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Listen to nature. The challenge of lifestyle medicine

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When Robert Koch entered the field of medicine, the world was ravaged by a host of infectious diseases. By keen observation of disease processes such as those in anthrax, cholera, and tuberculosis, Koch recognized that each infectious disease is induced by a specific "necessary" organism. In turn, his discoveries led to preventive strategies.

While avoiding infectious diseases presents a challenge to the individual, physicians and other health professionals have a special responsibility for their control and prevention. However, most of the major diseases prevalent in today's industrialized societies are not caused by infectious agents; rather, they result from deliberate use of tobacco, from alcohol abuse, illicit drug use, nutritional excesses or deficiencies, and unsafe sexual practices, which each individual could largely control.

Here then lies one of the dilemmas of modern medicine. There are several obstacles to personal control: the illusion of immortality most people suffer from, the insufficient interest of health professionals in motivating people to change their lifestyles, and a campaign of persuasive contrary messages from commercial interests about products potentially harmful to the health of the consumer.

Criteria for causation

Robert Koch has given us guidance in establishing proof of causation for communicable diseases by his classic postulates. Following our first epidemiological study linking smoking to lung cancer, we used Koch's postulates as a basis for establishing proof of causation for chronic diseases[1–3]. These postulates stated: (I) The factor has to increase the risk of cancer. (II) Its global distribution should be consistent with the rate of cancer. (III) After its removal or reduction in a given population, the cancer rate should decline after a suitable latent period. These postulates applicable to chronic diseases differ from those of Koch in two distinct ways.

For one, a risk factor for cancer is not "necessary" cause, although science may yet discover endogenous factors that may play such a role. Thus, any foreign agent, such as tobacco smoke, can but does not have to cause cancer in a particular individual, even though the occurrence of lung cancer in the absence of tobacco use is rare.

For another, experimental evidence in laboratory animals is not required to prove that a given factor can cause human cancer. Nevertheless, such experimental data can provide useful support for the human observations, as was the case when we first produced skin cancer in mice with topical application of tobacco tar in 1953, and similarly, cancer of the ear in rabbits in 1957[4,5]. These experiments, especially the proof of their dose-dependency, although not necessary evidence, supported the causative role of tobacco smoke in the development of lung cancer.

In line with postulate II, the global distribution of lung cancer is clearly consistent with smoking habits in various populations. Furthermore, (Postulate III) upon cessation of smoking, or even when tar yield in the smoke of cigarettes is reduced, the lung cancer rate tends to decline after an interval of about 5 years and continues to decline for another 10 to 20 years thereafter, even though the low risk of never smokers will not be reached[6,7].

The first Report on Smoking and Health by the Surgeon-General of the United States (1964) contained an important discussion about "Criteria of Judgment", which emphasized the various pieces of evidence required to conclude the presence of a causative association[8]. These criteria, which included such characteristics as dose response, consistency, and biological plausibility, should always be considered whenever causation is contemplated and investigated.

The role of lifestyle factors

Epidemiological studies and clinical observations by astute physicians have made it clear that most types of cancer and many other noncommunicable diseases are not an inevitable consequence of aging, but are a result of lifestyle, and at times, general environmental influences. In my experience, comparing cancer rates between the US and Japan, both based upon equally good vital statistics, has been especially constructive[9] (Table 1). An examination of these data in 1980 and even more their comparison to data from 1960, gives us valuable clues about cancer etiology. These differences are, with some exceptions (i.e., skin cancer), not due to genetics as studies of Japanese migrants to the US have demonstrated. Rather, lifestyle differences are the basis for these findings.

Metabolic overload

Excessive exposures to exogenous carcinogens have been established to increase risk for many cancers

Tab. 1. Age standardized incidence rates of cancer of selected sites in the US and Japan, 1980.

Males			Females	
US	*Japan*		*US*	*Japan*
64.0	30.0	Lung	25.3	8.7
15.0	3.0	Oral Cavity	5.5	1.2
8.0	2.0	Larynx	1.7	0.2
5.0	3.0	Esophagus	1.5	3.1
9.1	9.0	Pancreas	5.5	5.1
25.0	6.0	Bladder	7.4	1.9
10.8	79.6	Stomach	4.3	36.0
2.5	11.2	Liver	1.1	4.0
34.1	9.8	Colon	26.1	9.4
18.0	9.9	Rectum	11.4	7.4
6.1	5.4	Leukemia	6.1	4.4
17.3	6.0	Lymphoma	12.8	3.5
46.8	4.3	Prostate		
		Breast	77.8	22.0
		Ovary	12.0	1.9
		Cervix	6.8	10.0
		Corpus	19.3	2.8

Source: IARC Sci Publ Monographs, 1960–80.

(Figure 1). We have estimated the preventive potential of various exposures, which are largely lifestyle-related factors [10].

In medical school, my Anatomy Professor, Dr. Edward Cowdry, taught that one could learn much about the etiology of disease by "listening to nature". It would seem obviously against nature to expect the human lung to defend itself in a span of 30–40 years against inhalation of 6–7 kg of tobacco "tar", when it has been known since 1775 from Perceival Pott's observation among chimney sweeps that combustion products can cause cancer [11]. Analogously, it would appear unnatural to assume that the human liver, efficient as it is, can detoxify a half a liter of whiskey a day, and that the lumen of the narrow coronary arteries can accommodate heavy deposits of cholesterol without compromise of circulation.

From epidemiological studies among different populations, we have deduced the significance of dietary composition in the initiation, promotion, and progression of cancer. In the developing countries, malnutrition may be a key determinant of disease; yet, in industrialized countries, nutritional excesses cause chronic diseases at unparalleled rates with the notable exception until now of Japan. Malnutrition among the poor is principally due to a low intake of proteins and essential micronutrients, conditions that can and should be alleviated by improving standards of living. Malnutrition among the affluent, however, has existed for a long time and is an outgrowth of psycho-cultural practices, which are almost perceived as the norm.

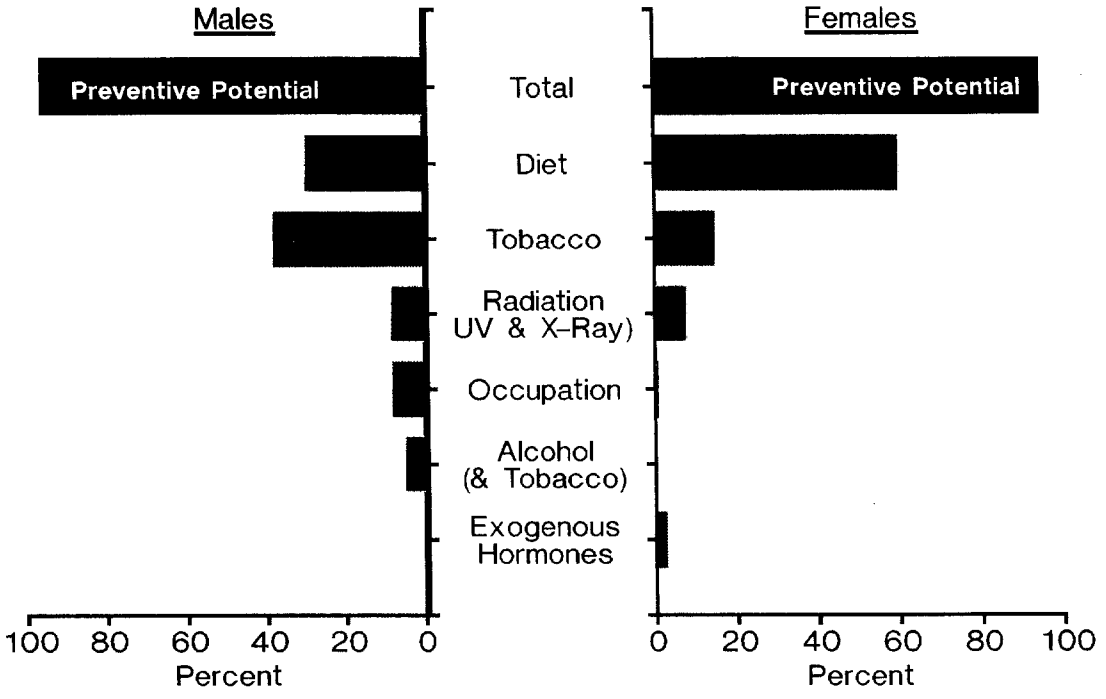

Fig. 1. Percent of cancer incidence attributable to specific environmental factors – USA.

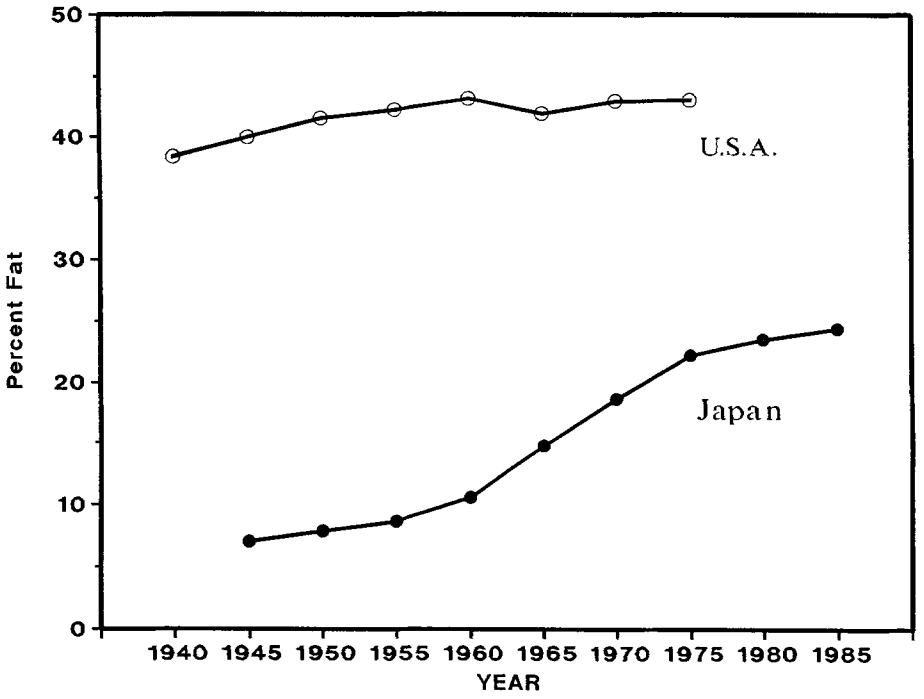

Fig. 2. Percent of fat in total energy, USA vs. Japan, 1940–1985. Source: Japanese Ministry of Health and Welfare, 1987; US Bureau of Census, 1971 and 1985.

We observe the high prevalence of obesity with its social and medical consequences but have no national program toward its prevention. We know that consumption of saturated fat is a principal factor for atherosclerosis and its major sequela, myocardial infarction[12]. Half a million people in the US die each year from cardiovascular diseases mainly relating to elevated levels of low-density lipoproteins (LDL) with cigarette smoking and hypertension importantly adding to this risk. Yet, not enough is done to encourage changes in dietary patterns or discourage cigarette smoking or reduce hypertension. We need to strive for "optimal" rather than "normal" or average blood cholesterol levels. We define ideal (optimal) levels for adult men to be <160 mg/dl (4.14 mmol/l), feasible levels to be <180 mg/dl (4.65 mmol/l), while US current levels average at 210 mg/dl (5.43 mmol/l). For children, the respective levels are 110, 140, and 165 mg/dl (2.84, 3.62, and 4.27 mmol/l). Our current so-called normal levels clearly are the cause of the high prevalence of coronary artery disease in many of the developed countries.

While we can readily see the "abnormalcy" of tobacco use, alcohol abuse, illicit drug use, and obesity, we find it difficult to determine the level of "optimal" fat intake, "optimal" being the amount needed to maintain sound physical and mental development and remain free of avoidable disease. As we examine the fat intake in the US and Japan (Figure 2), we observe differences that are the likely reason for the lower rates of certain cancers and coronary artery disease in Japan. At the same time, we note that normal neurological development is supported by a fat intake at a level as low as 10% of calories consumed.

Another question to consider is that of the optimal proportion of the different types of fat. When the total fat intake is at 25% of calories or less, the type of fat appears not to be of great importance. Saturated fats are the main factor in the development of atherosclerosis as can also be seen from studies on obligatory vegetarians (vegans) by our Institute[13]. While their overall fat intake is not low (about 30%), their low intake of saturated fat leads adults to have an average serum cholesterol level of 130 mg/dl (3.36 mmol/l). In respect to cancers of the breast and prostate, increasing evidence suggests that the percentage of linoleic acid, an omega-6 polyunsaturated fatty acid, may play a decisive role. We need to resolve whether the increase in breast cancer, particularly among postmenopausal women, in the US and the rising incidence of prostate cancer may relate to increasing amounts of oils based on linoleic acid in the US diet. As we examine the optimal percentage of fat in the diet, it might ideally be 10% of total calories with a P:S:M (polyunsaturated: saturated: monounsaturated fats) of 1:1:2. Such a diet, however, is unlikely to be consumer-acceptable, or even economically feasible, except in special clinical situations. As a feasible diet, we recommend 25% of calories as fat, with a P:S:M ratio of 1:1:1 (Figure 3).

Epidemiology shows that breast cancer also is not an inevitable conequence of aging, but is a preventable disease. The low rate of breast cancer among

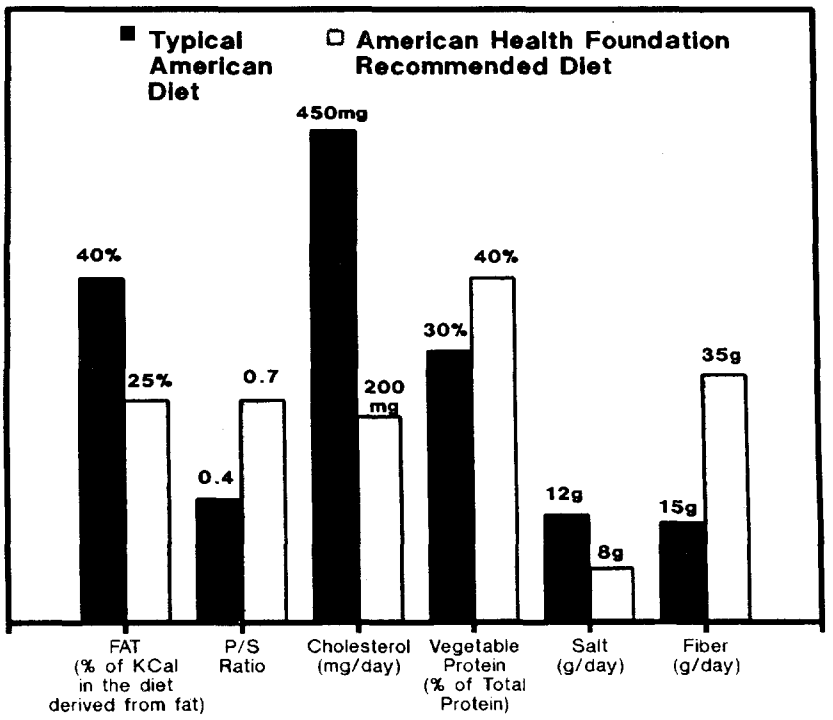

Fig. 3. American Health Foundation Food Plan. AHF recommended dietary intakes vis. those of the typical American diet.

Japanese women, especially those postmenopausal, is not maintained in their descendants in the US who adopt Western nutritional customs. After two generations, the cancer patterns of Japanese-American women are similar to those of other American women [14]. Increasingly, traditions in Japan itself are gradually giving way to more Western dietary patterns. The total fat intake, however, is still only 24% of calories consumed (as opposed to 38% in the US), although in 1955, it was only 9% [9] (Figure 2). Reflecting such changes, breast cancer rates are beginning to rise in Japan [9].

Studies with laboratory animals by Carroll, Cohen, Ip, and others, confirm the dynamics and trends of the relationship of dietary fat to breast cancer, and have shown that the amount of fat consumed as well as the type is apparently important in the promotion as well as the progression of breast cancer [15–20]. The development of chemically-induced mammary tumors in laboratory animals is not increased by diets containing oils rich in monounsaturated fatty acids, medium-chain saturated fatty acids, or long chain omega-3 fatty acids, but with oils rich in the omega-6 essential fatty acid linolate, tumor yields increase in proportion to the amount of linoleic acid in the diet up to about 40% of total calories. In cell culture, Rose in our Institute has shown linoleic acid to enhance growth of breast cancer cells while docosahexaenoic and eicosapentaenoic acids, the principal omega-3 fatty acids in fish oils, have pronounced inhibitory effects [21,22]. These studies support the hypothesis that the fat effect is due to the chemical nature of specific fatty acids and not to a general caloric effect [23,24].

These observations have suggested that breast cancer patients might derive therapeutic benefit from dietary modifications. An ongoing intervention trial, supported by the US National Cancer Institute, randomizes postmenopausal breast cancer patients, who have had surgery and subsequent adjuvant therapy, to determine differences in recurrence rates and survival as a result of maintaining these patients on a low-fat diet (20% of calories from fat) versus a standard US diet (about 40% of calories from fat). One would predict that women in the low-fat group would benefit, since such a low-fat diet causes a reduction in the plasma estradiol [25,26], decreases the biologically available estradiol [26], and may affect the interaction of protein hormones with their cell membrane receptors, as well as affecting prostaglandin synthesis and the immune system [27,28]. Confirmation of this hypothesis not only would explain why Japanese women have a lower incidence of breast cancer but also would clarify why those who do develop breast cancer postmenopausally have better survival rates than Western women [29]. Future studies of this type will have to consider the different types of fatty acids, especially the omega-3 fatty acids, in the diet.

This study represents but one example of the wide-ranging possibilities for cancer prevention through dietary modification. Other studies at our Institute are evaluating the interaction between dietary fat and fiber in both breast and colon carcinogenesis, with particular emphasis on the utility of insoluble wheat fibers as a regulator of the effects of dietary fat on circulating estrogens and intestinal bacterial enzymes [29,30]. Similarly, the role of fats in prostate

cancer, which follows a similar epidemiological pattern as breast cancer, is of ongoing interest to our group.

As we "listen to nature", we must sometimes strain our ears to hear fainter signals. One of these suggests that dietary fat may even play a role in tobacco-induced lung cancer. Our finding of a strong ecological correlation between dietary fat and lung cancer was biologically plausible and was supported by other evidence indicating that dietary fat could affect pulmonary carcinogenesis through several mechanisms [31]. Yet, this observation did not draw sufficient attention. The signal has become stronger as a result of a specific epidemiologic lead. The 1987 lung cancer rate in the US (72/100000) and Japan (39/100000) may reflect a striking difference in nutritional factors, and to fat consumption in particular (Figure 2). There has been a consistently higher percentage of smokers among Japanese men, as compared with US, male smokers since 1955, while the number of cigarettes smoked per day in the US and Japan are about equivalent. We also need to consider, however, that the age of onset of smoking in Japan has been several years later than that in the US in age groups 50–79 (Personal communication, Ikuko Kato, Aichi Cancer Center Res. Inst.) and that the lung tissue should be especially susceptible to carcinogenic stimuli during adolescent growth, similar to observations relating breast cancer to radiation in Hiroshima [32]. Yet, analysis of our data demonstrates a close association of age of onset of smoking to number of cigarettes smoked per day, the key determinant for lung cancer risk. Thus, the differences in onset of smoking between US and Japanese men could not explain the divergent lung cancer rates in the two countries. We are exploring possible pathways by which dietary fats may affect chemical carcinogenesis. Further delineation of mechanisms, especially those involving different fatty acids, may provide appropriate preventive strategies.

As we "listen to nature", we must also reflect on our evolutionary heritage. Our metabolic processes were "programmed" eons ago when our ancient forebears were physically far more active; when diets contained far less fat, probably not exceeding 10% of total calories; when the diet contained little saturated fat, and a relatively high proportion of monounsaturated fats, principally derived from nuts and seeds; and when the fiber content of food was high and diverse, perhaps as much as 100 g/day. Also, in those days, there was no stroke of the clock to signal that a meal had to be eaten, but food was consumed in small amounts throughout the day whenever hunger demanded, which probably affected production of insulin and other hormones [33]. Metabolic overload, the modern phenomenon and the cause of so many of the diseases of our time, overwhelms our organism, and against which we do not have sufficient defenses.

Low-level exposures

It seems reasonable to conclude that although excessive exposure would overwhelm our defensive systems, molecular, physical, and biological defenses can protect us against low-level stimuli. Evolutionary processes no doubt contributed to our ability to withstand exposure to low-level irritants in our respiratory environment as we can deduce from the intricate structure of nasal turbinates of rodents and other animals who inhale air close to the ground, and from the remarkable development of ciliated mucus-producing columnar or respiratory epithelium, which Dietrich Hoffman and I investigated in the sixties [34, 35]. Plants have natural-occurring pesticides, as shown by Ames, without which they may not have survived the millenia, and against which plant-eating species developed defensive postures [36].

Modern analytical technology permits us to identify trace compounds in our environment and in food. Such compounds, given to laboratory animals in excessive doses, may cause cancer, but the question remains whether such a finding implies a measurable human risk. Weisburger and Williams [37] introduced the distinction between genotoxic and epigenetic agents in this context. Genotoxic carcinogens, such as the cooked food pyrolysis products, studied by Sugimura, Weisburger, and Felton [38–40], can be active at low levels, whereas epigenetic agents such as hormones require high and subtantial exposures after leading to increased cell proliferation.

For example, certain airborne chemicals in occupational environments may cause lung cancer, but the same chemicals, occurring in dilute concentrations in air, may not exert their effect because the natural defenses of the respiratory system will prevent their absorption. Forceful inhalation of a concentrated aerosol of mainstream cigarette smoke will not only paralyze the respiratory cilia and thus damage the respiratory defense mechanisms, but will deliver a damaging dose of tumor initiators, tumor promoters, toxins, and highly reactive chemicals to the lungs. This is certainly different from exposure to dilute pollutants from a respiratory environment inhaled under normal breathing conditions. In this instance, we need to "listen" to the anatomical nature of our defense system.

In considering the significance of human exposure to harmful agents, public health authorities need to safeguard the environment, but they can only function responsibly when the benefits of removing a questionable hazard, especially a nongenotoxic one, outweigh the total cost incurred to society. Therefore, the toxicologist's role in determining what constitutes a health hazard is of great importance and care must be taken that our judgement is not clouded by a wish bias. Scientists may tend to overinterpret their findings; manufacturers, on the

other hand, prefer to pretend that a problem does not exist. Sound, well-documented facts on a given product provide an objective means of reaching decisions in the interest of public health.

The science of applying preventive strategies

Scientific discoveries are often easier to achieve than is the application of measures derived from them. This is particularly true for discoveries relating to disease prevention. The history of preventive medicine is replete with examples showing that the incidence of a disease can be reduced, if not eradicated, without a full comprehension of the processes involved in its pathogenesis. The history of Snow and cholera, and of Semmelweis and childbed fever serve as examples. Thus, as important as the contribution of biomedical science is to cancer and other disease entities, preventive measures can be developed to reduce the disease incidence without necessarily understanding all facts from the underlying science base. Scientific and financial resources must be judiciously apportioned between health promotion and laboratory research, and we must recognize the limitations that exist in each of our respective disciplines. Molecular biology may some day be able to aid us in diagnoses and even in the prevention of cancer, atherosclerosis, and other chronic diseases at the cellular level. Further knowledge in immunology may help us to protect those exposed to the virus associated with AIDS. In the meantime, however, through preventive measures already on hand, we can curb the incidence of many of the diseases related to tobacco use, excessive intake of fat, alcohol abuse and drug use, and unsafe sexual practices. Toward this end, we must place greater emphasis on the application of preventive measures.

As emphasized, the practice of lifestyle medicine deals with variables for which the individual must take responsibility. To enhance this process, the social, political, and public health leadership must provide encouragement that should lead to enhancing each person's self-esteem. The leadership must encourage industry to produce more healthful (or less harmful) products. For food products, this means that farmers and food processors lower the fat content of meat and dairy products, that they produce and market more foods with higher fiber content. The optimal human diet can better be attained if national focus would be on a diet such as that presented in Figure 3. Clearly, we need to increase the consumption of fruits and vegetables as evidenced by the important field of chemoprevention to which Wattenberg has greatly contributed, as have Hecht and Chung, Reddy, and others at the American Health Foundation[41–43]. The work with omega-3 fatty acids already referred to also relates to this concept.

To reduce the incidence of tobacco-related cancers, we must in increasing order of importance, envoke the following strategies: less harmful cigarettes, smoking cessation programs for adults, and preventing initiation of smoking among children and young people[44]. Strategies for general disease prevention also involve legislative measures, such as setting permissible levels for environmental pollutants, an upper level for the amount of fat in different food products, or limits on the tar yield of cigarettes.

Comprehensive school health education

"As the twig bends so the tree grows" is a commonly stated saying that represents a concept that certainly applies to child development including young people's health behavior. Thus, any national program of health promotion must include comprehensive school health education beginning in first grade since so many risk factors have their beginning in early childhood. For example, among 8 to 20 year olds in the US, one third already have serum cholesterol levels >180 mg/dl (4.65 mmol/l)[45,46]. Kunze has found similar levels of 8 to 10 year olds in Munich (Detlev Kunze, Univ. of Munich, personal communication). Because hyperlipidemia is relatively common at young ages, the American Health Foundation has recommended baseline screening for all children starting between the ages of two and six[45]. If cholesterol levels are found to be high, appropriate dietary intervention can be carried out early in life before the process of atherosclerosis has set in.

For more than a decade, the American Health Foundation has developed the Know Your Body Comprehensive School Health Promotion Program (KYB), which it is now disseminating[47,48]. It is a unique comprehensive, student-centered, skills-based program, beginning in grade K and continuing through high school. It follows the guidelines suggested by the National Professional School Health Education Organization (Table 2). KYB involves an annual screening (designed not only to personalize the program but also to identify children at high risk); provides developmentally-appropriate workbooks for every child and guides for every teacher; and conducts an annual evaluation for attitude, knowledge, and health behavior – all of which is coordinated by a fulltime health education teacher. The program not only involves the entire school but also its community environment (Table 2). We have shown that the program can help to reduce plasma cholesterol levels, and the onset of smoking[49,50]. The program, while offering knowledge, attitude and skills development principally focuses on behavioral strategies. With the involvment of parents, media, and community and use of extra-curricular and interdisciplinary activities in the school, we can achieve optimal re-

Tab. 2. The Comprehensive Health Education Components by the National Professional School Health Education Organization *

1. School Health Instruction (Curriculum)
   - A well-planned, sequential, developmentally appropriate program of learning experiences for students from K-12
   - Opportunities for students to develop and demonstrate their health-related knowledge, skills, and attitudes
   - Integration of physical, mental, social and emotional dimensions of health as a basis for study in the following areas:
     community health, consumer health, environmental health, family life, growth and development, nutritional health, personal health, prevention and control of disease and disorders, safety and accident prevention, tobacco and alcohol as well as substance use and abuse
   - Ongoing teacher and key school personnel inservice
   - Outreach programs to inform and involve parents
   - Workbooks for students
   - Teacher guides
2. Full-time health education teacher/coordinator
3. Annual assessment for attitudes, knowledge, and behavior
4. School health services (for students, teachers, and staff)
   The School should provide health services in association with community agencies that include: case finding through annual health screening, early intervention programs, and first aid
5. School health environment (health-related extra-curricular activities)
6. Integrated community and school health promotion efforts
7. School physical education
8. School food service (modification of cafeteria menus)

* Adapted with some modification by the "Know Your Body" Program from National Professional School Health Education Organizations: Comprehensive school health education – a definition. Journal of School Health 54(8):312–315, 1984.

sults. The program is comprehensive and thus, covers diverse subjects ranging from dental hygiene to nutrition, from smoking to alcohol abuse, from illicit drugs to sex education (Table 2).

If properly conducted in terms of quality, dose, and intensity, it can fill all children with the ability to make responsible health decisions and act accordingly. For the program to succeed, all of us – parents, teachers, and society as a whole – must actively be involved with our children. As one of my favorite mottos holds "Tell me, I forget; show me, I remember; involve me, I understand".

When the question of the program's cost is raised, we counter by asking the cost to treat a single cocaine-addicted or AIDS-afflicted baby, or how much society pays for teenage pregnancy, alcohol abuse, and drug use by our children. How much will early onset of smoking and hyperlipidemia contribute to premature mortality and morbidity? How much will children with low self-esteem cost a nation by becoming unproductive adults? Social support or the neglect thereof significantly affects disease rates throughout the world. If we are effective in enhancing the environment and the self-respect of all children everywhere, and create the recognition that they themselves are largely responsible for their physical, social, and mental well being, a major impact will have been made on disease prevention, and thus, on the health and economic state of our society.

Outlook

Lifestyle medicine as well as preventive medicine, of which it is an integral part, has long been neglected by health professionals, medical schools, and governmental health care planners. This holds true particularly for Germany, which has no Schools of Public Health and few Departments of Preventive Medicine in its medical schools. The barriers are apparent: lack of academic recognition, lack of immediate response, and the fact that a "preventive medical-industrial complex" does not exist. Yet, as nature tells us, disease is not an inevitable consequence of living and aging. Preventive medicine, particularly as it relates to lifestyle, presents unique challenges including health economics. In the US the 1989 cost of "disease care" was about 600 billion and is expected to increase to one trillion dollars by 1995. The costs are similarly high in Germany. How much better could we spend our money than on what we know to be preventable diseases and disabilities.

Specific to my theme, I propose Germany establish departments of preventive medicine, including sections in epidemiology, as mandatory components in each of its schools of medicine. Some of these departments could in time develop into schools of public health. By so doing, not only would we serve the public better but also would attract physicians and other health professionals who would thus see a career in this so vital medical practice.

I also recommend that comprehensive school health education along the lines described become an integral part of German school curricula. In the face of increasing health care costs, much of which relates to illness-producing behavior, it would seem imperative that every child has the opportunity and in fact the right to a comprehensive school health education program.

The ultimate challenge for the prevention of acute or chronic diseases to health professionals is the same – to reduce the physical, social, and economic toll of avoidable illness so that each of us can live at our optimal capacity and pass from life only

when the genetic time clock as set by hereditary processes has run its course. Such a goal is medically feasible and is socially and fiscally imperative. In brief, " It should be the function of medicine, to help people die young, as late in life as possible." The work, so well achieved by Robert Koch on infectious diseases about a century ago, and currently, the deliberately planned application of existing knowledge for the prevention of noncommunicable diseases will hasten the day toward this desired future for mankind.

Abstract

Unlike the days of Robert Koch when infectious diseases were the principal contributors to morbidity and mortality, today's illness and death are most often caused by noncommunicable diseases that have the special characteristics of resulting largely from one's own lifestyle, especially tobacco use and nutritional excesses. By "listening to nature", we can detect and identify risk factors for various types of cancer, explore their mechanisms of action, and execute preventive strategies leading to their reduction or modification, thereby, decreasing the incidence and mortality of disease. An example of the role of metabolic overload in carcinogenesis is the impact of an excessive intake of dietary fat on the development and progression of breast cancer. For the general pathogenesis of cancer, the risks associated with metabolic overloads are contrasted with those of low-level exposures. To broaden the impact of preventive medicine beyond factorial nutritional education in Germany, we recommend that (a) every medical school have a department of preventive medicine with emphasis on epidemiology and health promotion, and (b) all schools beginning in first grade have a comprehensive school health education program coordinated by a full-time health education teacher.

Résumé

Alors que du temps de Robert Koch, les maladies infectieuses étaient les principaux déterminants de la morbidité et de la mortalité, la mort et la souffrance sont aujourd'hui causées le plus souvent par des maladies non transmissibles. Ces dernières ont la caractéristique d'être la conséquence de notre propre style de vie, en particulier l'usage du tabac et les excès alimentaires. En étant attentifs à la nature, nous pouvons identifier les facteurs de risque des cancers, explorer leur mécanisme d'action, et mettre en place des stratégies préventives permettant leur réduction ou leur modification: en somme, on peut diminuer l'incidence des maladies et la mortalité. Un exemple du rôle de la surcharge métabolique dans la carcinogenèse est l'effet de la surconsommation de graisses alimentaires sur le développement et la progression du cancer du sein. La pathogenèse générale du cancer, les risques associés avec les surcharges métaboliques sont comparés aux conséquences d'une sous-consommation. Pour augmenter l'impact de la médecine préventive au-delà de l'éducation alimentaire en Allemagne, nous recommandons que a) chaque faculté de médecine ait un département de médecine préventive actif dans les domaines de l'épidémiologie et la promotion de la santé, b) tous les écoliers devraient avoir dès le début de leur scolarité une éducation sanitaire globale, dont l'enseignement serait coordonné par un spécialiste de l'éducation à la santé.

Zusammenfassung

Zur Zeit von Robert Koch waren Infektionskrankheiten die Hauptverursacher der Morbidität und Mortalität; heute sind Krankheiten und Todesfälle vorwiegend durch nicht übertragbare Krankheiten verursacht. Charakteristisch für solche Krankheiten ist, dass sie die Konsequenz des eigenen Lebensstiles darstellen z. B. Tabakgebrauch, Fehlernährung. Indem wir in einem „listening to nature" auf die Signale unseres Körpers achten, können wir die Risikofaktoren für die verschiedenen Arten von Krebskrankheiten aufspüren und identifizieren und ihren Wirkungsmechanismus erforschen. Wir werden Strategien zur Prävention entwickeln können, die diese Risikofaktoren vermindern oder verändern und somit zur Senkung der Inzidenz und der Mortalitätsrate beitragen. Welche Rolle eine Stoffwechsel-Überbelastung im Krankheitsverlauf eines Karzinomes haben kann, zeigt das Beispiel einer übermässig fetthaltigen Ernährung auf die Entwicklung und den Krankheitsverlauf bei Brustkrebs. Zum Verlauf einer Krebskrankheit im allgemeinen lässt sich feststellen, dass jene Risikofaktoren, die mit einer Stoffwechselüberbelastung assoziiert sind, sich stark von solchen unterscheiden, welche Expositionen mit geringen Schadstoffmengen haben. Um in Deutschland den Einfluss der Präventivmedizin zu verstärken und zwar über eine Ausbildung in Ernährungslehre hinausgehend, empfehlen wir, dass a) jede medizinische Ausbildungsstätte eine Abteilung „Präventivmedizin" mit dem Schwerpunkt Epidemiologie und Gesundheitsförderung hat und dass b) alle Schulen vom ersten Schuljahr an eine umfassende Einführung in ein Gesundheitsprogramm anbieten, welches durch einen Spezialisten koordiniert wird.

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