes" represents the genetic contribution of a child's father in the sperm.

After explaining these concepts, divide the class into small groups and give each group of students two containers of beads, one labeled "Father's Genes" and one labeled "Mother's Genes." Indicate that each container contains an equal number of red and white beads.

Next, instruct each student to withdraw a single pop-bead from the container labeled "Father's Genes," and another pop-bead from the container labeled "Mother's Genes," as shown in Figure 1. After each draw, replace the bead with a bead of the same color from your stock of extra beads. This will guarantee that the containers contain an equal number of red and white beads for the next student's draw.

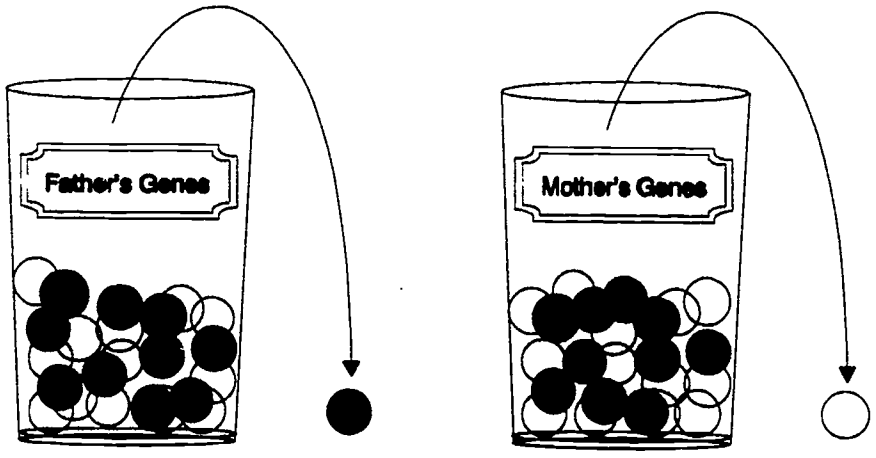
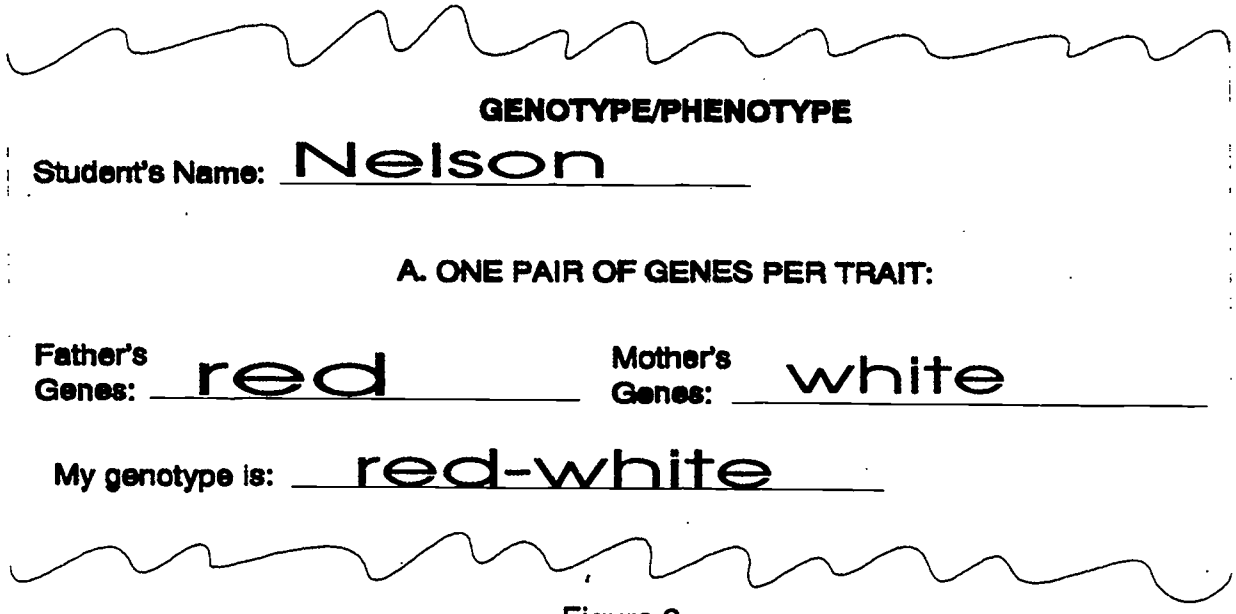


Figure 1

The students should then record their results (either red or white) in the blanks beside "Father's gene" and "Mother's gene" in Section A on their "Genotype/Phenotype" worksheet. Refer to Figure 2.



GENOTYPE/PHENOTYPE

Student's Name: Nelson

A. ONE PAIR OF GENES PER TRAIT:

Father's Genes: red **Mother's Genes:** white

My genotype is: red-white

Figure 2

Once the students have recorded their results on their worksheets, introduce the term "genotype."

1. Genotype refers to the combination of genes a person inherits. In this exercise, "red-red," "red-white," and "white-white" represent the three possible genotypes, or gene combinations. Emphasize that both parents contribute equally to the genotypes of their children.

At this point in the lesson, the students should push their two pop-beads together to illustrate their "genotypes." Students should then record their results, either red-red, red-white, or white-white, in the blank following "My genotype is:" on their worksheets.

Next introduce the concept of "phenotype."

2. Phenotype refers to the observable physical, biochemical or physiological characteristics that are determined by a person's genotype and the environment in which they develop. The number of possible phenotypes is determined, in part, by the number of genes involved in the expression of a trait.

To introduce the concept of phenotype, have your students imagine that each bead represents a gene that codes for a person's height. The red gene codes for growth up to 6 feet, and the white gene codes for growth up to 5 feet. You may want to write this on the board so your students can refer back to this information as the discussion proceeds.

LESSON 3

Begin by asking your students how tall they think a person will be if that person inherits two red genes. Emphasize that the effect of these two genes is not additive. For example, each red gene does not add 6 feet to a person's height. Instead, a person who inherits at least one red gene will grow to be 6 feet tall.

Then ask them how tall they think a person will be if that person inherits two white genes. If they are unsure about either of these answers, refer back to your discussion about genes and the information you recorded on the board about the significance of these two colors.

Once your students have concluded that a person who inherits two red genes will be 6 feet tall, and that a person who inherits two white genes will be 5 feet tall, record these responses on the "Genotype/Phenotype Key" in the column marked "Phenotype: One" in Section A. Then ask your students what they think the phenotype of the person who inherits one red gene and one white gene will be if the red gene is dominant. You may have to explain that "dominant" means that the expression of this gene will cover up or mask the presence of the second gene in the pair. When your students have concluded that a person who inherits one red gene and one white gene will be 6 feet tall, record this answer on the "Genotype/Phenotype Key." Then have your students fill in the blank on their worksheet following the statement, "If the red gene is dominant, my phenotype is: Example One."

If these traits follow the classical Mendelian pattern of inheritance as is suggested in this first exercise, the relationship between the genotypes and phenotypes will be as follows:

BEAD GENOTYPES	EXAMPLES OF PHENOTYPES HEIGHT
Red-Red	6 feet tall
Red-White	6 feet tall
White-White	5 feet tall

Once your students are comfortable with the concepts of genotype and phenotype, ask them to predict what the phenotype of a person would be if both genes were equally expressed, or co-dominant. Again, begin your discussion by asking about the height of a person with two red genes and a person with two white genes. Record their answers in the "Phenotype: One" column in Section A2 on the "Genotype/Phenotype Key." Then ask your students to consider how tall a person would be if that person inherited one red gene and one white gene. Your students should conclude that this would result in an intermediate phenotype. In our hypothetical model, this person would be 5 1/2 feet tall.

If both genes in a pair are equally expressed, the relationship between the genotype and phenotype will be as follows:

BEAD GENOTYPES	EXAMPLES OF PHENOTYPES HEIGHT
Red-Red	6 feet tall
Red-White	5 1/2 feet tall
White-White	5 feet tall

Once your students have recorded the results on their worksheets, ask them to compare their results with the results of other students in their group. In this exercise, some students will have drawn two red genes. They will be 6 feet tall. Other students will have one red gene and one white gene. Phenotypically, these students will be 5 1/2 feet tall. The third possible combination is two white genes, and these students will be 5 feet tall.

Then ask each group to consider the following questions: Did each student in your group draw the same color beads? Does each child in a family inherit the same set of genes from the mother and the father? Does each child have the same physical characteristics as his or her brothers and sisters?

They should reach the conclusion that children can inherit different combinations of genes from the same parents. Encourage your students to think about couples they know who have children with different physical traits.

Conclude this discussion by asking your students if they think that a person's height is determined by one gene, or set of instructions. Do they expect to grow to the same height as their parents or siblings? Is it possible that a person's height is determined by a number of different genes, or sets of instructions? What are some of the other things that can affect how tall a person becomes? (Examples might be nutrition, whether or not a person was sick as a child, etc.)

EXAMPLE TWO

Begin the next segment of this exercise by asking your students to consider the possibility that the genes they have selected will affect their health. Tell them that the white beads represent genes that code for disease and that the red beads represent genes that code for the normal, or healthy condition. Again, you may want to write this on the board so that your students can refer back to this information throughout the class period.

Begin by asking your students if they think that a person who inherits two red genes will be healthy. Then ask them if they think that a person who inherits two white genes will be healthy. Your students should conclude that a person with two red genes will be healthy and that a person who has two white genes will get sick. These responses should then be recorded in the first section of the "Genotype/Phenotype Key" below the space labeled "Two."

Next, ask your students to predict the phenotype of a person with one red gene and one white gene if the red gene is dominant. They should recall that a dominant gene will cover up or mask the expression of the second gene in the pair. The correct response would be that a person with one red gene and one white gene will be healthy. Record this answer on the "Genotype/Phenotype Key" and ask your students to fill in the blank on their worksheets following the statement; "If the red gene is dominant, my phenotype is: Example Two."

BEAD GENOTYPES	EXAMPLES OF PHENOTYPES HUMAN DISEASE
Red-Red	Healthy
Red-White	Healthy
White-White	Gets Disease

Next, ask your students to list the possible phenotypes if these two genes were co-dominant. In this scenario, a person with two red genes would still be healthy, and a person with two white genes would still get the disease. When you have recorded these responses on the key, ask your students to consider what the phenotype of a person would be if that person inherited one red gene and one white gene. Your students should conclude that this gene combination may result in a person who is genetically predisposed to the disease, but who will not necessarily develop the disease. They may decide that this person is at risk.

If the genes are co-dominant, the relationship between the genotype and the phenotype will be as follows:

BEAD GENOTYPES	EXAMPLES OF PHENOTYPES HUMAN DISEASE
Red-Red	Healthy
Red-White	At risk to get disease
White-White	Gets disease

After this discussion, the students should return to their worksheets and complete the blank that follows the statement "If the genes are co-dominant, my phenotype is: Example Two." The choices are "I will remain healthy," "I am predisposed to, or at risk to get the disease," or "I will eventually get the disease."

After all of the students have recorded their genotypes, and predicted their phenotypes, encourage them to discuss what these results mean. They should compare classical Mendelian patterns of inheritance to co-dominant patterns of inheritance. In the classical Mendelian genetics example, only two phenotypes are possible, either healthy or ill people. There are no intermediate traits such as, "At risk to get disease."

If time permits, you might want to point out that many genetic conditions are recessive in nature, examples include; cystic fibrosis, albinism (an absence of skin, hair and eye pigment), and a large number of enzymatic disorders, or inborn errors of metabolism.

Everyone carries between four and eight recessive genes that code for potentially lethal genetic disorders. Most people, however, never know which recessive genes they carry unless they have a child with a recessive genetic condition. For example, parents with normal skin and eye pigmentation may not know they carry the gene for albinism until they give birth to a child who lacks skin and eye pigmentation. Because the gene for albinism is recessive, a child with albinism must possess two copies of this gene.

In such families, each parent carries one gene for normal pigmentation (the dominant gene, which expresses itself in the parents) and one gene for albinism (the recessive gene, whose expression is masked by the dominant gene). There is a 25% chance with each pregnancy that both parents will pass on the gene coding for albinism and that a child will be born with this condition. There is a 75% chance that at least one parent will pass on a copy of the gene coding normal pigmentation and that a child will be born with normal skin and eye color. This particular pattern of inheritance is graphically represented in Figure 3.

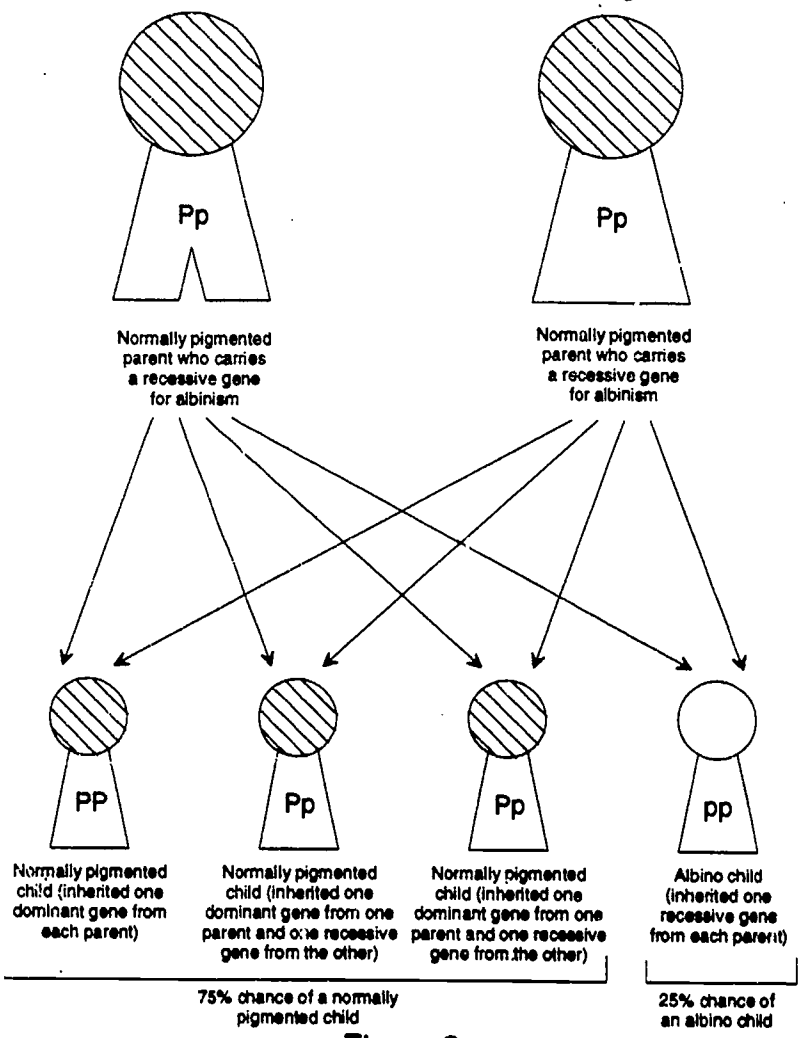


Figure 3

Human disorders due to a single dominant gene are far less common; examples include many skeletal anomalies, such as achondroplasia (a form of dwarfism), and Marfan syndrome, (a connective tissue disorder resulting in tall stature and potential heart problems). Familial polyposis of the colon is a dominant single gene disorder that results in the formation of multiple polyps in the colon which, if not removed, can become cancerous. Neurofibromatosis (NF) is another dominant disorder that causes small lumps, referred to as "neurofibromas," to appear beneath the skin and around nerves in the body. People with NF also have numerous cafe-au-lait spots (brown patches of skin), and small tumors on their irises.

Many human traits, however, are not inherited in classic Mendelian patterns. Physical traits can be the result of genes which act in a co-dominant manner. When co-dominant genes are involved, a third intermediate phenotype appears. In our examples, 5 1/2 feet is the intermediate height between 5 feet and 6 feet, and "At risk to get disease" falls in-between "Healthy" and "Gets disease."

Once the students understand the inheritance of single gene traits, move on to the next exercise, which involves two independent pairs of genes that code for the same trait.

SECOND EXERCISE: Inheritance of Two Pairs of Genes

Instruct each group of students to turn in their beads to your stockpile. Then explain that this exercise will illustrate the possible genotypes and phenotypes that can occur if each parent passes on two genes that code for a specific trait. In this example, each parent will contribute two genes to the embryo, for a total of four genes.

Your students should conclude from this discussion that a number of genes might be involved in the expression of certain physical traits, or the predisposition to a particular disease. When they have grasped this concept, instruct each students to withdraw two pop-beads from each container, as shown in Figure 4. After withdrawing each bead, students should record their results (either "red" or "white") in the appropriate blanks following "Father's genes" and "Mother's genes," in Section B, on their worksheets. After all four beads have been withdrawn, students should record their genotypes (see chart below) on their worksheets. Because each parent contributes two genes to his or her child, each student will finish with a four-bead genotype. (Note: Be sure to replace each pop-bead drawn with a bead of the same color in order to maintain an equal number of red and white beads in each container.)

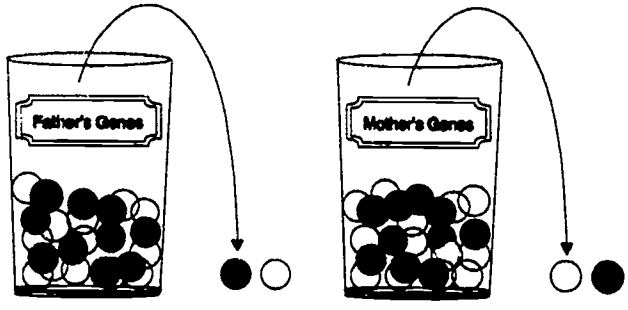


Figure 4

LESSON 3

In this exercise, there are five possible gene combinations, or genotypes, and five different phenotypes, if you assume that the genes in each pair are co-dominant.

Have the students in each group compare their genotypes. Then record the five possible genotypes in the "Genotype" column in Section B on the transparency. Next have them predict the phenotype of an individual who inherited four red genes, or four white genes. Again, they should conclude that a person who inherits four red genes will be healthy, and that a person who inherits four white genes will develop the disease.

As your students predict the phenotype for the three remaining genotypes, they should begin to appreciate that there can be varying levels of susceptibility to disease when more gene pairs are involved in the expression of a single trait.

The possible genotypes and phenotypes for this portion of the lesson are listed below:

BEAD GENOTYPES	EXAMPLES OF PHENOTYPES HUMAN DISEASE
4 Reds, 0 White	Healthy
3 Reds, 1 White	Low risk of getting disease
2 Reds, 2 Whites	Moderate risk of getting disease
1 Red, 3 Whites	High risk of getting disease
0 Red, 4 Whites	Gets disease

After you have finished filling out Section B on the "Genotype/Phenotype Key" and your students have recorded their phenotypes on their worksheets, encourage your students to compare these results (four genes per trait) with the previous results (two genes per trait). Guide the discussion so that your students discover the following:

1. One pair of co-dominant genes per trait yields three possible genotypes (R-R, R-W, and W-W) and three possible phenotypes (healthy, moderate risk of getting disease, and gets disease);

2. Two pairs of co-dominant genes per trait yields five possible genotypes (R-R-R-R, R-R-R-W, R-R-W-W, R-W-W-W and W-W-W-W) and five phenotypes (healthy, low, moderate and high risk categories, and gets disease);

3. Increasing the number of genes involved in the expression of a particular trait produces a greater range of phenotypes;

4. Students should also understand that a person with a "low risk phenotype" might need repeated exposures to certain environmental agents over a long period of time before he or she develops the disease;

5. A "high risk" person, on the other hand, may develop the same disease sooner, with more limited exposure to the same environmental agent. You might mention that during the

next period they will explore some of the non-genetic factors that influence gene expression.

THIRD EXERCISE: Inheritance of Three Pairs of Genes

This next activity is optional. If you feel that your students understand all of the concepts presented in the last two exercises, continue with this activity.

In this exercise, each student will draw three beads from each container and record his or her results on the blanks following "Father's genes," and "Mother's genes" in Section C. They will also fill in the section, "My genotype is:" on their worksheets.

BEAD GENOTYPES	EXAMPLES OF PHENOTYPES HUMAN DISEASE
6 Reds, 0 White	Healthy
5 Reds, 1 White	Very low risk of getting disease
4 Reds, 2 Whites	Low risk of getting disease
3 Reds, 3 Whites	Moderate risk of getting disease
2 Reds, 4 Whites	High risk of getting disease
1 Red, 5 Whites	Very high risk of getting disease
0 Red, 6 Whites	Gets disease

As before, ask your students to list the possible genotypes and predict what each phenotype would be. When you have completed filling in the "Genotype" and "Phenotype" columns in Section C on the "Genotype/Phenotype Key," instruct each student to complete the blank following "My phenotype is:" on his or her worksheet.

After the students have finished filling out their worksheets, point out that when there are three pairs of genes per trait, a much greater range of possible phenotypes exists. Use your discretion about how thoroughly to discuss these results.

Recall that one pair of genes produced three phenotypes, and that two pairs of genes produced five phenotypes. Then point out that three pairs of genes produce seven phenotypes. Ask them to consider also that the phenotypes are beginning to overlap. For example, it would be very difficult to distinguish a person at "High risk for getting disease" from a person with a "Very high risk of getting disease."

LESSON 3

SECTION II: NON-GENETIC FACTORS

OBJECTIVES

By the end of this section, students will be able to explain:

1. That gene expression is influenced by environmental conditions and lifestyle choices;
2. That only rarely does inheriting a particular combination of genes automatically mean that a person will inevitably develop a particular disease;
3. That people can reduce their risk of disease by altering their environment, diet and lifestyle.

MATERIALS AND ADVANCE PREPARATION

1. Before class, create one string of pop beads for each student. Join four beads in the following patterns, making approximately equal numbers of each "genotype":
   a. Three Red and 1 White pop-beads (Representing a "low risk" phenotype);
   b. Two Red and 2 White pop-beads (Representing a "medium risk" phenotype);
   c. One Red and 3 White pop-beads (Representing a "high risk" phenotype).
2. Then fill a series of containers with slips of paper that describe the non-genetic factors that influence a person's chance of getting the disease. The text for these strips is found on pages S-19, S-20 and S-21 in the "Student Manual" (one page for each of the three diseases studied in Lesson 2). Select the pages that describe the diseases (diabetes, heart disease or alcoholism) that your students researched in Lesson 2. Make enough copies of these pages so that you have at least one slip of paper, in each of the four categories, for each student who studied the disease.

The number of containers necessary for each disease and the strips that should be included in each container are listed below. The scores are not meant to be taken as absolute risk values. If your students want more information about their own personal risk for disease refer them to their family doctor or to a health care professional in your area.

A. If your students studied heart disease, you will need four containers. Label one container "FAMILY HISTORY OF HEART DISEASE," one "EATING HABITS," one "SMOKING HABITS" and one "EXERCISE HABITS."
   1. The "FAMILY HISTORY OF HEART DISEASE" container will contain equal numbers of slips of paper with the following phases and scores:
      a. No relatives with a history of heart disease: Score=0
      b. One relative with a history of heart disease: Score=2
      c. Two or more relatives with a history of heart disease: Score=4

2. The "EATING HABITS" container will contain equal numbers of each of the following slips of paper:
   a. Strict vegetarian: Score=0
   b. Eat turkey, chicken or fish, but no beef or pork: Score=1
   c. Eat beef or pork only once or twice a week: Score=2
   d. Eat beef or pork daily: Score=4
   e. Eat beef or pork more than once every day: Score=6
3. The "SMOKING HABITS" container will also contain equal numbers of the following slips of paper:
   a. Never smoke: Score=0
   b. Smoke less than five cigarettes a week: Score=1
   c. Smoke one to four cigarettes daily: Score=2
   d. Smoke less than one pack of cigarettes daily: Score=3
   e. Smoke more than a pack per day: Score=5
4. The "EXERCISE HABITS" container will contain the following slips in equal numbers:
   a. Vigorous daily exercise at both school (or work) and at home: Score=0
   b. Vigorous daily exercise at school or at home, but not both: Score=1
   c. Some daily exercise at both school and home: Score=2
   d. Exercise every other day or so: Score=3
   e. Exercise once a week or less: Score=5

B. If your students studied diabetes mellitus, you will need four containers. Label one container "FAMILY HISTORY OF DIABETES," one "EATING HABITS," one "EXERCISE HABITS," and one "WEIGHT."
   1. The "FAMILY HISTORY OF DIABETES" container will contain equal numbers of slips of paper with the following phrases and scores:
      a. No relatives with a history of diabetes: Score=0
      b. One relative with a history of diabetes: Score=2
      c. Two or more relatives with a history of diabetes: Score=4
   2. The "EATING HABITS" container will be filled with equal numbers of the following slips:
      a. Eat sweets once a month or less: Score=0
      b. Eat sweets once a week or less: Score=1
      c. Eat sweets two or three times a week: Score=3
      d. Eat sweets daily: Score=5

3. The "EXERCISE HABITS" container will contain the following slips in equal numbers:
   a. Vigorous daily exercise at both school (or work) and at home: Score=0
   b. Vigorous daily exercise at school or at home, but not both: Score=1
   c. Some daily exercise at both school and home: Score=2
   d. Exercise every other day or so: Score=3
   e. Exercise once a week or less: Score=5
4. The "WEIGHT" container will contain equal numbers of each of the following slips of paper:
   a. Normal weight for my height: Score=0
   b. 5 pounds or less overweight for my height: Score=1
   c. 6-10 pounds overweight for my height: Score=2
   d. 11-19 pounds overweight for my height: Score=3
   e. 20 or more pounds overweight for my height: Score=5

C. If your students studied alcoholism, prepare four containers. Label one container "FAMILY HISTORY OF ALCOHOLISM," one "FAMILY HISTORY OF DEPRESSION," one "DRINKING HABITS," and one "SMOKING HABITS."
   1. The "FAMILY HISTORY OF ALCOHOLISM" container will contain equal numbers of slips of paper with the following phrases and scores:
      a. No relatives with a history of alcoholism: Score=0
      b. One relative with a history of alcohol abuse: Score=2
      c. Two or more relatives with a history of alcohol abuse: Score=4
   2. The "FAMILY HISTORY OF DEPRESSION" container will contain equal numbers of the following slips of paper:
      a. No relatives have a history of depression: Score=0
      b. One relative with a history of depression: Score=2
      c. Two or more relatives with a history of depression:. Score=4
   3. The "DRINKING HABITS" container will contain equal numbers of the following slips of paper:
      a. Never drink. Disregard all other scores. A person who never drinks alcohol will not become an alcoholic."
      b. One drink per week: Score=1
      c. Two drinks per week: Score=2
      d. One drink every day: Score=4
      e. Two or more drinks per day: Score=6

4. The "SMOKING HABITS" container will contain equal numbers of the following slips of paper:
   a. Never smoke: Score=0
   b. Smoke less than five cigarettes a week: Score=1
   c. Smoke one to four cigarettes daily: Score=2
   d. Smoke less than one pack of cigarettes daily: Score=3
   e. Smoke more than a pack per day: Score=5

DIRECTIONS FOR CONDUCTING THE ACTIVITY

In the first section, students learned that a person's chance of getting certain diseases varies from none to unavoidable, depending on the genes that person inherits. They also learned that the number of phenotypes depends on how many pairs of genes are involved in the expression of a particular trait. The current exercise focuses on the non-genetic factors that cause some at-risk individuals to get the disease.

Distribute one pop-bead genotype to each student in a random manner. Divide your students into groups according to the diseases they studied in Lesson 2. Then place the four containers you prepared for each group on a table or counter-top. Each container should contain slips of paper that list specific non-genetic factors that influence gene expression and a score.

Instruct each student to withdraw (without looking) one slip of paper from each container. Each student now possesses one genotype plus several non-genetic factors that interact with that genotype to determine his or her phenotype. The students should add up the scores written on each slip of paper and then predict whether they think they will develop the disease based on their genotype and score. Refer to Figure 5.

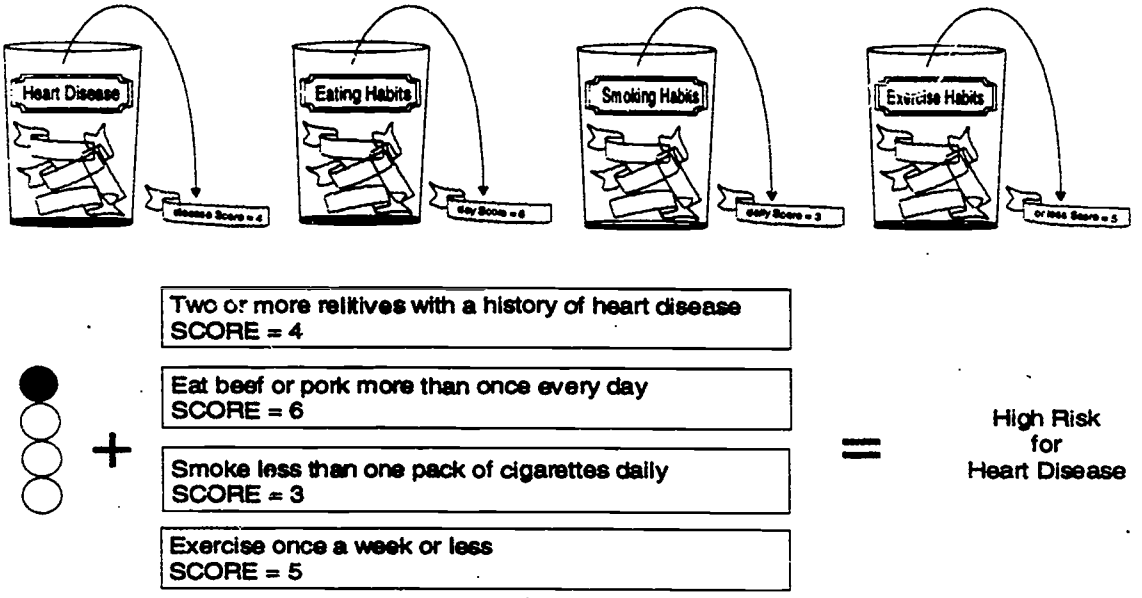


Figure 5

If it is not immediately clear to your students that the higher the score the more likely it is they will develop the disease, have those students who share the same "genotype" form a group and compare their scores. They should determine who is most likely to develop the disease and who is least likely to develop the disease, even though their genetic background is identical.

When it is clear that your students understand that a person's susceptibility to disease is determined by a combination of his or her genotype, family history, and lifestyle choices, then ask the students in each group to arrange their slips of paper in two piles. One pile will include those non-genetic factors over which they feel they have no control (such as family history); the other pile will include factors that students feel they can exert control over (such as, what they drink and eat, whether they smoke or chew tobacco).

You can then ask, "Can you change your risk of getting the disease by modifying your lifestyle and behaviors?" "What specific changes in behavior would lower your risks?"

At some point in this discussion, emphasize that some people who "do everything right" will still get some diseases because of factors they have no control over. Mention also that some people who engage in many "risk-increasing" behaviors will not get the disease because they have not inherited genes which predispose them to the disease. For example, almost everyone claims to have a relative who smoked every day of his life and lived well into his 80's or 90's. Conclude by reminding your students that, for the vast majority of people, eating a balanced diet, exercising regularly, using alcohol in moderation and cutting down or eliminating cigarette smoking can reduce the risk of dying prematurely from these diseases.

HEALTH IN THE YEAR 2100

LESSON 4

WHAT ARE CHROMOSOMES?

INTRODUCTION

Through a hands-on activity and classroom discussion, students will learn that chromosomes are microscopic structures made up of sequences of genes. Normally, a person inherits a total of 46 chromosomes. Twenty-three chromosomes are passed on by the father in the sperm, and 23 chromosomes are passed on by the mother in the egg. Twenty-two of the 23 chromosome pairs are similar in both males and females. The 23rd chromosome pair differs between the sexes.

GOAL

Students will learn that each person normally inherits 23 chromosomes from his or her mother in the egg and 23 chromosomes from his or her father in the sperm, and that each chromosome consists of a sequence of genes.

OBJECTIVES

By the end of this lesson, students will:

1. Be able to define "karyotype," and describe how a karyotype is constructed;
2. Understand that the normal human karyotype consists of 46 paired chromosomes (that is, two sets of 23 chromosomes, one set being passed on by each parent);
3. Understand that the autosomes are numbered from 1 through 22, and that the sex-chromosomes are labeled X and Y;
4. Understand that genes are sequentially arranged along these chromosomes.

MATERIALS AND ADVANCE PREPARATION

Make enough copies of the handouts "Chromosomes Contributed by the Mother in the Egg," "Chromosomes Contributed by the Father in the Sperm," "Karyotype Form" and "What are Chromosomes?," for each student in you class. These handouts are located on pages S-22, S-23, S-24 and S-25 of the "Student Manual."

Obtain a pair of scissors for each student, and have clear tape available so that your students can tape their paired chromosomes to the appropriate spaces on the "Karyotype Form." (Note: The completed karyotype is located on page 4-5 of your manual.)

LESSON 4

Prepare by reviewing the "Directions for Conducting the Activity" section. This section describes the preparation of the karyotype. If you have difficulty understanding the procedures for pairing and arranging the chromosomes, the students may also become confused.

Obtain two cans. Fill one can with 50 separate beads. Each bead in this can represents a gene. The second can will also contain 50 beads linked together in four groups of 12 to 14 beads. Each chain of beads represents a single chromosome made up of 12 to 14 genes. These two cans will be used in the demonstration described below to introduce the concept of chromosomes.

DIRECTIONS FOR CONDUCTING THE ACTIVITY

Begin by reviewing the fact that genes are passed on from parents to children at the time of conception, when the egg and sperm join. Remind students that, in most instances, a person inherits at least two genes coding for each trait. (The exception to this rule occurs in males who inherit only one copy of the genes on the X and Y chromosomes.)

Then ask your students how many genes they think a person has. If their initial predictions are low, ask them to consider how many physical characteristics a person has. The final consensus should be that there are between 50,000 and 100,000 genes in the human genome.

Next, have your students consider how their parents were able to pass on only one copy of each gene in the egg or the sperm. Your students should appreciate the fact that it would be virtually impossible to accurately separate 50,000 to 100,000 individual gene pairs. This can be dramatically demonstrated as follows:

Ask your students to imagine how unmanageable it would be if each gene were contributed to offspring independent of all other genes. Ask them to imagine 50,000 to 100,000 genes moving independently as a cell divides in two (that is, if there were no chromosomes to link large numbers of genes together into a single physical unit).

Demonstrate how unmanageable this would be by taking the can containing 50 separate beads, and upending it on the table. Your students should react as the 50 beads roll across the table and floor. Ask your students to imagine the chaos if there were 50 thousand beads, instead of just 50. Next, upend the other can which contains the same number of beads, linked together to form four "chromosomes." The four "chromosomes" should land on the tabletop without rolling all over the floor. Discuss how much easier it is for cells to divide when 50,000 to 100,000 separate genes are linked together into 23 chromosomes.

Introduce the term "chromosome" and the fact that chromosomes are passed on in the egg and the sperm at conception. Reinforce the idea that each chromosome is made up of thousands of individuals genes, and that it is the separation of the 23 chromosome pairs that greatly improves the accuracy of gene pair separation.

When it is clear to you that your students understand these concepts, divide the class into groups of three or four students so they can consult with each other as they work. Give each student a photocopy of the "Chromosomes Contributed by the Mother in the Egg" worksheet. Instruct the students to cut out and arrange these chromosomes from the longest chromosome to the shortest. After they have arranged the 23 chromosomes contributed by the egg, distribute the worksheet "Chromosomes Contributed by the Father in the Sperm." These 23 chromosomes should also be cut out and arranged from the longest to the shortest.

Continue by engaging your students in a discussion, during which the following points are mentioned:

1. Each parent contributes 23 chromosomes to his or her child. This means that each person inherits a total of 46 chromosomes. Chromosomes numbered 1 to 22 are autosomes, and the 23rd pair is the sex chromosomes.

2. Some observant students might notice that, in our example, the mother's and father's contributions are not exactly identical. The mother contributes eight medium-sized chromosomes from the C group and two very short G group chromosomes. The father, on the other hand, contributes seven medium-sized and three very short chromosomes. The extra chromosome in the C group from the mother is the X chromosome, and the extra G group chromosome from the father is the Y chromosome. These two "mis-matched" chromosomes determine the sex of the fetus.

3. Children who inherit two X chromosomes are female, while those who inherit one X and one Y chromosome are male. Lead a discussion revealing that the father's contribution determines the sex of the fetus. This discussion should bring out the following points: Both of the mother's sex chromosomes are X chromosomes, therefore, she contributes an X chromosome to all of her children; daughters and sons. The father possesses one X chromosome and one Y chromosome. Children who receive an X from their father are females (they have inherited two X chromosomes, like their mother). Children who receive a Y from their father are males (they have inherited one X and one Y chromosome, like their father). Hence, it is the father's contribution that determines the sex of a couple's children.

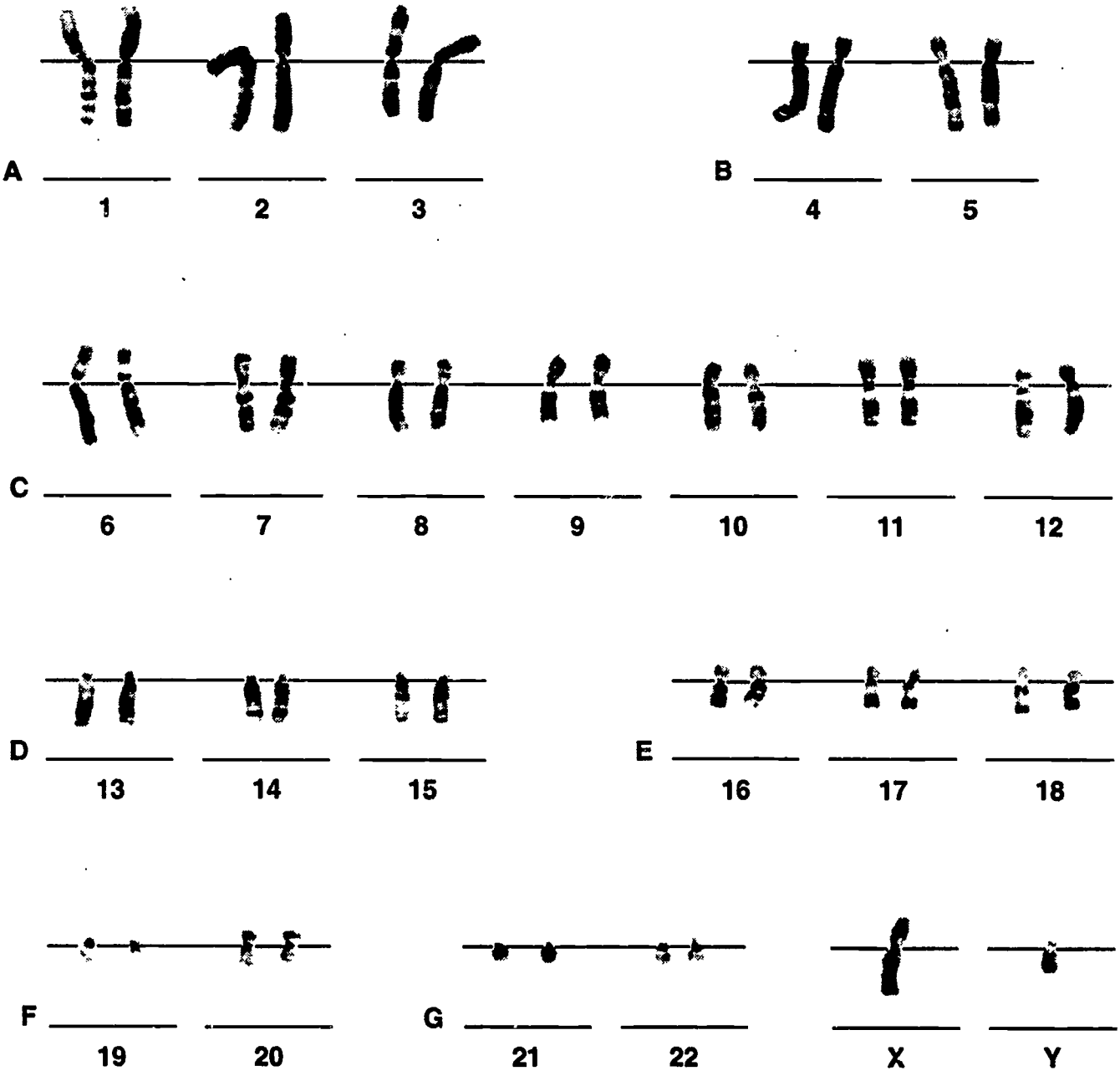
4. Students might also notice that each parent contributes chromosomes with similar banding patterns. The banding patterns on the chromosomes, the series of light and dark horizontal stripes, are not natural features of the chromosomes. They are the result of diagnostic staining procedures. Untreated chromosomes do not show this banding pattern. Furthermore, the bands are not genes. Individual genes are extremely small and are not visible through a light microscope.

After discussing these points, have students identify the chromosome pairs, and place each pair on the "Karyotype Form" handout. They should begin with the largest chromosome (presumably autosome number one) from the sperm and the largest chromosome from the egg. This pair of chromosomes should be taped on the "Karyotype Form" handout, with the

centromere, or constricted region, positioned on the line above the space labeled "1." Then they should pair the next largest chromosomes (presumably autosome number two), and tape them on the line above the space labeled "2." In this manner, the students should continue working their way down to the shortest chromosome pairs (group G, pairs 21 and 22). After they have taped these 22 pairs of chromosomes to their papers, there should be two unpaired chromosomes left over; these are the sex chromosomes and should be placed above the spaces labeled "X" and "Y." (A photograph showing the correct matching and placement of these chromosome pairs is on the following page.)

LESSON 4

CLINICAL CYTOGENETICS LABORATORY
KARYOTYPE FORM



KARYOTYPE: _____

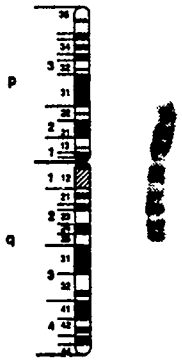
DATE: _____

As they arrange each pair of chromosomes, the students should take note of the sizes and shapes of the chromosomes in each group. Specifically:

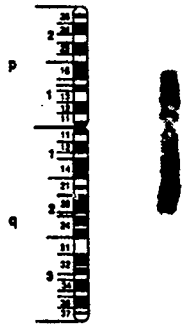
Group A: Consists of the three longest chromosomes, numbers 1 through 3. All of these chromosomes have arms nearly equal in length. The shorter of the two arms is identified with the letter "p." This notation is derived from the French word for small, "petite." The letter "q" follows "p" in the alphabet and is used to refer to the long arm of the chromosome.

Each arm of the chromosome is subdivided into as many as four major regions, each of which is designated with a number, 1 through 4. Notice, for example, that the largest chromosome, number 1, is subdivided into seven major regions, p1-p3 representing three regions on the short arm, and q1-q4 representing four regions on the long arm. A much smaller chromosome, number 15, on the other hand, is subdivided into only three regions, one region, p1, on the short arm and two regions, q1 and q2, on the long arm.

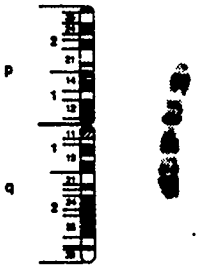
Each of these major regions is also subdivided into smaller units. For example, the terminal region of the short arm of chromosome 1 is subdivided into six smaller regions, p31-p36; the terminal region of the long arm is subdivided into four smaller regions, q41-q44.



NUMBER 1



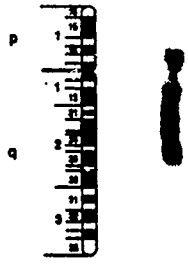
NUMBER 2



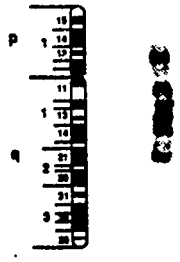
NUMBER 3

CHROMOSOME GROUP A

Group B: Consists of the next two longest chromosomes, numbers 4 and 5. Each of these possesses a long arm and a short arm. When arranging chromosomes to form a karyotype, the short arm should be above the line and the long arm should be placed below the line.



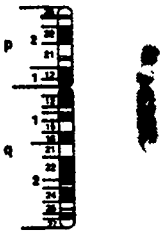
NUMBER 4



NUMBER 5

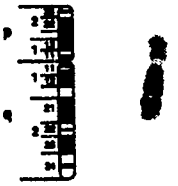
CHROMOSOME GROUP B

Group C: Consists of seven autosomal chromosomes, numbers 6 through 12. Because there are so many chromosomes in the C-group, this is usually the most difficult group for students to identify correctly.



NUMBER 6

NUMBER 7



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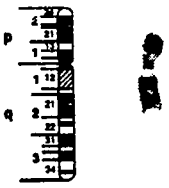
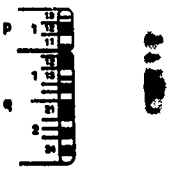


NUMBER 9

NUMBER 10

NUMBER 11



NUMBER 12

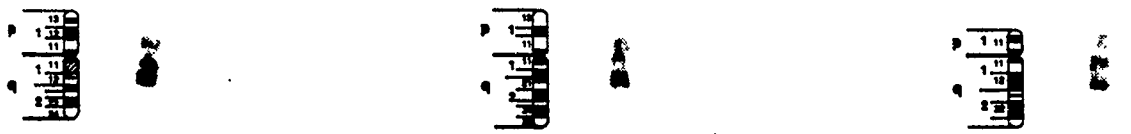
CHROMOSOME GROUP C

Group D: Consists of three medium-sized chromosomes, numbers 13 through 15. Each of these has a very long arm and a very short arm.

NUMBER 13 NUMBER 14 NUMBER 15

CHROMOSOME GROUP D

Group E: Also consists of three pairs of medium-sized autosomes. The arms of chromosome number 16 are nearly equal in length. The arms of chromosomes 17 and 18 are of unequal length.



NUMBER 16 NUMBER 17 NUMBER 18

CHROMOSOME GROUP E

Group F: Two pairs of very short chromosomes, numbers 19 and 20, having arms of nearly equal length, make up the F group of chromosomes.

NUMBER 19 NUMBER 20

CHROMOSOME GROUP F

LESSON 4

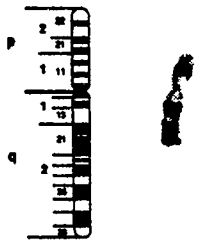
Group G: The G group chromosomes are the shortest autosomes, and, like the D group, they have a long and a very short arm.

NUMBER 21

NUMBER 22

CHROMOSOME GROUP G

Sex Chromosomes: After all of the above autosomes have been paired and taped into place, there should be two chromosomes left over. These are the sex chromosomes. The extra chromosome from the mother will be an X chromosome, about the size of a C group chromosome. The extra chromosome from the father could be an X or a Y chromosome, the Y chromosome being about the same size as a G group chromosome. In our example, the father contributes a Y chromosome. Note that all paired chromosomes are of the same size and shape, except the X and Y. These sex chromosomes are the only "mis-matched" pair of chromosomes in the karyotype. The two sex chromosomes should be taped into the spaces labeled "X" and "Y."



X CHROMOSOME

Y CHROMOSOME

SEX CHROMOSOMES

Note: Many genetic disorders are not due to errors in individual genes, but to errors in the structure or number of chromosomes passed on in the egg or the sperm. Structural chromosomal abnormalities result in the addition or deletion of multiple genes. It is the loss or gain of multiple sets of instructions that disrupts the normal development process. Several of the most common chromosomal abnormalities are discussed in Section II of Lesson 5.

Upon completion of this lesson, distribute the handout "What are Chromosomes?" to each student.

LESSON 4

BACKGROUND INFORMATION

At conception the egg and the sperm join to form a single cell with a total of 46 chromosomes. Twenty-three chromosomes come from the father in the sperm. The other 23 chromosomes are passed on by the mother in the egg. The chromosomes present in this cell are copied and the fertilized egg divides to make two identical cells. The chromosomes in these cells are copied and the two cells divide, creating four cells. This process of cell proliferation continues throughout life, and copies of the original 46 chromosomes are present in all but a few cells in the body.

Twenty-two of the 23 chromosome pairs are similar in both males and females. These chromosomes are referred to as autosomes. They have been assigned numbers based on their length and banding pattern. The longest chromosome is number 1, the shortest is number 22.

The sex chromosomes, X and Y, make up the 23rd pair of chromosomes. The X is the larger of the two sex chromosomes. A female inherits two copies of the X chromosome, one from her mother and one from her father. A male inherits one X chromosome from his mother, and a Y chromosome, with genes coding for the male sex characteristics, from his father.

A normal female karyotype is written as 46, XX. The number "46" indicates that there are 46 chromosomes present in each cell. The "XX" indicates the person is female. The normal male karyotype is 46, XY. If a person has a chromosome abnormality, the number will change to reflect the total number of chromosomes in the cell, the sex chromosomes will be recorded after the number, and the specific abnormality will be defined by a series of symbols and numbers following the sex chromosomes. For example, the karyotype of a male with an extra chromosome 21, or Down syndrome, is written as 47, XY, +21. The karyotype of a female with Turner syndrome is 45, X.

Each chromosome pair is separated when the eggs and sperm are formed. The resulting gametes contain one sex chromosome and one copy of each autosome. You cannot tell, simply by looking at the chromosomes, which chromosomes were passed on by the mother and which chromosomes came from the father.

When looking at a karyotype, it is also impossible to identify a person who is genetically predisposed to a particular disease. The genes are invisible under the microscope. New technologies, however, are being developed to study genes and their effects on development and health. These discoveries will lead to a better understanding of human development, and new treatments or cures for genetically related health problems.

THE HUMAN GENOME PROJECT

The Human Genome Project is a collaborative effort of the National Institutes of Health, The National Center for Human Genome Research and the Department of Energy. The goals of this project include identifying and mapping each gene to a specific location on a

chromosome; determining the role each gene plays in human health and development; and developing technologies that can be applied to other research endeavors[1].

Today, many geneticists are concentrating on "mapping" the human genome. As you can imagine, the human gene map grows more detailed with each passing month. Thousands of genes have been located; tens of thousands of genes remain to be found. A few examples of gene loci (gene positions) are listed below[2]:

Chromosome 1:

A gene for one form of cataracts is located on the long arm. Genes for the Rh factor and for malignant melanoma, a cancer of the skin, are located on the short arm.

Chromosome 2:

A gene coding for susceptibility to liver cancer is located on the long arm. The gene for IgG, the major type of antibody produced by the immune system, is located on the short arm.

Chromosome 3:

The gene for retinitis pigmentosa, a childhood form of blindness, is located on the long arm. A gene for a form of lung cancer is located on the short arm.

Chromosome 4:

The gene for acute lymphocytic leukemia is located on the long arm. The gene for Huntington disease, a degenerative disease of the central nervous system that affected the folk singer Woody Guthrie, is located on the short arm.

Chromosome 5:

A gene for one type of schizophrenia, and a gene that conveys susceptibility to diphtheria, are located on the long arm.

Chromosome 6:

A gene that conveys susceptibility to coronary heart disease is located on the long arm. A gene for one form of atrial septal defect (a hole in the wall between the right and left upper chambers of the heart), and several genes involved in tissue transplant rejection, known as the major histocompatibility complex, are located on the short arm.

Chromosome 7:

The gene for cystic fibrosis, a disease of the respiratory system, and one of the most common lethal birth defects among European-Americans, is located on the long arm.

Chromosome 8:

The gene for Burkitt lymphoma, a cancer of the lymphatic system that appears to be associated with a virus, is located on the long arm. A gene for one form of genetic high blood lipid levels (hyperlipoproteinemia) is located on the short arm.

Chromosome 9:

The genes for the A-B-O blood groups are located on the long arm. The gene for interferon, an antiviral protein produced by tissue cells, is located on the short arm.

Chromosome 10:

A gene for one form of leukemia is located on the long arm.

Chromosome 11:

The gene for albinism, the total lack of body pigment, is located on the long arm. The genes for insulin production and for sickle cell anemia, a disease common among Africans and African-Americans, are located on the short arm.

Chromosome 12:

A gene for an intolerance to alcohol is located on the long arm.

Chromosome 13:

Genes for some blood-clotting disorders and for another form of retinoblastoma, a childhood cancer of the eye, are located on the long arm.

Chromosome 14:

Genes for one form of leukemia and for part of the antibody molecule are located on the long arm.

Chromosome 15:

The gene for Tay-Sachs disease, a disease of the brain common among Jews from Eastern Europe, and a gene coding for a form of leukemia are located on the long arm.

Chromosome 16:

A gene for one type of cataract is located on the long arm. The gene for polycystic kidney disease is located on the short arm.

LESSON 4

Chromosome 17:

The gene for a type of growth hormone deficiency is located on the long arm. The gene coding for neurofibromatosis is located on the short arm.

Chromosome 18:

Genes for one form of cancer of the large intestine, and for a form of leukemia are located on the long arm.

Chromosome 19:

A gene coding for susceptibility to poliomyelitis is located on the long arm. A gene for an insulin-resistant form of diabetes mellitus is located on the short arm.

Chromosome 20:

A gene for a type of growth hormone deficiency is located on the long arm.

Chromosome 21:

Genes for one form of Alzheimer disease and for resistance to some flu viruses are located on the long arm.

Chromosome 22:

A gene associated with one form of leukemia is located on the long arm.

Chromosome X:

On the long arm of the X chromosome there are genes coding for a form of cleft palate, two major forms of hemophilia, several forms of color blindness and fragile X syndrome. Genes for two forms of muscular dystrophy (Duchenne and Becker) are located on the short arm.

Chromosome Y:

Genes for initiating male sexual development in the fetus are located on the long arm.

REFERENCES

1. Watson JD. The Human Genome Project: past, present and future. Science 1990;248:44.

2. McKusick VA. The human gene map. Cytogenetics and Cell Genetics 1989;51:1-1147.

HEALTH IN THE YEAR 2100

LESSON 5

WHAT CAUSES BIRTH DEFECTS?

INTRODUCTION

Every year 250,000 children in the US are born with birth defects. Some birth defects are caused by chromosome abnormalities, others by single gene disorders. Prenatal exposure to certain drugs and environmental agents can disrupt normal fetal development and cause birth defects. Abnormalities can also result from a combination of genetic and environmental factors. In most cases, however, the cause of a birth defect is unknown.

In this lesson, students will learn about the various stages of fetal development and about the causes of birth defects. They will learn that every woman has a 3 to 5% chance of having a child with a birth defect. They will also learn that some birth defects, like fetal alcohol syndrome, are preventable, and that there are steps a couple can take to improve pregnancy outcome.

GOAL

Students will demonstrate an understanding of normal fetal development, the causes of birth defects and steps a couple can take to reduce their risk of having an adverse pregnancy outcome.

OBJECTIVES

The students will be able to:

1. Explain what happens during normal fetal development;
2. Define birth defects and classify their different causes;
3. Identify environmental factors that affect normal fetal development;
4. Demonstrate an understanding of how the consumption of alcohol might affect the developing fetus;
5. Demonstrate an understanding that, although some birth defects are preventable, the majority are not;
6. List the various things a couple can do to improve their chances of giving birth to a healthy baby.

LESSON 5

MATERIALS AND ADVANCE PREPARATION

Confirm that the videotape *Fetal Development: A Nine Month Journey* has arrived and make arrangements to show it. This video is available through the South Dakota State Library Video Lending Program.

Make enough copies of the handouts, "Fetal Development: A Nine Month Journey," "All About Birth Defects," and "Critical Periods of Development," for each student. These handouts are located on pages S-26, S-27 and S-28 of the "Student Manual."

Collect articles, fact sheets and patient literature on some of the known chromosome abnormalities, single gene disorders and multifactorial traits for your classroom files; also, collect information about known teratogenic agents such as alcohol, inhalants and rubella (German measles). Fact sheets on genetic diseases and information about teratogens can be obtained from the March of Dimes and the other organizations listed at the end of this lesson.

If there are students in your school or individuals in your community with known genetic conditions or identifiable syndromes, be sure to have information about these conditions in your classroom resource materials. Your students will most likely include these conditions in their list of known birth defects.

DIRECTIONS FOR CONDUCTING THE ACTIVITY

DAY ONE

This lesson is too long to be covered in a single class period, so it is subdivided into three sections. On Day One, hand out the videotape worksheet, "Fetal Development: A Nine Month Journey," and give your students time to read and become familiar with each question. Then, show the videotape. Following the video, have students divide into small groups and answer the questions on the worksheet. When the small groups have completed their worksheets, ask each group to share its answer to at least one of the questions. Invite the class to comment on each answer. If an answer seems unclear, or is incorrect, and none of the other student groups have the correct answer, replay the videotape.

The role and responsibilities of a father prior to the birth of his child are not clearly defined in the videotape. Therefore, it will be necessary to spend a few minutes at the end of the class period going over the answers to Question 9. Your students should understand that there are many things men can do to promote normal fetal development. For instance, men can avoid exposure to certain environmental agents and chemicals like lead, x-rays, certain solvents and pesticides that can adversely affect the male reproductive system. They can stop smoking in the presence of pregnant women, as cigarette smoke increases the risk of having a low birth weight baby. Men can take responsibility for the heavy household chores and the cleaning jobs that require the use of aerosol sprays, as inhalation of fumes may be harmful to the developing fetus. By cleaning out the cat litter box, men can protect women from exposure to toxoplasmosis, an organism that can disrupt normal fetal development. A pregnant woman's

partner can also provide transportation to prenatal visits, encourage avoidance of alcohol and drugs, and promote good nutrition.

When you are satisfied that your students understand the steps involved in normal fetal development, and the role that men can play in this process, hand out the worksheet "All About Birth Defects." Ask students to be prepared to discuss their answers to these questions on Day Two.

DAY TWO

On Day Two, find out how many students answered "yes" to Question 1 on the worksheet, and how many students answered "no." If everyone in your class does not agree on the answer to this question, ask a number of students to explain how they came up with their answers. Reach a consensus before completing the class review of the worksheet.

Ask students to share their responses to Question 2. If your students have varying opinions about the percentage of babies born with birth defects, ask for volunteers to research this question when you divide the class into working groups. Encourage this volunteer group to look up information on the number of babies born with specific categories of birth defects, such as single gene disorders and chromosome abnormalities. You may want to suggest that these students talk to the resource people you have identified at the local health clinic or hospital. The clinic staff should be able to provide your students with excerpts from medical texts, or articles that will help them answer these questions.

Next, create a list of all of the birth defects your students wrote down in response to Item 3. Then, write the following causes of birth defects on the board: chromosome abnormalities, single or multiple gene disorders, exposure to known teratogens, and cause unknown. Ask your students to determine in which category each birth defect on the class list belongs.

In the "Background Information" section, you will find a list of common chromosome abnormalities, single gene defects and multifactorial traits. This is not meant to be an exhaustive list of birth defects. If a student mentions a birth defect that you are not familiar with and the cause is not immediately apparent, record this birth defect in the "cause unknown" category. It is not unreasonable to think that most of the birth defects that your students are familiar with will fall into this category. Approximately 60% of all birth defects occur for unknown reasons[1].

Once your class list of birth defects has been categorized by cause, ask your students to identify which birth defects are preventable and which birth defects are not preventable. They should conclude that those birth defects resulting from exposure to known teratogenic agents, such as alcohol, lead, infectious agents and inhalants, are preventable. Those birth defects caused by chromosome abnormalities and single or multiple gene defects should be placed in the "not preventable" category.

Next, divide your students into small groups. Have each group choose a birth defect from the list and write down all of the things the group wants to learn about this birth defect. The list of

topics might include the physical and intellectual characteristics associated with the condition, the incidence in the community, the cause of the birth defect, the risk of recurrence, or things a couple can do to decrease their chance of having a child with this particular problem. Encourage your students to use the materials available in the classroom and outside resources for their research. Each group should prepare a brief presentation on the birth defect it is researching.

DAY THREE

Some students may need an additional day to complete their research and prepare for a class presentation. Other student groups may be ready to give class presentations on Day Three. Use your discretion when planning this portion of the lesson.

DAY FOUR

Each group should make a brief presentation to the class on Day Four. Leave a few minutes at the end of each presentation for questions. If there are facts about a particular condition that were not addressed, ask the group members to elaborate on the topic. If, for instance, a group chooses to study neural tube defects and fails to mention the benefits of folic acid supplementation, ask them what they know about this research. If a student group researches Down syndrome and does not mention that the chance of having a baby with a chromosome abnormality increases with age, ask if the risk of having a child with Down syndrome is greater for a woman in her 20's or a woman in her late 30's. This might inspire someone in the class to share what they have learned about risks associated with advanced maternal age.

The groups that research birth defects caused by teratogenic agents will most likely mention at least one of the principles of teratology (refer to the "Background Information" section), in their presentations. If not, ask group members if they think every woman who is exposed to the agent in question will have a baby with an identifiable birth defect. Is there a spectrum of abnormalities seen in children who are exposed to the agent? What causes this variation in effect? Does the amount of drug or chemical a woman uses affect fetal outcome? What birth defects are seen when exposure occurs in the first trimester? Are these different from the birth defects you would expect to see if the exposure occurred later in pregnancy? Are some women more sensitive to drugs than others? What effect do you think this has on pregnancy outcome?

When each student group has completed its presentation, conclude Lesson 5 with a discussion about fetal alcohol syndrome (FAS). If none of your student groups chose to research a teratogenic agent, review the principles of teratology at this time.

Begin this discussion by asking your students to list some of the physical features and psychological problems associated with prenatal alcohol exposure. If your students are not familiar with FAS, spend time reviewing the signs and symptoms associated with alcohol-related birth defects. Point out that there is a spectrum of abnormalities associated with alcohol use during pregnancy.

LESSON 5

If your students are knowledgeable about alcohol-related birth defects, distribute the handout "Critical Periods of Development." Ask them to predict what might happen to the fetus if alcohol were consumed early in pregnancy, when the heart and facial features are forming, before a woman even knows she is pregnant. Students may predict that the child would be born with a heart defect, a cleft lip or palate, or the characteristic facial features associated with FAS. They might also predict that the use of alcohol early in pregnancy would have no effect fetal development, and in some cases they would be right. Approximately 60% of all infants born to chronic alcoholic women have no identifiable physical defects[2].

Next, have students consider what would happen if alcohol were consumed during the second or third trimester. If they are having problems generating ideas about the possible effects, ask them if the child would have a heart defect or a cleft lip. Given that these structures are formed during the first trimester, students should conclude that the consumption of alcohol later in gestation would not adversely affect the formation of these structures. Then, ask them to consider whether drinking alcohol later in pregnancy would adversely affect fetal brain development. They should conclude that the time during pregnancy when a particular exposure occurs will determine, to a large extent, what effect, if any, alcohol will have on fetal development.

The amount of alcohol a woman consumes during pregnancy must also be considered when predicting fetal outcome. It makes sense that the more alcohol a woman consumes, the more likely it is that she will have a child with alcohol-related birth defects. Begin by asking your students to consider whether a woman who does not use alcohol during pregnancy will have a child with alcohol-related birth defects. Then ask them to consider whether a woman who drinks occasionally will have a child with alcohol-related birth defects. If your students are knowledgeable about FAS/FAE, they should tell you that there is no safe level of alcohol consumption during pregnancy.

In follow-up to this discussion, ask your students to predict how many pregnant women who drink more than 2 ounces of alcohol per day will give birth to a child with FAS/FAE. If your students do not know the answer to this question, tell them that the risk is less than 50%, and ask them to propose a hypothesis to explain why the risk is not 100%. They should come to the conclusion that people are physically and genetically different. Therefore, alcohol will affect each pregnant woman and her fetus differently.

If time permits, talk about things a couple can do to improve their chances of having a healthy baby. Your class list might include planning a pregnancy, taking vitamins with folic acid prior to conception, getting immunized against rubella, seeking prenatal care early in pregnancy, and avoiding teratogenic agents (alcohol, drugs, x-rays, etc.). Your students should also mention those things a father can do to reduce the risk of an adverse pregnancy outcome.

LESSON 5

BACKGROUND INFORMATION

SECTION I: FETAL DEVELOPMENT

On average, a pregnancy lasts 40 weeks, or 280 days. This nine-month term begins with the first day of the last menstrual period, about 14 days before conception, and is divided into three 3-month periods known as trimesters. A summary of fetal development during pregnancy follows[3,4].

THE FIRST TRIMESTER

The first three months of pregnancy, from Week 1 through Week 12, is called the first trimester. This trimester is subdivided into the pre-embryonic stage, the embryonic stage, and the beginning of the fetal stage.

1. The Pre-Embryonic Stage: The ovary releases an unfertilized egg into the fallopian tube (a process called "ovulation") on the 14th day of a woman's 28-day menstrual cycle. The term "pre-embryonic" refers to events that happen between ovulation and fertilization. Fertilization of the egg by the sperm normally takes place as the egg moves down the tube toward the uterus.

2. The Embryonic Stage: The embryonic stage begins on Day 15 of the menstrual cycle (or Day 1 of pregnancy) and continues until Week 8. During this time, various cells and tissues begin to form the major internal organs and external features. During this critical period of development, embryos are extremely sensitive to teratogens (chemicals or physical agents that cause birth defects). By the end of the embryonic period, the embryo clearly has a human form.

   Week 1: By definition, fertilization occurs on Day 1 of pregnancy. Within 36 hours the fertilized one-cell egg (zygote) divides into a two-cell embryo. Continued cell division will produce a four-cell embryo at 60 hours after fertilization, an eight-cell embryo at 72 hours, and a solid ball of 120 or so cells (called the morula) by the fourth day. This is transformed to a fluid-filled ball called a blastocyst.

   By the end of Week 1, the blastocyst has traveled down the fallopian tube, entered the uterus, and begun the implantation process in the lining of the uterus. The external cells of the blastocyst will form the placenta and sac of fluid that surrounds the embryo. Only the cells inside the blastocyst will develop into the embryo.

   Week 2: Implantation continues. The embryonic placenta interacts with the lining of the uterus to establish a means of nourishing the developing embryo. Small pools of blood in the maternal tissue bathe the embryonic tissue. Oxygen and nutrients will pass from the mother's circulation into the developing embryo. By the end of Week 2, implantation is completed and fetal blood is circulating through the placenta.

Week 3: The first missed menstrual period occurs at about Day 15 of pregnancy. The neural tube and digestive tube begin to form. The most advanced organ at this early stage of development is the heart. By the end of Week 3, the placenta has grown and covers about 20% of the uterus.

Week 4: A tail bud is seen; structures which will form the lower jaw and throat are visible; the heart begins to beat; small cubes of tissue (called somites) form along both sides of the neural tube (brain and spinal cord) which will develop into the vertebrae, muscles, bones and other structures; arm buds become visible. By the end of Week 4 (the first month) the embryo is "C" shaped and now measures 4 to 5 mm (less than 1/5 of an inch) from the top of the head to the buttocks (this measurement is called the "crown-rump length").

Week 5: Depressions called optic cups (future eyes) are visible on the sides of the head; nasal pits are visible; a primitive mouth begins to form; leg buds develop; the brain subdivides into different regions. By the end of the fifth week, the crown-rump length is approximately 8 mm, or 1/3 of an inch.

Week 6: The head, arms and legs are maturing; the jaw, upper lip, palate, nostrils and ears are clearly recognizable; fingers and toes are distinct; the heart is well-formed and the liver begins to produce blood cells; the palate is developing. If the embryo is a male, a gene on the Y chromosome is switched on and the process of masculinization begins; otherwise, the embryo will continue to develop as a female. The embryo is approximately 13 mm, or 1/2 inch in length.

Week 7: The eyes are moving toward the front of the head, and the eyelids are developing; the digestive, reproductive and urinary tracts are forming. By the end of Week 7 the initial stages of all body organs and structures have appeared; the embryo has reached 18 mm, or nearly 3/4 inch in length.

Week 8: The face continues to develop; the external genitalia is developing; bones of the arms and legs are developing and some muscles are capable of contracting. By the end of the eighth week, the embryo is 30 mm, or 1 1/4 inches in length. The beginnings of all the major organ systems and structures have been established, ending the embryonic period of development.

3. Fetal Stage: The fetal period extends from the ninth week of pregnancy until birth. Fetal development generally consists of growth and differentiation of structures that have already formed during the embryonic period. Very few new structures are formed during the fetal stage of development.

Week 9: Although the genitalia continue to develop during the third month of pregnancy, differences between male and female genitalia are not

particularly obvious. The fetus moves its arms, kicks its legs, and develops a sucking reflex. The hands are well developed and fingerprints can be obtained. The head is large, about half the size of the fetus. The fetus is about 50 mm, or 2 inches in length, and weighs 8 grams (1/4 ounce).

Weeks 10-12: The eyelids close during the 10th week, and do not reopen until the 28th week. Tooth buds for all 20 deciduous (baby) teeth form. The face has a human profile, with a protruding nose, receding chin, well-formed ears and a well-defined neck. The arms are proportionate in relation to the body, but the legs are less well-developed. By the end of the 12th week it is possible to differentiate between male and female genitalia. The fetus is now 87 cm, or 3 1/2 inches in (crown-rump) length, and weighs 45 grams, or more than 1 1/2 ounces.

THE SECOND TRIMESTER

The second trimester begins at the end of the third month and continues until the end of the sixth month. The fetus increases dramatically in size. The head, which is about half the size of the fetus at the beginning of the second trimester, grows more slowly than the body, so that by the end of the second trimester, the head and body approach normal proportions.

Weeks 13-16: A period of rapid growth begins during the 13th week. Fine, fuzzy hair (called "lanugo," from the Latin word for "down") covers the body, especially the head. The skin is so transparent that the blood vessels are visible. By the end of the 16th week, the fetus measures 140 mm, or 5 1/2 inches, and weighs 200 grams, or 7 ounces.

Weeks 17-20: Rapid overall growth of the fetus continues. By the end of the 20th week, the fetus measures 190 mm or more, almost 7 2/3 inches, and weighs 460 grams, slightly greater than one pound.

Weeks 20-25: By the end of the 25th week, the fetus measures 240 mm, or 9 1/2 inches, and weighs 900 grams, or 2 pounds. This marks the end of the second trimester.

THE THIRD TRIMESTER

The third trimester begins at the end of the sixth month and continues until birth. During this last trimester, the brain becomes even more complex. The lungs and respiratory system mature. The fetus can often survive if born prematurely after the 26th week, but the death rate is high (primarily because the respiratory system is unable to function efficiently). Premature babies who weigh more than 1000 grams (about 2 1/4 pounds) stand a fair chance of surviving.

Weeks 26-29: The eyes open during this time, the head is covered with hair, and the skin is slightly wrinkled. By the end of the 29th week, the fetus measures 275 mm, or nearly 11 inches, and weighs 1500 grams, or 3 pounds.

Weeks 30-34: The skin becomes smooth, fingernails and toenails are present, and the body fills out. In boys, the testes descend from the abdomen into the scrotum. By the end of the 34th week, the fetus measures 320 mm, or 12 inches, and weighs 2500 grams, or 5 1/2 pounds.

Weeks 35-38: Growth slows down during these last four weeks of pregnancy, with male fetuses growing more rapidly than females. The fetus produces about 14 grams (about half an ounce) of fat per day. By the end of the 38th week, the fetus is 19 to 21 inches in length, and weighs 3400 grams, or 7 1/2 pounds.

SECTION II: BIRTH DEFECTS

Every pregnant woman has a 2 to 3% chance of giving birth to a child with a serious, possibly life-threatening birth defect. When minor defects, such as clubfoot, or extra fingers and toes are included, the risk of having a baby with a birth defect increases to 5%.

The term "birth defects" refers to any abnormal physical or mental conditions that are present at birth. Some birth defects occur as the result of single gene defects, multiple gene defects, or chromosome abnormalities. Other birth defects are caused by exposure to physical, chemical, or biological factors. The majority of birth defects, however, have no known cause.

It is also worth mentioning that not all genetic (inherited) diseases are obvious at birth. Some cancers, for instance, have a genetic basis and may not be clinically apparent until later in life. Other examples include genetic diseases, like Huntington disease (a progressive neurologic disease that results in mental deterioration, involuntary muscle movements and eventually death), that occur in the 4th or 5th decade of life.

SECTION III: GENETIC CAUSES OF BIRTH DEFECTS

The causes of inherited birth defects can be subdivided into three broad categories:

1. Abnormalities in the structure or number of chromosomes;
2. single gene disorders;
3. multifactorial disorders. Information about each of these categories of genetic birth defects is provided below.

1. CHROMOSOMAL ABNORMALITIES

Approximately 6% of all infants born with birth defects have chromosomal abnormalities[1]. For your convenience, we have classified the kinds of chromosomal defects as follows.

LESSON 5

ABNORMALITIES IN CHROMOSOME NUMBER

Extra sex chromosomes

The human body tolerates extra sex chromosomes (X and Y) better than extra autosomes (chromosomes 1 to 22). The most common syndromes involving extra sex chromosomes include Klinefelter syndrome (47, XXY), XYY males (47, XYY), and triple X females (47, XXX).

Extra autosomes

The body does not tolerate abnormal numbers of autosomes. One of the few exceptions to this rule involves one of the shortest autosomes, number 21. Inheriting three copies of chromosome 21 causes Down syndrome. Inheriting three copies of most other autosomal chromosomes results in a miscarriage or stillbirth.

Missing sex chromosomes

Females with Turner syndrome have only one X chromosome, instead of two. A fetus with a single Y chromosome, and no X chromosome, will be miscarried.

Missing autosomes

Embryos that inherit fewer than 44 autosomes are usually miscarried.

ABNORMALITIES IN CHROMOSOME STRUCTURE

Duplications and deletions

Many birth defects result from errors within a single chromosome. In some cases, a portion of the chromosome is present in multiple copies (duplication). In other cases, birth defects are caused by the loss of a small segment (deletion) of chromosome material.

Translocations

Chromosomes are said to be translocated when a segment of one chromosome is attached to another chromosome. During the exchange process, chromosome material can be duplicated or deleted. Single gene abnormalities can also occur if a chromosome is broken at the site of a specific gene during the exchange process.

Balanced translocations also occur. Individuals who have a balanced translocation have not lost or gained genetic material. They are, however, more likely to create gametes (eggs or sperm) with an abnormal complement of chromosome material and have miscarriages or children with birth defects.

LESSON 5

COMMON CHROMOSOME ABNORMALITIES RESULTING FROM EXTRA OR MISSING CHROMOSOMES

Down Syndrome

Down syndrome (named after the physician who first described the recognizable pattern of malformations) is the most common autosomal chromosome abnormality. Children with Down syndrome have inherited an extra copy of chromosome number 21.

The majority of individuals with Down syndrome have three separate copies of chromosome 21. This condition is referred to as trisomy 21 ("tri" means three, "somy" means bodies). The presence of this extra chromosome suggests that an error occurred in the chromosome separation process as the egg or sperm were formed. Rather than being pulled apart, the two copies of chromosome 21 were drawn to the same cell, creating a gamete with a total of 24 chromosomes. When fertilized by a gamete carrying 23 chromosomes, the resulting embryo inherited a total of 47 chromosomes. The parents of a child with trisomy 21 have a 1% chance of having a second affected child.

Three to 4% of the children with Down syndrome have a translocated chromosome. In these children, the extra copy of chromosome 21 is attached to another chromosome. If the translocation occurred sporadically, at the time the gametes were formed, the chances are low that the couple will have a second affected child. If, however, one of the parents is a balanced translocation carrier, the chance they will have a second child with Down syndrome could be as high as 15%.

One percent of the individuals with Down syndrome have a mosaic cell population. This means that some of their cells contain the normal complement of 46 chromosomes, and that other cells contain an extra copy of chromosome number 21. The chances are low that a couple who has one child with the mosaic form of Down syndrome will have a second affected child.

Regardless of how it happens, the presence of this extra chromosome disrupts normal fetal development, and individuals who inherit three copies of chromosome 21 will have some of the characteristic features associated with the diagnosis of Down syndrome. Individuals with Down syndrome tend to have a low nasal bridge, folds of skin that cover the inner corners of their eyes, up-slanting eyes, low set ears, a single crease across the palm of their hand, wide-spaced toes, and decreased muscle tone. They are more likely to have heart defects and intestinal abnormalities than other children. Individuals with Down syndrome are also moderately mentally retarded.

Note: Because of the characteristic eye folds and mental retardation associated with Down syndrome, Dr. Down coined the term "Mongolism." Because of the racial overtones, people are encouraged not to use this term.

Trisomy 13

Infants who inherit an extra copy of chromosome number 13 have trisomy 13, or Patau syndrome. Common signs of this condition include a cleft lip and palate, abnormalities of the ears, small malformed eyes, extra fingers and toes, and internal organ abnormalities. Eighteen percent of the infants born with trisomy 13 will survive the first year of life. The infants who survive are severely mentally retarded. They often have seizures and fail to thrive.

Trisomy 18

Infants with three copies of chromosome number 18 have Edward syndrome, or trisomy 18. Affected infants have multiple physical abnormalities including a small jaw, low set and malformed ears, overlapping fingers, prominent heels, decreased muscle mass and adipose tissue, a short sternum, heart defects and other internal organ abnormalities. Ninety percent of infants with this condition die within the first year of life. Those children who survive are severely mentally retarded.

Klinefelter Syndrome

Male individuals who have two X chromosomes and one Y chromosome (47, XXY) have Klinefelter syndrome. The presence of the extra X chromosome disrupts normal testicular development and function. Many individuals with this condition come to medical attention in their teens when they fail to go through normal pubertal development. In most males with Klinefelter syndrome, the testes do not produce a sufficient amount of the hormones necessary to promote development of the secondary male sex characteristics. Males with this condition are likely to have female-like breast development, an incomplete masculine body build, and sparse body hair. They tend to be taller that average and some have social and learning problems.

Sterility is the most common characteristic of Klinefelter syndrome. Adolescents and adults have normal sexual function but they cannot produce sperm and are unable to father children. Some men with Klinefelter syndrome are not diagnosed until they are seen in infertility clinics as adults.

Turner Syndrome

Females who are missing one X chromosome (45, X) have Turner syndrome. The two main characteristics associated with this condition are short stature and the absence of functional ovaries. Some individuals with Turner syndrome have a short wide neck, puffy hands and feet, up-turned nails and arms that turn out at the elbows. Heart defects and kidney abnormalities are also common in Turner syndrome.

Given that the ovaries are responsible for producing the hormones necessary for the development of secondary sexual characteristics, girls with Turner syndrome do not usually go through puberty. Without hormone replacement therapy, they have poor breast

development and generally do not menstruate. The ovaries do not produce eggs, and, as a rule, women with Turner syndrome cannot have their own biologic children.

Most women with Turner syndrome are intellectually normal. They may, however, have lower than average non-verbal skills, resulting in problems conceptualizing three dimensional objects, manipulating materials or following directions.

COMMON EXAMPLES OF PROBLEMS ASSOCIATED WITH ABNORMAL CHROMOSOME STRUCTURE

Cancer

Some inherited forms of cancer occur because of deletions of chromosome material. A deletion in the long arm of chromosome 13, for example, causes retinoblastoma, a childhood cancer of the eyes. A deletion in the short arm of chromosome 11 results in Wilms' tumor, a childhood cancer of the kidneys. One form of colorectal carcinoma, a cancer of the large intestine that usually occurs in older adults, is due to a deletion in the long arm of chromosome 18.

Cri du Chat Syndrome

Another deletion syndrome involves the loss of a portion of the short arm of chromosome number 5. This condition is called cri du chat, or cat cry syndrome. Babies who are missing the short arm of chromosome number 5 usually have a weak, high-pitched cry that sounds like a cat's meow. These babies also have characteristic facial abnormalities, a small head circumference, slow growth, poor muscle tone and mental retardation.

Translocation Down Syndrome

Translocations account for 3 to 4% of all children who are born with Down syndrome. In these children, the extra chromosome 21 is usually translocated to the top portion of a chromosome in the D group.

WHEN DO CHROMOSOME ABNORMALITIES USUALLY OCCUR?

Recall that fertilization unites a sperm and an egg, each carrying 22 autosomes and a sex chromosome. The resulting embryo inherits 46 chromosomes (44 autosomes and 2 sex chromosomes). It is not uncommon, however, for errors to occur when the eggs and sperm are formed. If the chromosome pairs do not separate properly, a gamete is created with an abnormal number of chromosomes. Embryos that inherit too many or too few chromosomes are more likely to be miscarried. Some, however, survive fetal development and birth.

Children who are born with an abnormal number of chromosomes generally have an unusual physical appearance and some degree of mental retardation. These problems are related to the fact that they have extra or missing copies of genes, not that the genes themselves are abnormal.

LESSON 5

WHAT ARE THE CHANCES OF HAVING A CHILD WITH A CHROMOSOME ABNORMALITY?

Every pregnant woman has a chance of giving birth to a child with a chromosome abnormality, regardless of her race, ethnic background or age. The chance of having a child with a chromosome abnormality, however, increases with a woman's age. A woman in her 20's, for instance, has a 1 in 500 chance of giving birth to a child with a chromosome abnormality. At 30 a woman's risk is 1 in 385, at 35--1 in 179, at 40--1 in 63, and at 45--1 in 15.

In addition to advanced maternal age, other factors can increase a woman's chance of having a child with a chromosome abnormality. Women under the age of 20, for instance, have a risk greater than 1 in 500. A couple who has already given birth to a child with a chromosome abnormality also has an increased chance of having a second affected child.

WHY ARE OLDER WOMEN MORE LIKELY TO GIVE BIRTH TO CHILDREN WITH CHROMOSOME ABNORMALITIES?

The answer to this question is unclear. Some people suggest that a woman's chance of having a child with a chromosome abnormality increases with age because her eggs are chronologically older. The eggs are formed prior to birth. Therefore, a 20-year-old woman has 20-year-old eggs, and a 40-year-old woman has 40-year-old eggs.

Eggs do not complete meiosis until they are ovulated and fertilized. The second mitotic division, which results in the formation of a mature egg with 23 chromosomes, is not completed until after fertilization. It has been suggested that because the eggs of older women are older at the time of ovulation, the mechanism that causes chromosome pair separation is more likely to be faulty.

Another hypothesis was proposed by Dr. James German to explain why older women are more likely to have babies with chromosome abnormalities. He suggests that older women are less likely than younger women to have intercourse on a regular basis and that, at the time of fertilization, the egg or sperm is old relative to the time it was ovulated, or deposited. If fertilization does not occur immediately at the time of ovulation, Dr. German suggests that the gametes begins to deteriorate thus increasing the risk of abnormal chromosome pair separation. This particular hypothesis would also explain why younger women, who are more likely to have irregular cycles and infrequent intercourse, are also more likely to have children with chromosome abnormalities.

CAN CHROMOSOME ABNORMALITIES BE PREVENTED?

There is no way to prevent the conception of a chromosomally abnormal fetus. The majority of chromosome abnormalities occur sporadically.

CAN CHROMOSOME ABNORMALITIES BE DIAGNOSED PRENATALLY?

If a woman is over the age of 35, or has given birth to a child with a chromosome abnormality, she may choose to pursue prenatal testing. A sample of fetal cells can be obtained through

chorionic villus sampling or amniocentesis. These cells are cultured in the laboratory and chromosome analysis is completed in 1 to 2 weeks.

The option of prenatal diagnosis is also offered to individuals known to carry balanced translocation chromosomes, and to women with low levels of alpha fetoprotein (AFP) in their blood. Women with low maternal serum AFP levels are more likely to be carrying a fetus with Down syndrome or another chromosome abnormality.

2. SINGLE GENE DISORDERS

There are over 3000 known single gene disorders. Some cause changes in development that are obvious in the newborn period. Other single gene disorders results in changes that occur later in life.

Approximately 7.5% of the children with recognizable birth defects have problems related to a single pair of genes[1]. Single gene disorders fall into three main categories: dominant, recessive, and X-linked disorders.

DOMINANT SINGLE GENE DISORDERS

Genes are inherited in pairs. A gene is said to be dominant if the presence of only one copy of the gene results in the expression of a particular genetic condition or physical trait. If a person has a gene coding for a dominantly inherited single gene disorder, there is a 50% chance that he or she will pass on the dominant gene and have a child with the same genetic condition.

Neurofibromatosis

One example of a dominantly inherited single gene disorder is neurofibromatosis. This condition affects approximately one in 4,000 babies. The common signs of neurofibromatosis include benign tumors referred to as neurofibromas, multiple café au lait spots, and Lisch nodules, or pigmented bumps on the iris.

The neurofibromas typically develop on, or just beneath the surface of the skin. Neurofibromas can also develop within the body. They usually begin developing around puberty and continue to form throughout life.

Café au lait spots are light brown spots that are usually present at birth. People with NF almost always have six or more café au lait spots of varying size. The number of spots may increase in childhood and occasionally later in life.

The number of neurofibromas, café au lait spots and Lisch nodules varies widely among affected individuals. There is no way to predict how many neurofibromas or café au lait spots will develop. Approximately 60% of the people with NF have a mild form of the disorder. Twenty percent have correctable problems, and 20% have more serious and persistent problems which may include seizures, mental retardation and rare malignant tumors.

Neurofibromatosis is commonly, but erroneously, called "elephant man's disease." John Merrick, whose severe malformations have been dramatized in movies and books, apparently had another condition. It is important to stress that the vast majority of people with neurofibromatosis are not malformed; indeed, their symptoms may be so mild that they are overlooked altogether.

Marfan Syndrome

Another dominantly inherited single gene disorder is Marfan syndrome, a condition resulting in the abnormal formation of connective tissue. Individuals with this syndrome are usually taller than average. Their arms and legs may be long when compared to their trunks. They may have lax ligaments, tendons and joints, and the lenses in their eyes may be displaced.

Connective tissue is also a component of the blood vessels. If this tissue is formed incorrectly it is possible that, over time, the walls of the aorta (the large artery leaving the heart) may become thin, and balloon. If the walls of the aorta tear, the aorta may rupture and cause sudden death.

If your students follow sports, they may be familiar with Marfan syndrome. Following the sudden death of the Olympic volleyball player Flo Hyman, a number of college and professional athletes were diagnosis as having Marfan syndrome. If your students choose to research this particular condition they should learn that, with close medical management, the risk of sudden death due to a ruptured aortic aneurysm can be reduced.

Familial Hypercholesterolemia

Familial hypercholesterolemia (FH) is another example of a dominantly inherited single gene disorder. Individuals with FH have a gene that disrupts the cells' ability to bind and remove low density lipoprotein (LDL) in the blood. Elevated levels of LDL and cholesterol in the blood, in turn, increase the risk of coronary heart disease.

Approximately 50% of the people who inherit the FH gene will develop signs of coronary heart disease by 50 years of age. A decrease in LDL receptors also increases the risk of a stroke and peripheral vascular disease.

RECESSIVE SINGLE GENE DISORDERS

Recessive genes are expressed only when two copies of the gene are present. In other words, a recessive trait will become apparent only when a child inherits the same recessive gene from each parent. Presumably, each person carries a single copy of four to eight recessive genes that code for serious genetic disorders. In most cases, a person will never know which recessive genes they carry unless they have a child with a recessive single gene disorder.

Some of the more common recessive disorders like PKU are evident in the newborn period. Other recessive single gene disorders are expressed later in life.

LESSON 5

Phenylketonuria

Approximately one in 20,000 Americans is born with phenylketonuria (PKU). Individuals with PKU do not produce the enzyme phenylalanine hydroxylase, which breaks down the essential amino acid, phenylalanine. If phenylalanine is not removed from the diet, it will build up in an affected individual and cause brain damage.

A blood test, done in the newborn period, identifies individuals with PKU. Babies who have PKU are placed on a special low phenylalanine diet, and it is recommended that they remain on this diet for life.

Women with PKU are at risk to have children who are mentally retarded if they do not maintain a low phenylalanine level during pregnancy. High levels of phenylalanine also increase the risk of congenital heart disease and low birth weight.

Cystic Fibrosis

Cystic fibrosis (CF) is the most common lethal genetic disease among Europeans and European-Americans. Approximately one in every 25 European-Americans (a total of 12 million people) carries the CF gene, meaning that one in 2,500 white babies are born with cystic fibrosis. The majority of individuals with CF die in their late teens or 20's, usually from recurrent lung infections. Because of a faulty enzyme, mucus in the digestive system and respiratory tract becomes extremely thick and interferes with normal functions.

While cystic fibrosis is not common in the Native American population, your students might be familiar with this disorder, given the number of articles that have appeared in the popular press following the discovery of the gene. If this condition is included by your students on the class list of birth defects, information can be obtained from the Cystic Fibrosis Foundation, referenced in the "Resource" section.

Wernicke-Korsakoff Syndrome

Wernicke-Korsakoff syndrome is another autosomal recessive single gene disorder. Individuals who inherit two copies of the gene coding for this condition produce an abnormal form of transketolase, an enzyme which binds thiamin pyrophosphate.

Individuals who inherit two copies of this gene will remain symptom-free unless they experience an episode of thiamin deficiency. Thiamin (vitamin B1) is vital to carbohydrate metabolism in the heart and central nervous system. Individuals who inherit two copies of the gene for Wernicke-Korsakoff syndrome will experience episodes of confusion, ataxia (unstable gait), and paralysis of eye movement if their diet is lacking thiamin.

In the United States, thiamin deficiency is most commonly seen in the chronic alcoholic population. Therefore, this condition is sometimes referred to as alcohol-induced encephalopathy. With treatment, the signs of vitamin deficiency are frequently reversed within a few weeks. Even with treatment, however, the mortality rate of patients with Wernicke-Korsakoff syndrome is about 15%.

X-LINKED RECESSIVE GENETIC CONDITIONS

The above diseases are caused by recessive genes located on autosomal chromosomes. In addition, several recessive disorders have been linked to single genes located on the X chromosome. They include conditions like Duchenne muscular dystrophy, fragile X syndrome, hemophilia, and some immune disorders.

Recall that females inherit two X chromosomes. Thus, women can carry an X-linked recessive gene without exhibiting signs of the disease (because the dominant normal gene on the other X chromosome produces a sufficient amount of the normal gene product). Males born to women who carry an X-linked recessive gene, however, have a 50 percent chance of inheriting the X chromosome with the abnormal recessive gene. If they inherit the recessive gene these males will develop signs of the disorder.

Fragile X Syndrome

Fragile X syndrome is one of the most common forms of inherited mental retardation (1 in 1000 males; 1 in 2000 females). The presence of a repeated DNA sequence alters the cells' ability to produce a protein commonly found in the brain. Males with fragile X syndrome are mentally retarded. They tend to have significant delays in speech and language development. They may be hyperactive, have difficulty adjusting to change, and exhibit autistic-like behaviors.

The physical characteristics associated with this syndrome are subtle. As young children, affected males may have large heads and prominent ears. Over time they may develop large jaws, broad noses, prominent foreheads and large testicles.

One third of the females who carry the fragile X gene are intellectually normal. A third of the females who carry the gene have learning problems, and a third of the carrier females are mentally retarded.

Duchenne Muscular Dystrophy

Duchenne muscular dystrophy (DMD) is a condition that affects young boys. The early signs of DMD include a tendency to fall, difficulty rising from a sitting or lying position, and a waddling gait. Another classic sign is the enlargement of the calf muscles due to an accumulation of fat and connective tissue within the muscle. Progression of the disease will vary from child to child. However, most boys with this condition are in a wheelchair by age 12, and die of respiratory problems in their twenties.

Recent studies have shown that males with Duchenne muscular dystrophy are unable to make the muscle protein, dystrophin. The gene coding for this protein is located on the X chromosome.

3. MULTIFACTORIAL DISORDERS

Approximately 20% of all birth defects are inherited as multifactorial traits[1]. A multifactorial trait is a trait that is determined by a combination of factors, both genetic and non-genetic. Each factor has a minor and an additive effect on the trait. Multifactorial inheritance is believed to account for much of the normal variation in families. It also accounts for a number of common disorders.

Multifactorial disorders tend to cluster in families, but they do not show any particular genetic pattern. Disorders inherited in this manner include neural tube defects, cleft lip and palate, congenital hip dislocation, and various heart defects, to name a few.

Neural Tube Defects

The incidence of neural tube defects in the general population is 1 per 1000 live births. Neural tube defects occur when the neural tube fails to develop and fuse properly between the 18th and 27th day of pregnancy. If the top third of the neural tube does not form and fuse properly, the skull and brain will not form correctly, and an infant will be born with anencephaly. Most anencephalic infants are stillborn or die within the first few hours of life.

If there is a defect in the lower two thirds of the neural tube, a child will be born with spina bifida. Individuals with spina bifida have abnormalities characterized by defective formation of the vertebra. The degree of problems observed in individuals with spina bifida depends on the location of the defect; whether the lesion is covered or uncovered by skin; and whether a portion of the spinal cord protrudes from the open spine.

No matter where the lesion is, almost all children with spina bifida will have some degree of paralysis of the legs and problems controlling bowel and bladder functions. Approximately 90% of children with spina bifida will also have hydrocephalus, or an accumulation of fluid in the brain.

A couple who has had one child with an isolated neural tube defect has a 4% chance of having a second affected child. Studies have shown that this risk can be decreased by as much as 70% if 4.0 milligrams of folic acid is taken daily prior to conception[5,6]. Vitamin supplementation has also been shown to be effective in reducing the risk of neural tube defects in the general population. In September of 1992, the US Public Health Service issued a statement recommending that every woman of childbearing age consume 0.4 mg of folic acid per day to reduce her risk of having a child with a neural tube defect[7].

A prenatal screening test for neural tube defects is also available. This test involves measuring the level of a fetal protein (alpha fetoprotein) in the blood of a pregnant woman between the 15th and 18th week of pregnancy. If the level of alpha fetoprotein is high, further testing is recommended to rule out a neural tube defect. This particular test will identify 85% of the fetuses with open neural tube defects.

A low maternal serum alpha fetoprotein (MSAFP) value is associated with Down syndrome. Women with low MSAFP values are offered the option of amniocentesis.

Cleft Lip and Palate

Cleft lip and palate are of particular interest because these defects occur more frequently in Native Americans than among other ethnic groups. The incidence of clefting in the Native American population is 3.6 in 1,000 births, while the incidence in the European-American population is one in 1,000 births[8].

Clefting occurs in the fetus between the 5th and 7th week after fertilization, when the upper lip and palate are forming. If an individual has a child with an isolated cleft lip or palate, his or her chance of having another affected child is increased to 3 to 5%. The more affected family members a person has, the greater his or her risk of having a child with a cleft.

SECTION IV: NON-GENETIC CAUSES OF BIRTH DEFECTS

INTRODUCTION

Fetal development is a complicated and delicately balanced series of interactions. By now, it should be clear that certain events can occur during pregnancy which disrupt normal fetal development. Disruptive events may have no obvious effects, or they may cause mild, moderate or severe consequences. In this section, we will examine some effects of non-genetic factors on fetal development.

The science of teratology is replete with technical terminology. A short list of definitions is given here. We are not suggesting that your students memorize these definitions; we are simply providing this list for your convenience.

Birth defect:	Any abnormality present at birth, or biochemical abnormality manifest at or near the time of delivery.
Genetic disorder:	Any problem determined by genes. Not to be confused with congenital anomalies.
Congenital Anomaly:	The same as birth defect.
Teratogen:	Any agent that interrupts normal fetal development and causes the production of abnormalities.
Carcinogen:	Any agent that causes cancer; many carcinogens are also teratogens.
Mutagen:	Any agent that causes a change (or mutation) in the structure of genes or chromosomes; many mutagens are also teratogens.

Radiation: When applied to molecules, radiation refers to atoms giving off subatomic particles, such as electrons, protons, etc. "Ionizing radiation" refers to subatomic particles that contain so much energy that they strip off subatomic particles from other atoms.

WHAT ARE TERATOGENS?

Teratogens are agents that interfere with normal fetal development. The list of teratogens includes chemicals, drugs, radiation and infectious agents. Exposure to teratogenic agents can result in a variety of reproductive outcomes including spontaneous abortions, stillbirths, prematurity, congenital defects, growth retardation and functional disabilities. The effects of exposure, however, depend on the properties of the agent, the dose and timing of exposure, and the genetic susceptibility of the fetus[9].

THE PRINCIPLES OF TERATOLOGY

Timing of Exposure

Tissues and organs are not always vulnerable to teratogens at all times during embryonic and fetal development. They are most sensitive to disruption and damage during times of rapid cell division and tissue growth. Therefore, the potential effects of exposure to a teratogenic agent depends on when, during pregnancy, the exposure occurred.

1. The "All-or-None Period" refers to the first two weeks of pregnancy (after fertilization and before the placenta is functional). Exposure to a teratogen at this time will either damage the embryo so severely that a miscarriage occurs, or it will only affect a few cells, and the embryo will recover and continue to develop normally.

   Your students might wonder why there is no "intermediate" level of damage at this stage of development. Some reasons follow:

   a. Until the placenta is functional, there is no direct contact between the maternal circulation and the embryonic cells. The embryo is not directly exposed to chemicals circulating in the mother's bloodstream.

   b. During these first weeks of development, the embryonic cells are undifferentiated. Every cell has the potential to mature into any tissue. The death, then, of one or two cells will not result in the abnormal development of specific tissues or organ systems. This type of recovery is not possible later in fetal development. For example, if the fetal brain cells are damaged, other cells are too specialized to replace the damaged cells.

2. Organogenesis (the period of organ development) is recognized as the period during which the embryo is most sensitive to the teratogenic effects of toxic agents. During this period (Weeks 2 - 8), embryonic cells become highly specialized. They are "programmed" to take on specific roles, and they begin to group together to form organs. The central nervous system is the first organ system to develop, followed by

the heart, ears, eyes, arms and legs. The following are the most critical weeks, following conception, for the development of various organs and structures:

Heart	3.5 to 6.5 weeks
Ears	4 to 10 weeks
Eyes	4.5 to 8 weeks
Arms and Legs	4.5 to 7 weeks
Teeth and Palate	7 to 8 weeks
External genitalia	7 to 11 weeks[10]

The embryonic period ends between 55 and 60 days after fertilization. By this time, the external structures are recognizably human, and most organs and structures have formed. The major exceptions are the external genitalia and the brain. Although it is the first organ to develop, the central nervous system is the last to mature. Maturation continues throughout the first year of life.

3. Following organogenesis is a period of tremendous growth in both size and weight. During this period, the organs mature and begin to function. The most common feature of exposure to teratogenic agents during the later half of the fetal period is a reduction in the size of the fetus and the fetal organs.

Dose Response

If an agent is teratogenic, the incidence of adverse pregnancy outcomes will increase as the dose increases. In animal models, virtually all teratogenic dose-response curves display a threshold. Doses just below the threshold will cause no observable adverse effects. For every teratogenic agent, there is also an amount above which no fetus can survive. Therefore, it is important to know how much of the agent a fetus has been exposed to when predicting possible pregnancy outcomes.

Genetic Susceptibility

The type and severity of abnormalities caused by a teratogenic agent are also dependent on the genotypes and phenotypes of the pregnant woman and the fetus. For example, the way a woman's body metabolizes a particular drug will determine what metabolites the fetus is exposed to and the duration of exposure. Differences in placental membranes, placental transport, and biotransformation will affect fetal exposure. The genetic susceptibility of the fetus to a particular teratogenic agent will also have an effect on pregnancy outcome.

LESSON 5

WHICH AGENTS ARE TERATOGENIC?

Many agents are suspected teratogens, but few have been proven conclusively to cause birth defects. Known or suspected teratogens include environmental chemicals, radiation, infectious agents, physical agents, drugs and maternal conditions.

The following is a list of a few known or suspected teratogenic agents. For information about other agents, refer to the "Resources" section at the end of this lesson.

Environmental Chemicals

The federal government and many private organizations are investigating the possible harmful effects of environmental chemicals, such as lead, copper, mercury, pesticides and chlorinated hydrocarbons. Many of these chemicals cause mutations and cancer, as well as birth defects; consequently, everyone, not just pregnant women, should take appropriate precautions when contact with these agents is unavoidable[11].

Radiation

"Ionizing radiation" refers to the emission of high-energy particles from atoms. This activity is called "ionizing" because the emissions possess enough energy to tear electrons away from other atoms. Such disruption in living cells can cause gene mutations, chromosomal damage, and other damage that can lead to birth defects[12].

Radiation is a known teratogen. In women treated for malignancies during pregnancy, radiation therapy has caused microcephaly, mental retardation, and physical malformations in exposed fetuses. No definitive proof is available, however, that congenital malformations have been caused by the low doses of radiation used in diagnostic X-rays, or emitted from video display terminals. It is also unlikely that exposure to radon causes birth defects[11].

Infectious Agents

Certain microorganisms that infect a pregnant woman can cross the placenta and adversely affect fetal development. Some common examples of these infectious diseases include:

1. Rubella, or German measles, is a viral infection. Over two-thirds of the women who have rubella have some evidence of infection. The signs of rubella include a discrete rash that begins on the face and spreads, enlarged lymph nodes, a slight fever, headache and fatigue.

   The possible effects of an infection include a miscarriage, a stillbirth, or the birth of a child with multiple birth defects. The more common birth defects associated with a prenatal rubella infection include eye defects, heart defects, deafness, growth retardation and mental retardation.

Keep in mind that the risk to the unborn baby will vary based on the time of the rubella infection. Approximately 85% of the babies born to women who have an infection during the first eight weeks of pregnancy will have detectable birth defects. Between weeks nine and twelve, only 52% of the exposed fetuses will have birth defects. If a woman has an infection between the thirteenth and twentieth weeks after fertilization, the risk to the fetus is only 16%[13].

It is also important to note that the birth of a baby with congenital rubella is preventable. A woman who is vaccinated prior to conception will not become infected during pregnancy and will not put her fetus at risk.

2. *Toxoplasma gondii* is a protozoan (a single-celled animal). It is transmitted to humans in raw meat and the feces of infected cats. The incidence of maternal toxoplasmosis during pregnancy is 0.5%. If it is acquired in the first trimester, the infection is transmitted to the fetus 15% of the time. Twenty five percent of the fetuses exposed in the second trimester are infected. In the third trimester, the risk of fetal infection is 60%[13].

   The signs of congenital toxoplasmosis are likely to be most severe when the infection occurs in the first trimester. The effects of a prenatal infection, however, are not always immediately apparent at birth. Only 20% of the newborns with congenital toxoplasmosis have clinically recognizable signs at birth. They may include a small head size, convulsions, eye abnormalities, a rash, an enlarged liver and spleen, jaundice, a fever and inflammation of the lungs[13].

   To prevent congenital toxoplasmosis, pregnant women should avoid eating meat that has not been thoroughly cooked or thoroughly frozen. They should also avoid contact with infected cats, and exposure to soil or litter that may contain infectious feces[13].

3. Human immunodeficiency virus (HIV) causes AIDS (acquired immunodeficiency syndrome). Women who are infected, whether symptomatic or not, can transmit the infection to their children in utero, at delivery, or by breast feeding. Evidence accumulated so far indicates that less than 50% of the babies born to women infected with HIV will develop AIDS. About 50% of those are diagnosed as having AIDS by 9 months, 80% are diagnosed by two years, and 85% develop AIDS by 3 years of age[13].

   Signs of AIDS in babies can include failure to thrive, an enlarged liver or spleen, enlarged lymph glands, pneumonia, fever and diarrhea. In addition, infected babies are extremely vulnerable to viral and bacterial infections, and commonly die within 11 months after diagnosis.

Drugs and Medications

Any chemical that is ingested, inhaled, absorbed or injected into the mother can cross the placenta, enter the bloodstream of the embryo or fetus, and interfere with normal development. Information about some of the more common exposures is provided below.

LESSON 5

1. The effects of alcohol on the developing fetus are described in detail in "Section V: Fetal Alcohol Syndrome and Fetal Alcohol Effects."

2. Caffeine is a stimulant found in coffee, tea, chocolate, cocoa, soft drinks and over 200 over-the-counter medications. While caffeine does cross the human placenta, prenatal exposure to caffeine is not thought to cause birth defects.

   Concern about caffeine developed following reports that caffeine increased the risk of low birth weight, skeletal abnormalities and other birth defects in rats[14,15]. The fact that rats metabolize caffeine differently than humans, that the dose of caffeine used was equivalent to 80 cups of coffee, and that the caffeine was injected rather that consumed, makes it impossible to draw conclusions about risks to humans from this study.

   The results of human studies are inconsistent. Some studies suggest that caffeine consumption increases the risk of miscarriage, stillbirth and premature birth. Other studies fail to confirm these findings. While there are no data to suggest that modest amounts of caffeine adversely affects fetal development, decreasing caffeine consumption may be the safest option[16].

3. Women who smoke are more likely to give birth to children with low birth weights; and a low birth weight increases the risk for medical complications. The rate of premature births is two times higher in women who smoke. They are more likely to have placental complications and bleeding disorders. The risk of a miscarriage is 30% to 70% higher among pregnant smokers. There is also an increased incidence of stillbirths and infant deaths in the smoking population. Women who smoke more than a pack per day are 36% more likely to give birth to children who die in infancy[9].

   The risks associated with smoking during pregnancy can be reduced, or even eliminated, if cigarette smoking is discontinued. Therefore, pregnant women should be encouraged to reduce the number of cigarettes they smoke, or quit smoking altogether.

4. Inhalation of paint products and solvents is not recommended during pregnancy. Pregnant women who use large amounts of these substances for recreational purposes may give birth to children with problems similar to those seen in fetal alcohol syndrome. Three children born to women who abused paint thinner during pregnancy had small heads, short palpebral fissures, small midfaces, low-set ears, small chins, flattened fingertips with small fingernails, decreased muscle tone, and increased reflexes[17]. In a second report, three of the five infants born to women who were sniffing toluene-containing spray paints during pregnancy were growth-retarded. Other reported anomalies included malformed ears, a heart defect, a small chin and kidney abnormalities[18].

Maternal Conditions

There are a number of maternal conditions that can affect fetal development:

1. The risks associated with maternal PKU were discussed in the section on single recessive gene defects.

2. The incidence of congenital anomalies in children born to diabetic women is 2 to 4 times that of the general population. Abnormalities occur in the spine, lower limbs, heart, kidneys and genitourinary system. The incidence of neural tube defects is approximately 2%. Diabetic women are 200 times more likely to have infants with the caudal regression sequence, the incomplete development of the lower back leading to small abnormal lower limbs and neurologic problems. Heart defects include transposition of the great vessels, ventricular septal defects, and coarctation of the aorta. Tracheoesophageal fistula, bowel atresia, imperforate anus and a narrowed colon have also been described[9].

   Babies born to diabetic mothers are more likely to weigh over 10 pounds. There is an increased incidence of respiratory distress syndrome, hypoglycemia, hypocalcemia, erythema and high amounts of bilirubin. The incidence of miscarriages and stillbirths is also increased.

   Most of the major malformations arise prior to the 7th week of gestation. The most convincing evidence suggests that high blood sugar levels are largely responsible for the poor pregnancy outcomes observed in insulin dependent diabetic women. By measuring the level of glycosylated-hemoglobin, researchers demonstrated that women whose diabetes is poorly controlled during the first trimester are more likely to give birth to infants with abnormalities. This suggests that diabetic women who are able to control their blood glucose levels can decrease their risk of having babies with birth defects[9].

SECTION V: FETAL ALCOHOL SYNDROME AND FETAL ALCOHOL EFFECTS

WHAT ARE FAS AND FAE?

Fetal alcohol syndrome (FAS) refers to a lifelong disability caused by prenatal exposure to alcohol. The three main characteristics of FAS include growth retardation, characteristic physical abnormalities and impaired intellectual abilities. Individuals with fetal alcohol effects (FAE), are less severely affected than individuals with FAS. The characteristics commonly associated with FAE include learning disabilities, behavior problems and subtle physical abnormalities.

Alcohol-related birth defects occur in all races and ethnic groups, and among all socioeconomic classes. Between 3,800 and 7,600 babies are born with FAS in the US each year. This amounts to a rate of one or two FAS babies per 1,000 live births[19]. The incidence of FAE is 1 in 300 nationwide. The incidence of FAS among Native Americans, however, is estimated to be anywhere from 4 to 26 per 1,000 live births[2].

LESSON 5

WHAT ARE THE SYMPTOMS OF FETAL ALCOHOL SYNDROME?

Although the features of FAS vary from one individual to the another, there is a general pattern of growth retardation, characteristic physical features and intellectual impairment that is common to all affected children.

Central Nervous System Function

As infants, it is not unusual for individuals with FAS to have poor sucking and swallowing abilities, a hyperactive startle response, and a history of frequent hospitalizations for failure to thrive. As they grow, developmental delays may become apparent. Children with FAS are hyperactive and distractible. They may have difficulty grasping abstract concepts such as time and making change.

Lifelong problems associated with FAS include poor impulse control, poor judgment, an inability to learn from past experiences, difficulty solving problems, and poor adaptation to change. The average IQ of individuals with FAS is 70. Socially, affected individuals tend to function at the 7 to 9 year old age level. Individuals with FAS who are moderately or severely mentally retarded require supervised living situations as adults.

As a rule, children born to social drinkers are not mentally retarded. They may, however, have decreased IQ's and academic abilities, especially in the area of math. They may also have attention problems, memory problems, and learning disabilities.

Growth Deficiencies

Children with FAS experience both pre- and postnatal growth retardation. Even with proper nutrition, they are shorter and weigh less than other children their age. For example, a five-year-old boy with FAS may have the height of a three-year-old and the weight of a two-year-old. Many doctors refer to this below-average growth as "failure to thrive."

Facial Characteristics

The facial characteristics associated with FAS include small, wide spaced eyes, a low nasal bridge and flattened facial profile, a short nose with upturned nostrils, a wide, smooth philtrum, and a thin upper lip. An affected individual may also have a small head circumference, a small jaw and low set or malformed ears. Approximately 7% of individuals with FAS are born with a cleft lip or palate.

Other Physical Characteristics

Some infants with FAS are born with heart defects, such as holes in the wall between the heart chambers, and defective heart valves. In some cases, these heart defects cause critical health problems.

Other malformations include abnormal joints, patches of lighter or darker areas of skin, excessive hair growth on the face and body, and abnormalities of the kidneys, the liver, and other internal organs.

You should be aware that these physical characteristics are not due exclusively to prenatal alcohol exposure. Most of these birth defects can be caused by factors other than drinking alcohol during pregnancy.

HOW MUCH ALCOHOL CAN A PREGNANT WOMAN SAFELY DRINK?

Alcohol is absorbed by the digestive tract. It goes directly into the bloodstream and from there travels across the placenta into the fetal circulation. The more a pregnant woman drinks, the more likely it is she will have a child with alcohol-related birth defects.

There is no "safe amount" of alcohol during pregnancy. Any alcohol consumed, no matter how small the amount, will enter the fetal bloodstream within minutes. If a woman is a chronic drinker, alcohol will be present in the fetal circulation for days or weeks.

RESOURCES

There are many support groups and organizations that have educational materials about specific birth defects. The most familiar national organization is the March of Dimes. The Alliance of Genetic Support Groups also has information about many of the known genetic disorders, and they will accept calls for information from teachers using this text. For information about teratogens, refer to Christine Kelley-Buchanan's book, *Peace of Mind During Pregnancy.*

March of Dimes Birth Defects Foundation
PO Box 1657
Wilkes-Barre, PA 18703
Phone: 1-800-367-6630

March of Dimes Office/ Sioux Falls
2600 S. Minnesota, Suite 200
Sioux Falls, SD 57105
Phone: 1-800-275-1021

Alliance of Genetic Support Groups
35 Wisconsin Circle, Suite 440
Chevey Chase, MD 20815
Phone: 1-800-336-GENE

National Marfan Foundation
382 Main Street
Port Washington, New York 11050
Phone: 516-883-8712

The National Fragile X Foundation
1441 York Street, Suite 215
Denver, Colorado 80203
Phone: 1-800-688-8765

National Down Syndrome Congress
1605 Chantilly Drive, Suite 250
Atlanta, Georgia 30324-3269
Phone: 1-800-232-NDSC

National Neurofibromatosis Foundation, Inc.
141 Fifth Avenue, Suite 7-S
New York, New York 10010
Phone: 1-800-323-7938

Cystic Fibrosis Foundation
6931 Arlington Road
Bethesda, Maryland 20814
Phone: 301-951-4422 or 1-800-FIGHT-CF

American Cleft Palate Association/
The Cleft Palate Foundation
1218 Grandview Avenue
Pittsburgh, Pennsylvania 15211
Phone: 1-800-24-CLEFT

American Heart Association
Dakota Affiliate, Inc.
1005 12th Ave. SE
Box 1287
Jamestown, ND 58402-1287
Phone: 701-252-5122

Birth Defects Genetics Center
USD School of Medicine
314 Julian Hall
414 East Clark Street
Vermillion, SD 57069
Phone: 1-800-962-1642

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HEALTH IN THE YEAR 2100

LESSON 6

THE KINSHIP SYSTEM AND MARRIAGE

INTRODUCTION

In this lesson, students will talk about the traditional kinship systems of the Lakota, Dakota and Nakota people, and about how marriage partners were selected in the past. Students will suggest hypotheses to explain why the rules governing the selection of marriage partners were developed. They will learn that related individuals are more likely to carry similar recessive genes and give birth to children with recessive genetic conditions. Students will also learn that marriage to people outside the kinship group improved the chances of survival, and prevented disruption of the social relationships within the kinship group.

GOAL

Students will demonstrate an understanding of the traditional kinship system, and why rules governing the selection of a marriage partner may have been developed.

OBJECTIVES

The students will:

1. Listen to a traditional member of the community speak about kinship belief systems;
2. Be able to explain that people who are related are more likely to have genes coding for the same traits or conditions, and are at an increased risk of having children with birth defects;
3. Discuss why they should respect traditional beliefs;
4. Discuss the consequences of not respecting traditional beliefs.

MATERIALS AND ADVANCE PREPARATION

Identify a member of the community, preferably an elder, who is willing to share information about the traditional kinship system and about how marriage partners were chosen. Ask this person to talk about how and why the kinship system evolved, and to share the Iktomi stories about incestuous relationships. You may also want to ask your speaker if you can videotape the presentation. This will provide you with an in-class resource for future use.

Confirm the appearance of your speaker prior to beginning this lesson. If you have difficulty finding a local resource person to talk to your students, visit with members of the community who work at your school, your school administrators, or staff members from your community college or from the Institute of American Indian Studies at the University of South Dakota (605) 677-5209. You may also want to contact the South Dakota Humanities Council. The Council has a list of native resource speakers, and will usually fund speaker appearances.

The Council's office phone number and address are (605) 688-6113, Box 7050, University Station, Brookings, SD 57007.

Copy the worksheet, "Understanding the Kinship System." Make enough copies for each student and small group. Make copies of the handout, "Tiospaye Kinship System," to distribute at the end of this lesson. The worksheet and the handout are located on pages S-29, and S-30 of the "Student Manual."

DIRECTIONS FOR CONDUCTING THE ACTIVITY

DAY ONE

This lesson requires two days to complete. On Day One, students will listen to a speaker talk about the traditional kinship system and the selection of marriage partners.

If a traditional elder is asked to speak, encourage your students to listen rather than ask questions, as it is usually inappropriate for young people to ask direct questions of an elder. Traditionally, young people were taught to listen and not ask questions. Even today, it is often considered rude and inappropriate to ask direct questions of others.

If a non-traditional speaker is asked to make this presentation, encourage your students to ask questions. Some students may wonder how the kinship system corresponds with genealogy. Others might want to know what happens if kinship beliefs are disregarded in traditional societies.

At the conclusion of the presentation, distribute the worksheet "Understanding the Kinship System." Encourage your students to visit with their parents and relatives about the answers to these questions.

DAY TWO

On Day Two, break students into small groups and hand out one copy of the worksheet "Understanding the Kinship System" to each group. When it appears that the groups have completed the worksheet, ask each group to share its answer to at least one of the questions. Ask the other groups if they agree with the answer that was given. If there is no consensus, identify volunteers to research the question and present the answer to the class on the following day.

There are a number of possible answers to Question 6. Some students might recognize the importance of expanding the family support system by marrying people outside their own kinship. There might also be an economic gain if one marries into a family that has access to different resources. Some students might also mention the emotional disruption caused by incestuous relationships as another reason for these social rules. If this subject is brought up, handle the resulting discussion with care.

If your students have a difficult time answering Question 7, ask them how many genes they have in common with their parents. From the previous lessons, it should be clear that children inherit half their genes from their mother and half their genes from their father.

Then ask them how many genes they share in common with their biologic aunts and uncles. They should reason that brothers and sisters share one half of their genes; therefore, half of their parents' genes are similar to their aunts' and uncles' genes. Your students have one-half of their parents' genes. The number of genes, then, that they share in common with their aunts and uncles is 1/2 X 1/2, or 1/4.

Your students do not have to know how to calculate the coefficient of inbreeding to answer this question. They do, however, need to understand that people who are closely related are more likely to carry similar recessive genes. When they understand this concept, they should then reason that the risk of having children with birth defects increases for couples who are more closely related.

At the end of this lesson distribute the handout, "Tiospaye Kinship System."

BACKGROUND INFORMATION

Throughout history, social institutions have been created to guarantee the survival of each individual within the group. Cooperative living, however, requires an elaborate system of rules and regulations governing the social behavior of each group member in order to keep friction and strife at a minimum. The people of this region developed strategies for communal living that involved the creation of an intricate kinship system.

The kinship group, or *tiospaye*, was an all-inclusive system that provided rewards and obligations for every member of the group. Membership in a *tiospaye* implied that the individual would abide by the established practices of the group and maintain a specific attitude toward the other group members. Members shared common goals. They were duty-bound to assist each other and place the group's interests and needs above their own.

Observance of the kinship rules and the rules for being a good relative were the ultimate goal of the members of the group. The kinship system provided a discipline for group living. There were occasional violations of the rules, but the sanctions were severe enough to keep serious violations minimal. Individuals who did not conform to the group norms were subject to severe criticism, ridicule and gossip. This might eventually drive them from the group. In other instances, offending individuals were exiled from the kinship group.

Outside of the *Hunka* ceremony, one of the seven sacred rites of the Lakota, the kinship group consisted of only those individuals who were related by blood or marriage. With the *Hunka* ceremony, an addition to the family was made which increased the size of the *tiospaye*. Those who were made *Hunka* became related. Such an arrangement created a permanent relationship among the individuals and families involved.

As a child, every person was taught to know to whom he or she was related, and how to interact appropriately with each member of the *tiospaye*. Children learned there were certain

levels of communication between people of different ages and of the opposite sex, and that these protocols were based on respect and honor. They were taught that sisters and brothers did not talk directly to one another after reaching a certain age. They learned that, to show respect, fathers-in-law should never talk to or be in the presence of daughters-in-law, and mothers-in-law should never talk to or be in the presence of their sons-in-law. They also learned to address others using kinship terms and not formal names. The kinship terms indicate a person's sex, birth order and relationship to others in the family. These terms are still used by many older people today.

Members of a kinship group camped, lived, and worked together throughout the year, and were often referred to as a "band" or "camp circle." The group might, at certain times of the year, camp with other bands, especially those with whom they had friendly and cooperative relations because of intermarriage, trade and geographic proximity.

As in most cultures, there were strict rules governing the selection of a marriage partner. It was a commonly accepted practice that a person should not marry anyone in his or her own kinship. If, by chance, a person was unable to properly identify others in relationship terms, he or she could ask the elders of the group for assistance. Each *tiospaye* had elders who were astute genealogists. Elders were highly proficient in keeping track of relationships. It was said jokingly that before one became attracted to a member of the opposite sex, they should consult with Grandma to make sure the potential mate was not a relative.

THE GENETICS OF INBREEDING

Throughout history, most cultures have developed rules prohibiting the marriage of genetically related individuals. One reason for these social regulations has been concern for the health of the family, both physical and mental. Experience has shown that children born to blood relatives are more likely to have birth defects than children born to unrelated couples.

WHY ARE THE CHILDREN BORN TO RELATED INDIVIDUALS MORE LIKELY TO HAVE BIRTH DEFECTS?

To answer this question, it is necessary to review some basic genetic concepts:

1. Presumably, each person carries four to eight recessive genes that code for serious genetic conditions, and a copy of the normal, dominant gene.

2. A person can pass on only one gene coding for each trait to his or her child.

3. A person who has one copy of the recessive gene and one copy of the normal, dominant gene has a 50% chance of passing on the recessive gene with each pregnancy.

4. If each parent carries the same recessive gene, for each pregnancy there is a:

    a. 25% chance that their child will inherit both dominant genes. This child will not develop the disease, nor is he or she at risk to pass on the recessive gene.

b. 25% chance that their child will inherit both copies of the recessive gene and will develop the disease. If this child is able to reproduce, all of his or her children will inherit one copy of the recessive gene.

c. 50% chance that their child will inherit one copy of the recessive gene and one copy of the normal dominant gene. This child will "carry" the recessive gene like his or her parents, but will not develop the disease. He or she will also have a 50% chance of passing on the recessive gene.

Looking at this another way, there is a 75% chance that a child born to parents who carry the same recessive gene will be normal, and a 25% chance that the child will develop the disease. If the child is physically normal, there is a two thirds chance that he or she will carry one copy of the recessive gene just like his or her parents.

IF TWO PEOPLE ARE RELATED, WHAT ARE THEIR CHANCES OF HAVING A CHILD WITH A SPECIFIC RECESSIVE GENETIC CONDITION?

Although it makes sense that having children with someone you are related to increases your risk of having children with two copies of the same recessive gene, the answer to this question is not simple. The exact risk depends on how closely related the parents are to one another, and the frequency of the recessive gene in the community. People who are closely related by blood share more genes in common than individuals who are more distantly related. All children , for example, inherit half of their genes from their mother, and half of their genes from their father. Brothers and sisters also share one half of their genes.

"Second degree relatives" is a phrase used to describe a person's biological aunts, uncles, and half siblings (children who have only one biological parent in common). On average, a person shares one out of four genes in common with a second degree relative.

"First cousins" is a European-American term referring to the children of a person's aunts and uncles (the children of a person's parents' brothers and sisters). In this context, first cousins are third degree relatives. The number of genes a person shares in common with a first cousin is one out of eight. If, for example, a man chooses to have children with his first cousin (such as the daughter of his father's brother), there is a one-eighth chance that the mother of this child will pass on the same genes. For any gene the father gives to his child, then, the chance that the mother has the same gene and will transmit it is 1/8 X 1/2 or 1/16 (6.25%). This is the coefficient of inbreeding for first cousins, and equals the probability that an individual has received the same gene from an identical ancestral source.

The following table provides some examples of the coefficient of inbreeding:

Degree of Relationship	Genes in Common	Coefficient of Inbreeding
First degree relatives	1/2	1/4
Second degree relatives	1/4	1/8
Third degree relatives	1/8	1/16
Fourth degree relatives	1/16	1/32
Fifth degree relatives	1/32	1/64

Note that the coefficient of inbreeding applies to blood relatives only, to people who are genetically related. They do not apply to persons who are adopted or otherwise accepted into an extended family or *tiospaye*. Nevertheless, many traditional societies do not distinguish between blood and non-blood relatives.

Many studies have compared aspects of health and survival with different coefficients of inbreeding. The traits that have been studied include congenital malformations, mental illness, IQ, fertility, and mortality within given time periods. Examples of the latter include miscarriages, stillbirths, neonatal deaths (that is, within one month of birth), and deaths during childhood and teens. All of these traits are influenced in many ways by both genetic and environmental factors.

In one of the few prospective studies[1], Baird and McGillivray followed 21 women who, at the time of contact, were pregnant from a relationship with a first degree relative. Nine of the 21 children (43%) born to these women had severe malformations or mental retardation. In 8 cases the cause of the birth defect was unknown. One child had a specific autosomal recessive disorder.

This study supports the findings of other researchers, and suggests that the empiric risk for abnormalities in children born to first degree relatives is high. The risk for second and third degree relatives is lower than the risk for first degree relatives, but it is not negligible[2].

OPTIONAL ACTIVITY

As a follow up to this activity, you might suggest that your students construct their own family trees. To do this, instruct each student to draw the outline of a tree. The names of the student's great-grandparents should be written on the trunk of the tree. From the trunk, branches should extend to represent each of the student's great-grandparents' children. The names of the student's great-grandparents' children should be recorded on these branches, along with the names of their partners. From each of these branches, other smaller branches should be drawn to represent the children born to each couple. The names of each couple's children and their partners should be recorded on these branches. From each of these branches should extend a leaf for each child born to that couple. The names of your student's siblings and cousins should be recorded on these leaves.

LESSON 6

RESOURCES

To gain an appreciation of the kinship system read the books *Waterlily* and *Dakota Texts* by Ella C. Deloria. Both may be purchased from Dakota Press, University of South Dakota. *Waterlily* is a story of plains people and relationships from a female perspective. *Dakota Texts* is a compilation of Dakota stories. Included are at least two stories directly related to marrying a relative, "Iktomi Marries His Daughter" (p. 7) and "Incest" (p. 81).

Other resources include:

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HEALTH IN THE YEAR 2100

LESSON 7

LIFE CHOICES AND THE FUTURE

INTRODUCTION

When making decisions, people are guided, both consciously and subconsciously, by their beliefs, customs and values. Societal rules and regulations, and the behavior of peers, are also weighed in the decision-making process.

In this lesson, students will examine the traditional beliefs and values that governed the behavior of their ancestors. They will compare these beliefs and values to the beliefs and values they use when making choices. They will also predict what effect their life choices will have on the health of the Native American community in 100 years. If your students' predictions are not similar to the class vision, they will create a plan to make their class vision for health in the year 2100 become a reality.

GOAL

To have students look to the past for strategies to promote the health of their community in the future.

OBJECTIVES

The students will:

1. Listen to a community member or scholar talk about the traditional beliefs and values that governed the choices made by Native Americans 200 years ago;

2. Think about present-day lifestyles and the effect these lifestyles have on the health of individuals and the community;

3. Discuss what they can do to make their vision for health in the year 2100 become a reality.

MATERIALS AND ADVANCE PREPARATION

If you are unfamiliar with the Seven Sacred Rites of the Dakotas and the concept of the Medicine Wheel, visit with a traditional elder in your community, or read *The Sacred Pipe*, by Joseph Brown and *The Sacred Tree*, by Judie Bopp, Michael Bopp, Lee Brown and Phil Lane prior to beginning this lesson. To conduct Lesson 7, identify a traditional elder or a native scholar who can speak to your class about the traditional beliefs, customs and values that have shaped the decisions made by individuals native to this region.

If you chose to do the "Optional Activity" in Lesson 1 remind your students that they will need to bring in their completed grocery store worksheets on Day Three. Each student will also need a copy of the brochure, "Daily Values and You," and the "Food Guide Pyramid."

LESSON 7

DIRECTIONS FOR CONDUCTING THE ACTIVITY

DAY ONE

Lesson 7 can be completed in two to three days. On Day One, begin by asking your students to list some of the things that were taken into consideration by their ancestors when they were making decisions. If it is difficult to get class participation, break students into small groups and have them generate a list of traditional beliefs that might have influenced how decisions about personal conduct were made. Once each group has generated a list of beliefs, values or customs, have each group share its list with the class. For the benefit of those students in the class who have not been instructed in the traditional beliefs, ask the groups to share some specific information about the beliefs or customs they have listed.

If the majority of your students are not familiar with the traditional beliefs of the Dakota, Lakota and Nakota people, instruct each group of students to research a topic of interest to them. Topics might include the spiritual beliefs of their ancestors, the concept of the Medicine Wheel, or the values of generosity, freedom, bravery, wisdom and harmony. Upon completion of the research, each group should share what it has learned with the class.

Then encourage your students to explore what things they consider when making decisions about their own personal conduct. Do they consider what members of their families might think about their actions? Do they consider societal rules and regulations when they make life choices? Do they consider the teachings of their religion; or how their choices will affect their community and future generations?

Have students compare their two lists; the list outlining the traditional way of making decisions, and their own decision-making strategies. Encourage them to identify the similarities and differences between these two lists. Have the students discuss the advantages and disadvantages of the different decision-making strategies that have been suggested. Your students can record their thoughts in a personal journal if they find it difficult to share their thoughts on these questions with their classmates.

DAY TWO

On Day Two, invite a traditional elder or Native American scholar to speak to your class about the traditional values of generosity, freedom, bravery, wisdom and harmony. Also ask the speaker to share information about the Medicine Wheel, and the beliefs and customs that guided the decisions made by members of the early Native American societies

DAY THREE

On Day Three, ask students to break into small groups and discuss how their life choices will affect themselves, their families, and their vision for health in the year 2100. Will the foods they are choosing to eat increase their risk for heart disease or diabetes? Are they at risk to get sexually transmitted diseases or have unplanned pregnancies? Are their chances of developing lung cancer increased because of their smoking habits, or the smoking habits of the people they live with? Can they speak Lakota? Do they participate in Indian ceremonies? Are their children going to be instructed in the traditional ways?

LESSON 7

If students are reluctant to talk about themselves and about the decisions they have made, present a number of different scenarios for class consideration. For instances, ask you students to predict what might happen to a person who eats a well balanced diet, exercises regularly, and who abstains from smoking and drinking. Is that person likely to have a heart attack, or develop diabetes? What might happen to a person who chooses to live in the traditional way? Is there support in the community for young people who choose to live in this manner? Are the extended family systems in place? Are there enough people in the community who are knowledgeable about the traditional ways to instruct a young person? If a student chooses not to incorporate traditional teachings into his or her life; what impact might this decision have on the Native American community?

(Note: If you chose to do the "Optional Activity" in Lesson 1, refer to the "Optional Activity" section below to complete this lesson.)

Conclude Lesson 7 by asking your students to consider the vision statement they created in Lesson 1. Ask them to generate a list of things they can do to see that their vision for health in the year 2100 becomes a reality. Record this list on a piece of newsprint and hang it in your classroom next to your students' vision statement.

OPTIONAL ACTIVITY

DIRECTIONS FOR CONDUCTING THE ACTIVITY

One of the choices each person makes is what type of food to eat, and what type of food to serve to his or her family. To help students focus on the food choices they make, ask each student to list one food item from each of the six food groups they recorded on the "Grocery Store Worksheet."

Write the name of each food group on the board and record each student's response under the appropriate heading. Try to elicit as many different responses as possible. If a student collected information on one of the food items already included on the class list, place a mark by that item and keep track of how many times it is listed. Refer to Figure 1.

Bread Group	Vegetable Group	Fruit Group	Milk Group	Meat Group	Fats & Sweets
bread III	green beans	oranges	Skim milk	tuna fish	candy bar
rice	peas IIII	canned pears	cheese	chicken IIIIII	pop IIIIIIIII
noodles	celery	apples	yogurt II	ground beef	potato chips

Note: In this example, three students mentioned that they collected data on some type of bread. Four students have information on peas, two students researched yogurt, etc.

Figure 1

When each of your students has contributed an item to each list, circle those items in the "Bread Group" that were listed more than once. (In the above example, this would be bread.)

Then, have each student who listed this item record the information he or she collected from the "Nutrition Label" on the black board or an overhead transparency.

Have the class compare the available nutritional information and decide which brand of bread has the least fat, which brand has the least sodium, and which brand has the most fiber. Then, ask your students which brand of bread they would choose to eat if they were genetically predisposed to heart disease. Next, circle an item that was listed more than once in the "Vegetable Group" and repeat this process.

When it appears that your students understand how to compare food labels, distribute copies of the brochure "Daily Values and You." Ask your students to find their own daily calorie level and determine their nutrient needs. They should record this information on the "Quick-Reference Card" provided on the back panel.

Next, give your students a copy of the "Food Guide Pyramid" and ask each one to plan menus for one day that will meet his or her daily calorie level and nutrient needs. For this portion of the activity students should pool all of their "Grocery Store Worksheets." They should then select their food items from this combined list.

When your students have made their food selections for breakfast, lunch and dinner, ask them if they were able to create menus that would meet their caloric and nutritional needs. If they were not able to create such menus, ask them to explain what factors made this impossible.

Your students might conclude that their choices were limited by the number of food items they had to choose from. If this is the case, ask them if there were other food items available at the local store that they could have researched. If they say that the food items on their class list accurately reflect the food items available at the local store, ask them what they can do to improve the selection of food items in their area. Are they willing to ask the store owner to stock foods that have more of the nutrients they need? Would they purchase these foods if they were available in their community?

If your class responds that there were other food items to choose from at the local store, ask them to consider whether they will look at the labels on these other food items next time they go grocery shopping. Are they willing to develop menus that will help them meet their daily calorie and nutrient needs? If they have children, will they plan meals that will meet their children's nutritional needs? Do they participate in any community functions where food is served, and if so, will they make menu selections that include items from each of the six food groups?

Conclude Lesson 7 by asking your students to consider the vision statement they created in Lesson 1. Will any of their visions be realized if they make a conscious effort to change their diet? Will the health of the community be significantly altered if your class opens a health food store? Will teaching their children about good nutrition have an impact on the future health of people in their community?

LESSON 7

Ask your students to list other things they can do to see that their vision of health becomes a reality in the year 2100. Record this list of strategies on a piece of newsprint and post it in your classroom next to the vision statement.

BACKGROUND INFORMATION

In early Dakota, Lakota and Nakota societies, survival depended on the attitude of cooperation that was embodied in the kinship system. From birth, children learned that they could rely on their mother and the other members of their extended family to provide for their basic needs. Infants were never far from their mothers, strong attachments were formed, and a sense of security developed from this constant interaction.

Ideally, children were treated with dignity. They were thought of as rational human beings and allowed to exercise personal choice. This permissive attitude fostered a strong sense of individual freedom in children. Physical punishment was avoided. When a reprimand seemed appropriate, it was done in a gentle manner to avoid hurting the child's feelings or subjugating his or her spirit.

With the right to choose, children also learned responsibility. Parents had a responsibility to teach their children that the actions of every person were essential to the survival of the group and reflected on the entire kinship. Disobedient children were often reminded of their duty to their family and their social group, a reprimand that often resulted in feelings of shame.

Young children were given light chores and informal instruction in the skills they would need to participate fully in the community. Quite early in their training, children were taught to endure physical hardship without whimpering. Children learned to display self control in both their actions and speech. They were encouraged to develop their powers of observation. They were instructed in the legends and history of the People. Children also learned the correct use of the language and the ways to show proper respect for age and wisdom.

The socialization of children depended on the internalization of the principle virtues of Dakota, Lakota and Nakota life. Through example, children learned that honor was the most important virtue in the society. Individuals who performed great feats of bravery or outwitted their enemies by stealth held great places of honor in their community. People who exhibited such traits could be relied on to fight against great odds to protect their community.

Elders who achieved positions of leadership on the council were also respected and honored. They served as advisors to the community in matters pertaining to such activities as fishing and hunting.

The second greatest virtue in the traditional society was fortitude. Courage was required to accept the continued hardships of life, or suffer pain without complaint.

Generosity was the third virtue of traditional society. The importance of generosity was taught through the "giveaway" ceremony. Children who observed this practice learned that people who gave away their possessions were honoring others and gained in social prestige. This practice made it possible for everyone to feel included in the group and to receive those things needed for survival without having to depend on false charity.

LESSON 7

Wisdom was the fourth essential virtue of traditional life. In families, grandparents were respected for their knowledge and were expected to pass on to their grandchildren what they had learned. Leaders, too, were chosen for their wisdom.

Interactions between members of the kinship group were also governed by a strict code of behavior. At a young age, sisters and brothers were taught that they should no longer speak, or sit next to one another. Girls learned to show respect for their brothers by applauding their bravery and making gifts for them. Females were taught not to trouble or embarrass the males. Likewise, males were expected to respect females. Boys were taught to show respect for their sisters by giving them the best trophies they won or captured.

This pattern of avoidance served as a model for adult relations. To show respect for aunts and uncles and nieces and nephews, a person was required to maintain an attitude of restraint and deference. The degree of reserve depended on the sex of the person and the degree of relatedness. Partial avoidance, for instance, was required between a man and his sisters, female cousins, and father-in-law. Women were expected to avoid their brothers, male cousins, and mothers-in-law. The ideal expression of respect and love was found in the complete avoidance between a man and his mother-in-law and between a woman and her father-in-law.

More intimate and friendly relationships were maintained between cousins of the same sex, between husbands and wives, and between brothers-in-law and sisters-in-law. The greatest love and devotion was shown between brothers and male cousins. It was believed that more dependence could be placed upon a brother during times of need than upon any other relative.

Sisters were also devoted to one another, and presented a united front to outsiders. This attitude of devotion was essential if a sister lost her husband. By custom, a widow could marry her sister's husband, or simply move in with her sister's family. Under these circumstances, their devotion to one another allowed sisters to live together in harmony. Other members of the nuclear family were also treated with tenderness and affection. Grandparents often played an important role in child-rearing, and married couples showed affection for their parents by taking responsibility for their care as they grew old.

Traditional native people realized that strict adherence to the kinship patterns of behavior were essential for the survival of the group. People who demonstrated proper kinship behaviors were honored and gained respect from other bands.

Instructions for living were also spelled out in the Seven Sacred Rites, the first of which was given to Chief Standing Hollow Horn by the White Buffalo Cow Woman, and through the symbolism of the Medicine Wheel and the Sacred Tree.

RESOURCES

Bopp JB, Brown M. Brown L, Lane P. The sacred tree. Four Worlds Development Press, Alberta, Canada; 1984.

Brown JE. The Sacred pipe. Norman: University of Oklahoma Press; 1971.

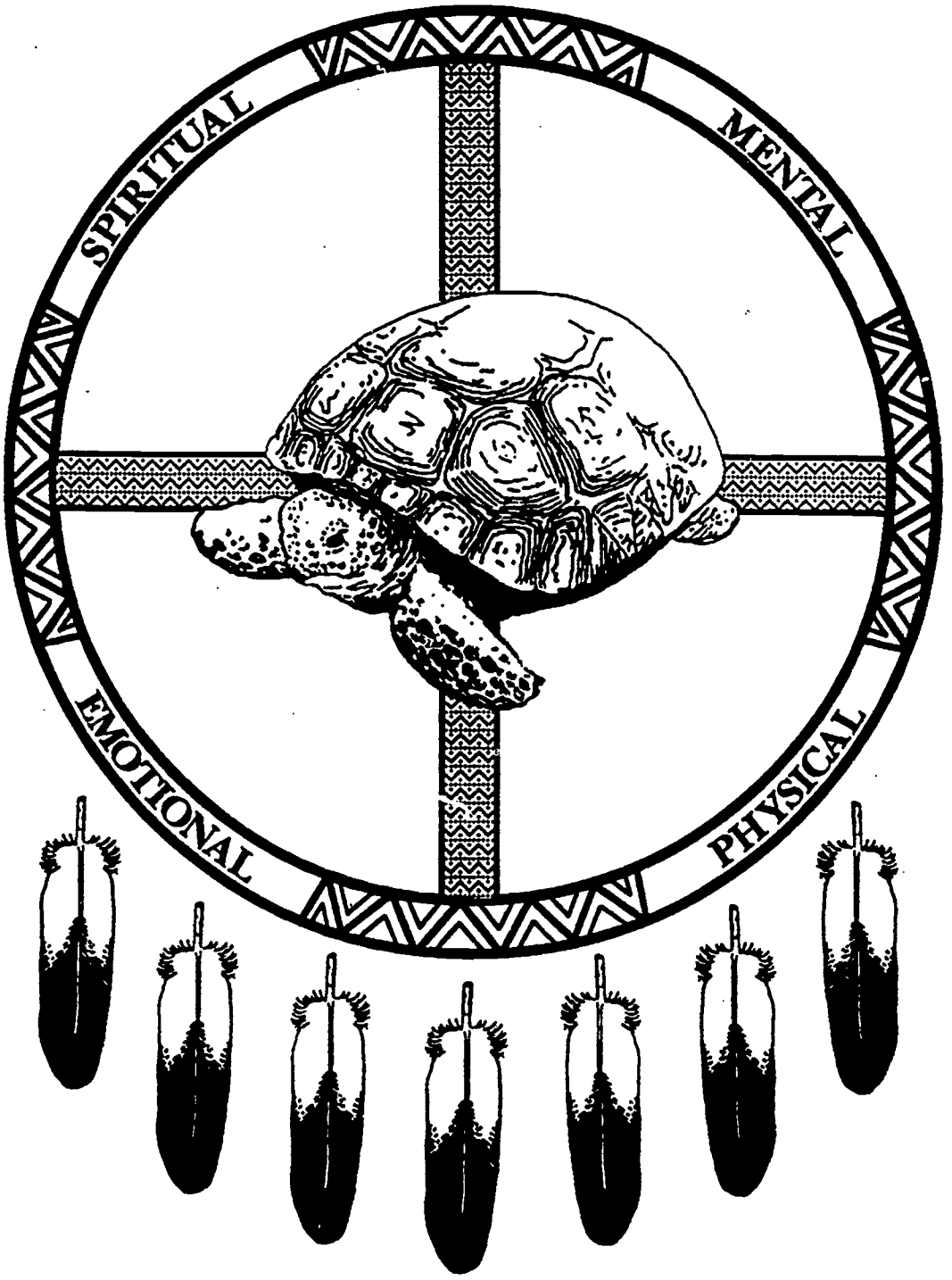
Malan VD. The Dakota Indian family. Rural Sociology Department, Agricultural Experiment Station, South Dakota State College, Brookings, SD. Bulletin 470; May 1958.

OPTIONAL ACTIVITY

To capitalize on the preferred learning style and strengths of each student, ask each one to develop a project that demonstrates an understanding of the concepts presented in this module. Ask each student to present a formal project proposals, and a timeline of when he or she plans to complete the project. If a student's project seems to be too narrow in its scope, or too ambitious, help them modify the project idea or timeline.

Students may do any number of projects. One student may choose to research a birth defect mentioned in Lesson 5 and write a brief report. Another student may choose to research his or her family history and the health problems of relatives. Students may also want to write stories, produce plays, or paint pictures depicting some of the concepts that were presented in this module. The list of projects will be as varied as the students in your class

HEALTH IN THE YEAR 2100:
WHAT'S HEREDITY GOT TO DO WITH IT?



STUDENT MANUAL

TABLE OF CONTENTS

LESSON 1

HEALTH IN THE YEAR 2100

A person develops from a fertilized egg into a fetus, from a fetus into a baby, and from a baby into an adult. This transformation from egg to adult is shaped by two major factors: heredity and the environme nt. Environment refers to the world in which we live, both our physical world and our social world. Heredity refers to those factors that are passed on from our parents and ancestors, and which we will pass on to our children and descendants.

Some inherited factors, such as the language we speak at home, the traditions our parents and elders teach us, and the beliefs and practices we share with members of the community are cultural. Some inherited factors are biological, or what scientists call genetic. Genetics is the study of how genes and chromosomes are passed on from generation to generation, and how they influence growth and development.

A person's health is also influenced by the interaction between the environment and inherited traits. The goal of the module *Health in the Year 2100*, is to examine the roles that the environment and heredity play in the development of disease, in birth defects and in the future health of your community.

LESSON 1

HEALTH 200 YEARS AGO

Over the course of the next three weeks you will learn about factors that can affect health. You will identify environmental and social factors that affected the health of your ancestors, and compare them to factors that affect health today. You and your classmates will then create a vision for health in the year 2100.

To learn more about the health of your ancestors, talk to the elder members of your family, or other people in your community. Find answers to the following list of questions.

1. What types of health problems were common 200 years ago?

2. Do people still have these health problems today?

3. Do people today have health problems that your ancestors did not have? Please list.

4. How did your ancestors care for people who were ill?

5. Do the health care practices used 200 years ago, differ from the health care practices used today? Please explain.

6. What were the common causes of death 200 years ago?

7. What are the common causes of death in your community today?

8. What aspects of life 200 years contributed to good health?

9. List those things that you can do today to promote good health in your community.

OPTIONAL ACTIVITY

FOOD AND YOU

The next time you go to the grocery store, collect information on items from the following food groups. Remember, gather information from the nutrition labels on the specified number of items from each group. Be sure to record any label claims such as "fat free" or "high fiber" in the space provided for each item you choose.

Bread Group

Collect data on a total of 6 different items from the bread group. Examples of items in this group include breads, cereal, rice and pasta.

Vegetable Group

Collect data on a total of 3 different types of vegetables.

Fruit Group

Collect data on 3 different types of fruit

Milk Group

Collect data on 3 different items from the milk group. Examples of items in this group include milk, cheese, and yogurt. Be sure to include information about the type of milk (i.e. 2%, skim) and whether or not the cheese is natural or processed.

Meat Group

Collect information on 3 items from the meat group. Examples include any type of meat, fish, poultry, dry beans, eggs, nuts and peanut butter.

Fats and Sweets Group

Collect information about the type of butter or margarine you use, your favorite salad dressing or mayonnaise, and 3 or your favorite snack foods, such as pop, potato chips, and ice cream.

GROCERY STORE WORKSHEET

Food Group												
Brand Name												
Serving Size												
Calories/Serving												
Calories from Fat												
	Amt	% DV	Amt	% DV	Amt	% DV	Amt	% DV	Amt	% DV	Amt	% DV
Total Fat												
Saturated Fat												
Cholesterol												
Sodium												
Total Carbohydrate												
Dietary Fiber												
Sugar												
Protein												
Label Claim												

AMT = Amount %DV = Percent daily value

LESSON 2

DIABETES MELLITUS

WHAT IS DIABETES?

Diabetes mellitus, or sugar diabetes, is a disease that occurs when the body cannot process glucose (a sugar) properly. The word "diabetes" comes from the Greek word meaning "to pass through." This word is used because people with diabetes urinate more often than non-diabetic individuals. "Mellitus" comes from the Latin word for "honey." It is used because the urine of diabetic patients contains large amounts of sugar.

There are several types of diabetes. The most familiar forms of diabetes mellitus are called Type I and Type II.

A. TYPE I DIABETES MELLITUS

Type I diabetes mellitus usually occurs in children, teenagers or young adults. It is sometimes called juvenile-onset diabetes. The pancreas of an affected person does not make insulin. Insulin helps the muscles, liver and other tissues break down sugar.

People with Type I diabetes must take insulin all their lives. For this reason, some doctors call this form of the disease insulin-dependent diabetes mellitus (IDDM).

Approximately 10 to 15% of all Americans with diabetes have Type I diabetes. The rate is much lower among Native Americans. Less than 2% of American Indians in the Aberdeen Area with diabetes have Type I diabetes.

B. TYPE II DIABETES MELLITUS

Type II diabetes mellitus usually affects people later in life. It is called maturity-onset, or adult-onset diabetes mellitus. Most people with Type II diabetes are overweight and physically inactive when they are diagnosed with this disease. Many doctors treat this form of diabetes by prescribing low-calorie diets and daily exercise.

Some people call Type II diabetes non-insulin-dependent diabetes mellitus (NIDDM). This can be confusing because some people with Type II diabetes take insulin in addition to their dietary and exercise therapies. In the Aberdeen Area, 98 to 99% of Native Americans with diabetes have Type II diabetes.

The pancreas of a person with Type II diabetes usually makes insulin, but the insulin does not work properly. In some cases the pancreas does not make enough insulin. In other cases, the cells are resistant to the insulin. Being overweight is a major factor in increasing the cells' resistance to insulin.

LESSON 2

COMPARISON OF DIFFERENT TYPES OF DIABETES MELLITUS

	TYPE I DIABETES	TYPE II DIABETES
When do the symptoms of diabetes usually appear?	During childhood or early adulthood	In adults older than 35 years of age
How common is this form of diabetes in the US?	10%-15% of all Americans with diabetes	85%-90% of all Americans with diabetes
How common is this form among Native Americans in the Aberdeen Area?	1%-2% of Native Americans with diabetes	98%-99% of Native Americans with diabetes
Is normal insulin present in affected people?	No, either not enough insulin is made, or the insulin is abnormal in shape and cannot work properly.	Yes, normal insulin is made, but the body's cells have become resistant to its action.
Is this form of diabetes associated with excess weight?	No, many children with Type 1 diabetes are normal or underweight.	Yes, more than 80% of adults diagnosed are overweight by 20% or more.
Are daily insulin injections required for treatment?	Yes, along with dietary and weight control.	Sometimes, but most doctors focus on weight loss through diet and exercise, or sugar reducing pills.

HOW COMMON IS DIABETES AMONG NATIVE AMERICANS?

Diabetes mellitus affects one in 20 Americans (10 to 12 million people). One in 10,400 people will die of this disease each year. The death rate among Native Americans, however, is much higher. In 1989, one in 1,700 people on the Pine Ridge reservation died of Type II diabetes mellitus. This is more than six times higher than the national average.

Historically, diabetes was not a major health problem for Native Americans. Diabetes was rare in all tribes before the 1940's. In 1900, for example, the census reported only two Native American deaths from diabetes in a population of about 266,000. In 1955, the proportion of American Indians dying from diabetes was equal to the proportion of diabetes deaths in the Caucasian population. Now, the proportion of Native American deaths is much higher, and

increasing. This rapid increase in the incidence of diabetes makes it one of the most common chronic diseases among Native Americans.

WHAT ARE THE SIGNS AND SYMPTOMS OF DIABETES MELLITUS?

The most common characteristics of diabetes mellitus are:

1. Sugar in the urine. Doctors call this condition glucosuria.
2. Excessive urination.
3. Excessive thirst. Because the body is losing more water through the urine than usual, diabetics are constantly thirsty and drink large amounts of water.
4. Increased appetite. The major source of fuel for the body is sugar. Given that people with untreated diabetes do not use sugar efficiently, they may eat more to make up for this lack of fuel.
5. Long-term effects of diabetes mellitus may include blurred vision, kidney problems and poor circulation.

IS DIABETES INHERITED?

Diabetes mellitus runs in families. Nationwide, about 17% of the people with diabetes have a diabetic mother, and another 8% have a diabetic father. If a person develops diabetes, the chance that his or her children will become diabetic increases. If both a parent and a child develop diabetes, the risk to other children becomes even greater.

Some geneticists have suggested that "diabetes genes" probably exist among certain groups of Native Americans, Mexican Americans, Australian aborigines, migrant Asian Indians, urbanized Pacific Islanders and some other non-Western populations. Such genes are thought to have been essential to the survival of these peoples.

HOW COULD GENES THAT CAUSE DIABETES BE BENEFICIAL?

Geneticists hypothesize that "diabetes genes" make cells more efficient at turning the sugar a person eats into body fat. According to this hypothesis, people who inherit these genes store more fat when they eat. When food is scarce, this fat serves as an extra source of energy, to sustain these people until more food becomes available. People who do not have these genes (and are therefore lacking the extra fat) are more likely to starve when food is not available for long periods of time.

This hypothesis suggests that the native people who had these genes were more likely to survive periods of drought, or harsh winters, when food was scarce. Those who survived were, in turn, more likely to have children and pass on this particular genetic trait to future generations.

During the past century, the diets and lifestyles of Native Americans have changed. For example, meals today are low in fiber and high in fats and sugars. In addition, most people

rarely exercise on a daily basis. Nevertheless, people who have inherited these genes continue to store fat. As droughts and harsh winters no longer affect the availability of food, this weight is not lost. Rather, people with these genes risk becoming overweight and, eventually, diabetic.

OTHER RISK FACTORS

In addition to excess weight and a lack of exercise, advanced age is a risk factor for diabetes mellitus. Only 1 in 900 people less than 20 years of age have diabetes. The rate increases to 1 in 200 between the ages of 41 and 50. Over the age of 61, the risk of diabetes increases to 1 in 50.

A person's chances of developing Type II diabetes mellitus are greatest when he or she has two or more of the above risk factors. For example, a person who has a diabetic relative and is overweight is far more likely to develop diabetes than a 20-year-old with no family history of this disease.

REDUCING THE RISK OF DIABETES

Your genes do not doom you to diabetes. Adopting a healthy lifestyle and behavior patterns may prevent this disease. The behaviors most often recommended include:

1. Keeping your weight appropriate for your age and height.
2. Reducing fat intake by choosing lean meats (lean beef and chicken) and low fat dairy products, and by decreasing the use of butter and shortening.
3. Reducing the number of calories you eat by avoiding foods like candy, sugar, chocolate and ice cream.
4. Increasing the amount of fiber and complex carbohydrates you eat by adding more vegetables, whole grain cereals, grains, beans, peas and other legumes to your diet.
5. Exercising regularly by walking, swimming, running or doing other aerobic activities.

RESOURCES

Information is available from state and national organizations. Consider writing or phoning for information about diabetes from the following organizations:

Diabetes Control Program
South Dakota Department of Health
445 East Capitol
Pierre, SD 57501-2080
Phone: 605-773-3737

American Diabetes Association
National Service Center
1660 Duke Street
Alexandria, VA 22314
Phone: 1-800-232-3472

Juvenile Diabetes Foundation International
60 Madison Ave., 4th Floor
New York, NY 10010
Phone: 1-800-223-1138

National Diabetes Information
Clearinghouse
Box NDIC
9000 Rockerville Pike
Bethesda, MD 20892
Phone: 301-468-2162

LESSON 2

HEART DISEASE

WHAT IS HEART DISEASE?

Heart disease, is a general term for several different conditions that can lead to heart failure. There are five major forms of heart disease:

A. CORONARY HEART DISEASE OR CORONARY ARTERY DISEASE

The major blood vessels that supply food and oxygen to the heart are called coronary arteries. Damage to these arteries can decrease the blood flow to the heart. Decreasing the blood flow reduces the amount of food and oxygen reaching the heart muscle and causes heart attacks.

The primary cause of coronary artery damage is the long-term build up of fatty deposits on the artery walls. Doctors call this condition atherosclerosis. This build up results from high levels of cholesterol in the blood.

People who have heart attacks often have a diet that is high in fat and cholesterol. This increases the chance that fatty deposits will build up on the walls of the coronary arteries. The build up of fatty deposits decreases the size of the arteries. This, in turn, results in an increased risk of a heart attack.

B. CONGESTIVE HEART FAILURE

Congestive heart failure refers to the mechanical failure of the heart to pump blood efficiently. This condition may or may not be life-threatening, depending on what is causing the heart to pump less blood. Things that may cause the heart to pump blood less efficiently include a damaged heart valve, an irregular heartbeat and high blood pressure.

C. CONGENITAL HEART DISEASE

Some babies are born with heart defects. In most cases, the defect involves a change in the heart structure, such as a hole in the wall that separates the right and left chambers, or an abnormal heart valve. Heart abnormalities that are present at birth are referred to as congenital heart defects.

D. RHEUMATIC HEART DISEASE

Rheumatic fever usually occurs in school-aged children following a strep throat infection. Normally, the body makes antibodies to remove the invading bacteria. In some children, however, these antibodies also attack normal tissues. The most common tissues that are attacked are the joints. Less commonly, heart tissue is attacked. Swelling of the joints rarely causes permanent damage, but swelling of heart tissue can damage heart valves so that they no longer work properly.

Antibiotics can prevent the long-term consequences of rheumatic heart disease. Now rheumatic fever rarely causes heart failure, and most children who have had this disease can lead normal or near-normal lives.

E. HYPERTENSION

The term "blood pressure" refers to the pressure blood exerts against the artery walls. It is defined by two numbers, for example 120/80. The larger number, 120, is the pressure of the blood against the artery walls when the heart contracts. The smaller number, 80 in our example, represents the blood pressure when the heart relaxes between beats. Doctors and nurses call the larger number systolic blood pressure, and the smaller number diastolic blood pressure.

When either systolic or diastolic values rise above normal, a person is said to have high blood pressure, or hypertension. An increased blood pressure can damage the blood vessels and cause heart failure, strokes, kidney failure, and other life-threatening conditions.

CAUSES OF HIGH BLOOD PRESSURE

Many people have high blood pressure for no obvious reason. This form of high blood pressure is called essential hypertension, and tends to run in certain families.

In many cases, people with essential hypertension can lower their blood pressure by simply changing their diet and lifestyle. Recommended changes include losing weight, exercising on a regular basis, avoiding fatty foods, avoiding salty foods and learning to use salt-free seasonings on foods.

When the cause of high blood pressure is known, it is called secondary hypertension. Some causes of secondary hypertension include kidney disease and certain hormonal disorders. About 5% of the women who use the "pill" will develop high blood pressure. Secondary hypertension may also occur during pregnancy.

SYMPTOMS OF HIGH BLOOD PRESSURE

In the earliest stages, most people do not know they have high blood pressure because there are no symptoms. In the later stages of the disease, symptoms of hypertension include headaches (usually restricted to the back of the skull), episodes of dizziness and weakness, nosebleeds and blurred vision. These symptoms are <u>not</u> the result of increased pressure itself, but of damage to the blood vessels and other tissues caused by high blood pressure over a long period of time.

LONG-TERM EFFECTS OF HYPERTENSION

People with untreated high blood pressure can develop health problems that may cause early death. People who follow their doctors' orders carefully can lead normal or near-normal lives.

LESSON 2

The most common causes of death resulting from untreated hypertension are heart failure, stroke and kidney disease.

These long-term consequences of uncontrolled high blood pressure are preventable. Because there are no symptoms associated with the early stages of hypertension, everyone should have their blood pressure checked at least once a year. If diagnosed early, people with high blood pressure can lower their risk of heart failure, stroke and kidney disease by following their doctors' orders, watching their weight and diet, and taking their medications as prescribed.

HOW COMMON IS HEART DISEASE AMONG NATIVE AMERICANS?

Heart disease accounts for nearly one-third of all deaths among Europeans and European-Americans. This means that one out of 571 Americans nationwide dies of heart disease each year, most often as the result of coronary heart disease or high blood pressure. The death rate among Lakota people living on the Pine Ridge reservation is one in 305, nearly double the national rate.

WHAT ARE THE SIGNS AND SYMPTOMS OF HEART DISEASE?

Symptoms of heart disease and high blood pressure do not usually appear until after a great deal of damage has already occurred. The first noticeable sign of a heart attack is often chest pain that spreads to the arms, back, throat or jaw. During the attack, chest pain may vary from a tight feeling to a bursting sensation. The pain may be continuous or it may come and go. Other symptoms include dizziness, shortness of breath, sweating, an upset stomach and fainting.

IS HEART DISEASE INHERITED?

Heart disease runs in families. The more relatives a person has with heart disease, the greater the risk is for developing heart disease. One study showed that if two members of a person's immediate family (biological parents, grandparents, brothers and sisters) has a heart attack before the age of 55, that person's risk of developing heart disease is five times the risk of someone who has no family history of heart disease.

As people grow older, their chance of having a heart attack also increases. Historically, men were at a greater risk for coronary heart disease than women. This trend, however, seems to be changing, at least in industrialized countries.

REDUCING THE RISK OF HEART DISEASE

None of these factors, family history, age or sex, are under your control. You can, however, reduce your chances of heart disease by:

1. Not smoking cigarettes. Nicotine increases the heart rate and intensifies the effects of high blood pressure. Heavy smokers who have heart attacks are much more likely to die than non-smokers who have heart attacks.

2. Keeping your weight appropriate for your age and height. Being overweight causes the heart to work harder to pump blood to all the tissues in the body.

3. Reducing the amount of food you eat that contains high amounts of saturated fats, such as fatty meats, butter, bacon, cream and whole milk cheeses. These foods contribute to the build up of fatty deposits on the artery walls.

4. Reducing the amount of sugar-rich foods you eat, such as pies, cakes, cookies, ice cream, candy, soft drinks, fruit drinks, fruit packed in syrup, jams, jelly, doughnuts and sweet rolls. These foods contribute significantly to obesity.

5. Reducing the amount of salt in your diet. Foods that are high in salt include potato chips, pretzels, salted nuts and popcorn, soy sauce, steak sauce, cheese, pickled foods and cured meats. Salty foods contribute to high blood pressure, which increases the risk of heart attacks.

6. Exercising regularly.

In most cases, the risk of heart disease is determined by three things: the genes one inherits, the environment one lives in, and the lifestyle one chooses. By eating a balanced diet, exercising regularly, and eliminating cigarettes and alcohol, even a person with a family history of heart disease can significantly reduce his or her risk of heart disease.

RESOURCES

The American Heart Association publishes and distributes educational materials on a wide variety of topics related to heart disease. The National Heart, Lung and Blood Institute also sends out information upon request.

American Heart Association
1005 12th Ave. SE
PO Box 1287
Jamestown, ND 58402
Phone: 1-800-437-9710

NHLBI Information Center
PO Box 30105
Bethesda, MD 20824-0105
Phone: 301-951-3260

LESSON 2

ALCOHOL AND ALCOHOLISM

WHAT IS ALCOHOL?

Alcohol is a small molecule that acts as a depressant, and interferes with the activity of the brain and spinal cord. It is a mind- and mood-altering drug. If consumed in small amounts, alcohol usually has little or no apparent effect on the drinker. In moderate amounts, alcohol can produce an exaggerated sense of happiness and well-being. In large amounts, alcohol acts as a sedative.

Not only does alcohol affect the mind, it also affects the body. Alcohol can damage the stomach, pancreas, liver, heart, blood vessels, and the nerves throughout the body. There is also a high correlation between alcohol abuse and sugar diabetes among Native Americans.

WHAT IS ALCOHOLISM?

There are many different definitions of alcoholism. Most definitions, however, include the following factors:

1. An inability to control one's drinking habits, resulting in excessive alcohol consumption.
2. Drinking at inappropriate times.
3. A preoccupation with ways of obtaining and drinking alcohol.
4. An increased tolerance to alcohol.
5. Chronic, or repeated consumption of alcohol, leading to impairment of health and interference with normal functioning.
6. A physical dependency on alcohol, resulting in withdrawal symptoms when alcohol is no longer consumed.

HOW COMMON IS ALCOHOLISM AMONG NATIVE AMERICANS?

Native Americans exhibit a higher rate of problems associated with alcohol abuse than any other ethnic group in the United States. Death rates from chronic liver disease and alcoholism are 6 to 10 time higher than the national rate of one per 15,600. Deaths due to injuries (18%), suicide (3%), and homicide (3%) resulting from alcohol abuse are among the top ten causes of death reported by the Indian Health Services.

HOW DOES ALCOHOL AFFECT THE BODY?

THE LIVER

The stomach and small intestine absorb alcohol quickly. Alcohol is carried to the liver, where it is converted into another molecule called an acetaldehyde. This molecule causes hangover symptoms and "the shakes" associated with heavy drinking.

Next, the acetaldehyde is converted into an acid. This process uses oxygen needed for other functions, and causes an abnormal build-up of various chemicals in the liver.

With repeated drinking, fatty deposits build up in the liver. These deposits crowd out normal liver cells and interfere with their functions. Eventually the liver becomes swollen (a condition called hepatitis) and scarred (cirrhosis).

THE HEART AND BLOOD VESSELS

Alcohol causes blood vessels to dilate, or increase in diameter. This creates both a mild drop in blood pressure and an increased heart rate. Large amounts of alcohol will increase blood pressure, although how this happens is not clear. Continued drinking may eventually damage the heart muscle itself, resulting in heart enlargement and an abnormal heart rate. This damage can contribute to heart failure. Strokes, the rupture of blood vessels in the brain, can also follow heavy drinking episodes.

THE CENTRAL NERVOUS SYSTEM

Perhaps the most familiar effects of alcohol abuse are altered behavioral patterns. People who drink may have an artificial sense of well-being or euphoria, followed by depression and sometimes hostility. Alcohol can lower inhibitions and interfere with judgment, so that people will say or do things they would not say or do if they were sober. Alcohol use may cause staggering and slurred speech. Excessive drinking may also lead to episodes of unconsciousness or "blackouts."

Continued drinking can interfere with normal nutrition, causing malnutrition and vitamin deficiencies. In some people this leads to double vision, a lack of muscular coordination and decreased mental function. Individuals addicted to alcohol may also experience delirium tremens when they stop drinking. The symptoms include tremors, hallucinations and delusions.

IS ALCOHOLISM INHERITED?

The tendency to become alcohol-dependent is most likely determined by the interaction between several genes and a number of non-genetic factors. Non-genetics factors that influence a person's risk of becoming an alcoholic include:

1. A family history of alcoholism. If relatives are heavy drinkers, other family members have a higher chance of becoming alcoholic.

2. A family history of depression. Families with a high incidence of depression, especially among female members, tend to have more alcoholic members than other families.

3. A family history of total abstinence. Ironically, families that forbid drinking alcohol and that enforce strict moral controls are more likely to have alcoholic children. Children who feel the need to rebel against regulations at home (or school) may deliberately set out to

engage in behaviors that they know will upset their parents or other adults in the community.

4. A family history of divorce and parental discord. Children raised by single parents are more likely to become alcoholic than children raised by two parents.

5. Being a member of certain ethnic groups. The rate of alcoholism is higher among certain ethnic groups than among others. Those that are most vulnerable include Native Americans, Northern Europeans (including Irish and Scandinavians), the French, Mediterranean peoples (Italian, Greek and Jewish), and the Chinese.

6. Being a heavy smoker. Heavy smokers are at a higher risk for abusing alcohol. Apparently, many people who find nicotine and tobacco calming also tend to drink alcohol.

REDUCING THE RISK OF ALCOHOLISM

In most cases, a person's risk of alcoholism is caused by an interaction between three factors: the genes one inherits, the environment one lives in, and the lifestyles one chooses. By choosing to live an alcohol-free life, one can avoid the medical and social problems associated with alcohol abuse.

RESOURCES

There has been a great proliferation of organizations set up to advise, counsel and treat persons suffering from alcoholism and substance abuse. Perhaps the most familiar national organization is Alcoholics Anonymous (AA). For information from a local resource, contact Anpetu Luta Otipi in Kyle, on the Pine Ridge Reservation.

Alcoholics Anonymous National Office
General Service Board
Box 459
Grand Central Station
New York, NY 10163
Phone: 212-870-3400

Anpetu Luta Otipi
PO Box 275
Kyle, SD 57752
Phone: 605-455-2331

Other organizations include:

Alateen World Service Headquarters
PO Box 862
Midtown Station
New York, NY 10018-0862
Phone: 212-302-7240

Division of Alcohol and Drug Abuse
500 East Capitol
Pierre, SD 57501
Phone: 605-773-3123

LESSON 3

GENOTYPE / PHENOTYPE

Student's Name: _____

SECTION A: ONE PAIR OF GENES PER TRAIT

Father's genes: _____ Mother's genes: _____

My genotype is: _____

Genotype--the combination of genes a person inherits.

If the red gene is dominant, my phenotype is: Example One: _____

Example Two: _____

Phenotype--a person's observable physical characteristics that are determined by the interaction between his or her genes and the environment.

If the genes are co-dominant, my phenotype is: Example One: _____

Example Two: _____

SECTION B: TWO PAIRS OF GENES PER TRAIT

	1st Gene	2nd Gene
Father's genes:	_____	_____
Mother's genes:	_____	_____

My genotype is: _____ My phenotype is: _____

SECTION C: THREE PAIRS OF GENES PER TRAIT

	1st Gene	2nd Gene	3rd Gene
Father's genes:	_____	_____	_____
Mother's genes:	_____	_____	_____

My genotype is: _____ My phenotype is: _____

LESSON 3

GENOTYPE / PHENOTYPE KEY

SECTION A1: THE RED GENE IS DOMINANT

GENOTYPES	PHENOTYPES	
	ONE	TWO
Red-Red	_____	_____
Red-White	_____	_____
White-White	_____	_____

SECTION A2: THE GENES ARE CO-DOMINANT

GENOTYPES	PHENOTYPES	
	ONE	TWO
Red-Red	_____	_____
Red-White	_____	_____
White-White	_____	_____

SECTION B: TWO GENES PER TRAIT

GENOTYPES	PHENOTYPES
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

SECTION C: THREE GENES PER TRAIT

GENOTYPES	PHENOTYPES
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

LESSON 3

DIABETES MELLITUS

No relatives with a history of diabetes SCORE=0 A	One relative with a history of diabetes SCORE=2 A
Two or more relative with a history of diabetes SCORE=4 A	Eat sweets once a month or less SCORE=0 B
Eat sweets once a week or less SCORE=1 B	Eat sweets two or three times a week SCORE=3 B
Eat sweets daily SCORE=5 B	Vigorous daily exercise at both school (or work) and at home SCORE=0 C
Vigorous daily exercise at school or at home, but not both SCORE=1 C	Some daily exercise at both school and home SCORE=2 C
Exercise every other day or so SCORE=3 C	Exercise once a week or less SCORE=5 C
Normal weight for my height SCORE=0 D	5 pounds or less overweight for my height SCORE=1 D
6-10 pounds overweight for my height SCORE=2 D	11-19 pounds overweight for my height SCORE=3 D
20 or more pounds overweight for my height SCORE=5 D	

Divide the number of students in your class who studied diabetes by three. This is the number of copies of the this page you will need to make. Cut along the solid lines and sort each slip of paper by the letter in the lower right-hand corner. Place all of the slips of paper with the letter "A" in the container labeled "Family History of Diabetes." Place the slips of paper with the letter "B" in the "Eating Habits" container. The letter "C" slips of in the container labeled "Exercise Habits." Place the letter "D" slips in the container labeled "Weight."

LESSON 3

HEART DISEASE

No relative with a history of heart disease SCORE=0 A	One relative with a history of heart disease SCORE=2 A
Two or more relatives with a history of heart disease SCORE=4 A	Strict vegetarian SCORE=0 B
Eat turkey, chicken or fish, but no beef or pork SCORE=1 B	Eat beef or pork only once or twice a week SCORE=2 B
Eat beef or pork daily SCORE=4 B	Eat beef or pork more than once every day SCORE=6 B
Never smoke SCORE=0 C	Smoke less than five cigarettes a week SCORE=1 C
Smoke one to four cigarettes daily SCORE=2 C	Smoke less than one pack of cigarettes daily SCORE=3 C
Smoke more than a pack per day SCORE=5 C	Vigorous daily exercise at both school (or work) and at home SCORE=0 D
Vigorous daily exercise at school or at home, but not both SCORE=1 D	Some daily exercise at both school and home SCORE=2 D
Exercise every other day or so SCORE=3 D	Exercise once a week or less SCORE=5 D

Divide the number of students in your class who studied heart disease by three. This is the number of copies of this page you will need to make. Cut along the solid lines and sort each slip of paper by the letter in the lower right-hand corner. Place all of the slips with the letter "A" in the container labeled "Family History of Heart Disease." Place the slips of paper with the letter "B" in the "Eating Habits" container. The letter "C" slips go in the container labeled "Smoking Habits." Place the letter D slips in the container labeled "Exercise Habits."

LESSON 3

ALCOHOLISM

No relatives with a history of alcohol abuse SCORE=0 A	One relative with a history of alcohol abuse SCORE=2 A
Two or more relatives with a history of alcohol abuse SCORE=4 A	No relatives with a history of depression SCORE=0 B
One relative with a history of depression SCORE=2 B	Two or more relatives with a history of depression SCORE=4 B
Never drink. Disregard all other scores. A person who never drinks alcohol will not become an alcoholic. C	One drink per week. SCORE=1 C
Two drinks per week SCORE=2 C	One drink every day SCORE=4 C
Two or more drinks per day SCORE=6 C	Never smoke SCORE=0 D
Smoke less than five cigarettes a week SCORE=1 D	Smoke one to four cigarettes daily SCORE=2 D
Smoke less than one pack of cigarettes daily SCORE=3 D	Smoke more than a pack per day SCORE=5 D

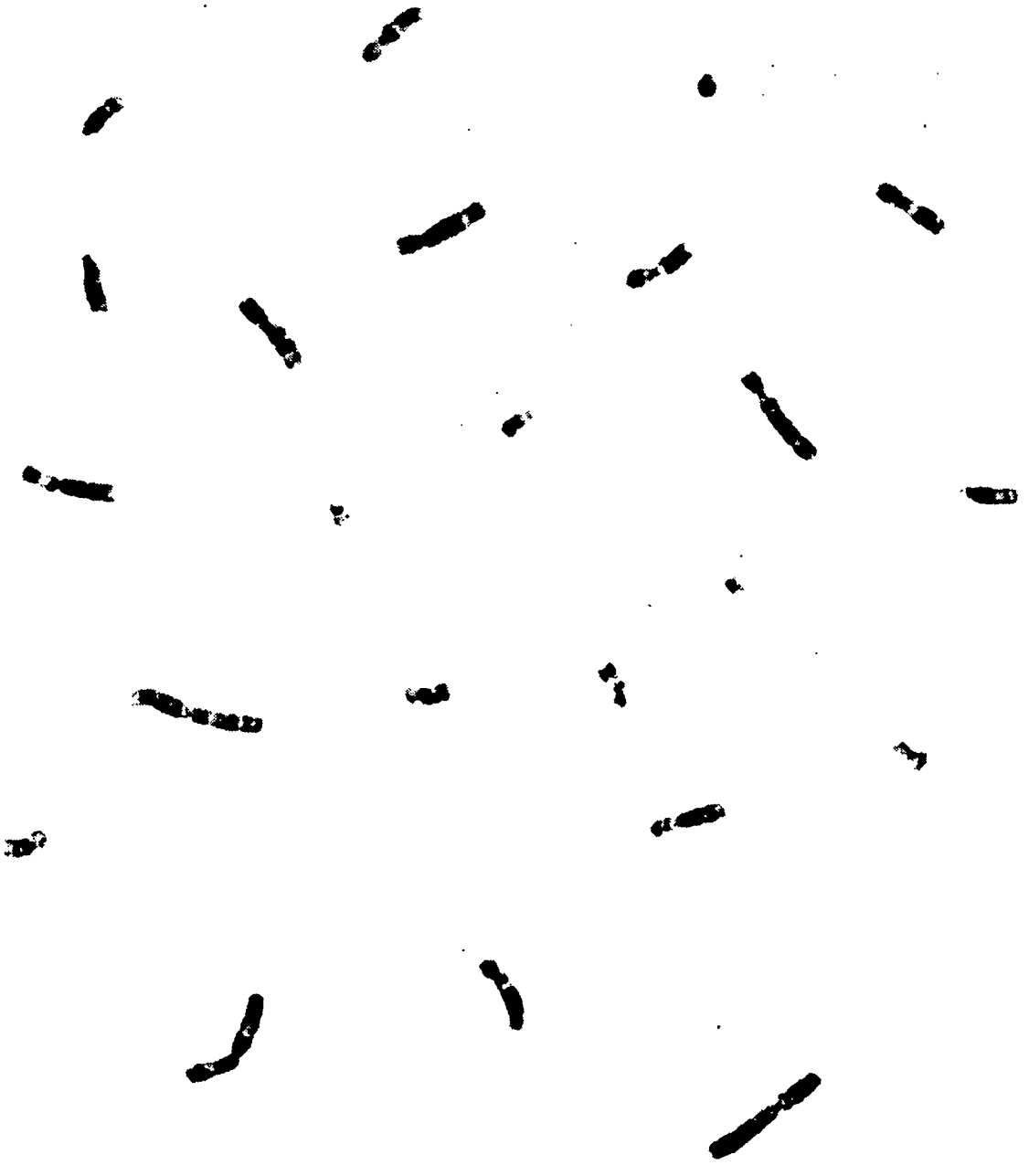
Divide the number of students in your class who studied alcoholism by three. This is the number of copies of this page you will need to make. Cut along the solid lines and sort each slip of paper by the letter in the lower right-hand corner. Place all of the slips of paper with the letter "A" in the container labeled "Family History of Alcoholism." Place the slips of paper with the letter "B" in the "Family History of Depression" container. The letter "C" slips go in the container labeled "Drinking Habits." Place the

LESSON 4

CHROMOSOMES CONTRIBUTED BY THE MOTHER IN THE EGG

LESSON 4

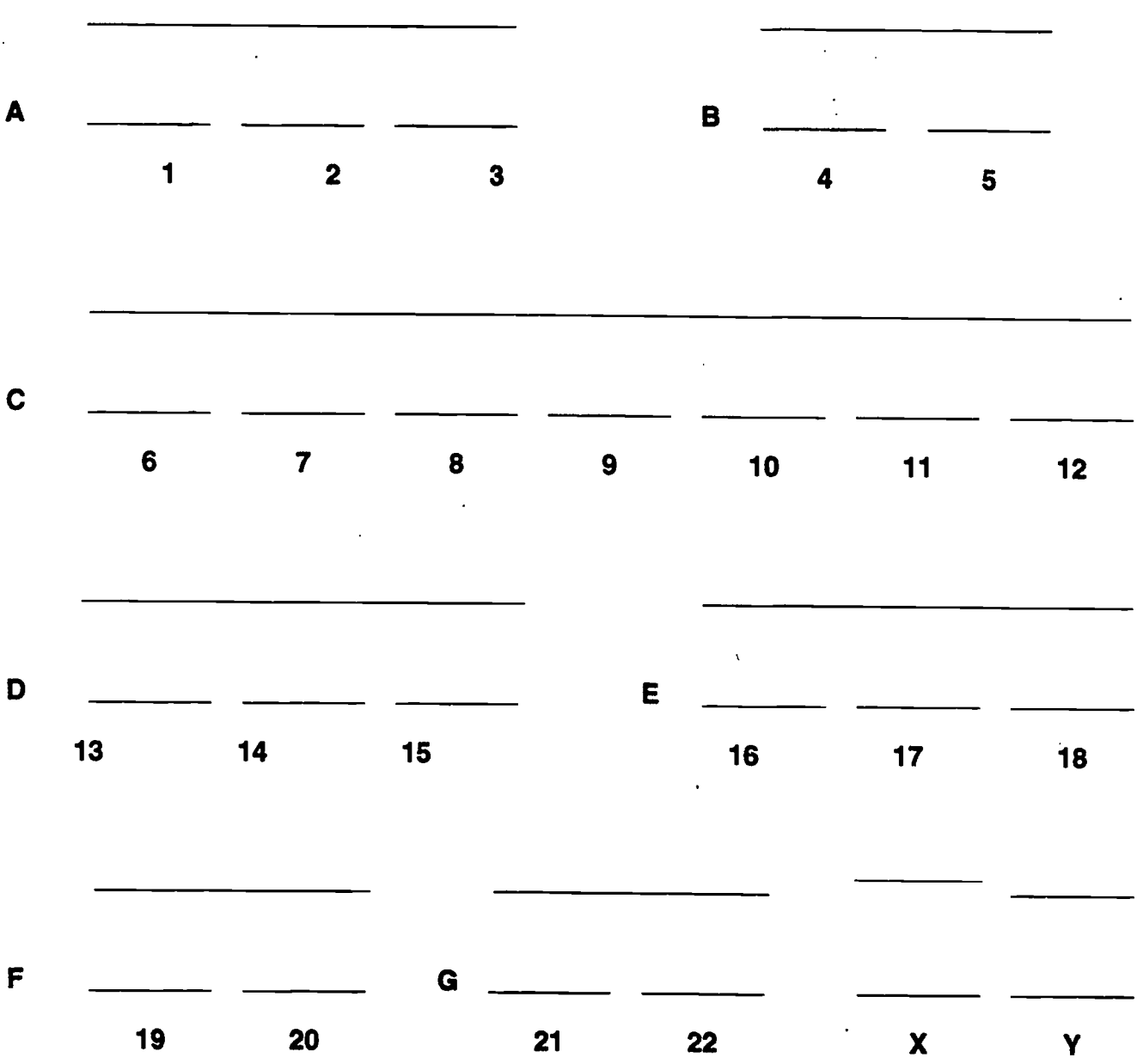
CHROMOSOMES CONTRIBUTED BY THE FATHER IN THE SPERM



LESSON 4

CLINICAL CYTOGENETICS LABORATORY

KARYOTYPE FORM



KARYOTYPE: _____

DATE: _____

LESSON 4

WHAT ARE CHROMOSOMES?

Chromosomes are the packaging units for genes, the small segments of genetic material that transmit traits from generation to generation. One common analogy compares a chromosome to a single strand of pearls. Each pearl can be thought of as a single gene, and the entire necklace as a chromosome.

Normally, a person inherits a total of 46 chromosomes. Twenty-three chromosomes come from the father in the sperm. The other 23 chromosomes are passed on by the mother in the egg. At conception, the egg and the sperm join to form a single cell with a total of 46 chromosomes. These chromosomes are copied and the fertilized egg divides to make two identical cells. The chromosomes in these cells are copied and the two cells divide, creating four cells. This process of cell proliferation continues throughout life, and copies of the original 46 chromosomes are present in all but a few cells in the body.

Twenty-two of the 23 chromosome pairs are similar in both males and females. These chromosomes are referred to as autosomes. They have been assigned numbers based on their length and banding pattern. The longest chromosome is number 1, the shortest is number 22.

The sex chromosomes, X and Y, make up the 23rd pair of chromosomes. The larger of the two sex chromosomes is the X chromosome. The smaller sex chromosome is referred to as Y. A female inherits two copies of the X chromosome, one from her mother and one from her father. A male inherits one X chromosome from his mother, and a Y chromosome from his father.

Each chromosome pair is separated when the eggs and sperm are formed. The resulting reproductive cells contain one sex chromosome and one copy of each autosome. You cannot tell, simply by looking at the chromosomes, which chromosomes were passed on by the mother and which chromosomes came from the father.

When looking at a karyotype, it is also impossible to identify a person who is genetically prone to a particular disease. The genes are not visible under the microscope. New technologies, however, are being developed to study genes, and their effects on development and health. These discoveries will lead to a better understanding of human development, and new treatments or cures for genetic-related health problems.

LESSON 5

FETAL DEVELOPMENT: A NINE MONTH JOURNEY

1. What happens during the first 12 weeks of pregnancy?

2. When does implantation occur?

3. When does the heart start beating?

4. When are the internal organs fully developed?

5. What does the placenta do?

6. Do drugs and chemicals pass through the placenta?

7. What happens between the 16th and 20th weeks of pregnancy?

8. What happens during the last trimester, between the 28th week and birth?

9. What is the role and the responsibilities of males in the childbearing process?

LESSON 5

ALL ABOUT BIRTH DEFECTS

1. Do all babies develop normally?

   If you answered "no," please answer the following questions:

2. What percentage of children do you think are born with birth defects?

3. List all of the different birth defects you know.

4. What do you think caused the birth defects you have listed above?

5. Are any of these birth defects preventable?

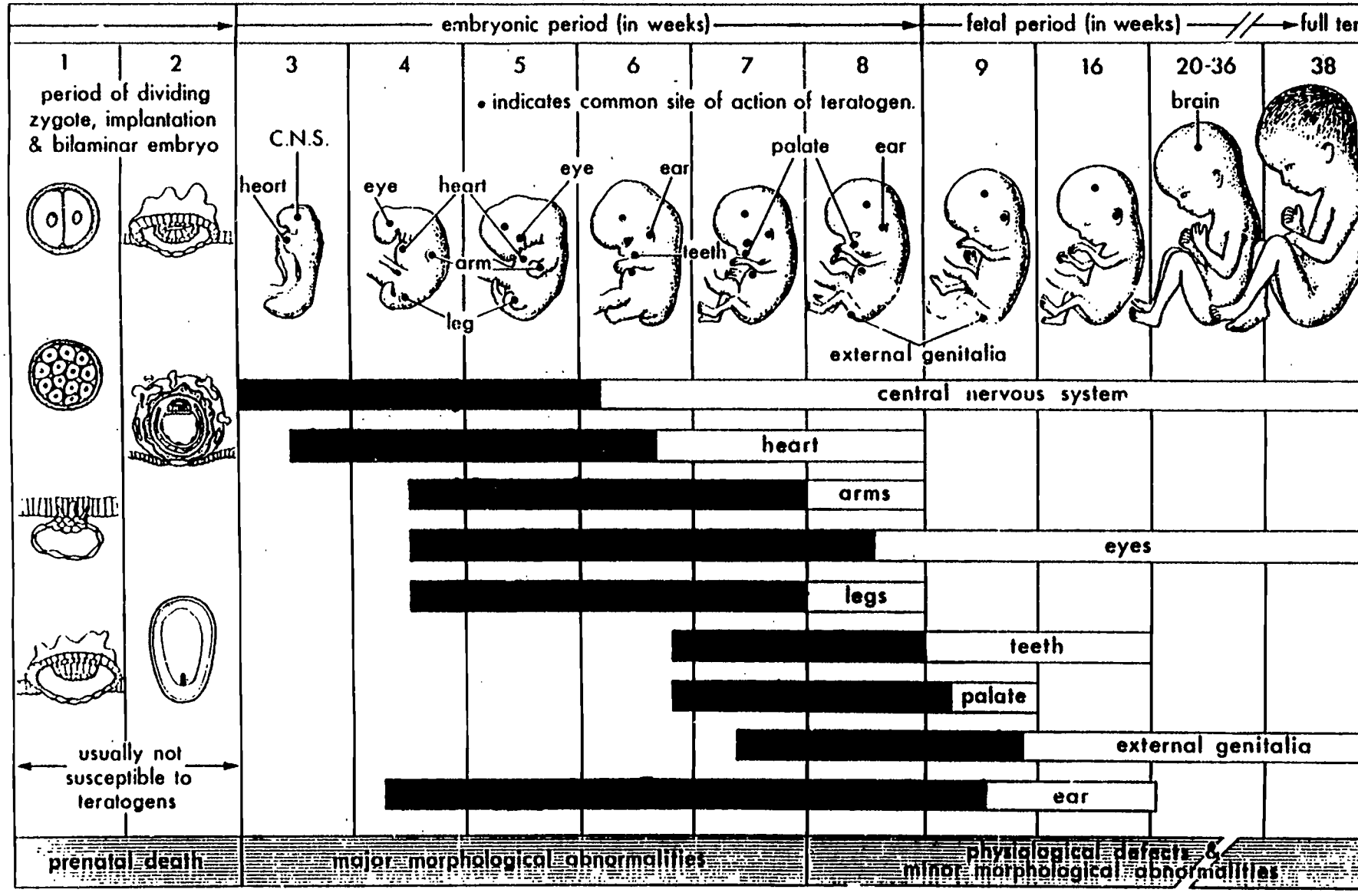
6. Which of the birth defects listed above are not preventable?

7. Can you think of other causes of birth defects?

LESSON 5

CRITICAL PERIODS OF FETAL DEVELOPMENT

(SOLID BARS DENOTE HIGHLY SENSITIVE PERIODS)



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LESSON 6

UNDERSTANDING THE KINSHIP SYSTEM

1. How are relationships defined in the traditional kinship system? For example, who is a person's mother, who is his or her father, who are his or her aunts and uncles?

2. Who kept track of relationships in traditional societies? Who keeps track of relationships today?

3. How were marriage partners chosen?

4. Were there rules governing who a person could or could not marry?

5. Why do you think these rules were created?

   a. Are there any social reasons you can think of to explain why these rules were created?

   b. Are there any biologic reasons you can think of that might explain why people created rules about who a person could marry? You may find it helpful to think about the children born to people who are related. Are these children more likely to have health problems? (Explain your answer.)

LESSON 6

TIOSPAYE KINSHIP SYSTEM

Human relationships:

Having many relatives has always been equated with cultural richness and is very important in the traditional Lakota/Dakota/Nakota societies.

Mother:

A person's biological mother and all her sisters are referred to as mother. They all share in the responsibility of caring for and nurturing the young children. A girl's mothers and one or more respected and/or spiritual elder women also take responsibility for her continued education. They provide teaching and direction as she matures. The women primarily teach in indirect ways. They tell stories to teach morals. They model appropriate behaviors and they lead her through important rituals.

Father:

A person's biological father and all his brothers are referred to as father. These men play an important role in the education of the male children. As a male child becomes older, his fathers, with the assistance of one or more respected and/or spiritual elder men, teach him the morals, values, behaviors and skills necessary to live as a member of the community. They also lead him through important rituals.

Brothers and sisters:

All the children from all the mothers and fathers are brothers and sisters.

Adopted brothers and sisters:

Persons of appropriate ages can be adopted as brothers or sisters. Adopted brothers and sisters are not differentiated from biological brothers and sisters in Lakota/Dakota/Nakota family systems. Adoption of brothers and sisters can occur at any point in a person's life.

Brothers and sisters through marriage and adoption:

All children from an adopted sister or brother become brothers and sisters. Marriage and adoption brings the adopted individual's relatives into relative status with the adopter's relatives at all appropriate levels.

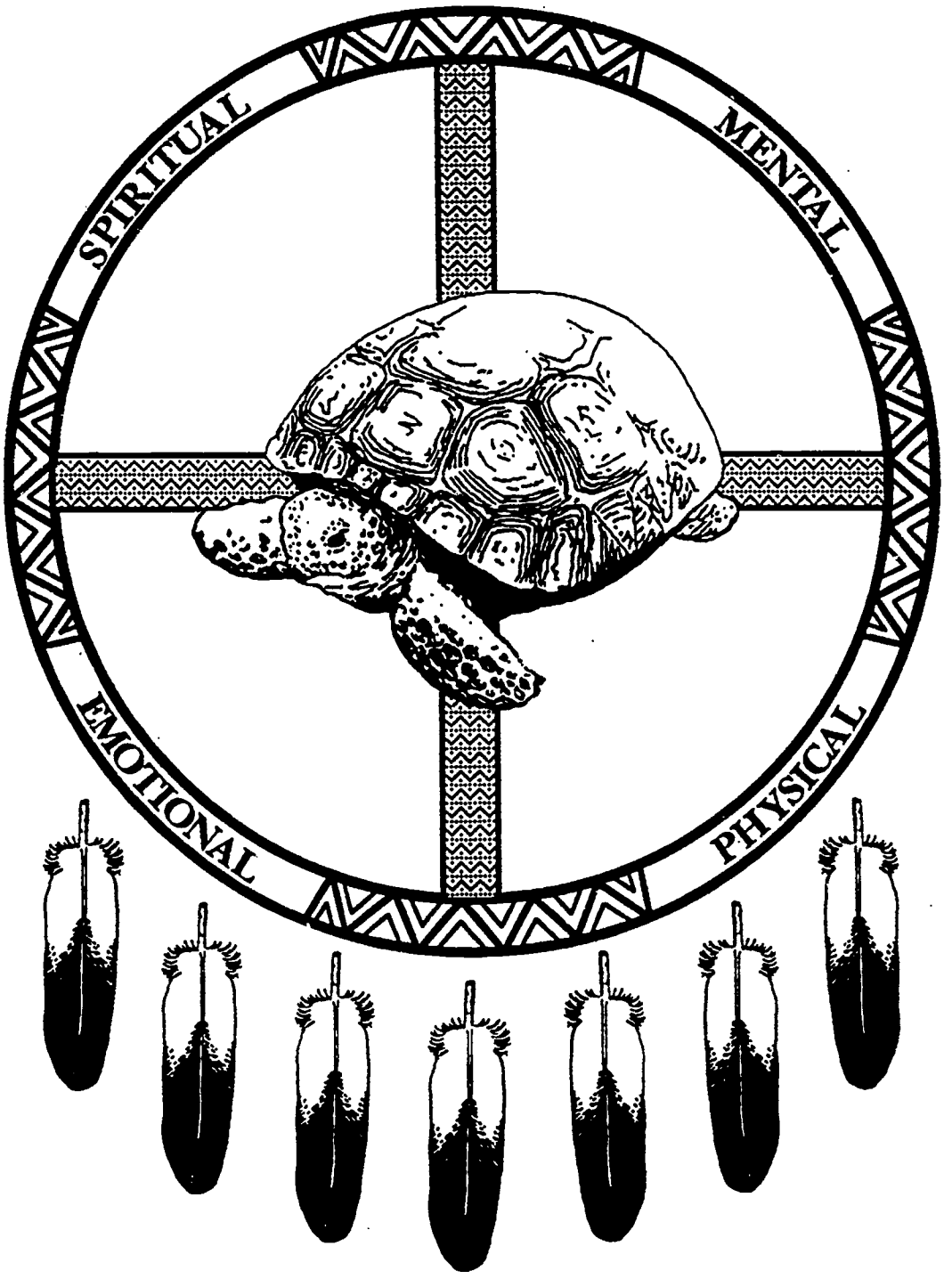
Grandparents:

Grandparents are individuals who are biologically related and any other persons adopted as grandparents It is honorable to call anyone of appropriate age "grandfather" or "grandmother."

Grandchildren:

All children from the family described above are grandchildren. In addition, any child may be called "grandchild" by an elder as a way of honoring the child.

HEALTH IN THE YEAR 2100:
WHAT'S HEREDITY GOT TO DO WITH IT?



APPENDIX

The Indian Burden of Illness and Future Health Interventions

EVERETT R. RHOADES, MD
JOHN HAMMOND
THOMAS K. WELTY, MD, MPH
AARON O. HANDLER
ROBERT W. AMLER, MD

Dr. Rhoades is the Director of the Indian Health Service (IHS). Mr. Hammond is a Health Economist with the Indian Health Service. Dr. Welty is a Medical Epidemiologist assigned to the IHS's Aberdeen Area. Mr. Handler is the Chief of the Vital Events Staff in the IHS's Division of Program Statistics. Dr. Amler, a Medical Epidemiologist employed by the Centers for Disease Control, Public Health Service, is currently on detail to the Carter Center of Emory University. Tearsheet requests to Mr. Hammond at Rm. 5A-27, Parklawn Bldg., 5600 Fishers Lane, Rockville, MD 20857.

Synopsis

This article describes the burden of illness of Indians eligible for services from the Indian Health Service (IHS) and discusses strategies for reducing morbidity and mortality related to those conditions. To improve health to an extent that parallels the IHS's past achievements, the illnesses that now are prevelant among Indians require changes in personal and community behavior rather than intensified medical services. Analysis of these conditions leads to the conclusion that much of the existing burden of illness can be reduced or eliminated.

IHS is responding to this challenge by continuing to ensure Indians' access to comprehensive health care services, by increasing educational efforts aimed at prevention, and by enlisting the support of other government and private organizations in activities that have as their purpose (a) treating diseases if intervention will lessen morbidity and mortality (such as diabetes and hypertension) and (b) encouraging of dietary changes, cessation of smoking, exercise, reduction in alcohol consumption, and other healthy behavior.

DISEASES AND CAUSES OF DEATH among Indians in reservation States today are different from those prevalent two generations ago. Infectious diseases such as tuberculosis and gastroenteritis have been superseded by injuries, violence, cardiovascular disease, alcoholism, diabetes, and mental illness as the major Indian health problems. As a consequence, the Indian Health Service (IHS) currently faces diseases that are less susceptible to correction by the measures adopted in the past 30 years such as provision of safe water, sanitary waste disposal, and primary medical care. In this article, we describe the health conditions that constitute the current burden of illness borne by the IHS service population, identify factors common to many of those conditions, and propose various strategies to diminish the adverse consequences of these conditions.

Indian Mortality and IHS Services

Following the lead of the Carter Center of Emory University (*1*), IHS officials recently initiated analysis of the following health problems to determine the relative contribution of each to the burden of illness borne by Indians:

Unintentional injuries	Chronic renal failure
Violence	Maternal health
Cardiovascular diseases	Pediatric conditions
Musculoskeletal diseases	Cancer
Substance abuse	Dental disease
Digestive diseases	Respiratory diseases
Infectious diseases	Diabetes mellitus

All 14 health conditions are prevalent, costly in terms of both suffering and dollars, and susceptible to known interventions.

Analysis of these conditions has employed cause-specific mortality data for calendar years 1981–83 for Indians in counties where IHS has responsibility and cause-specific patient care data for fiscal years 1982–84 for the IHS Areas. Because deaths are infrequent, the IHS normally uses a 3-year period to analyze Indian deaths. The number of hospital days is the total from 1982 through 1984 provided by the IHS directly, through its tribal projects, and under contract with local hospitals and practitioners. The number of outpatient visits is the total of ambulatory services rendered by IHS

Table 1. Mortality and clinical services related to selected Indian health conditions, 1981–83

Condition	Productive life lost[1]		Deaths		Hospital days[4]	Clinical impressions[5]
	Number of years	Percent of total	Number[2]	Rate[3]		
IHS total (all causes)	94,321	100.0	5,207	695.1	529,376	3,251,170
Unintentional injuries	31,050	32.9	957	116.5	76,277	198,221
Infant mortality[6]	17,593	18.7	270	12.6	...	...
Violence[7]	12,704	13.5	363	43.1	12,015	14,664
Cardiovascular diseases	6,620	7.0	1,362	192.3	32,780	143,723
Alcoholism	6,156	6.5	342	52.7	22,596	13,694
Cancer	4,036	4.3	615	92.9	17,051	6,213
Respiratory diseases	3,428	3.6	329	42.2	41,838	412,641
Digestive diseases	2,163	2.3	170	24.2	54,346	81,315
Infectious diseases	1,667	1.8	101	13.6	16,132	119,808
Diabetes mellitus	882	0.9	165	25.5	16,398	121,071
Chronic renal failure	624	0.7	81	11.7	3,961	3,195
Pregnancy and childbirth	75	0.1	2	0.0	73,634	239,404
Musculoskeletal diseases	...	...	...	...	20,373	133,533
All other	7,322	7.8	450	60.4	141,975	1,763,688

[1] Average years of productive life lost, 1981–83.
[2] Average annual deaths, 1981–83.
[3] Age-adjusted deaths per 100,000 population, 1981–83.
[4] Average annual direct and contract hospital days, 1982–84.
[5] Total primary care provider outpatient clinical impressions, 1983.
[6] Years of productive life lost through infant mortality excludes 73 infant deaths reflected in the other listed causes. The infant mortality rate, which includes those deaths, is based on the number of infant deaths per 1,000 live births.
[7] Self-inflicted and by others.
NOTE: 0.0 rounds to zero.
SOURCE: National Center for Health Statistics and the Indian Health Service Division of Program Statistics.

physicians, physician assistants, and certain other IHS clinical personnel in 1983. Because IHS's California Area Office operates no hospitals and reports no contract hospital workload, the inpatient data that follow cover 11 of the 12 IHS Areas. IHS hospital discharge and day rates are adjusted for patients' age to permit comparisons with the U.S. population and among IHS Areas. Data on patient care funded privately or by third party resources are unavailable, thereby understating Indians' true rates for outpatient and inpatient care.

Indians who are eligible for and who periodically use IHS services constitute the IHS user population. The IHS user population is the denominator employed to calculate rates of inpatient and outpatient services provided by the IHS because the Service has patient care data only on the services it funds. The user population is a more appropriate denominator than the entire population eligible for IHS services. The entire population is estimated from the census of American Indians, Eskimos, or Aleuts who reside in the geographic areas where IHS has responsibility.

Table 1 displays Indian mortality and the number of services provided by IHS for the conditions analyzed. Together, these conditions account for approximately 92 percent of the years of potential life lost (YPLL) before age 65, 91 percent of all deaths, 46 percent of all IHS outpatient visits, and 73 percent of total IHS hospital days. These data reflect the relationship between the morbidity and mortality among the IHS service population and socioeconomic and environmental conditions. They illustrate the important combined effects of living conditions, environment, and personal behavior on the current burden of illness of the IHS service population. Changes in these conditions, therefore, have great potential for preventing many early deaths and reducing much of the burden of illness among Indians.

The YPLL rates presented in table 2 for all causes reflect the excessive numbers of deaths of young Indians in all IHS Areas. Unintentional injuries, diseases of infancy, violence, cardiovascular disease, cancer, and sequelae of alcoholism account for 80 percent of the productive life lost by the IHS service population.

Unintentional injuries and violence. In combination, unintentional injuries, homicides, and suicides account for 25 percent of the deaths among the service population of the Indian Health Service, roughly 1,300 annually. The age-specific death rate for Indians is approximately double the U.S. rate (all races) for the 15- to 45-year age group, and the rate of years of productive life that the IHS service population loses each year from nondisease causes is greater than that for any other cause of death.

Infectious diseases. IHS has had major success in reducing the incidence of infectious diseases among

Table 2. Rates of years of potential life lost before age 65 for selected causes of death, United States, all races 1982, and American Indians and Alaska Natives in Indian Health Service Areas, 1981–83[1]

Geographic area	All causes[1]	Unintentional injury	Infant mortality[2]	Violence	Cardiovascular	Alcoholism	Cancer	Respiratory disease	Digestive disease	Infectious disease	Diabetes	Renal failure
United States	60.6	11.9	11.6	6.6	10.0	1.5	9.3	2.0	2.2	0.8	0.6	0.3
IHS Areas[3]	113.9	37.5	21.2	15.3	8.0	7.4	4.9	4.1	2.6	2.0	1.1	0.8
Aberdeen.....	183.0	55.0	47.5	27.0	12.4	10.3	7.0	6.9	2.5	2.2	1.6	0.8
Alaska	166.2	64.5	31.5	23.4	8.8	4.2	8.0	8.9	1.7	2.6	0.2	0.1
Albuquerque	98.5	31.8	15.3	18.4	3.6	9.1	4.0	2.5	2.9	1.0	0.8	0.4
Bemidji.....	115.7	41.4	23.8	15.0	13.6	2.9	6.7	3.2	0.8	2.2	1.1	0.8
Billings.....	176.5	64.5	25.2	25.0	12.2	17.3	5.1	6.6	4.7	2.5	1.3	2.0
Nashville	113.0	29.5	26.5	12.6	11.7	2.9	7.4	3.8	2.4	2.7	2.1	0.3
Navajo	120.7	48.1	18.5	10.7	6.5	5.7	4.1	4.7	3.9	2.9	0.9	1.0
Oklahoma	72.2	20.5	16.2	6.2	6.6	4.8	4.3	1.9	2.0	1.1	1.0	0.6
Phoenix	134.4	36.3	16.0	25.9	10.3	16.7	4.9	6.2	3.3	3.2	1.9	0.7
Portland.....	109.4	33.2	23.6	16.1	7.5	9.6	3.5	2.6	2.0	1.6	1.3	1.0
Tucson.....	113.9	29.0	22.7	30.7	7.0	14.5	5.5	6.0	8.7	4.4	0.4	2.0

[1] Rates of years of productive life lost per 1,000 persons between birth and 65 years old at the time of death.
[2] Excludes infant deaths reflected in other listed causes.
[3] Data for California are not shown separately because Indian race on death certificates there is underreported. California data are included in the overall Indian rate.
SOURCES: National Center for Health Statistics and the Indian Health Service Division of Program Statistics.

Indians since 1955. Immunization has nearly eradicated the vaccine-preventable diseases, but the mortality rate for infectious diseases is still more than two times higher for Indians than for the U.S. population in general. Inadequate water and sanitation facilities in Indian households contribute to high rates of infectious enteric, respiratory, and skin diseases and to high post neonatal infant mortality. Despite a dramatic 96 percent decrease since 1955, tuberculosis still occurs two to seven times more frequently among Indians than in the U.S. population. Meningitis, hepatitis, and sexually transmitted diseases also cause significant morbidity. Adequate housing, sanitation, water supplies, and new vaccines, not medical services, are the key to reducing these diseases among Indians.

Maternal and child health. Maternity care accounts for the majority of hospital admissions and outpatient visits at IHS and its contract care facilities among Indian women. Later pregnancies and more frequent pregnancies, both risk factors, occur more often among Indian women, although overall maternal mortality rates are similar for U.S. and Indian women. The prevalence of diabetes in certain tribes creates the need to ensure excellent preconception control of the disease.

The overall infant mortality rate is no longer higher for the IHS than for the U.S. population; the birth trauma and asphyxia rates, however, are 2.5 times the U.S. rates, and fetal alcohol syndrome occurs up to six times more frequently. The disproportionate number of Indian deaths after the first 27 days of life reflects adverse environmental factors rather than the events of pregnancy and birth. Deaths from gastroenteritis (three times the U.S. rate) reflect poor sanitation and living conditions and perhaps delays in obtaining medical care. Sudden infant death syndrome is almost twice as common among American Indians and Alaska Natives as in the U.S. population.

Chronic diseases. Indian life expectancy at birth (71.1 years for reservation States, 1979–81) is approaching that for the United States as a whole (73.7 years, 1980). As the Indian lifespan increases, the disease pattern of the IHS service population more closely resembles that of other Americans. For example, even if the age-specific incidence of cancer in the IHS service population remained the same, the increasing proportion of elderly would increase the number of patients with cancer. Age-specific rates of cardiovascular disease in the service population tend to resemble those in the general population, and diseases of the heart are now the leading cause of death for Indians. Although cancer is the third leading cause of death, age-adjusted cancer death rates among Indians are only 70 percent of the U.S. rate. Possible explanations include lower prevalence of smoking and other cancer risk factors, greater responsiveness to treatment of the types of cancer which afflict Indians, prompt seeking of services, and the availability of appropriate services.

Approximately 15 percent of the IHS user population aged 45 or more years is diabetic, and half of the adults ages 45–64 in some southwestern tribes are diabetic. Age-adjusted diabetes death rates are more than two times higher for Indians than for the general population. That fact alone, however, does not convey the relationship of diabetes to other health problems—coronary heart disease, blindness, peripheral vascular disease, and kidney failure. In fiscal year 1983, diabetes was second only to upper respiratory infection as the leading reason for IHS outpatient visits, and 76 percent of all IHS hospitalizations for lower extremity amputations involved diabetes.

Musculoskeletal diseases, including arthritis, low back pain, and diseases of the spine and connective tissue, are frequent causes of severe illness and disability. The risk factors of obesity, diabetes, and alcoholism put Indians at high risk for low back pain, and the prevalence of stress, another risk factor, may be increasing with acculturation and sociocultural development.

Oral diseases. Inadequate access to preventive services and basic dental care is partly to blame for the high rates of tooth decay and gum disease among American Indians and Alaska Natives. Indian children have more decayed, missing, and filled teeth than other U.S. children, and nursing caries in preschool children are common.

Substance abuse. Indians have the highest frequency of drinking-associated problems of any ethnic group (*2*). Two-thirds of the alcoholics under treatment in Alaska, for example, are Eskimos and Aleuts; they constitute one-seventh of the State's population but experience three-fifths of all reported alcohol-related deaths. Indian alcohol-related deaths occur at more than four times the age-adjusted rate for U.S. population, and alcohol misuse results in a rate of years of productive life lost nearly five times that of the U.S. population. The increase over the past 15 years in the age-specific incidence of fetal alcohol syndrome is important because many mothers who produce one affected child also produce others (*3*). Although Indians are infrequent abusers of intravenous drugs, abuse of inhalants and other drugs, such as marijuana and stimulants, is common among Indian adolescents.

Respiratory diseases. Influenza, pneumonia, and chronic obstructive pulmonary disease are the most common respiratory diseases among American Indians. Mortality rates for influenza and pneumonia have decreased 82 percent since 1955, but rates for all respiratory diseases suffered by Indians are still 31 percent higher than the U.S. rates, and the rate of years of potential life lost is three times as high. Respiratory disease is closely linked to smoking prevalence. Occupational exposure to hazardous materials (asbestos, uranium) has not been a problem except in small groups, but the hazards may increase due to building and mining on Indian lands.

Digestive system diseases. The three major digestive diseases among Indians—gallbladder disease, appendicitis, and ulcers—together account for only a small part of the IHS workload and mortality in the IHS population. Overall, rates of Indian mortality and morbidity attributable to these diseases compare favorably with the U.S. rates; the rates for gallstones and cholecystitis among Indians, however, are up to six times higher than U.S. rates. Since ulcer disease is linked to both smoking and stress, acculturation may tend to increase the rate among Indians, which is currently slightly below the U.S. rate.

Proposed Interventions

Most of these conditions are largely unnecessary and unnecessarily costly, and deaths related to them may be regarded as "premature." Because they typically lead to health service encounters, these conditions are responsible for a large portion of IHS costs. Each condition has been analyzed in light of what can be done to reduce it by employing existing technology and participation by tribal, State, and local governments and the private sector. The interventions recommended in response to these health problems are summarized in the box. Some consist of objectives for the nation tailored to the IHS population (*4*). For conditions such as low birth weight and cancer, the challenge for the IHS is to maintain the currently favorable rates for Indians. Indians compare so poorly for such conditions as gastroenteritis, however, that the 1990 objectives for the nation are unrealistic for the IHS. At best, such national objectives become long-range goals for the Indian population: at existing levels of environmental health services, for example, the disproportionately high rates of enteric diseases and avoidable infant deaths will persist.

The infant mortality rates presented in table 3

Summary of objectives proposed for the Indian Health Service

Health status

Lower specific morbidity and mortality rates for the IHS service population

Awareness

Ensure that the IHS service population has a general knowledge of

- Stress reduction
- Parenting
- Nutrition
- Sanitation
- Symptoms requiring treatment
- Sources of information and care
- Specific health conditions; for example, diabetes
- Availability of mutual support and self-help for selected conditions, such as alcoholism

Ensure that IHS personnel have

- Appropriate training and experience
- Knowledge of Indian culture
- Continuing education
- Skills for taking patient histories that detect particular conditions
- Structured treatment protocols
- Self-awareness as role models

Advocate Indian health issues

Risk factors

Collaborate with tribal, other government, and private organizations to encourage healthful behavior including smoking cessation, diet, exercise, avoidance of substance abuse, injury avoidance, positive parenting attitudes, and so forth

Services

Screen for such conditions as hypertension and diabetes

Immunize the Indian population against vaccine-preventable diseases

Ensure universal accessibility to preventive primary care services for Indians, including well-child care, developmental screening, and prenatal care

Tailor specific programs to Indian culture

Coordinate regional service delivery with other community health resources

Surveillance

Establish and continue systems to monitor particular health conditions, including consistent reporting of notifiable diseases

Conduct periodic health status and health care delivery surveys

Improve current data gathering

Track specific health problems—for example, diabetes, hypertension, and pregnancy—and events indicating potential health or service delivery problems

Establish standards for all IHS programs and conduct periodic reviews

Table 3. Estimated preventable Indian infant mortality

Age at death	1 IHS 1982–84	2 U.S. 1983	3 Parity difference	4 U.S.[1] achievable	1–4 Preventable difference
Infant mortality[2]	11.7	11.2	0.5	4.0	7.7
Neonatal mortality[3]	5.2	8.3	−2.1	2.8	2.4
Postneonatal mortality[4]	6.5	3.9	2.6	1.2	5.3

[1] "Carter Center Interim Report: Closing the Gap." Estimate of the lowest achievable mortality rates. The achievable rate is the rate that could be realized nationwide under the best possible circumstances, including reduced maternal risk factors, optimal services, and so forth.
[2] Deaths in the first year of life per 1,000 live births.
[3] Deaths during the first 27 days of life per 1,000 live births.
[4] Deaths of infants aged 28 days to under 1 year of life per 1,000 live births.
SOURCES. National Center for Health Statistics and IHS Division of Program Statistics.

illustrate the concepts of parity and preventable gaps in Indian health status. The first and second columns present Indian infant mortality rates for Areas served by IHS for 1982–84 and the corresponding U.S. rates for 1983. The difference, which appears in the third column, is the amount that the Indian rate would have to be reduced in order for it to equal the U.S. rate. If Indian death rates for each age and sex group were the same as for the U.S. population, there would be 1,200 fewer Indian deaths annually—23 percent of all deaths within the service population. The last column of table 3 indicates that Indian rates are almost triple those which could be realized if all available scientific and technological capabilities could be brought to bear, which would result in preventing more than 8 deaths per 1,000 live Indian births. That much of this burden can be prevented by applying existing knowledge suggests that the IHS should aim for the reduction and eventual elimination of all preventable disease, not simply for parity with the nation.

Future IHS efforts will be focused on (*a*) ensuring adequate levels of demonstrably effective prevention and clinical services and (*b*) attacking specific health problems with targeted programs of health promotion and disease prevention.

Alcohol abuse. Alcohol is the leading and perhaps the most costly risk factor among Indians. Alcohol misuse underlies many major causes of Indian deaths in reservation States and contributes to an array of physical conditions treated by the IHS. Four of the top 10 causes of death among Indians are alcohol-related—injuries (18 percent of all deaths), chronic liver disease and cirrhosis (5 percent), suicide (3 percent), and homicide (3

'The disproportionate number of Indian deaths after the first 27 days of life reflects adverse environmental factors rather than the events of pregnancy and birth . . . . Sudden infant death syndrome is almost twice as common among American Indians and Alaska Natives as in the U.S. population.'

percent). An estimated 75 percent of all traumatic deaths and suicides among Indians involve alcohol, making it a major contributor to premature death. Strategies should be developed to target alcohol abuse among Indians; elements of such strategies should include public education aimed at heavy drinking, especially during pregnancy, and attempts to enhance community support for those at risk for alcohol problems. The IHS role will be largely limited to that of facilitator for the community. More and more Indian communities are beginning to recognize that only their members can adequately deal with the problem.

Obesity. The recent upsurge of obesity in some Indian populations makes them disproportionately susceptible to diabetes, hypertension, and cardiovascular disease; all conditions that are aggravated by sedentary living and high-fat diets. Manual labor, hunting, and travel on foot have given way to mechanization, and highly refined processed foods have supplanted a high-fiber diet. Most obesity among Indians can be controlled by diet and exercise. The IHS, therefore, should initiate aggressive interventions to prevent the projected increased incidence of diabetes by almost a third among the IHS user population within the next decade. At present, community fitness programs appear to offer the greatest opportunity for attacking this problem.

The Zuni Diabetes Project, for example, is trying to prevent diabetes on the Zuni Indian reservation through weight reduction and minor changes in eating habits. Seventeen aerobic exercise classes are offered each week at central locations within the pueblo. Since July 1983, 140 Zunis have been involved in the project; most had been obese and sedentary for their entire adult lives. Not only are these formerly nonathletic adults now embracing fitness, but 36 participants have lost an average of 12 pounds each. The 12 diabetics who required medication to control blood sugar levels at the beginning of the program have all lost weight and been taken off medication (*5*).

Tobacco use. The burden of illness that the use of tobacco creates for Indians varies by locale; Indians in the Southwest, for example, smoke little relative to those in northern States, including Alaska. Smokeless tobacco also is an ascendent problem among Indian youth. In Areas where smoking and using smokeless tobacco are uncommon, IHS should encourage Indians not to start, and in Areas where tobacco use is widespread, Indians should be encouraged to stop.

In 1983, the Keams Canyon Public Health Service Indian Hospital became totally free of tobacco smoke, and with the approval of the tribes, nearly all IHS facilities are now smoke-free.

Maternal and child health. The age of the mother, birth interval, and number of children affect fetal development and birth outcomes. Improved family planning services, prenatal care that includes preconception control of diabetes, and education about nutrition and health risks can improve maternal and infant mortality rates. Postneonatal mortality and infectious disease statistics likewise reflect the continued need for environmental health services.

Program planning. In recognition that sound epidemiologic studies are essential to the refinement of its present health care delivery system, IHS is developing the structure and procedures necessary to conduct regular epidemiologic analyses of the use of health services. The IHS will develop standards that permit refined analyses of community health problems and determine where to target IHS resources. The IHS will also develop epidemiology-based models with the ability to predict the impact of developments in the private and public health service sectors. Specific service, enrollment, cost, and revenue assumptions will enable IHS management to evaluate its policy alternatives routinely.

Additional headquarters and field positions are being filled by medical epidemiologists; analytic and administrative procedures to ensure routine epidemiologic analyses by IHS will be developed; and the results of these analyses will be integrated with IHS's ongoing statistical, planning, budget-

ing, and evaluation activities. An immediate benefit will be more appropriate allocation of resources based on need, rather than allocations made according to historic demand. Allocation based partly on epidemiology will improve the existing system.

Health professionals representing a variety of disciplines and IHS Areas have developed a strategy to improve Indian health status over the next several years. The proposed strategy consists of 14 goals derived from national objectives for 1990 that focus on the most common health problems of Indians. Most Areas are already addressing the national objectives relevant to IHS, and many have subsequently incorporated elements of the proposed strategy in their program plans. By concentrating on health status, the strategy provides clear direction for future programmatic efforts but avoids the additional reporting burden that would be created by process-oriented objectives; achievement of IHS goals can be monitored from data that are generated by existing data systems.

Extramural activities. The creative involvement of other organizations can provide substantial technical and financial assistance to the IHS and its service population. None of the three health block programs, for example—preventive health; maternal and child health; and alcohol, drug abuse, and mental health—has a specific set-aside either for tribes that previously had received categorical funds or for nonreservation Indian health programs. In the case of block grants, IHS will serve as an advocate of Indian interests with State and local governments to help ensure that, as citizens, Indians receive appropriate types and amounts of services.

IHS also proposes to expand its collaborative efforts within the Health Resources and Services Administration and with the Centers for Disease Control, the National Institutes of Health, Bureau of Indian Affairs, and other Federal agencies to provide funding, technical assistance, training, and consultation.

The joint IHS–Centers for Disease Control attack on hepatitis B in Alaska Native villages is an example of the potential of such collaboration. Beginning in 1981, all Native residents were screened; their communities were ranked by the incidence of the disease; and all susceptible residents, including newborns, were vaccinated. The incidence of hepatitis B dropped from 240 to 10 new cases per 100,000 population after 2 years, and it is expected that hepatitis B and its lethal complications will eventually disappear in Alaska Native villages.

IHS has enjoyed similar success in collaboration with other Federal, State, and local organizations to promote Indian safety, curb alcohol and substance abuse, and so forth. Likewise, leaders of individual IHS programs and Areas will actively explore alternative and innovative ways of participating with the private sector in consortia that share facilities and coordinate services and otherwise involve business and industry, civic and social groups, and broadcast and print media in IHS activities.

Applied research. Steady improvement of Indian health status requires the deliberate, coordinated application of existing technology and research to programs that promise substantial dividends. Ways to effect behavioral and environmental change are increasingly understood; many causes of chronic disease are known; some effects of health programs have been isolated from the results of other factors; and the impact of modifying several proven risk factors has been demonstrated adequately. What remains is to apply this knowledge to the unique social and cultural circumstances in which many Indians live. As regards alcohol and substance abuse, for example, IHS needs to improve the level of knowledge about alcohol and drugs and their prevention and to rehabilitate casualties more successfully (*6*). IHS's research agenda therefore includes participation in special information-gathering activities such as the National Medical Expenditure Survey conducted by the National Center for Health Services Research and Technology Assessment.

Information systems. Historically, lack of complete information made it difficult for IHS to manage its resources efficiently and to respond readily to requests for data about its operations. IHS has adopted a strategic plan for information systems that is designed to promote effective and efficient resource management and delivery of health care services. The plan has defined, as a basic requirement of information systems, the need to satisfy both the clinical and administrative needs of the organization. The plan also focuses on several broad initiatives that include improving the usefulness of financial management information systems, support of the efficient use of third party resources, and promotion of local control and responsibility for data. As a result, known deficiencies in data collection are being corrected, and

improved methods of data retrieval and presentation are being implemented.

The Resource and Patient Management System (RPMS), for example, represents a major departure for IHS toward decentralization of data system operations; when fully implemented, RPMS will place multiuse computers at IHS Areas, Service Units, hospitals, and health centers to support patient care, administration, hospital and clinic management, and local data collection and processing. Other systems, such as patient registration already in use by the IHS, are generating much of the information necessary to plan services, allocate resources, and monitor program activities.

Conclusion

The recommendations produced by this analysis of Indian health conditions and possible interventions will be used to stimulate widespread discussion about ways to effect desired service and environmental and behavioral changes. Implementation of the recommendations is both practicable and consonant with the longstanding IHS goal of raising Indian health status to the highest possible level. The proposed interventions provide direction to IHS services in the future and strongly support the Service's longstanding emphasis on disease prevention, primary care, and health promotion. Prevention services that include family planning, immunization, and water fluoridation are highly responsive to the needs of IHS's relatively young service population. Services particularly important to the specific health problems of the Indian population are early prenatal care, well-child care and developmental screening, hypertension screening and treatment, and education about diet, weight control, exercise, smoking, alcohol abuse, and vehicular and environmental safety. When these services are not readily available or accessible, the IHS has a continuing responsibility to develop the capacity to fill in the gaps.

The next step is for IHS and Indian tribes to develop a course of action that formally evaluates, according to the following principles, each recommendation within the context of local health problems:

1. Full participation by tribal governments is essential.
2. There is multilevel, multidisciplinary responsibility within IHS to ensure that each objective is fully considered.
3. Areas must have maximum flexibility in tailoring program actions responsive to each objective to local conditions.
4. Each Area is responsible for ensuring that its services address each objective satisfactorily.

Deliberation about the national health strategies of the United States, of other countries (*7*), and recent efforts at risk factor control (*8*) will produce insights useful in weighing the utility, costs, disadvantages, and benefits of each recommendation.

As an advocate for improved Indian health, the IHS will work with tribes, other public agencies, and private organizations to create a more healthful environment. Improved education and recreation, meaningful employment and, ultimately, enhanced self-esteem, will make IHS interventions most successful. The results of many of these efforts, however, will not necessarily be manifest in the short term. Motor vehicle accident deaths will decline as soon as Indians use seatbelts regularly and control drinking, but cancer rates related to cigarette smoking may take years to respond. Nonetheless, successful health promotion and disease prevention programs eventually should reduce the demand for therapeutic services and substantially improve the quality of life for Indian people.

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