Part 1

The context of health psychology

Part contents

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The traditional biomedical model emphasised biological factors such as viruses, lesions or bacterium as the main causes of illness, and its treatments focused on medication or surgery. From this perspective psychological factors were seen as possible consequences of illness but their role in its etiology was considered minor; having a heart attack may be distressing and being diagnosed as having cancer may make the person depressed but such psychological factors were conceptualised only as the result of the illness. Over the past century, however, such a perspective has been challenged and psychological variables are increasingly seen as relevant at all stages along the continuum from health to illness, and central to this shift has been the recognition that behaviour is at the core of any understanding of why someone becomes ill, how they manage their illness and how their illness progresses. Furthermore, as the main causes of death these days are chronic illness such as heart disease, cancer and diabetes, which are linked to behaviours such as eating, drinking, smoking and adherence to medication, behaviour is seen as central to both the onset of illness and its management. This focus on psychological factors, particularly behaviour and the cognitions and emotions that relate to behaviour, is the domain of health psychology. This part presents two papers that clearly highlight the importance of behaviour and provide the context to health psychology:


Mokdad et al. (2004) present an analysis of the role that behaviours such as smoking, diet, activity, alcohol consumption, car crashes and sexual behaviour played in the deaths of the 2.4 million people who died in 2000 in the USA. Their analysis is based upon a search of papers that identified a link between risk behaviours and mortality to give them an estimate of how many of the disease-related deaths could be accounted for by a particular behaviour. They then multiplied this by the data on actual causes of deaths in the year 2000. For example, from the literature they estimated what proportion of deaths by cancer could be accounted for by diet. They then multiplied this estimate by the number of people who actually died from cancer in the year 2000. Then, by collating all these figures, they arrived at the total number of deaths (regardless of disease) attributable to each behaviour. This paper is interesting as it illustrates how to analyse secondary data and provides an insight into the language, statistics and methods of epidemiology and statistical modelling. It also illustrates how to answer a question that is central to much health psychology literature as the awareness of the link between behaviour and mortality forms the starting point for much research.

Mokdad et al. (2004) therefore emphasise behaviour as a predictor and cause of illness and mortality. In contrast, while still highlighting the importance of behaviour, the paper by Kaplan (1990) describes the importance of behaviour as an outcome variable. In particular, this theoretical piece describes how traditional research focuses on the impact of interventions on outcomes such as cell pathology, blood pressure, lesion size and cardiac output. Kaplan then argues that such outcomes are flawed in several ways and that behavioural outcomes are more useful. First, he suggests that biological outcomes often lack validity and reliability; second, he suggests that they rely on behavioural measures to give them their validity; third, he suggests that a focus on biological outcomes has sometimes led researchers down
the wrong and occasionally dangerous path; and finally, he argues that even classic outcomes such as mortality and morbidity are essentially behavioural outcomes in disguise. In addition, he suggests that a behavioural conceptualisation of health does not negate a medical model but offers a broader perspective, and that medical interventions such as medicines and surgery should be seen as complementing those that aim to change behaviour. This paper is much earlier than that by Mokdad et al. (2004) and was published when health psychology was still in its early stages. In its time it was ground breaking as it presented a challenge to contemporary biomedical perspectives on health research. It remains highly relevant today, however, as many researchers, journal editors and clinicians still prioritise medical explanations and medical outcomes and see these as more ‘real’ than those that focus on behaviour. Furthermore, in a discipline such as health psychology which often sits on the edge of medicine, a researcher has to decide whether to ‘go native’ with medicine or fight the corner for psychological perspectives. This paper provides ammunition for a more fighting approach.

In summary, these two papers provide a context to health psychology, and illustrate why much of health psychology research is concerned with predicting, explaining or changing health-related behaviours.
Actual causes of death in the United States, 2000

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Abstract

Context: Modifiable behavioral risk factors are leading causes of mortality in the United States. Quantifying these will provide insight into the effects of recent trends and the implications of missed prevention opportunities.

Objectives: To identify and quantify the leading causes of mortality in the United States.

Design: Comprehensive MEDLINE search of English-language articles that identified epidemiological, clinical, and laboratory studies linking risk behaviors and mortality. The search was initially restricted to articles published during or after 1990, but we later included relevant articles published in 1980 to December 31, 2002. Prevalence and relative risk were identified during the literature search. We used 2000 mortality data reported to the Centers for Disease Control and Prevention to identify the causes and number of deaths. The estimates of cause of death were computed by multiplying estimates of the cause-attributable fraction of preventable deaths with the total mortality data.

Main Outcome Measures: Actual causes of death.

Results: The leading causes of death in 2000 were tobacco (435,000 deaths; 18.1% of total US deaths), poor diet and physical inactivity (400,000 deaths; 16.6%), and alcohol consumption (85,000 deaths; 3.5%). Other actual causes of death were microbial agents (75,000), toxic agents (55,000), motor vehicle crashes (43,000), incidents involving firearms (29,000), sexual behaviors (20,000), and illicit use of drugs (17,000).

Conclusions: These analyses show that smoking remains the leading cause of mortality. However, poor diet and physical inactivity may soon overtake tobacco as the leading cause of death. These findings, along with escalating health care costs and aging population, argue persuasively that the need to establish a more preventive orientation in the US health care and public health systems has become more urgent.
In a seminal 1993 article, McGinnis and Foege described the major external (nongenetic) modifiable factors that contributed to death in the United States and labeled them the “actual causes of death.” During the 1990s, substantial lifestyle pattern changes may have led to variations in actual causes of death. Mortality rates from heart disease, stroke, and cancer have declined. At the same time, behavioral changes have led to an increased prevalence of obesity and diabetes.

Most diseases and injuries have multiple potential causes and several factors and conditions may contribute to a single death. Therefore, it is a challenge to estimate the contribution of each factor to mortality. In this article, we used published causes of death reported to the Centers for Disease Control and Prevention (CDC) for 2000, relative risks (RRs), and prevalence estimates from published literature and governmental reports to update actual causes of death in the United States – a method similar to that used by McGinnis and Foege.

**Methods**

Our literature review used a MEDLINE database search of English-language articles that identified epidemiological, clinical, and laboratory studies linking risk behaviors and mortality. Our search criteria were to include all articles including the following key words: mortality, smoking, physical activity, diet, obesity, alcohol, microbial agents, toxic agents, motor vehicle, firearms, sexual behavior, illicit drug use. Our search allowed for words with similar meaning to be included (ie, exercise as well as physical activity). The search was initially restricted to articles published during or after 1990, but we later included relevant articles published in 1980 to December 31, 2002 (search strategies are available from the authors on request). For each risk factor, we used the prevalence and RR identified by the literature search. To identify the causes and number of deaths, we used mortality data reported in 2000 to the CDC. We used no unpublished information or data.

We used the following formula to calculate attributable fractions for each disease: 
\[
\frac{P_0 + \sum P_i (RR_i) - 1}{P_0 + \sum P_i (RR_i)},
\]

in which \(P_0\) is the percentage of individuals in the United States not engaging in the risk behavior, \(P_i\) is the percentage engaging in separate categories of the risk behavior, and \(RR_i\) is the RR of death for each separate category relative to none. For instance, in the case of smoking, \(P_0\) is the percentage of persons who never smoked, \(P_i\) is the percentage of former smokers, \(P_2\) is the percentage of current smokers, \(RR_i\) is the RR of a certain type of death for former smokers compared with those who never smoked, and \(RR_2\) is the RR of death for current smokers compared with those who never smoked. We then multiplied estimates of the cause-attributable fraction of preventable deaths by total mortality data. Whenever possible, we used RRs of death and mortality data by other variables such as age, sex, and race.

We estimated ranges for our estimated number of deaths by using the smallest and highest RRs and their boundaries when available. When data were available, we used specific underlying causes of death in deriving some of our estimates (ie, firearms, motor vehicles, and illicit drug use). Further details of these methods may vary due to availability of data and are presented in each section below. We used SAS (version 8.2, SAS Institute Inc, Cary, NC) and SUDAAN (version 8.0, Research Triangle Institute, Research Triangle Park, NC) statistical software.

**Results**

The number of deaths in the United States in 2000 was 2.4 million, which is an increase of more than 250 000 deaths in comparison with the 1990 total, due largely to population growth and increasing age. Leading causes of death were diseases of the heart (710 760), malignant neoplasms (553 091), and cerebrovascular diseases (167 661) (Table 1).
Tobacco

We used methods and software used in previous CDC reports to compute the annual smoking-attributable mortality for 2000. As in previous reports, we used RRs for each cause of death from the American Cancer Society’s Cancer Prevention Study II and included deaths due to secondhand smoking.

We used data from the Behavioral Risk Factor Surveillance System (BRFSS), a cross-sectional telephone survey conducted by state health departments with the CDC's assistance, to determine changes in US smoking prevalence from 1995–1999 to 2000. A detailed description of survey methods is available elsewhere. A slight decline in smoking was observed from 1995–1999 to 2000. The prevalence of smoking in 1995–1999 was 22.8% for current smokers (males: 25.1%; females: 20.6%), 24.1% for former smokers (males: 28.3%; females: 20.3%), and 53.1% for never-smokers (males: 46.5%; females: 59.2%). In 2000, these estimates were 22.2% for current smokers (males: 24.1%; females: 20.5%), 24.4% for former smokers (males: 28.3%; females: 20.7%), and 53.4% for never-smokers (males: 47.6%; females: 58.8%).

We estimate that approximately 435,000 deaths were attributable to smoking in 2000, which is an increase of 35,000 deaths from 1990 (Table 2). This increase is due to the inclusion of 35,000 deaths due to secondhand smoking and 1000 infant deaths due to maternal smoking, which were not included in the article by McGinnis and Foege.

Poor diet and physical inactivity

To assess the impact of poor diet and physical inactivity on mortality, we computed annual deaths due to overweight. Recent articles have reported that overweight increased in all segments of the US population. To derive the attributable number of deaths due to overweight, we used estimates from the CDC’s 1999 and 2000 National Health and Nutrition Examination Surveys. We used the same procedure reported by Allison et al to estimate annual overweight-attributable deaths. We used the body mass index (BMI) range of 23 to 25 as our reference category to match the method used by Allison et al. Body mass index is calculated as weight in kilograms divided by the square of the height in meters. Using data from the 1999 and 2000 National Health and Nutrition Examination Surveys, the percentages for BMI cut points

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>No. of Deaths</th>
<th>Death Rate per 100,000 Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease</td>
<td>710,760</td>
<td>258.2</td>
</tr>
<tr>
<td>Malignant neoplasm</td>
<td>553,091</td>
<td>200.9</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>167,661</td>
<td>60.9</td>
</tr>
<tr>
<td>Chronic lower respiratory tract disease</td>
<td>122,009</td>
<td>44.3</td>
</tr>
<tr>
<td>Unintentional injuries</td>
<td>97,900</td>
<td>35.6</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>69,301</td>
<td>25.2</td>
</tr>
<tr>
<td>Influenza and pneumonia</td>
<td>65,313</td>
<td>23.7</td>
</tr>
<tr>
<td>Alzheimer disease</td>
<td>49,558</td>
<td>18.0</td>
</tr>
<tr>
<td>Nephritis, nephrotic syndrome, and nephrosis</td>
<td>37,251</td>
<td>13.5</td>
</tr>
<tr>
<td>Septicemia</td>
<td>31,224</td>
<td>11.3</td>
</tr>
<tr>
<td>Other</td>
<td>499,283</td>
<td>181.4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2,403,351</strong></td>
<td><strong>873.1</strong></td>
</tr>
</tbody>
</table>

* Data are from Minino et al.
were less than 23 (22.3%), 23 to less than 25 (15.09%), 25 to less than 26 (7.49%), 26 to less
than 27 (7.36%), 27 to less than 28 (6.23%), 28 to less than 29 (6.30%), 29 to less than 30
(5.94%), 30 to 35 (16.95%), and more than 35 (12.62%).

We used hazard ratios reported previously 13 to recompute annual deaths for 6 major popu-
lation-based studies. The mean estimate of the total number of overweight-attributable deaths
in 2000 was 494 921. For the Alameda County Health Study, the estimated number of over-
weight-attributable deaths in 2000 was 567 683; Framingham Heart Study, 543 981; Tecumseh
Community Health Study, 462,005; American Cancer Society Cancer Prevention Study I,
451 708; Nurses Health Study, 504 602; and the National Health and Nutrition Examination
Survey I Epidemiologic Follow-up Study, 439 548.14–19

As in the study by Allison et al, the estimate for the attributable number of deaths for non-
smokers or never-smokers was higher than the estimate for the total because smoking is associ-
ated with both lower body weight and higher mortality. Also in 2000, the mean estimate of the
total number of overweight-attributable deaths among nonsmokers or never-smokers was
543 797. For the Alameda County Health Study, the estimate of overweight-attributable deaths
among nonsmokers or never-smokers was 639 026; Framingham Heart Study, 583 913; Tecum-
seh Community Health Study, 457 460; American Cancer Society Cancer Prevention Study I,
466 729; Nurses Health Study, 570 855; and the National Health and Nutrition Examination
Survey I Epidemiologic Follow-up Study, 544 798. Our estimates indicate an increase of 76.6%
over the 1991 estimate of overweight-attributable deaths, with more than 80% of excess deaths
occurring among individuals with class 2 and 3 obesity.

The prevalence of overweight used in this study is based on data from 1999–2000. Because
the effects of overweight on mortality may not appear until some years after a person becomes
overweight, it is likely that the increase in prevalence of overweight in the 1990s overestimates
the current actual number of deaths. However, the total number of deaths from the 1999–2000
data may well be the expected number of deaths in the next few years. Thus, we believe a more
accurate and conservative estimate for overweight mortality in 2000 such as 385 000, which
is the rounded average of 2000 and 1991 estimates (494 921 and 280 184).

Overweight would account for the major impact of poor diet and physical inactivity on
mortality.20 Diet may have a minor additional effect on mortality mainly from lack of certain
essential nutrients.21,22 Consumption of fruits and vegetables increased in the 1990s,23 and fat
intake as a percentage of calories declined.24 Physical activity has increased slightly.25 We esti-
mate that poor diet and physical inactivity will cause an additional 15 000 deaths a year, although

<table>
<thead>
<tr>
<th>Actual Cause</th>
<th>No. (%) in 1990*</th>
<th>No. (%) in 2000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>400 000 (19)</td>
<td>435 000 (18.1)</td>
</tr>
<tr>
<td>Poor diet and physical inactivity</td>
<td>300 000 (14)</td>
<td>400 000 (16.6)</td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td>100 000 (5)</td>
<td>85 000 (3.5)</td>
</tr>
<tr>
<td>Microbial agents</td>
<td>90 000 (4)</td>
<td>75 000 (3.1)</td>
</tr>
<tr>
<td>Toxic agents</td>
<td>60 000 (3)</td>
<td>55 000 (2.3)</td>
</tr>
<tr>
<td>Motor vehicle</td>
<td>25 000 (1)</td>
<td>43 000 (1.8)</td>
</tr>
<tr>
<td>Firearms</td>
<td>35 000 (2)</td>
<td>29 000 (1.2)</td>
</tr>
<tr>
<td>Sexual behavior</td>
<td>30 000 (1)</td>
<td>20 000 (0.8)</td>
</tr>
<tr>
<td>Illicit drug use</td>
<td>20 000 (&lt;1)</td>
<td>17 000 (0.7)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>1 060 000 (50)</strong></td>
<td><strong>1 159 000 (48.2)</strong></td>
</tr>
</tbody>
</table>

* Data are from McGinnis and Foege.1 The percentages are for all deaths.
this too may be conservative. Nutritional deficiencies alone (International Classification of Diseases, 10th Revision [ICD-10] codes E40–E64) were reported as the causes of 4242 deaths in 2000.

We estimate that 400 000 deaths were attributable to poor diet and physical inactivity, an increase of one third from 300 000 deaths estimated by McGinnis and Foege,¹ and the largest increase among all actual causes of death. However, poor diet and physical inactivity could account for even more deaths (> 500 000) when the 1999–2000 prevalence estimates of overweight have their full effect.

**Alcohol consumption**

We used 2 large nationally representative surveys to determine US alcohol consumption. The National Health Interview Survey, a household survey that measured alcohol intake in 1999 and 2000, and the BRFSS, a telephone survey that measured alcohol intake in 1999.⁸,²⁶

We used RRs from the Australian National Drug and Safety Report that were based on mortality rates derived from pooled data of several studies.²⁷,²⁸ The RR values were 1.33 for hazardous drinking (4.01–6.00 drinks/d for males and 2.01–4.00 for females) and 1.47 for harmful drinking (≥6.01 drinks/d for males and ≥4.01 for females) in contrast to low levels of drinking (0.26–4.00 drinks/d for males and 0.26–2.00 for females) and abstinence (0–0.25 drinks/d for both males and females).

We used BRFSS data to compute the number of alcohol-attributable deaths for the US population aged 18 years or older. The BRFSS also asked questions about binge drinking (ie, ≥5 drinks per occasion). To account for the effect that respondents appeared not to include binge drinking in their reported regular drinking, we reran our analyses, adding 5 drinks per binge occasion to average drinks per day. The total number of deaths attributable to alcohol was 103 350.

We also used 3 other recent studies to estimate alcohol-attributable mortality. Two studies were based on the National Health Interview Survey²⁹,³⁰ and the National Alcohol Survey.³¹ Using all-cause mortality and RRs from these studies, we estimated approximately 60 000 deaths per year. This difference in number of deaths is mainly due to the fact that BRFSS respondents report a higher percentage of heavy drinking than do respondents in a household survey such as the National Health Interview Survey.

In another approach, we aggregated alcohol-related deaths from specified ICD codes that were summed to provide an overall estimate of deaths. In 2000, 18 539 deaths were reported as alcohol-induced (ICD-10 codes F10, G31.2, G62.1, 142.6, K29.2, K70, R78.0, X45, X65). In addition, 16 653 persons were killed in alcohol-related crashes.³² We estimate another 34 797 deaths in 2000 using BRFSS alcohol consumption data and disease-specific RRs from the Australian study for oropharyngeal, esophageal, liver, laryngeal, and female breast cancers; stroke; hypertensive heart disease; and other chronic liver disease and cirrhosis (ICD-10 code K73-74). This totals to 69 989 deaths in 2000 from these factors alone. In the Australian study, all-cause mortality was also higher than the summation of cause-specific mortality.

Total alcohol-attributable deaths would reach about 140 000 if mortality among previous alcohol drinkers were included. It is unclear whether excess mortality among former alcohol drinkers is due to damage or illness from past alcohol consumption.

Taking these various numbers into account, our best estimate for total alcohol attributable deaths in 2000 is approximately 85 000, based on the conservative estimate from cause-specific deaths and the high estimate using all-cause mortality. This is a reduction of 15 000 deaths from the 1990 estimates.
Microbial agents

We excluded human immunodeficiency virus (HIV) from this category and included it with sexual behaviors to be consistent with the analysis by McGinnis and Foege. In the past, infectious agents were the leading cause of mortality. These agents still present a major threat to the nation’s health and are associated with high morbidity. Several improvements in the health system have led to a decline in mortality from infectious diseases. The increase in US immunization rates led to a decline in mortality from many vaccine-preventable diseases. Several laws ensure this high immunization rate for children by requiring vaccination for school and day-care enrollment. There also have been substantial improvements in sanitation and hygiene, antibiotics and other antimicrobial medicines, and hospital-infection control.

In 2000, influenza and pneumonia accounted for 65,313 deaths, septicemia for 31,224, and tuberculosis for 776. In general, mortality from infectious and parasitic diseases has declined since 1990. Because pneumonia and septicemia occur at higher rates among patients with cancer, heart disease, lung disease, or liver disease, some of these deaths really are attributable to smoking, poor diet, and alcohol consumption. We estimate that approximately 75,000 deaths were attributable to microbial agents in 2000 from all ICD-10 codes for infectious and parasitic mortality. The major cause of the decline was a decrease in deaths from influenza and pneumonia probably reflecting at least in part an increase in immunization in older adults against vaccine-preventable diseases. This contrasts with 90,000 deaths attributed to microbial agents in 1990 estimates.

Toxic agents

Estimating the number of deaths due to toxic agents is more challenging than any of the other risk factors due to limited published research and the challenges of measuring exposure and outcome. In the 1990s, many improvements were made in controlling and monitoring pollutants. There is more systematic monitoring of pollutants at state and county levels, and exposure to asbestos, benzene, and lead have declined. In fact, the US Environmental Protection Agency reported a decline of 25% from 1970 to 2001 in 6 principal air pollutants: carbon monoxide, lead, ozone, nitrogen dioxide, sulfur dioxide, and particulate matter.

Toxic agents are associated with increased mortality from cancer, respiratory, and cardiovascular diseases. We used the National Morbidity, Mortality, and Air Pollution Study to estimate mortality due to air pollution. The study assessed the association between air pollution and mortality and morbidity in 90 cities in the United States. Only particulate matter (PM) was associated with a significant increase in mortality – an approximate 0.5% increase in total mortality for each 10-µ/m³ increase in PM₁₀. Previous studies reported a range of 0.4% to 1% for that association. We used 23.8 µ/m³ as the daily average of PM₁₀ concentration in 2000, which results in an estimate of 24,000 deaths per year (range, 22,000–52,000 deaths) from air pollution alone.

The National Institute for Occupational Safety and Health (NIOSH) estimates that about 113,000 deaths are due to occupational exposure from 1968 to 1996. The number of deaths caused by occupational exposure has declined during that period. In 1996, NIOSH estimated 3119 deaths from pneumoconiosis and 1176 from asbestosis. Although, particulate air pollution accounts for the majority (about 60%) of mortality related to toxic agents, indoor air pollution, environmental tobacco smoke, radon, lead in drinking water, and food contamination are associated with increased mortality. We estimate that toxic agents (excluding environmental tobacco exposure) were associated with 2% to 3.5% of total mortality in 2000. We estimate approximately 55,000 deaths attributable to toxic agents in 2000. This estimate is our least certain of the various causes.
Motor vehicles

Motor-vehicle crashes involving passengers and pedestrians resulted in 43,354 deaths in 2000.4 This decline from 47,000 deaths in 1990 represents successful public health efforts in motor-vehicle safety.5,6 Deaths from alcohol-related crashes declined from 22,084 in 1990 to 16,653 in 2000.7 Major contributing factors include the use of child safety seats and safety belts,8,9 decreases in alcohol-impaired driving,10 changes in vehicle and highway design,11,12 and national goals to reduce motor-vehicle-related mortality and injury.13 We estimate that approximately 26,500 deaths in 2000 were attributable to motor-vehicle crashes in which alcohol was not a factor. This is an increase of 1,500 from the 1990 report because both estimates were not adjusted for the number of registered vehicles, number of crashes, nor miles of travel. We included alcohol-related deaths to stress that efforts to educate the public and enforce laws against driving while intoxicated have accounted for most of the decline in deaths related to motor-vehicle crashes.

Firearms

Firearm-related incidents resulted in 28,663 deaths among individuals in the United States in 2000.4 This is a decline from approximately 36,000 deaths in 1990. The largest declines were in deaths from homicides and unintentional discharge of firearms. In 2000, 16,586 deaths were due to intentional self-harm (suicide) by discharge of firearms (ICD-10 codes X72–X74). Assault (homicide) by discharge of firearms (ICD-10 codes X93–X95) resulted in 10,801 deaths. Unintentional discharge of firearms (ICD-10 codes W32–W34) resulted in 776 deaths, while discharge of firearms, undetermined intent (ICD-10 codes Y22–Y24), resulted in 230 deaths. The remaining 270 deaths were due to legal intervention (ICD-10 code Y35). These numbers were ascertained from death certificate reports.

Sexual behavior

Sexual behavior is associated with an increased risk of preventable disease and disability.14 An estimated 20 million persons are newly infected with sexually transmitted diseases each year in the United States.15,16 Mortality from sexually transmitted diseases is declining due to the availability of earlier and better treatment, especially for HIV.17,18 In 2000, HIV disease (ICD-10 codes B20–B24) resulted in 14,578 deaths. In 1990, HIV was the cause of 27,695 deaths for persons older than 13 years, indicating about a 48% decline in HIV mortality during the decade. Based on the sexual behavior-attributable fraction from the literature,19–21 we estimate that 20,000 deaths (range, 18,000–25,000 deaths) in 2000 were due to sexual behavior—mainly HIV; other contributors were hepatitis B and C viruses and cervical cancer. The decline of 10,000 deaths from the 1990 estimates1 was due to the decline in HIV mortality.

Illicit use of drugs

Illicit drug use is associated with suicide, homicide, motor-vehicle injury, HIV infection, pneumonia, violence, mental illness, and hepatitis.22–27 An estimated 3 million individuals in the United States have serious drug problems.28,29 Several studies have reported an undercount of the number of deaths attributed to drugs by vital statistics;30 however, improved medical treatments have reduced mortality from many diseases associated with illicit drug use. In keeping with the report by McGinnis and Foege,1 we included deaths caused indirectly by illicit drug use in this category. We used attributable fractions to compute the number of deaths due to illicit drug use.22,28,31 Overall, we estimate that illicit drug use resulted in approximately 17,000 deaths in 2000, a reduction of 3,000 deaths from the 1990 report.
Other factors
Several other factors contribute to an increased rate of death. There are factors that we do not know of such as unknown pollutants or perhaps exposures that may cause a considerable number of deaths. Poverty and low education levels are associated with increased mortality from many causes, partly due to differential exposure to the risks described above. However, controlling for differential exposure to risk factors is unlikely to explain the entire impact on mortality. Lack of access to proper medical care or preventive services is associated with increased mortality. Biological characteristics and genetic factors also greatly affect risk of death. In most studies we reviewed, low education levels and income were associated with increased risk of cardiovascular disease, cancer, diabetes, and injury. The Healthy People 2010 initiative has made the elimination of health disparities, especially racial and ethnic disparities, a primary goal.

Comment
We found that about half of all deaths that occurred in the United States in 2000 could be attributed to a limited number of largely preventable behaviors and exposures. Overall, we found relatively minor changes from 1990 to 2000 in the estimated number of deaths due to actual causes. Our findings indicate that interventions to prevent and increase cessation of smoking, improve diet, and increase physical activity must become much higher priorities in the public health and health care systems.

The most striking finding was the substantial increase in the number of estimated deaths attributable to poor diet and physical inactivity. We estimate that roughly 400,000 deaths now occur annually due to poor diet and physical inactivity. The gap between deaths due to poor diet and physical inactivity and those due to smoking has narrowed substantially. Because rates of overweight increased rapidly during the 1990s, we used a conservative approach to make our estimates, accounting for the delayed effects of overweight on mortality. In addition, overweight lessens life expectancy. However, it is clear that if the increasing trend of overweight is not reversed over the next few years, poor diet and physical inactivity will likely overtake tobacco as the leading preventable cause of mortality.

The most disappointing finding may be the slow progress in reducing tobacco-related mortality. A few states, notably California, have had major success in programs that led to reducing deaths from heart disease and cancer. However, efforts in most other states are too recent or short-term to have a similar effect. In response to the increase in tobacco use among youth in the early 1990s, state and national tobacco-control efforts increased their focus on prevention of initiation and recognized the importance of cessation on reducing smoking-related deaths. Thus, most national and state efforts now address comprehensive program strategies. Current tobacco-control efforts will also need strong cessation components to show a decline in tobacco deaths in a future assessment. Recent reports on the effects of telephone quit lines for smokers are encouraging. On the other hand, large state budget shortfalls are leading to large cuts in public health, with a corresponding diversion of resources from tobacco taxes and settlement dollars to cover deficits instead of tobacco-control programs.

Despite the call to action on these risk factors a decade ago, there has been little progress in reducing the total number of deaths from these causes. The progress that has occurred primarily involves actual causes of death that are less prominent. With the shift in the age distribution of the population, more adults now are in the age group at highest risk because of the cumula-
tive effects of their behavior. The net effect is that both total deaths and total burden due to the actual causes have increased.

Our analyses have several limitations. Our study reported actual causes of mortality in the United States. However, these causes are also associated with a large morbidity burden. In addition to premature death, years of lost life, diminished productivity, and high rates of disability, decreased quality of life is also strongly associated with these actual causes. A recent World Health Organization report finds these actual causes of death to be the leading causes of total disease burden, not just mortality, in the developed world. Because we used self-reported estimates for some risk behaviors, (i.e., prevalence of alcohol intake) they may have been underestimated. Finally, using all-cause mortality may result in overestimates of the number of deaths from specific causes. In addition, if the effect of the risk factor is age-dependent, then age- and sex-specific estimates are preferable.

Our analyses did not assess the effect of genetics. Genetic factors have been associated with several diseases discussed herein. Much of the impact of genetics is likely mediated through increased physical susceptibility to these behavioral and other modifiable risks. However, increases in obesity and diabetes cannot be due to widespread changes in the human genome over the last 10 years. Nevertheless, genetics offers great potential for treating and ameliorating risk. Identifying individuals at higher risk for a disease through genetic testing may promote lifestyle changes that can help prevent the onset of that disease.

In this study we also did not examine the effects of high blood pressure and cholesterol or lipid profile on mortality, although some of the effects of these factors are mediated through poor diet and physical inactivity. These risk factors are common among adults in the United States. More than 30% of US adults have high blood pressure or high cholesterol. Monitoring and controlling blood pressure and cholesterol is crucial to preventing premature mortality and morbidity.

One of the most difficult aspects of this analysis is that the attribution of the actual cause that led to death varies depending on perspective. We used similar methods to those used by McGinnis and Foege to allow comparisons. We tried when possible to use RRs that are fully adjusted for other risk factors in our analyses, but possibly not eliminating duplicate attribution of causes. We also explicitly included some deaths in more than 1 category (e.g., alcohol and motor vehicle crashes) when choosing another category seemed as though it might artificially constrain interpretation for future prevention programs.

In summary, smoking and the deaths attributed to the constellation of poor diet and physical inactivity currently account for about one third of all deaths in the United States. The rapid increase in the prevalence of overweight means that this proportion is likely to increase substantially in the next few years. The burden of chronic diseases is compounded by the aging effects of the baby boomer generation and the concomitant increased cost of illness at a time when health care spending continues to outstrip growth in the gross domestic product of the United States. In ancient times, Hippocrates stated that “the function of protecting and developing health must rank even above that of restoring it when it is impaired.” The findings in this study argue persuasively for the need to establish a more preventive orientation in health care and public health systems in the United States.
Notes

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References


Behavior as the central outcome in health care

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Abstract

A predominant justification for health psychology and behavioral medicine is that behavior or environmental conditions affect a biological process. Thus, many investigators focus attention on the effects of behavior on cell pathology and blood chemistry. This article argues that behavioral outcomes are the most important consequences in studies of health care and medicine. These outcomes include longevity, health-related quality of life, and symptomatic complaints. Traditional measures in biomedical science often have limited reliability and validity. Their validity is demonstrated only through relationships with longevity, role performance, behavioral functioning, and symptomatic experience, and these correlations are often modest. A model is proposed to guide future investigations. Biological, environmental, and psychological variables are included in the model as predictors or mediators of behavioral health outcomes. Recognizing that health outcomes are behavioral directs intervention toward whatever method produces the most health benefit at the lowest cost.

Health psychology and behavioral medicine are among the most rapidly developing areas of psychological research and practice. Although no one model has dominated the field, the biomedical disease model has guided most thinking. According to this model, syndromes expressed as signs and symptoms are associated with lesions or some underlying pathology. This pathology is the focus of research and the target of treatment. Interventions are made to eradicate the lesion or prevent its pathogenesis. The lesion, however, is the central focus of examination.

Reviews of the emerging field of behavioral medicine and health psychology often emphasize the role of behavior in the onset, maintenance, and treatment of disease (Miller, 1983). Many of these reviews characterize the role of stress on bodily processes. Krantz, Grunberg, and Baum (1985) emphasized the links between behavior and health through basic physiological mechanisms. Their review concluded with an emphasis on new technologies for assessing physiologic, rather than behavioral, health outcomes. For example, they pointed to the availability of portable blood-withdrawal pumps, blood pressure monitors, and biochemical assessment tools. Rodin and Salovey (1989) underscored the importance of disease end points. They encouraged

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health psychologists to focus on placement in specific disease categories such as cancer or coronary heart disease. These reviews characterize the field as emphasizing the impact of behavior on identified lesions or specific disease states.

Progressive versions of the medical model acknowledge that the cause of illness might be environmental or the lesion psychosocial. Even the biopsychosocial model (Engle, 1976), however, concentrates on sickness and its causes. Attention is directed toward the psychological or environmental etiology and the physiological lesion (White, 1988). These models have directed measurement toward assessment of disease categories, characteristics of lesions, and disease risk factors.

In their efforts to be in the mainstream, many behavioral research investigators also focus their studies on the health outcomes measured by physicians and other health care providers. Typically, these are measures of blood chemistry, physical characteristics, and blood or tissue sensitivity to medication. In this article I argue that the only important indicators of health and wellness are behavioral. Thus, outcome measures in health and medicine should be anchored in their relations with behavior. In this context the definition of behavior is general, as offered by Atkinson, Atkinson, Smith, and Hilgard (1987) in their widely used introductory psychology textbook. They define behavior as “those activities of an organism that can be observed by another organism or by an experimenter’s instruments” (p. 657). Included in behavior are verbal reports about subjective conscious experiences. In this article, I refer to biological measures as measures of physiological state. Biological measures and disease classifications are important precisely because they are predictors or mediators of behavioral outcomes.

In the following sections, I argue that there has been too much concentration on purely biological measures and that the importance of behavioral health outcomes has been undervalued.

**Behavioral health outcomes**

The conceptualization and measurement of health status has interested scholars for many decades. After the Eisenhower administration, a report of the President’s Commission on National Goals, (1960) identified health status measurement as an important objective. In his influential book, *The Affluent Society*, John Kenneth Galbraith (1958) described the need to measure the effect of the health care system on quality of life. In recent years there have been many attempts to define and measure health status.

The movement toward behavioral measures is an old one. When Sullivan (1966) reviewed the literature on health measurement nearly a quarter of a century ago, he emphasized the importance of behavioral outcomes. Bolstered by the accomplishments of behavioral scientists, Sullivan developed a convincing argument that behavioral indicators such as absenteeism, bed-disability days, and institutional confinement were the most important consequences of disease and disability. Ability to perform activities at different ages could be compared with societal standards for these behaviors. Restrictions in usual activity were seen as prima facie evidence of deviation from wellbeing. Health conditions affect behavior, and in this article behavioral health outcomes are conceptualized as observable behavioral consequences of a health state. Arthritis, for example, may be associated with difficulty in walking, observable limping, or problems in using the hands. Even a minor illness, such as the common cold, might result in disruptions in daily activities, alterations in activity patterns, and decreased work capacity.

Diseases and disabilities are important for two reasons. First, illness may cause a truncation of the life expectancy. In other words, those in specific disease categories may die prematurely. Death is a behavioral outcome. It can be defined as the point at which there is no observable
behavior. Second, diseases and disabilities may cause behavioral dysfunctions as well as other symptoms. Biomedical studies typically refer to health outcomes in terms of mortality (death) and morbidity (dysfunction) and sometimes to symptoms.

Mortality remains the major outcome measure in most epidemiologic studies and clinical trials. In order to make informed decisions about the nation’s health, Congress receives various reports of statistical indicators from the National Center for Health Statistics. These include the crude mortality rate, the infant mortality rate, and years of potential life lost. Although important, each of these measures ignores dysfunction while people are alive. The National Center for Health Statistics provides information on a variety of states of morbidity. For example, it considers disability, defined as a temporary or long-term reduction in a person’s activity. Over the last 15 years, medical and health services researchers have developed new ways to assess health status quantitatively. These measures are often called quality of life measures. Because they are used exclusively to evaluate health status, the more descriptive health-related quality of life is preferred (Kaplan & Bush, 1982). Some approaches to the measurement of health-related quality of life combine measures of morbidity and mortality to express health outcomes in units analogous to years of life. The years-of-life figure, however, is adjusted for diminished quality of life associated with diseases or disabilities (Kaplan & Anderson, 1988).

Modern measures of health outcome consider future as well as current health status. Cancer, for example, may have very little impact on current functioning but may have a substantial impact on behavioral outcomes in the future. Today, a person with a malignant tumor in a leg may be functioning very much like a person with a leg muscle injury. However, the cancer patient is more likely to remain dysfunctional in the future. Comprehensive expressions of health status need to incorporate estimates of future behavioral dysfunction as well as to measure current status (Kaplan & Anderson, 1988).

The spectrum of medical care ranges from public health, preventive medicine, and environmental control through diagnosis to therapeutic intervention, convalescence, and rehabilitation. Many programs affect the probability of occurrence of future dysfunction rather than alter present functional status. In many aspects of preventive care, for example, the benefit of the treatment cannot be seen until many years after the intervention. A supportive family that instills proper health habits in its children, for example, may also promote better health in the future, even though the benefit may not be realized for years. The concept of health must consider not only the ability to function now but also the probability of future changes in function or probabilities of death. A person who is functional and asymptomatic today may harbor a disease with a poor prognosis. Thus, many individuals are at high risk of dying from heart disease even though they are perfectly functional today. Should we call them healthy? The term severity of illness should take into consideration both dysfunction and prognosis. Comprehensive models that combine morbidity, mortality, and prognosis have been described in the literature (Kaplan & Anderson, 1988). A behavioral conceptualization of health status can represent this prognosis by modeling disruptions in behavior that might occur in the future.

Many medical treatments may cause near-term dysfunction to prevent future dysfunction. For example, coronary artery bypass surgery causes severe dysfunction for a short period of time, yet the surgery is presumed to enhance function or decrease mortality at a later time. Patients may be incapacitated following myocardial infarction and restricted to coronary care units. Yet the treatment is designed to help them achieve better future outcomes. Pap smears and hysterectomies are performed in order to decrease the probability of future deaths due to cancer. Much of health care involves looking into the future to enhance behavioral outcomes over the life span. Therefore, it is essential to divide health into current and future components.
In appraising the importance of behavioral outcomes, we must ask why there is concern about diseases, injuries, and disabilities. The behavioral perspective suggests that the only reasons are the following: (a) Life expectancy may be shortened, (b) quality of life may be compromised either now or at some time prior to death, or (c) some combination of a and b. A disease that has no impact on either life expectancy or life quality would be unimportant. In fact, disease states gain their importance precisely to the degree to which they correlate with decreased longevity or impaired health-related quality of life.

The importance of behavioral outcomes has not been disregarded by the traditional medical community. In fact, recognition of the centrality of behavioral outcomes has been emphasized in several articles and editorials recently featured in *The New England Journal of Medicine* (Ellwood, 1988; Greenfield, 1989; Shortell & McNerney, 1990). Despite the growing recognition of the importance of behavioral outcomes by the medical community (Advances in Health Status Assessment, 1987; Bergner, 1989; Institutes of Medicine, 1989; Quality of Life Assessment, 1988a, 1988b; Shumaker, Furberg, Czajkowski, & Schron, in press; Walker & Rosser, 1988), behavioral scientists manifest a paradoxical reluctance to follow this trend. Instead, the trend has been to focus on measures of biological process.

**Trend toward biological variables as opposed to behavioral indexes**

We are witnessing a trend toward the biologicalization of both behavioral and biomedical sciences. Reviews of the health psychology literature criticize studies that do not focus on some aspect of blood chemistry or those that do not use disease categories (Baum, Grunberg, & Singer, 1982). Measures of biological process are seen to be more pure, more reliable, and more valid than are behavioral indicators. Thus, an increasing number of studies assess health status through measures of blood cholesterol, blood pressure, or characteristics of the immune response, including natural killer-cell and t-cell activity. W. T. Kelvin created the doctrine that measurement is the prerequisite to science. For most of this century, scientists and clinicians followed the doctrine and attempted to use measures, even when the validity of the measures was unknown. Feinstein (1967) suggested that modern trends represent the “curse of Kelvin.” The fact that there is a measure for some variable does not always mean that the measure is useful. Clinicians have been more attracted to blood pressure and cardiac output than to headache and anxiety. Clearly, the former are easier to quantify, but are they more meaningful?

It is important to emphasize that not all biological variables are measures of health status or health outcome. They are, however, predictive of some health outcomes. Elevated blood pressure, for example, is important because it predicts premature mortality or behavioral dysfunction resulting from coronary heart disease and from stroke. If blood pressure were unrelated to these behavioral outcomes, it would be a matter of little concern. There are many aspects of blood chemistry that bear no relation to clinical outcomes. Even common clinical tests, such as urine analysis, serum phosphorus, and alkaline phosphatase, have only weak and inconsistent relations to outcomes in all but the most extreme cases. Amberg, Schneiderman, Berry, and Zettner (1982), for example, demonstrated empirically that the alkaline phosphatase screening test provides essentially no information relevant to health outcome. Elevated blood cholesterol may be predictive of future bad outcomes, including early mortality from heart disease. However, other lipids in blood such as very low-density lipoproteins or chylomicrons may bear little relation to health outcomes in all but the most extreme cases. Modest elevation in these fractions of blood lipids may be of little concern.
Recently there has been a significant growth of interest in the relation between stress and measurable aspects of immune function. Temoshok, Soloman, and Jenkins (1989) cautioned scientists against overinterpreting these immunologic measures. Normal oscillations in most immune parameters are still poorly understood. Immunologists are uncertain about whether absolute numbers or percentages of cell subtypes are most meaningful. Most important, the immune system is a genuine system in which various components adjust to changes in one another, and some important aspects of the system may remain to be identified. Our understanding of the relation between immune parameters and health is still very sketchy.

Biological measures are also assumed to be more reliable than behavioral tests. Often however, the reliability of these measures is not assessed. When data are available, the results can be discouraging. Many investigators, fascinated by blood pressure as an outcome measure, have criticized behavioral measures for being non-physiologic and unreliable. Yet the reliability of blood pressure is equally open to question because conventional sphygmomanometric measurements have poor test–retest coefficients. This leads to misclassification, incorrect diagnosis, and potentially damaging labeling (Hla, Vokaty, & Fuessner, 1986; Patterson, 1984). There are many sources of error in blood pressure measurement. These include misreading biases, time sampling problems (blood pressure changes minute to minute), and situational factors. For example, it has been demonstrated that some patients have specific arousal of blood pressure in the presence of physicians. The condition has now been given the diagnostic label white coat hypertension (Pickering et al., 1988). Low reliability is not limited to blood pressure. It also characterizes measures of blood cholesterol, glucose, and a large number of other biochemical assays.

One of the important appeals of biological measures is that they focus on objectively defined events. Behavioral outcomes are often not measured objectively. Observer bias common to behavioral measures may be less common with biological measures. However, at a conceptual level, behavioral outcomes can represent defined events such as exercise or role performance. Subjective events, such as pain or discomfort, are characterized in pain behaviors and through verbal behaviors. It is tempting to assume that biological measures are more valid and reliable because they have less observer bias. However, they may include several other sources of measurement error and they do not necessarily have evidence for validity.

To summarize, in order to avoid known problems with behavioral measures, researchers and clinicians have been attracted to outcomes that can be measured with biochemical assays, mechanical devices, or auto-analyzer machines. Although these measures are not subject to the same errors as behavioral tests, they have their own sources of error and often have low reliability and questionable validity. Establishing the validity of biological measures requires a model that relates them to health status.

Are medical measures more valid or meaningful?

It is often assumed that the relation between the biologic variables and health outcomes is nearly perfect. However, there is a remarkable variability in behavioral health outcomes within fixed levels of many biological variables. There are numerous examples, of which only three will be considered; biologic measures of arthritis, blood pressure, and blood cholesterol. The arthritis example emphasizes current behavioral health outcomes, whereas the latter two focus on future behavioral outcomes and mortality.
Arthritis

Clinical outcomes in studies of rheumatology have been difficult to evaluate (Deyo, 1988). Clinical measures often include joint tenderness, grip strength, and joint circumference. Some studies have shown that the reliability of these measures is often poor (Buchanan, 1982). Fries (1983) questioned the validity and reliability of a variety of traditional outcome measures, ranging from laboratory measures of erythrocyte sedimentation rate (ESR), latex fixation titer, and hemoglobin. It has been shown that rheumatoid arthritis patients may develop serological abnormalities that are poorly correlated with joint inflammation (McCarty, 1986). In addition, Fries suggested that traditional clinical measures such as grip strength, walking time, and patient global assessment are merely surrogates for the true outcomes in arthritis, which he argued are disability, physical discomfort, and financial loss. An elevated ESR means little to a patient who feels fine and can conduct his or her life without pain. The ESR characterizes current inflammation but does not give information about future dysfunction. Conversely, a patient with disabling arthritis pain is not well when the ESR is normal. Clinical tests are useful only when they identify treatment to remedy current dysfunction or predict future problems. Fries asserted that pain and functional outcomes are meaningful to the patients and that clinical measures are of less importance. As a result, a growing number of rheumatologists are focusing attention on behavioral or functional health outcomes (Anderson, Firschein, & Meenan, 1989).

Blood pressure

Elevated blood pressure is a serious problem in the United States and in most other developed countries. Following the Hypertension Detection and Follow-up Program (HDFP), elevated blood pressure came to be defined as systolic pressure exceeding 140 mmHg or diastolic pressure (DBP) exceeding 90 mmHg. Using these guidelines, it has been estimated that as many 58 million adults (about 30% of the adult population) have hypertension (Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, 1985). Investigators are concerned about elevated blood pressure because of its relation to future behavioral health outcomes. Several major epidemiologic studies have documented the relation between elevated blood pressure and both morbidity and mortality (National Heart, Lung and Blood Institute, 1984). In addition, evidence from the HDFP (1979) has demonstrated that reductions in blood pressure result in reductions in deaths due to heart disease. People are often not concerned about high blood pressure because it may produce no symptoms or current behavioral dysfunction. High blood pressure does have a bad prognosis, with affected individuals being at risk for behavioral dysfunction or death later in life. However, the relation between blood pressure and both mortality and morbidity are far from uniform. Severe elevated blood pressure is a severe risk for mortality, whereas blood pressure in the mild hypertension range (DBP = 90–104 mmHg) is a less significant risk (Rocella, Bowler, & Horan, 1987). Indeed, most of those with mild hypertension, even those untreated, have normal life expectancies with no complications.

Although high blood pressure is associated with risks, the treatment of high blood pressure may cause some problems. Significant numbers of patients experience dizziness, tiredness, and impotence when treated with medications (Breckenridge, 1988). Thus, the treatment of high blood pressure can cause undesirable health outcomes. Treatment, like hypertension, is a factor that may influence behavioral health outcomes, sometimes in the negative direction. Studies that measure only blood pressure and neglect these behavioral side effects will overestimate the net benefit. Conversely, too much focus on side effects might lead to an incorrect judgment that the treatment should be avoided. There may be considerable advantage in translating the side
effects and benefits into common behavioral units and weighing them against one another in the treatment decision process (Kaplan & Atkins, 1989). The role of the clinician is to balance carefully the benefits and consequences of treatment (Aderman & Madhavan, 1981).

In summary, blood pressure is an important risk factor for heart disease and stroke. Systematic efforts to reduce blood pressure are advisable and effective. Yet, blood pressure is not a health outcome. It gains its importance through validity studies that demonstrate the association between blood pressure and behavioral outcomes including mortality, dysfunction, and symptomatic disturbances. Blood pressure is important because it provides probabilistic information about behavioral outcomes.

**Cholesterol**

The United States is currently experiencing a massive societal response to the presence of cholesterol in the diet. Numerous commercial products are promoted because they have no cholesterol. The term _hypercholesterolemia_ suggests an increased concentration of cholesterol in blood. Total cholesterol values above 200 mg/dl are now considered to be diagnostic (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 1988). Several epidemiologic studies have identified elevated blood cholesterol as a risk factor for cardiovascular disease mortality (Kannel, Castelli, Gordon, & McNamara, 1971). Yet the connection between dietary cholesterol and serum cholesterol has been less clearly established. Studies in metabolic wards and selected experimental studies do demonstrate that dietary manipulation can reduce serum cholesterol in the short run, although longer term changes have not been clearly documented. Furthermore, although there have been ecological correlations between estimated total fat consumption and total heart disease mortality across cultures, correlations within countries have not been systematically observed (Kaplan, 1985). A variety of explanations can be suggested for these “nonfindings.” For example, measurement error in both dietary cholesterol and serum cholesterol may account for the null results (Jacobs, Anderson, & Blackburn, 1979). But the availability of an explanation for a nonfinding is not a demonstration that a significant association exists (Kaplan, 1988). Thus, the relation between dietary cholesterol and serum cholesterol is somewhat ambiguous.

Stallones (1983) criticized the diet–heart-disease connection, suggesting that there is no zero-order relation between diet and mortality. The problems with the cholesterol interventions have been reviewed in several earlier articles (Fries, Green, & Levine, 1989; Kaplan, 1984, 1985). These positions are regarded as controversial, but they are related to the current issue.

The reason that cholesterol is important is that elevated cholesterol is a risk factor for behavioral health outcomes. If it were not, why would one care? The important point is that the outcome itself must be considered. The role of the investigator is to determine the relation between modifiable habits (dietary patterns) and outcomes as mediated through the channel of serum cholesterol. Serum cholesterol can serve as a target for modification, but one must be assured that modifying serum cholesterol improves outcome and does not adversely affect health status.

**Deaths in general versus deaths from specific causes**

Some investigators, recognizing the problems with measures of disease process, turn their attention toward the ultimate medical outcome – death. As noted earlier in this review, vital status is considered to be a behavioral outcome. However, the behavioral approach differs from the tra-
ditional medical model in its emphasis on life–death status without reference to medical cause of death. The emphasis is on observable outcome rather than on disease category.

Many medical studies confuse outcome with placement in disease categories. Results from several recent clinical trials illustrate this point. In one widely cited study (Steering Committee of the Physicians’ Health Study Research Group (1988, 1989)), physician subjects were assigned to take aspirin (325 mg/day) or placebo in order to prevent myocardial infarctions. The ultimate aim, of course, was to reduce the number of deaths associated with heart disease. Indeed, there was a significant reduction in deaths from myocardial infarctions over an eight-year follow-up period. This result was highly publicized and even earned aspirin the description of a “miracle drug” on the cover of *Newsweek* magazine (Clark, Gosnell, Hager, Carroll, & Gordon, 1988). Yet closer inspection of the data reveals that there was no advantage of aspirin for the crucial behavioral outcome – life or death. The left panel of Figure 1 shows deaths from all cardiovascular categories in the Physician’s Health Study. This stacking histogram reveals that reductions in death from myocardial infarction were compensated for by increases in death from other cardiovascular causes. There was a trend toward more hemorrhagic strokes among those taking aspirin, and there was the suggestion that aspirin may cause these strokes because it reduces blood clotting. Overall, aspirin did not reduce the number of deaths but changed the distribution among categories (Kaplan, 1989b).

The center portion of Figure 1 shows a similar result for the Coronary Primary Prevention Trial (Lipid Research Clinics Coronary Prevention Trial Results, 1984). In this study, a group of about 1,900 men at risk for coronary heart disease was given cholestyramine, a resin that binds bial acids and lowers serum cholesterol, whereas another group of about 1,900 men was given a placebo. Among the 3,800 male participants, 38 (2%) in the control group and 30 (1.6%) in the experimental group died of heart disease. The study is widely cited as the crucial evidence for cholesterol reduction. In addition, data from the study have been used to argue that a 1% reduction in cholesterol results in a 2% reduction in mortality. This has come to be known in health promotion campaigns as the 1% to 2% rule. The exact calculation of this 1% to 2% rule, so often cited in public statements, is difficult to follow. Forming a ratio of these small percentages of deaths and subtracting from 1.0 gives the estimate of about a 21% reduction in mortality (1.0 – [0.0157/0.022]). This combined with an observed 12% reduction in cholesterol yields the 1% to 2% rule.

This important study represents the scientific basis for the current campaign toward cholesterol reduction. The stacking histogram does suggest a significant reduction in heart disease deaths among those randomly assigned to cholestyramine. Yet the entire height of the bars in the stacking histogram shows that there was no advantage of treatment for total mortality. According to the behavioral conceptualization, the treatment had no benefit. Reductions in death from heart disease were compensated for by increases in death from other causes.

The right portion of Figure 1 shows similar results from the Helsinki Heart Study, which evaluated a similar drug called gemfibrozil (Frick et al., 1987). In the Helsinki study, 2,051 men were randomly assigned to take gemfibrozil twice daily, whereas another 2,030 men were given a placebo. After six years, 19 of the men in the placebo group had died of ischemic heart disease, whereas only 14 of those in the drug-treated group had died. This significant difference led the authors to conclude that gemfibrozil caused a 26% reduction in ischemic heart disease deaths. The 26% is calculated as follows: In the drug group, 0.68% (14/2,051) died, whereas in the placebo group 0.93% (19/2,030) died of ischemic heart disease. The actual difference is about one fourth of one percent. However, the ratio (0.0068/0.0093) subtracted from 1.0 yields about a 26% reduction. Furthermore, the total number of deaths in the Gemfibrozil group was actually higher than those in the placebo group (45 vs. 42).
Some argue that cholesterol-lowering drugs should still be regarded as efficacious because there is no biological model that would explain why decreased cholesterol should lead to increased deaths in nondisease categories. However, the finding that cholesterol lowering does not reduce total mortality has now been reported in several different studies (Fries et al., 1989). The burden of proving benefit rests with the treatment advocates. Those who adopt the traditional disease-specific view might be satisfied with reductions in cardiovascular deaths. However, the more comprehensive behavioral model requires a reduction in total mortality. Adoption of this model might stimulate new research designed to explain the increased deaths in nondisease categories.

In all three of these important clinical trials, there was a highly publicized benefit of treatment. However, the benefit only occurs for a specific disease category. There was no benefit of treatment with regard to the important life-death outcome (see total height of columns in each section of Figure 1). Investigators and the lay press often focus on improvements in a specific cause of death. Yet families of the deceased may be more concerned that the subject is dead than they are about the specific cause of demise. Focus on specific categories can obscure the most important behavioral outcomes. Research directed toward specific disease categories or aspects of a biological process may not capture global concerns about health. That task requires a comprehensive behavioral model.

Figure 1  Total mortality in three clinical trials: Physicians Health Study (top), Coronary Primary Prevention Trial (center), and Helsinki Heart Study (right).

Note  
CVD = cardiovascular disease; sudden = sudden death; MI = myocardial infarction; CHD = coronary heart disease.
Model of health outcomes

A model of behavioral health outcomes is presented in Figure 2. In the center of the figure is tissue or organ pathology, which makes up most of the study of medicine and results in most of the serious illnesses. These illnesses might be caused by multiple sources, including inherited birth problems, various diseases, the aging process, defects in the genetic program, and accidents. Each of these problems may be caused by biochemical or structural problems. The sources listed in Figure 2 are selected examples and are not intended to be exhaustive. Investigators are concerned about these biological problems because they may eventually affect behavior. If a disease or condition had an impact on a tissue or organ system but had no effect on life expectancy, no effect on function, no effect on appearance or symptoms, would it be of concern?

Most models in health psychology and behavioral medicine have a biological measure on the right side (implying that biology is the outcome). It is often emphasized that behavior is important because it can affect biological process. For example, diet can affect serum cholesterol and stress can affect natural killer cells. Figure 2 suggests a different focus. Both biological and environmental events gain their importance because they affect behavior.

Can a behavioral conceptualization influence research and practice?

Focus on biological rather than behavioral outcomes has led many investigators down the wrong path. For example, elevated levels of protein in urine suggest that the kidneys are misfiltering and removing some proteins that the body needs. Many years ago, when physicians measured high levels of protein in urine, they advised their patients to eat less protein. That advice led to poorer health outcomes because the body was already protein deprived. Ultimately, identifying the manipulations that led to better behavioral health outcomes led to more effective treatments. Another example involves the diagnosis and treatment of back pain. Legal definitions of disability sometimes require a physical diagnosis, and the medical evaluation often identifies a structural problem. This leads to the incorrect conclusion that the prognosis is poor. Thus, the medical model reinforces dysfunction, even though rehabilitation is common.

![Diagram](image-url)

*Figure 2* Relation between biological events, physiology, and behavioral health outcomes.
A comprehensive view of health can reveal when new treatment approaches are needed, whereas focus on specific processes might misdirect treatment.

The traditional approach leads to a focus on risk factors rather than on health outcomes. Yet, modification of risk factors may not necessarily improve health. For example, many epidemiologic studies have failed to find a relation between coffee consumption and death due to heart disease. Several very thorough evaluations have shown that those who drink coffee have the same life expectancies as those who abstain (Wilson, Garrison, Kannel, McGee, & Costelli, 1989). On the other hand, some investigators have reported that coffee increases low density lipoprotein cholesterol or blood pressure (Thelle, Heyden, & Fodor, 1987). Because cholesterol and blood pressure are risk factors for heart disease, people are advised to give up the coffee they enjoy. The logic behind this advice might be challenged, however, as coffee does not increase the risk of heart disease or other behavioral health outcomes.

Treatment of factors suspected of causing undesirable behavioral outcomes is usually advisable. Yet change in these risk factors does not assure that the behavioral goal will be achieved. One recent example is the treatment of cardiac arrhythmias. Research had demonstrated that adults who had suffered a heart attack were at risk for sudden death if they experienced asymptomatic cardiac arrhythmias (Bigger, Fleiss, Kleiger, Miller, & Rolnitzky, 1984). Drugs were available to suppress these cardiac arrhythmias, and these products were used often. In what many thought was a demonstration of the obvious, the National Institutes of Health initiated a major clinical study involving 1,455 post myocardial infarction patients in a variety of major medical centers (Cardiac Arrhythmia Suppression Trial [CAST] Investigators, 1989). The patients were randomly assigned to take anti-arrhythmic medication or placebos. All of the participants had been screened and demonstrated to experience suppression of their arrhythmia in response to the medication. Over an average of 10 months of follow-up, however, those assigned to the active drug had a significantly higher rate of death from arrhythmia than those assigned to the placebo. In addition, those in the active medication group had a higher overall death rate. If the investigators had only measured the response of the heart rhythm to anti-arrhythmic drugs, they would have concluded that the drug produced a benefit. Following the patients through the behavioral outcome (mortality) inspired them to stop the trial early and declare the medications unsafe.

The important point is that physiologic and biochemical measures do not necessarily have meaning. They gain their meaning through systematic correlations with health outcome. Attention directed at behavioral health outcomes can clarify the importance of biological processes.

Pathways to health outcomes

In this article I have argued that the only important outcomes in health and illness are behavioral. Clearly, these outcomes are deserving of one’s attention. People expend tremendous resources in order to achieve better health status. In fact, in the United States, more is spent on health care than on food (Kaplan, 1989a). How might one realize the best return on one’s investments in terms of health outcomes?

There are at least two ways to achieve better health outcomes. The first is through the modification of mediators of the behavioral outcomes. This is accomplished by identifying tissue pathology and seeking its remedy. Thus, those with diabetes experience poor health outcomes because of a problem in insulin production or insulin action. By supplying more insulin or by tuning up insulin receptors, better health outcomes may be achieved. Those with tumors may experience better health outcomes with the tumors excised. There is nothing wrong with the
medical model. In fact, direct treatment of pathology (lesions) remains one of the best methods for improving health outcomes. The traditional practice of medicine and surgery should be viewed as a set of methods designed to improve behavioral outcomes.

Another pathway for improving health outcomes involves modification of behavior, independent of the disease pathway. For example, patients with chronic obstructive pulmonary disease may face a situation in which there are no known medical or surgical remedies. However, behavior modification programs may enhance functioning independent of improvement in disease state (Atkins, Kaplan, Reinsch, Lofback, & Timms, 1984; Kaplan & Atkins, 1988). Pain treatment may also benefit from this conceptualization. Substantial evidence now suggests that pain and suffering are distinct. Pain behavior can continue after the injury that initiated the pain has healed. Several studies have shown that behavior modification can alter behavioral health outcomes for those with back pain, even though it does not affect back physiology in measurable ways (Fordyce, 1988). Health outcomes are behavioral, and one way to improve them is to modify behavior.

The behavioral conceptualization does not disregard the traditional medical model. Indeed, medicines and surgeries are excellent methods for improving behavioral health outcomes. However, the behavioral model is broader. Medicines, surgeries, and behavioral interventions are complementary methods for enhancing behavioral health outcomes. Often one alternative is superior in terms of efficacy or efficiency. For example, hernias can be surgically repaired and it would be inappropriate to use behavioral treatment to modify outcomes related to these problems. On the other hand, several disabilities do not respond to medicines or surgeries. For these, behavioral interventions may be the best alternative for producing a health benefit. Using behavioral outcomes as the target of care allows different alternatives to compete. Ultimately, treatments should be favored if they produce the most benefit at the lowest cost.

**Summary**

Physicians have long recognized that disease categories provide minimal information about the impact of illness upon patient experiences (Ellwood, 1988). A diagnosis is important because it may identify a course of treatment. Yet within specific diagnoses, patients differ considerably in how they are affected. Multiple sclerosis, for example, may have essentially no impact on behavioral dysfunction or it could have devastating implications. The impact of the disease on the daily life of the patient may be more important than naming the condition. A recent editorial in the *Journal of the American Medical Association* concluded that physicians need to learn to “treat the patient, not the disease” (Riesenberg & Glass, 1989, p. 943).

There are only two health outcomes that are of importance. First, there is life expectancy. Second, there is function or quality of life during the years that people are alive. Biological and physical events are mediators of these behavioral outcomes. Individuals are concerned about cancer, high blood pressure, high cholesterol, or other problems because they may shorten the life expectancy or make life less desirable prior to death. There is a growing consensus that these behavioral outcomes are central in studies of health care and medicine. Yet refinement of these measures requires active participation of behavioral scientists. Although behavioral outcomes are gaining a stronger foothold in medical research, psychologists and behavioral scientists have shown minimal interest. A behavioral conceptualization of health outcomes may suggest important new directions for research and practice.
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