GENETIC SEEDS OF DISEASE

How to Beat the Odds

Dear Reader,

How often these days do you read that genes cause cancer? Probably nothing in biomedical science deserves more attention. In my view, it even needs attention in nutrition newsletters. Why? Because there's a peculiar line of reasoning going around that goes something like this: If genes are primarily responsible for determining when and what kinds of cancer we get, then what difference does it make what we eat?

Essentially, this is a very fatalistic view. Further, if this notion about genes is accepted as being valid, then many may be tempted to simply trade in "bad" genes for "good" genes. Regrettably, this is an idea that is very much alive in science and kicking up considerable funding support. Rather than succumbing fatalistically to gene research reports or trading our genes haphazardly, let's start to think more seriously about preventing cancer, quite literally, by getting at its "root."

Where is the Money Going?

I've previously commented in this newsletter on some of my concerns about this overemphasis on gene research (see August 1995 issue). While some of the new gene discoveries may provide hopeful opportunities, many are clearly harmful—especially when people are not properly informed. Tragically, some people have become so distraught after finding out that they or their loved ones have a particular bad gene, that they then take unconscionable action—I've been called twice by mothers seeking advice on possible mastectomies for their daughters.

Why do NIH and similar funding agencies provide far more research funding for investigations on the genetic control of disease rather than for the nutritional control of disease? Whose purpose is being served here? Consider this: Improper diets, when compared to genetic backgrounds, are responsible for perhaps 30–40 times more cancers (at least), yet genetic research, according to my estimate, gets 30–40 times more funds.

On the more hopeful side, however, I would say this. If genes only indicate potential, not certainty for disease, then knowledge of troublesome genes may motivate people to be more careful about their dietary and lifestyle habits.

New Study Reveals Major Research Flaws

My reason for returning to this subject yet again in this newsletter is a very important study published this past spring. The findings of this study make a particularly compelling point that genes matter little, practically speaking, in the causation of cancer. I can hardly imagine a study being more devastating to the idea that genes are responsible for cancer risk. The study I have in mind was undertaken by scientists within the National Cancer In-
Editorial — continued from page 1

stitute (NCI) of the NIH. The study group was headed by a very prominent and experienced senior investigator, Dr. Robert Hoover.

The investigators asked a very simple question about whether identical twins were more likely to get cancers than were other sibling pairs. You see, identical twins have essentially the same genes, thus they should get the same cancers if their genes are the primary cause. According to this reasoning, if one member of the twin pair gets a specific cancer, then the second member, who carries the same genes, should also get it.

The extent to which the same cancers occurred for both members of an identical twin pair (same genes) was compared with the extent to which both members of a non-identical twin pair got the same cancer. The genes of non-identical twin pairs are no more alike than any other pair of siblings.

The findings from this study are unusually revealing. First, this study was the largest of its kind ever undertaken. Cancer deaths among 5,690 identical twin pairs were compared with cancer deaths among 7,248 non-identical twin pairs. Second, careful attention was given to include the details of record keeping so that only those records with verifiable information would be used in the analysis. It was, in my view, a well-designed and well-executed study.

Briefly, without getting into all the detailed findings, these scientists concluded that "... knowledge of the cancer mortality experience of an individual will not commonly have strong predictive value for the experience of another individual who has identical genes."

**Probabilities vs. Realities**

What this conclusion says, basically, is that genes do not matter, at least for any significant proportion of cancers. To be specific, the probability of both identical twins getting cancer was only 1.4 times that for both non-identical twins getting cancer, a result of only borderline statistical significance. Even for one of the cancers

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**Cancer Deaths in Twins**

<table>
<thead>
<tr>
<th>Selected Cancers*</th>
<th><strong>Identical Twins</strong></th>
<th><strong>Non-Identical Twins</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><em>one</em></td>
<td><em>both</em></td>
</tr>
<tr>
<td>Stomach</td>
<td>27</td>
<td>0</td>
</tr>
<tr>
<td>Colon or rectum</td>
<td>90</td>
<td>1</td>
</tr>
<tr>
<td>Liver</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>Skin</td>
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<td>0</td>
</tr>
<tr>
<td>Prostate</td>
<td>29</td>
<td>0</td>
</tr>
<tr>
<td>Brain or central nervous system</td>
<td>23</td>
<td>0</td>
</tr>
<tr>
<td>Non-Hodgkin’s lymphoma</td>
<td>41</td>
<td>0</td>
</tr>
<tr>
<td>Leukemia</td>
<td>17</td>
<td>0</td>
</tr>
</tbody>
</table>

* Smoking-associated cancers not shown

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*T. Colin Campbell is the Director of the Cornell-Oxford-China Diet and Health Project. He was trained at Cornell (M.S., Ph.D.) and MIT (Research Associate) in nutrition, biochemistry, and toxicology. He presently holds the endowed chair of Jacob Gould Schurman Professor of Nutritional Biochemistry at Cornell University.*

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Dietary Fiber: Preventing Cancer in China

by Dr. T. Colin Campbell

The cancer-prevention properties of dietary fiber are hardly a secret. Indeed, in modern times the late Dr. Denis Burkitt (of Burkitt’s Lymphoma fame) made the so-called “fiber story” famous in his work among native Africans. This British researcher, with his colleagues Alan Walker and Hugh Trowell, spent many years in Africa tirelessly working to better understand why diseases typically found in Western countries were rare in Africa. What he discovered made worldwide news. Diets high in fiber appeared to be associated with reduced cancer incidence of the large bowel, as well as the incidence of many other diseases common to Western countries.

Looking at the Big Picture

In rural China, we took this lead to further explore the fiber–cancer association among people who were consuming very large amounts of fiber compared to Americans. In so doing, we incorporated two other understandings. First, dietary fiber is not a single chemical entity. There is an almost unlimited variety of dietary fibers. Second, our knowledge about the unique effects of specific fibers was, and still is, very sparse. So, to get a better feel for the big picture, we measured the consumption of 14 different kinds of dietary fiber.

As expected, we first noted that the average intake of dietary fiber was at least double, perhaps even triple, the intake in the U.S. Second, the rates of colon and rectal cancers in China were only about one-half the rates in the U.S., although in some areas this cancer was almost nonexistent.

Also as expected, we did not see any special differences in the cancer-related effects of these different fibers. For each of the 14 different fiber types, there was an inverse correlation. That is, the higher the fiber intake, the lower the rates of colon and rectal cancers (although almost all of these correlations were somewhat less than statistically significant).

Finally, we found a very strong association between colon cancer and the presence of the water-borne parasite that causes schistosomiasis. This little creature is carried by snails and has caused enormous epidemics in some tropical countries. In conclusion, it is our view that Burkitt’s dietary fiber theory remains intact and it is worth-while to consume high-fiber diets, not just to prevent large bowel cancers but also to promote a variety of other positive health conditions. And finally, remember that dietary fiber can only be found in foods of plant origin, once again pointing to the value of consuming plant-based diets.

Keeping Your Dietary Balance

By Dr. T. Colin Campbell

Because there are different nutrients in different parts of the plant, try to make sure you are getting some food from each part. One way to do this is to visualize the whole plant while making your food selection. For example, you can start with the roots of a plant and move upwards through the stems, leaves and flowers to the fruits and seeds.

Remember, each nutrient provides its own special function for the body, but no nutrient acts independently of others. All of the nutrients should be present in the diet, though in varying quantities, for the body to stay healthy.

ROOTS beets, potatoes, carrots, onions
STEMS celery, rhubarb, bok choy, asparagus
LEAVES lettuce, spinach, kale, chard
FRUITS apples, oranges, bananas, tomatoes
SEEDS shelled peas, shelled beans, grains
FLOWERS Brussels sprouts, broccoli, cauliflower
Making Waves in School

I have changed my favorite food from candy to broccoli.

by Antonia Demas, Ph.D.

As our nation’s school lunch program hovers at the brink of possible extinction in the U.S. Congress, a counter-effort has quietly arisen within the ranks of the United States Department of Agriculture. The USDA’s new School Meals Initiative for Healthy Children establishes a host of nutritional objectives in line with the new Dietary Guidelines, including the lowering of fat content for school children from 40 to 30% by 1998. Given the many political struggles that historically have beset our nation’s school lunch program, the two recent opposing legislative efforts are indeed ironic but not surprising.

Feeding Empty Stomachs

Once legislation for mandatory schooling was passed more than a century ago, and children started showing up with empty stomachs, it became immediately obvious that someone would have to feed them. Among the first to sound the battle cry for the schools to step in was Ellen H. Richards, who developed a food lab to educate the public about nutrition, and who steadfastly worked to establish school lunch programs. Richards had the foresight to realize that schools needed to teach more than the three R’s. If children were going to have the capacity to concentrate, schools also needed to address health and lifestyle issues.

Although various philanthropic groups heeded her call by creating several pilot feeding programs by the turn of the century, the U.S. government was characteristically slow to act. Only when one-third of those who enlisted for the military were rejected because of diseases ascribed to malnutrition did a shocked public convince the government to wait no longer. The result: in the form of the National School Lunch Program (NSLP), became law in 1946. Its dual objectives have been to make food available to hungry children while helping farmers sell surplus food.

Pending present legislative outcomes, the National School Lunch Program stands today as one of the largest feeding programs in the world. It is available to 92% of all U.S. children, with students from low-income households receiving school meals free or at reduced prices. Low-income children depend upon the NSLP for one third to one half of their nutritional intake. Over 25 million children eat school lunches daily in the United States, while another 25 million either bring lunch from home, buy from outside vendors (such as McDonald’s), or else skip lunch altogether. The USDA subsidizes school lunches by providing cash reimbursements and entitlement commodity foods. Unfortunately, typical school lunch foods such as processed cheese, butter, and hamburger are very high in fat, while other low-fat commodities, such as dried beans, lentils, bulgur wheat, and brown rice, remain stockpiled. The main reason these plant-based commodity foods are not in greater use is the faulty perception that children will not eat them. Educators must realize that children receive important messages about food (either by accident or design) while eating in school cafeterias. It is time to begin to use this valuable opportunity to help teach them about nutrition.

What Can Be Done — The Trumansburg Study

One way to get children to accept a healthier diet without added expense is to provide more plant-based commodity foods. Research done last year in Trumansburg, a rural community in upstate New York, demonstrated that children will eat diverse foods once they learn about them in the classroom. New foods were introduced as part of science, math, geography, and social studies classes. All recipes incorporated the more healthful commodities. The children cooked these foods with their peers and sampled them in the classroom. Within a week of classroom discussions, the same food was served as part of the standard hot lunch.
Spelling Trouble at an Early Age
by Charles R. Attwood, M.D., F.A.A.P.

In 1953, as I completed the final stages of my first autopsy as a medical student, I saw something I’ll never forget. While sectioning the heart of a nine-year-old girl who had died suddenly and unexpectedly of meningitis, I found yellow deposits within one of her coronary arteries. “Take a good look,” the pathology professor said, “you’ll probably never see this again during your entire career.” The yellow deposits proved to be cholesterol.

That same year, it turned out, the vast majority of apparently healthy young American soldiers killed in the Korean War—their average age was 22 years—were found to have coronary artery fatty deposits. Their Asian counterparts had none.

Today, 42 years later, nearly 50 million American children have abnormally high blood cholesterol levels, which have led to fatty deposits within their coronary arteries as early as age 3. These deposits grow thicker during the teens, and virtually all young adults have them by the age of 21. By the age of 12, two-thirds of all children, like the little girl on my autopsy table, have the beginning stages of coronary disease, which eventually accounts for a third of all adult deaths.

These early stages of coronary artery disease were documented and related to children’s dietary fat by a 23-year prospective study of children in a small Louisiana community. In 1972, the Bogalusa Heart Study began following the eating habits of children through early adulthood. Before they had finished, more than 14,000 children had been studied. Dr. Gerald Berenson, director of the study, concluded that coronary disease definitely begins very early in childhood and that dietary fat is directly linked to cholesterol levels and coronary artery deposits as found by examining children who had died in accidents.

Coronary artery disease doesn’t just magically appear in adults, he reported, but develops over a 20-40 year period. The study detected potentially fatal heart disease symptoms as early as 6 months of age. The fact that American children typically get 38-42% of their calories from fat undoubtedly leads to high cholesterol levels and coronary lesions. Studies show that new coronary artery deposits appear at levels as low as 30%, the recommended upper acceptable level for children set by the National Institutes of Health, the American Heart Association, the American Medical Association, and the American Academy of Pediatrics.

We have known for over 40 years that one out of two children in Western industrialized nations will develop heart disease as adults. One out of three will be a victim of blood (fat)-related cancer. These, along with other diseases related to dietary fat, such as stroke, hypertension, and diabetes, account for 70% of all U.S. adult deaths. By contrast, population studies have shown wherever children consume a low-fat diet of 10-20% of calories, such as rural China or Japan, adult coronary disease is rare.

Next month, I will introduce a step-by-step program for greatly reducing dietary fat in your children’s meals. Remember, the key to a healthy adult body begins when lifelong eating habits are just starting to form.

Charles R. Attwood, M.D., F.A.A.P., directs one of the nation’s largest solo pediatric private practices, with a primary emphasis on preventative medicine and children’s nutrition. He is the author of Dr. Attwood’s Low-Fat Prescription for Kids.

To test whether this educational program had a noticeable effect, 24 classrooms were split into two groups, with half the classes serving as a control group. The results: children who had been taught about the healthy foods ate up to 20 times as much of the new food when it was offered for lunch. The Trumansburg pilot project demonstrated that experiential learning about food in the classroom can be a powerful technique for gaining acceptance of diverse low-fat foods. An additional benefit came when many of the children’s families changed their dietary habits. Plans are in place to continue this program as well as companion projects in New Mexico and Massachusetts with different ethnic populations. A goal is to make food literacy an education priority.

Because many diet-related chronic diseases take 20 years or more to develop, children must learn at an early age how to protect themselves. Politicians who threaten to cut school lunch programs need to realize that potential health benefits make these programs economically effective. To make these programs succeed, however, educators must address the fact that children will not accept foods even when they don’t know what they are. Ellen Richards was right: our children deserve to be fed and they deserve to know what they’re eating.
FOOD MEDICINE

CHINESE MUSHROOMS: Coming to a store near you?

By Dr. Jeffrey Gates, D.H.Sc.

More than 37,000 mushroom species abound on China’s ecologically diverse continent. Some are edible, others clearly are not. The Chinese government claims that 400 of these can be eaten without risk and 270 are beneficial as medicine. However, such lines of distinction between culinary and medicinal mushrooms often blur. Many, if not most, mushrooms are both food and medicine. While some of you may have already discovered the shiitake, maitake, and reishi mushrooms, you may be unfamiliar with the following varieties that are likely to be showing up soon on U.S. supermarket shelves.

New and Exotic

Prized for centuries in China for its combination of exotic taste and unique medicinal properties, the famous monkey mushroom (Hericium erinaceus), otherwise known as “white beard,” is sure to attract many in the U.S. The white beard’s tangle of threadlike strings, called mycelia, can be found enmeshed in the rotting stumps in China’s dense forests. Once harvested, these potent tendrils are commonly used for the treatment of ulcers and chronic gastritis. Recent evidence indicating the bacterium H. pylori as the possible common link between these diseases suggests that the monkey mushroom’s traditional efficacy may be due to its ability to suppress this pathogen. Nutritionally speaking, the monkey mushroom has fewer calories, more vitamin C, and more calcium than the ever-popular button mushroom. However, its unusually high sodium content (175 mg/100g) calls for prudent consumption.

In the Nagano district of China, a 10-year study was carried out on 12,600 growers of enokitake mushrooms (Flammulina velutipes). It was concluded that enokitake consumption significantly protects against stomach and colon cancers. Study results, along with subsequent clinical trials, suggest that enokitake holds promise for the treatment of stomach and colon cancers. Additionally, a recent Beijing research report notes that enokitake mushrooms can both reduce and prevent fatigue after heavy exercise.

Among other Chinese gastronomic medicinals deserving attention in the West is the edible oyster mushroom (Pleurotus ostreatus), increasingly available in the gourmet sections of U.S. chain stores. This humble fungus has been used in traditional Chinese medicine in a formulation called “tendon-easing power” for treating back and leg pains. Additionally, oyster mushrooms have a proven track record against certain kinds of tumors.

Last on our culinary list is Tremella (fusciforms). This delicious fungus is commonly made into a sugary syrup at Fujian hospitals for the treatment of asthma, chronic tracheitis, and chronic cough.

MUSHROOM NUTRIENT COMPOSITION TABLE

<table>
<thead>
<tr>
<th>COMMON NAME</th>
<th>Enokitake</th>
<th>Monkey</th>
<th>Button</th>
<th>Oyster</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCIENTIFIC NAME</td>
<td>Flammulina velutipes</td>
<td>Hericium erinaceus</td>
<td>Agaricus bisporus</td>
<td>Pleurotus ostreatus</td>
</tr>
<tr>
<td>CHINESE NAME</td>
<td>Dong Hu</td>
<td>Hou Tou Gu</td>
<td>Mo Gu</td>
<td>Ping Gu</td>
</tr>
<tr>
<td>Calories/100g</td>
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<td>13</td>
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</tr>
<tr>
<td>Protein (g)</td>
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<td>2.0</td>
<td>2.7</td>
<td>1.9</td>
</tr>
<tr>
<td>Ascorbic Acid (mg)</td>
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<td>4</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>55</td>
<td>19</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>20.4</td>
<td>175.2</td>
<td>8.3</td>
<td>3.8</td>
</tr>
</tbody>
</table>

Nutrition Advocate
When I cook a meal for my family, I try to make it as flavorful as possible. In preparing the recipes on this page, I was delighted to discover an entirely new taste treat—the oyster mushroom. You'll discover that the hearty flavor of this mushroom makes it a great alternative to meat.

While the shiitake mushroom is much more familiar, I found that these mushrooms captured the subtle flavors in the savory shiitake sauce in a unique way, giving this dish a gourmet flair.

While I recommend using the mushrooms we tried, feel free to experiment with any mushrooms you can find in your grocery store that fit into your budget. I've found that some Chinese mushrooms can be costly. As an alternative to your usual grocery, you may want to locate a Chinese or Japanese specialty market, which will often have the same mushrooms at considerably lower prices.

**Wild Mushroom Barley Soup**

**Preparation Time:** 30 minutes  
**Serves:** 4

- 1 leek
- 1 cup water
- 3 garlic cloves (chopped)
- 0.75 oz. dried oyster mushrooms
- 0.75 oz. dried wild mushrooms
- 10–12 white button mushrooms
- 1/2 t. thyme
- 1/2 t. marjoram
- 1/2 t. savory
- 1/4 t. basil
- 1/2 t. rosemary
- 1/8 t. sage

1 carton (33.8 oz.) light soy milk  
3 T. wheat flour  
1/4 cups barley (uncooked)  
salt (or salt substitute) to taste

Cut and clean the leeks thoroughly. Chop both the white part and the tender green part. Place in a heavy kettle with 3/4 cup water. Steam until tender, stirring occasionally.

Soak the dried mushrooms in water until completely hydrated and soft (15–20 minutes). Remove the stems from the oyster mushrooms and cut the tops into bite-sized pieces. Chop the uncooked button mushrooms. Add all mushrooms to the kettle. Add spices, and cook mixture until mushrooms are tender.

Sprinkle the wheat flour into the leek/mushroom mixture and mix well. Then, add soy milk, stirring until thickened. Add salt and barley and cook on low until barley is tender.

**Options:** Feel free to mix and match the various mushrooms in this recipe to your own taste.

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**Savory Shiitake Sauce**

**Preparation time:** 30 min.  
**Serves:** 4

- 1 cup water
- 1/2 cup chopped onions
- 2-4 cloves garlic, chopped
- 1/4 t. cumin
- 1/2 t. coriander
- 1/2 t. chili powder
- dash pepper
- 1/4 cup red wine
- 1 t. sugar (or sugar substitute)
- 1 T. low sodium soy sauce
- 1 lb. shiitake mushrooms
- 1 1/2 T. of cornstarch mixed with
- 1/3 cup water (or thickener like arrowroot)
- salt (or salt substitute) to taste

Steam onions and garlic in 3–4 T. of water until tender. Add the remaining cup of water to skillet.

Add all other ingredients except mushrooms and thickener. Cook briefly (1–2 min.)

Add mushrooms and turn the heat to low. Cover and simmer 30 min.

Thicken mixture with water and cornstarch.

Serve over brown rice or your favorite grain.

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**Nutrients per serving**

**Calories ...... 217**  
**Fat ............. 11 %**  
**Cholesterol .. 0.0 mg**

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**Nutrients per serving**

**Calories ...... 61.5**  
**Fat ............. 2 %**  
**Cholesterol .. 0.0 mg**
known to have a definite gene linkage from the mechanism point of view (a certain type of colon cancer), there was essentially no evidence that the mere presence of this gene actually caused the cancer.

To put this 1.4 fold difference into further perspective, smokers are 8–10 times more likely to get lung cancer than non-smokers, while people chronically infected with hepatitis B virus are 20–40 times more likely to get liver cancer than non-infected people. This small 1.4 fold increase, if real, might even be due to the greater similarity of lifestyles for identical twins than for non-identical twins. And finally this: these results essentially agree with the findings of four other smaller studies previously published. In short, this difference, even if real, is so small that it virtually eliminates genes as an important cause of human cancer.

This brings up some very practical questions. Why is so little attention given to the idea that it is not the mere presence of “bad” genes but the expression of these genes that matters most? For genes to cause something, they cannot lie dormant; they must be switched on or expressed. Thus, why isn’t gene expression, especially by nutritional means, given more attention, both among researchers and between researchers and the general public?

**Genetic Deceptions**

In conclusion, even though I had often thought that genes might account for a small proportion of human cancers, I am surprised by this finding showing that genes appear to contribute little or nothing to cancer risk. I would have thought that we might have seen in this study at least a small genetic effect.

The subjects in this study were men who, in all likelihood, consumed a typical American diet. With such a diet, whatever cancer-causing genes might have been present should have been consistently and uniformly expressed as cancer. This was not seen. Therefore, I must conclude on the basis of these and other data—with considerable confidence—that differences in cancer rates between people or individuals cannot be attributed simply to genes.

Obviously, the “gene story” is about much more than genes. It’s about what we eat and what we don’t eat. It is also about the great reluctance on the part of so many of us to change our eating habits. Despite the fact that Americans are more nutrition conscious than ever before, significant change is painfully slow to come. During the past 30 years, and especially so in the last decade, the number of overweight children has more than doubled.

As you can see in this issue of *Nutrition Advocate* we are giving a great deal of thought to these matters, and particularly to the role of the family in forming lifelong eating habits. In coming issues, we will expand on this theme by including more articles about nutritional solutions for the changing family. As always, we very much appreciate hearing from you with your suggestions and comments. Our definition of family includes you and we welcome you to join with us by sending us your letters, comments and recipes.

Wishing you the best of health,

*Colin*

T. Colin Campbell