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Communicating the Health Effects of Consumer Products: The Case of Moderate Alcohol Consumption and Coronary Heart Disease

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Moderate consumption of alcoholic beverages (MAC) has been estimated to significantly reduce the risk of myocardial infarction. This paper examines effective ways to communicate this information to guide individuals and their physicians, who must weigh personal benefits and costs when deciding about drinking. It argues that presenting a scalar representation of the effect, life years saved, is much more effective than the ways such information is currently communicated. A simulation using data from the Framingham Heart Study and a conservative estimate of the effect of MAC calculates age-specific gains in mortality, and survival gains from MAC. They are roughly 0.75 and 0.63 years, respectively, for men and women ages 21–50. Any MAC decision should weigh the benefits of cardiac risk reduction against possible financial, lifestyle or offsetting health costs.

INTRODUCTION

Decisions about what to ingest, including food, drugs, and supplements, are the most important health-affecting choices most people make. The health effects of these choices are extensively studied, yet somehow only vaguely appreciated by the most important decision makers, individual consumers. We all know that broccoli is good for us and ice cream is bad. But how good and how bad? Given that this question does not allow for a precise answer, what information do consumers need in order to make optimal decisions?

This paper addresses the question of effective communication in the context of one aspect of one health-affecting consumer good, alcoholic beverages. Almost everyone who reads the newspapers knows there is some evidence that drinking provides some benefit for cardiac health. This is potentially useful information, but few know more details than are contained in this vague statement. Should we try to do better? If so, how can we do better?

It should be emphasized that this analysis focuses on consumer decision making and communication. We do not purport to offer new insight into the effect that alcohol consumption has on coronary heart disease (CHD). We do not know which of the many estimates of this effect is 'right', if indeed any of them are. There are many questions about these estimates in the medical literature, and elsewhere (Phillips, 1995), we suggest there may be a frequent statistical error in correcting for the endogenous effects of blood pressure.

The next section provides a background discus-

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sion of alcohol as a consumer choice and its effects on coronary health. The third section provides a method for converting existing statistics to a scalar—life years saved—that individuals can understand, and lists some related considerations. The fourth section looks at the tradeoffs a consumer faces in deciding to consume alcohol. The final section concludes.

ALCOHOL: A COMPLICATED CONSUMER DECISION

Consumption of alcoholic beverages is a particularly interesting consumer choice. Its costs include purchase price, the detrimental effects of intoxication, ethical implications under some belief systems, and some detrimental health effects.[1] Its benefits include the pleasure of intoxication, its role in social situations, and some health benefits. Calculating how the average person wants to trade off these costs and benefits—let alone providing recommendations that are applicable to everyone—is beyond the abilities of any analyst or policy maker. Thus, if effective decisions are to be made, they must be made by each individual. Ideally, these decisions can be well informed.

Alcohol consumption is made even more interesting by the fact that its well-known externalities and addictiveness lead to public health policies that are intended to reduce consumption. There has been an attitude in public health policy that discouraging alcohol consumption is a universally positive accomplishment. Yet it is far from self-evident that the costs of drinking always outweigh the benefits. This is true even if we consider only the health aspects of alcohol consumption.

The preponderance of evidence in the medical literature indicates that moderate drinking (generally defined in the range of half a drink to a few drinks per day) has a major beneficial effect on coronary heart disease. These studies and reports have generally shown that people who drink the optimal amount (for CHD benefits) reduce their risk of CHD by 10–60% compared to teetotallers. More than 100 studies have found that moderate alcohol consumption significantly reduces the risk of various forms of CHD. (For a list of relevant studies, see the bibliographies of Maclure, 1993, and Srivastava *et al.*, 1994.) Manson *et al.* (1992) review the literature to compare moderate alcohol consumption (MAC) to other methods of preventing myocardial infarction, and conclude that MAC reduces risk by 25% to 45% (a risk ratio of 0.55 to 0.75). This is substantially greater than the available gains from cholesterol reduction and just below those for reasonable levels of weight reduction and increased exercise.[2]

But what does this mean to the individual consumer? Probably little or nothing for most of them, beyond being good news because heart attacks are bad. The baseline probability of heart attack mortality, let alone the impact of the whole menu of risk factors, is likely to be unknown to most consumers and even many of the medical professionals giving advice in this area. We suggest an alternative presentation, one that shares many of the limitations of any scalar representation of a complicated set of data, but that is in units that people can understand.

More than most health interventions, consuming alcohol is a very individual decision. Many dimensions of the decision to drink depend on individuals' propensities, behaviors, and ideologies. This makes an intervention like MAC close to a traditional market good, if adequate information is provided. An intervention that is relatively close to a market decision has the advantage that in implementing it no central decision maker, such as the government, has to determine the levels of tradeoffs to impose on the population. In a first-best world (one without externalities[3] and with people who correctly process information), it would be sufficient to provide information to the public about all major benefits and costs. Having done so, individuals would make decisions that result in the optimal level of alcohol consumption across the population.

Many medical interventions introduce a principal–agent problem, where an individual depends on a health professional to assess most positive and negative indications. The health professional may have inadequate knowledge or incentive to assess the full list of possible benefits and contraindications. In the case of MAC, however, the individual making the decision should have self-knowledge of contraindications, such as religious objections to alcohol consumption, social discomfort with alcohol, a propensity toward excessive alcohol consumption, etc. This will tend to reduce the risk that someone would use MAC to reduce

CHD when, all factors considered, it does more harm than good.

For goods that are both consumer products and medical interventions, consumers need to know two things to make wise choices. First, they need to know how to properly employ the intervention, such as getting the dosage right. Second, they need to know the benefits of the intervention so they can make the cost-benefit tradeoff in deciding whether to consume the good.

How Much is Good?

The question of how to employ the intervention properly is relatively easy for alcohol, unlike many other health-affecting decisions. This contrasts with exercise, where the optimal level requires more time and energy than most people are willing to expend; total calorie intake, which varies widely across people; or vitamin C intake, where there are orders-of-magnitude disagreements in the literature. The level of consumption to achieve optimal CHD effects from alcohol appears to be inexpensive, wide enough to encompass most of the population, and generally agreed upon.

Most or all of the beneficial CHD effects of MAC are seen when consumption is as low as a few grams of alcohol per day. (As a rule of thumb, there are usually 12 to 15 grams of alcohol in a 12-ounce beer, a 5-ounce glass of wine, or a 2-ounce shot of liquor.[4] Hereafter each is referred to as one 'drink'.) The typical recommendation is one-half to a few drinks per day, distributed so that consumption is relatively even (e.g. one drink every two days is fine, four drinks every Saturday does not work). The shape of the dose–response curve for CHD risk due to alcohol consumption is variously found to be U-shaped, reverse-J-shaped, or L-shaped.[5] Most studies that have found a U-shaped risk curve find that it has a fairly flat bottom and does not begin to turn up until a fairly high level of consumption—5 to 7 drinks per day.[6]

It is important to remember that the high end of the dosage that provides maximal CHD reduction has a wide range of negative health effects. The upper limit for 'moderate drinking' suggested by Turner *et al.* (1981), based on possible negative health effects, is 0.8 grams of alcohol per kilogram of body weight per day, not to exceed 80 grams per day or 0.7 grams per kilogram as an average for three consecutive days (2 to 6 drinks per day depending on body weight).[7] Other analysts have tended to suggest lower limits. Stampfer *et al.* (1993) define moderate drinking to be 1 to 2 drinks per day. Klatsky (1994) defines heavy drinking to be 3 or more drinks per day.

How Good is it?

A second and more difficult consideration for consumers is how much benefit will come from optimal consumption? If risk ratios are not very useful data for most people's decisions—as even medical researchers would probably agree—what information might we use? The following section offers one possibility.

LIFE YEARS SAVED

A perfect assessment of the CHD benefit of MAC would require consideration and comprehension of many dimensions of data. People would need to assess the shifts in the probability distributions of outcomes. Ideally, researchers would report—and everyone would understand—interactions with factors such as gender, smoking, blood pressure, blood lipid profiles, diet, exercise level, and age. Such reports would, however, dramatically overstate the precision of the existing analyses. Interactions with other risk factors have been studied, but the literature is not sufficiently detailed and consistent to develop a model that allows for this level of detail. In most cases, the confidence intervals do not allow us to reject the null hypothesis that there is no interaction effect.

More importantly for present purposes, there is no way people could make sense of so many dimensions of data. A single dimension seems much more promising. Simplification is especially critical since the fundamental effect of MAC—a change in the probability of losing some number of life years—is abstract, intangible, and unfamiliar.

Two dimensions of data provide information that would clearly be lost in a single-dimension analysis, since the effect has such a high variance. (Roughly speaking, it consists of a fairly small probability of saving a large number of life years and a large probability of not saving any.) However, until consumers are used to having a comprehensible scalar on which they can base their decision, it seems overly optimistic to expect them

to be able to deal with two dimensions. While most everyone could rationally assess multiple dimensions for a typical single good, we have to remember that they are trying to make a satisfactory decision for this one of hundreds of consumer decisions they make every day.

Risk ratios are not a useful scalar, since they require an understanding of the underlying base risk, a figure that is not well known and even less well understood. The common alternative in the popular press, 'lives saved' over a given time period, is even less useful, since it requires knowledge and appreciation of several other figures to be intelligible. Furthermore, it totally de-emphasizes the relevant point, what consumption means to the individual. (Indeed, it is also rather ludicrous, since in the long run, only potential life years—not lives—are saved.)

Life years (or days) saved seems to be the obvious measure of a lifesaving intervention. Directed research may determine the optimal way to communicate this type of information, but simply presenting this scalar seems to be the obvious starting point and a clear improvement over reporting just risk ratios.

Choice of Risk Ratio

To calculate average life years saved due to the CHD effect of MAC versus teetotalling, it is necessary to choose a value for the risk ratio.[8] The broadest-based summary figures available are from a study by Maclure (1993), the only complete meta-analysis of the cohort studies available in the literature. He concludes that the dose–response curve is L-shaped. After certain adjustments to the data, using all the studies with useful data, he calculates a risk ratio of 0.76 for drinkers (half a drink per day or greater) as compared to nondrinkers, (that is, a 24% reduction in heart attack risk).

One major concern in the literature is that the population of nondrinkers may be contaminated by unhealthy ex-drinkers, thereby diminishing the health of nondrinkers as compared to moderate drinkers. Ex-drinkers might be less healthy either because they are reformed heavy drinkers who have already suffered acute heart damage from excessive alcohol consumption or because they quit drinking due to health problems. Maclure argues that the effect of this contamination is not significant, but concerns about reverse causation are persistent enough to make them worth considering. Restricting his analysis to studies that separated ex-drinkers from other nondrinkers generated a risk ratio of 0.83, representing a 17% reduction in the risk of CHD. This estimate is toward the conservative end of the range of figures commonly discussed in the literature. To obtain conservative results in this paper (and to keep the emphasis on risk communication rather than dramatic final results), we use this risk ratio.

Another variation on this concern is the persistent suspicion that self-reported alcohol consumption will result in systematic underestimates. Light or moderate drinkers may report that they are nondrinkers or occasional drinkers, due to faulty recall or beliefs about how much they think they should drink. Heavy drinkers may be embarrassed about reporting their actual consumption, and so report moderate drinking or even nondrinking. In contrast to the implicit concern of critics, however, this misreporting (except the last possibility, which may act like the ex-drinker effect) will tend to reduce the measured magnitude of reductions in CHD effects. Any bias would mean the real effect is higher than what is measured and reported. Furthermore, extrapolating the self-reported consumption figures in the literature for Americans shows that it is consistent with total US consumption (Phillips, Zeckhauser, and Shepard, 1993).

Methodology

To convert risk-reduction findings into output measures such as life years saved, we developed a model that simulated survival patterns over individuals' lifetimes. Individuals with optimal and nonoptimal levels of alcohol consumption were run through a hazard process. The model defines age cohort groups, with the transition probabilities from one cohort to the next based on the probability of non-CHD death and the probability of CHD death. Using the adjusted CHD death rates for the effect of MAC, the model generates the probabilistic lifetime benefits of MAC.

Using 18-year mortality data from the Framingham Heart Study (see, for example, Shurtleff, 1974) we calculate the level of risk for individuals who drink the optimal amount compared with those who do not. To calibrate the optimal and nonoptimal CHD rates to the observed mortality

rate, we define anyone drinking 1 to 80 grams of alcohol per day, the range with the lowest risk ratio, to be in the optimal category. Many studies in the literature suggest a narrower range. But a lower bound of 1 gram is convenient, since in most studies any very low positive level of consumption is the first consumption category above teetotalling. This calibration is conservative, since the wide range will tend to dilute the effects of MAC if a narrower range would lead to a greater average CHD reduction. Using tabulations of Framingham data reported by Gordon and Kammel (1983), we determined that 73% of males and 58% of females in the Framingham data were in the 1 to 80 gram range.[9]

With this calibration factor and the risk ratio for optimal drinking, we are able to calculate the absolute CHD risk levels for the optimal and nonoptimal subpopulations that generate the average risk that is observed for this population. The model then calculates the age-specific survival rates and age-specific mortality for the subpopulations. By comparing these results, we determine the expected survivorship gains.[10]

Assumptions and Simplifications

Our use of a nonparameterized binary model is a major, but necessary, simplification. The model does not assume a particular functional form for the relationship between alcohol consumption and CHD risk, but merely relates CHD risk to the binary choice of drinking the 'optimal' amount for reduction of CHD risk or a 'nonoptimal' amount. ('Nonoptimal' refers to either no alcohol or so much that any CHD gains are lost.) Because estimates of the dose–response curve in the medical literature are not precise (and seldom address the relative advantages of different consumption levels within the moderate range), modelling anything finer than a binary choice is not likely to be informative. A nonparameterized life-cycle model is consistent with any of the various proposals for the shape of the dose-response curve, since it can include heavy drinkers in the low-risk group (the right-hand side of the bottom of the 'L') or in the high-risk group (the upward-curving part of the 'U' or reverse-'J').

It is important to reiterate that this binary choice model should not be interpreted to mean that heavy alcohol consumption is beneficial. The 80-gram upper bound may exceed a reasonable definition for moderate consumption due to non-CHD negative effects. While CHD mortality may be low for high levels of drinking, total mortality increases due to the many health hazards of heavy drinking (e.g. Friedman and Kimball, 1986).

The model assumes that MAC's effect is a constant percentage reduction in CHD risk across all age groups. This is consistent with some findings (e.g. Benfante *et al.*, 1992) that the CHD benefit of drinking was the same for those aged 51 to 59 as for those aged 64 to 74. The results of other studies suggest that the risk reduction increases with age (e.g. Klatsky *et al.*, 1990; Colditz *et al.*, 1985), but we find that the data are not sufficiently consistent to warrant varying our calculations based on age-specific risk ratio differentials. Because the age-specific effects of MAC remain an open question especially for very advanced ages, our results for individuals over 80 should be interpreted merely as illustrative extrapolations.

Our discussion of the results implicitly assumes that the benefits of MAC on CHD risk are immediate, rather than accruing due to long-term MAC. The literature tends to assume immediate effects implicitly, omitting the effect of drinking histories (except for separating ex-drinkers). This is not surprising since quantifying current drinking is difficult enough, without also trying to quantify a lifetime of past drinking behavior. If MAC has a cumulative effect rather than immediate efficacy, the effect of starting MAC late in life will be substantially diminished compared to the model's predictions. In this case, the age we report for starting MAC should instead be interpreted as the age at which the effects of ongoing MAC manifest themselves.

Results

Table 1 shows the increase in age-specific life expectancy for those who use MAC starting at age 21, compared to teetotallers. For a 21-year-old, beginning a lifetime regime of MAC will result in an expected gain of 0.75 years for a man or 0.63 years for a woman, for a population average of 0.69 years (252 days) of life compared to nonoptimal drinking. The expected gains at age 0 can be interpreted as the average increase in total life expectancy from birth for someone who will use MAC from age 21. This life expectancy gain is roughly 1% of the American life expectancy. Life

Table 1. Increase in Age-specific Life Expectancy for MAC versus Teetotalling (years)

Age	Male	Female
0	0.72	0.62
21	0.75	0.63
30	0.76	0.63
40	0.76	0.64
50	0.73	0.63
60	0.64	0.61
70	0.52	0.56
80	0.38	0.44
90	0.27	0.31

With MAC starting at age 21 and assuming 0.83 risk ratio for MAC versus teetotalling.

expectancy gain provides the scalar representation of the effect that will allow consumers to make informed decisions.

The gain in expected future life years from MAC stays about the same (even increasing slightly) at age 30, 40, or 50. This is because non-CHD mortality (which takes away potential gains from CHD reduction) is high compared to CHD mortality during the earlier years. This suggests that commencing MAC as late as 50 is roughly as efficacious as starting at 21.[11] Thus, moderate consumption of alcohol is entirely a lifestyle choice rather than a coronary health issue for younger individuals. This has implications for targeting or excluding certain populations for programs that may affect choices about MAC, such as public health campaigns designed to reduce alcohol consumption. Younger people are the most dangerous drinkers for many reasons, while the health benefits are concentrated in older people.

Tables 2 and 3 provide possible ways in which the above-mentioned second dimension of information could be presented. Table 2 shows the result of the CHD effect of MAC on age-specific mortality rates. Table 3 shows the increase in age-specific survivors under MAC. These tables illustrate that, as with any CHD intervention, most of the extra survivors will be older individuals. The effect on total mortality is quite low until age 40 or 50, because CHD deaths are a relatively small portion of total deaths. Older individuals see a dramatic reduction in age-specific mortality due to the significant role of CHD in total mortality.

Naturally, the effects for any given person will differ from the average based on baseline risk of CHD and other interactions. The tables offer a cut at one important interactive factor: sex. The effect for women increases more slowly because CHD does not become a major risk for women until menopause, about 15 to 20 years after it has become a significant risk for men. Men have greater gains in life expectancy from MAC than women, because men have a higher overall CHD rate. For the oldest men and women, the relative impact reverses, with women gaining more. This is because at advanced ages, men's life expectancies are so short relative to women's that they have fewer remaining years of life to be saved.

As would be expected, the model is sensitive to the magnitude of the risk ratio from MAC. Any model of the mortality impact of MAC will be very sensitive to the estimate of the effect of MAC used. Altering the relative risk reduction of 17% by plus or minus 1 percentage point results in a 6.3% change in the life expectancy gains for men and a 6.4% change for women, with a roughly linear change for greater deviations.

Other Factors that Must Be Considered in Quantifying the Benefit

Several issues could affect the magnitude of the CHD benefit of MAC. We explore these in detail elsewhere (Phillips, Zeckhauser, and Shepard, 1993). This section briefly addresses a few of those issues.

Table 2. Reduction in Total Age-specific Mortality Rate from CHD effect of MAC versus Teetotalling (%)

Age	Male	Female
21	0.1	0.8
30	1.0	0.6
40	4.4	1.8
50	6.4	3.1
60	7.1	5.0
70	7.7	7.0
80	7.3	7.8
90	7.3	7.8

With MAC starting at age 21 and assuming a 0.83 risk ratio for MAC versus teetotalling.
Note: The reduction in total death rate is due to the CHD effect of MAC only. No attempt is made to quantify all mortality effects of alcohol consumption.

Table 3. Increase in Number of Age-specific Survivors for MAC versus Teetotalling (per 1000 births)

Age	Male	Female
21	0.0	0.0
30	0.1	0.0
40	0.6	0.2
50	3.4	0.9
60	10.4	3.3
70	20.6	9.8
80	24.3	22.0
90	11.7	21.9
100	1.0	3.6

With MAC starting at age 21 and assuming a 0.83 risk ratio for MAC versus teetotalling.

First, there will likely be a reduction in nonfatal CHD in addition to the mortality reduction, though the total magnitude of this benefit (perhaps measured in quality-adjusted life-days) will be small compared to the mortality gain.[12]

Second, current alcohol consumption may be associated with reduced CHD because it serves as a proxy for a lifetime of moderate alcohol consumption. If so, the independent variable for current drinking in the clinical studies will be imperfectly correlated with long-term alcohol consumption, and studies that are based on current consumption will have an attenuation bias. A lifetime of optimal consumption, therefore, would have a greater impact than those studies (and this model) report. Beginning MAC late in life, however, would have less effect than would be naively predicted. This has important implications for older people who take up MAC to reduce their CHD risk. More data is required to determine whether such an attenuation is present and, if so, its magnitude.

Third, the calculations in this analysis are based on a population whose members exhibit the average risk of CHD and receive average benefit from MAC for their age and gender. But the real-world population is very heterogeneous with regard to risk. Some individuals stand to gain more than the average from MAC.[13] At the same time, the heterogeneity of the population will reduce the total gains that could accrue to the population, since those individuals who have the most to gain from MAC still have relatively higher risk. Saving them from an earlier death results in a population that is less healthy on average than one in which those at higher risk die sooner.[14]

Fourth, there is a potentially large psychological benefit available from communicating the health benefits of commonly consumed goods such as alcohol. Current moderate drinkers will learn that their behavior is potentially beneficial, rather than suffering anxiety about the negative health effects that are generally associated with alcohol. We have labeled this type of benefit the 'assurance effect' (Phillips, Zeckhauser, and Shepard, 1993). The assurance effect is similar (though opposite in sign) to the more familiar anxiety effect, which is widely recognized to diminish utility, and is related to Elster's (1983) 'sour grapes' effect. From an economic point of view, the psychological nature of this benefit does not make it any less real, and it should be included in the list of available benefits.

Fifth, since the life years that are potentially saved come substantially later than the intervention, time preference becomes relevant. Similarly, quality adjustment of life years is important, since the suddenness of a heart attack means that individuals avoiding heart attacks will have more years of old age and hence more years of ill health. However, researchers and analysts need not try to draw conclusions about people's preferences over these factors. As long as it is clear that saved years are mostly late in life (which should be fairly obvious to anyone), individuals can make a rough cut at time and quality adjustment according to their own preferences. Because of the many uncertainties involved, no analyst could do any better than such a rough cut.

TRADEOFFS

The key to any consumer market decision is making optimal tradeoffs. In the case of nonhealth costs and benefits, the consumer already has the necessary data.

Financial and Lifestyle Costs

The dollar cost of MAC is low. Purchasing alcohol in the form of domestic beer or off-brand liquor in a store can cost as little as 50 cents per drink. At the rate of half a drink per day (the lowest cost level of MAC) this is only 25 cents per

day. Even if we double this expenditure and assume a very long intervention period of 50 years (that is, the individual engages in MAC, even for the younger years where it may not have substantial effect), and if the preceding figures about life years saved are correct, MAC's out-of-pocket cost to consumers is only about $25 000 per life year saved.[15] This estimate ignores the reductions in cost resulting from alcohol that an individual may have consumed for its consumptive value, which would reduce the cost of getting to the optimal level for health effects.

The extra effort required to consume the alcohol is small (and often may be zero, given the opportunities to drink at many social occasions). By staying at the minimum optimal level of consumption, people who wish to avoid intoxication could limit themselves to a very small inebriation effect, resulting in virtually no alteration in lifestyle. This compares favorably to interventions such as highly restricted diets or dramatic changes in activity levels that require great effort and possibly unwanted lifestyle changes. For most people, this cost is outweighed by the other benefits of alcohol, as revealed by the portion of the population that consumed alcohol when all the health effects were thought to be negative.

Again, trying to assess these myriad costs and benefits is beyond the abilities of policy makers. Even individual medical practitioners cannot offer much insight beyond helping a patient sort out his or her own feelings.

For some individuals, MAC might have an extremely high cost because they object to the notion of drinking alcohol. Consuming aspirin or a prescription drug to reduce CHD risk is a wholly medical activity to which few would object on religious or quasi-religious grounds. Alcohol, on the other hand, is stigmatized in our society.[16] Furthermore, some religious and social teachings could lead many people to refuse to drink, even if they were convinced of its health benefits. However, as high as these costs might be if everyone were forced to consume alcohol, such costs are easily avoided. Since alcohol is self-administered, those who feel that MAC will have a net negative result for them can simply avoid it and no loss will occur. The aversion some individuals have to alcohol consumption reduces the maximum available social reduction in CHD from MAC, but it does not affect the benefits to those who choose to undertake MAC.

Countervailing Health Effects

As with most health interventions, there are medical conditions that contraindicate MAC. When such contraindications exist, MAC obviously should be avoided. For the population as a whole, moderate alcohol consumption has been linked to diseases whose effects should be netted against CHD gains.[17] There are also suggestions in the literature that moderate alcohol consumption improves cardiovascular health along some dimensions (especially coronary artery disease) but decreases it along others (including cardiomyopathy and arrhythmia). None of the countervailing health effects of moderate consumption appear to be large enough to counteract the CHD effect. It is important that information about the negative health effects of alcohol be communicated in an understandable form and put into perspective. This would be true even absent alcohol's role in CHD, since consumers need to evaluate these costs when consuming alcohol for pleasure.

Many concerns expressed about the health effects of MAC are based on the costs of alcohol abuse, rather than the effects of moderate doses. It has not been established that information that encourages consumption of half a drink per day also encourages heavy or irresponsible drinking, but it is certainly conceivable. It would be irrational for someone to read results about the CHD benefits of MAC, assuming they were accurately communicated, as a reason to engage in heavy drinking. Again, effective communication is critical.[18]

More generally, countervailing health effects of any intervention can introduce a moral question about balancing positive health effects for some individuals versus negative effects for others. An example is the recent decision to put folic acid in flour, which is good for fetuses but potentially bad for the elderly (among others). In the case of MAC, though, the decision to implement is up to the individual. Of course, providing such information might prove detrimental to some individuals who misinterpret the information (accidentally or intentionally). But it seems contrary to basic democratic principles to restrict information of

this nature based on the paternalistic fear that some people will misuse it.

CONCLUSIONS

Tables 2 and 3 illustrate the extra complication that is introduced by adding more than one dimension of information. That information should certainly be available. But even sophisticated readers of this journal probably paused to make sense of all the data. That information cannot be captured in a news soundbite, and is not likely to be included in a newspaper article or popular health publication. This is not to conclude that a single dimension of data is optimal, but it clearly would be an improvement over so little hard data or so much information that consumers can only surmise 'it is good for me'.

There are undoubtedly alternatives to life years saved that would make sense to people. A scalar along the lines of 'percentage *point* reduction in the chance you will have a heart attack in the next X years' could be useful.[19] It would be easier to calculate from the risk ratio and does not require the consumer to have extensive background knowledge. However, the well-known biases in how people deal with small probabilities suggest that such a statistic would be misleading, one way or another.

Further reflection suggests that any choice of the statistic(s) presented will influence decisions. There is no standard for a neutral presentation.[20] But even conceding the inevitability of bias, it still seems impossible to defend presenting information in a form that is not readily understood. When we presented these results at the Harvard School of Public Health one professor objected to communicating results in the form of life years gained. If we reported the cardio-protective effects of moderate exercise in this form, it would also come out to less than a year. This, the professor argued, would be bad because few people would choose to exercise to achieve this modest gain, and we must encourage them to pursue the benefits of exercise. But if few people would make the choice to exercise when they are presented with a statistic they understand, perhaps the assumption that they would be better off exercising is incorrect. That is the advantage of health-affecting decisions that consumers can make as a choice of goods.

Alcohol consumption is heavily influenced by public policy, which is primarily aimed at discouraging excessive and irresponsible drinking. It would be naive and misleading to look at alcohol as just another consumer good, given its stigma, addictiveness, and externalities. But traditional public policy methods such as taxation and restricted availability are necessarily blunt, and inevitably discourage moderate consumption along with excessive consumption. Figuring out how to send just the right message to dampen consumption by the optimal amount is impossible. But since MAC is self-administered and self-financed, as long as sufficient information is available, no central decision maker has to determine the tradeoffs.

Economists generally believe that information will be transmitted through the marketplace. With all the popular press reports about the CHD effect of MAC, it is difficult to find anyone who is not aware of it. With or without an active and well-thought-out policy, information about individual health-affecting behaviors will be disseminated and will have an impact on people's choices. This is disturbing to public health officials and medical practitioners because some individuals misinterpret the information. Indeed, people cannot be expected to critically assess what they hear, recognize flaws in the research, or anticipate revisions of the existing knowledge. However, given that the intervention in question is a consumer good and information wants to be free, health professionals cannot control it, whether or not such control would be beneficial. Given that, it is important to communicate useful information.

The market does not optimally transmit nonmarket information, such as information about the health effects of various activities. There is reason for concern about the way information on alcohol is disseminated. The popular press usually reports the findings of individual technical studies as they become public, with particular emphasis on results that are counterintuitive or counter to the bulk of previous findings. The media does not try to assess the weight of the total evidence, and neither the media nor the researchers try to translate the findings into readily understood figures. Individuals cannot behave at all like the idealized

rational consumers that economists want them to be when they are forced to try to interpret and strike a balance among confusing pieces of information.

It is likely that future analyses will revise what we now know about the CHD effect of alcohol consumption. What will not change is that alcohol is self-administered and that people will respond to the available information only if they understand it. Individuals undertaking moderate alcohol consumption for health purposes do so at their own volition. Presumably they are best able to assess and weigh the benefits and costs. Government and the health professions should provide the citizenry with the most accurate possible information about the benefits and costs of health-related actions. Then people can choose wisely for themselves.

NOTES

1. The detrimental health effects of heavy alcohol consumption include higher risk of liver cirrhosis, pancreatitis, gastritis, trauma, suicide, cardiomyopathy, hypertension, cardiac arrhythmia, hemorrhagic stroke, and degenerative nervous system conditions (Klatsky and Friedman, 1995), as well as certain cancers, particularly breast cancer (Longnecker *et al.*, 1988). For light drinking, the increased risk for these conditions has not been shown to be large, and appears to be substantially outweighed by the reduction in CHD.
2. Many popular reports of the effects of MAC focus on the CHD benefits of red wine consumption, apparently because some of the early discussion focused on the 'French paradox'—the unusually low rate of CHD in France considering the generally unhealthy diet—and tried to explain it in terms of consumption of red wine in particular. Subsequent studies have found the CHD effect to be similar for a given level of alcohol, no matter what the source. For example, Maclure (1993) concludes in his meta-analysis of clinical studies that the source of the alcohol has no effect. Klatsky (1990, 1994) draws a similar conclusion based on his review of the findings and suggests that the effect may be due to collinearity with other lifestyle choices.
3. There are always major externalities that interfere with market provision of health-related goods. The greatest of these is the moral hazard problem resulting from most of the financial costs of health care not being borne by the patient. However, since a huge part of the costs of CHD are nonmonetary, this problem is probably less significant here than it is elsewhere.
4. However, generous portions and increasing alcohol concentration in wine and beer suggests that in practice the alcohol content of what people consider to be one drink may be higher than this (Taylor, 1994).
5. It is most commonly described as U-shaped, indicating that the risk reduction is completely lost at high consumption levels. A reverse-J shape indicates that some of the risk reduction is lost at higher consumption levels. An L-shape describes a high risk for zero consumption with roughly the same reduction in risk for any reasonable positive level of consumption. Many articles suggest a shape but few actually have results for sufficiently narrow ranges of consumption to actually trace out the curve. Instead, they categorize three or four levels of drinking and find that moderate drinking is the one (or is among those) associated with the lowest CHD risk.
6. We do not claim expertise in determining the exact optimal range of consumption for CHD health benefits, much less other considerations. Rather, we conclude from the literature that there is a fairly wide optimal level over which the reduction in CHD risk is nearly constant. (The broad categories of consumption used in most studies make it almost impossible to determine exactly when the beneficial effects reverse.)
7. A limit of 0.8 grams per kilogram comes out to about 3 drinks per day for someone who weighs 120 lb or 4 drinks for someone who weighs 150. The maximum of 80 grams represents just under 7 drinks.
8. The risk ratio, the standard method for reporting the effect of a health intervention on the probability of some event, is likelihood of the event occurring (in this case, dying of a heart attack) following the intervention, divided by the baseline risk (the probability of dying without the intervention).
9. Note that our calculated results are relatively insensitive to variations in the calibration range. Our results focus on changes in CHD risk, while the calibration directly affects only the absolute level of risk.
10. In their study of major factors affecting CHD, Taylor *et al.* (1987) created a logit model of the effects of four key factors (smoking, blood pressure, total cholesterol, and HDL cholesterol) on CHD. We have compared the results of our model to their model and found our results to be consistent with theirs. We modified their model to include the effects of MAC and ran it to confirm the consistency of our results. However, we concluded that our simpler model is more appropriate for our analysis because the functional form in their model created an implicit (nonlinear) interaction effect with the other risk factors in their study. The MAC literature is presented in terms of constant risk reduction and no definitive picture of the interaction of alcohol and other CHD effects has emerged.
11. If the CHD efficacy lags the starting of MAC by a long time, it would be necessary to start MAC earlier than age 50 to achieve the available gains.

Note that the numbers in Table 1 represent the gain from the given age forward for someone employing MAC for their entire adult life. Someone commencing MAC at a later age will have a *slightly* different life expectancy at that age due to population heterogeneity.

12. This could, however, save over $1 billion in health care costs annually (Phillips, Zeckhauser, and Shepard, 1993).
13. There is anecdotal evidence that some physicians are acting on this, recommending MAC selectively to their patients who are at high risk for CHD.
14. An important area for future study would be to reanalyze existing data for the specific effects of heterogeneity, based on interaction effects with characteristics that define different subpopulations. For more details on heterogeneity effects, see Shepard and Zeckhauser (1980, 1982).
15. Putting this in context, Viscusi (1992) cites a variety of other interventions, with social costs per averted fatality (in 1984 dollars) ranging from $100 000 to $72 billion. Some well-known safety interventions that are relative bargains include passive restraints in automobiles ($300 000 per life saved), and airplane floor emergency lighting ($700 000 per life saved). Many regulations relating to toxic or carcinogenic exposures achieve far less life-savings per dollar spent. For example, OSHA asbestos exposure limitations cost $89.3 million per life saved. If as many as 40 life years are saved by an intervention that costs $3 million, somewhere in the middle of this range and in the range of typical estimates for the 'value' of a life, the cost is $75 000 per life.

    Of course the cost of MAC is completely borne by the individual, while those of other interventions are largely externalized. But if the available figures are even roughly correct, it is still a relative bargain.
16. It is easy to find anecdotal evidence that some people react to the findings about the CHD advantages of alcohol by saying, 'The studies be damned; alcohol cannot be good for you'.
17. See note 1.
18. It has been argued that there is no known way to identify an individual's propensity for alcoholism (e.g. Blackburn *et al.*, 1991), which might suggest that anything that encouraged MAC would doom susceptible individuals to alcoholism. But, while there may be no good physiological test for alcoholism, this argument does not seem compelling. The individuals we need to worry about must have all four of the following characteristics: (a) a propensity for alcohol abuse that will be triggered by consumption at the level appropriate to reduce CHD risk; (b) current drinking that is low enough to avoid triggering that propensity; (c) a failure to suspect that they have this propensity; and (d) the inability, upon beginning drinking, to recognize this propensity and stop drinking. The existence of condition (a) appears to be the implicit cause of concern about MAC, but it is problematic only for individuals for whom (b), (c), and (d) all apply.
19. Someone with a baseline CHD risk of 4% would probably not be able to assess the value of a 25% risk reduction (since he probably would not know the baseline). A reduction in the chance of dying (due to the CHD benefit) of one percentage point is much more meaningful.
20. As Amartya Sen has put it, 'truth is insufficient and whole truth is impossible' (Personal communication, 1992). That is, any presentation that does not include all relevant information will include some bias, and we cannot present all relevant information.

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