Dietary Fat and Risk of Breast Cancer

To the Editor: Dr Holmes and colleagues1 reported that analysis of data from the Nurses’ Health Study found no evidence that a lower intake of dietary fat was associated with a decreased risk of breast cancer. These results, in a well-defined, prospective cohort study, differ from those of retrospective studies, international comparisons, meta-analyses of case-control studies, and laboratory animal studies.

Martinez et al,2 in a study of the relation of dietary calcium and vitamin D to the risk of colorectal cancer in the same group of women, found that vitamin D intake in these women was considerably higher than the US average, primarily due to their regular intake of multivitamin supplements. The average US woman consumed about 60 IU (1.5 µg) of vitamin D in 1987,3 while the women in the Nurses’ Health Study had a median daily intake of more than 250 IU (6.25 µg), about 4-fold higher.

Garland et al4 found a strong inverse correlation between breast cancer and the availability of effective solar radiation for in vivo skin production of vitamin D. Coupled with the low average dietary intake of vitamin D, they suggested that inadequate vitamin D in the United States may be a significant risk factor for breast cancer. In animal studies5 variation of dietary calcium and vitamin D had little or no effect on carcinogen-initiated breast cancer in low-fat diets. However, increases in dietary calcium and vitamin D reduced tumorigenesis several-fold in diets with high fat (20% by weight, equivalent to 40% of energy intake).6 Taken together, animal and epidemiological studies suggest that increased vitamin D intake should result in a decreased breast cancer risk. Further, the animal data suggest that adequate increased vitamin D intake sharply reduces the effect of high dietary fat as a promoter of breast cancer.5,6

Therefore, it appears that the article of Holmes et al,1 coupled with that of Martinez et al,2 offers an explanation for the lack of effect of variation of dietary fat intake in breast cancer risk in the Nurses’ Health Study, due to the higher vitamin D intake reducing the effect of fat on breast cancer risk. This cohort apparently is an educated, health-conscious population, as evidenced by their higher levels of multivitamin use, and resultant vitamin D intakes.

In at least this aspect of daily habits this particular study population, while large for a study cohort, is certainly not representative of the US female population as a whole. Careful consideration of differences between this study group and the average US female population must be made before generalized recommendations of public health policy can be formulated.

Harold Newmark, DSc
Rutgers, The State University of New Jersey
Piscataway


To the Editor: Although Dr Holmes and colleagues1 imply that a high-fat diet does not increase the risk of developing breast cancer,2 this interpretation may be misleading. After careful examination of both the summary data and model assumptions, we argue that their analysis suffers from multicollinearity, an artifact that may easily have nullified an effect of a high-fat diet on breast cancer.

The effect of multicollinearity can be easily seen in their tables.3 Table 3 shows significant protective effects at the 5% level for vegetable fat, polyunsaturated fat, monounsaturated fat, and trans-unsaturated fat. Such findings would be consistent with previous studies demonstrating the beneficial effects of these types of fats in other diseases. After mutually adjusting for other fats (Table 4), none of the above factors retains significance, because the different fats are not consumed independently. The same artifact is likely to have obscured their main analysis.

In their main analysis, the authors adjusted for energy intake, body mass index (BMI) at age 18 years, and weight change after age 18 years. While adjustment for confounding factors (age, age at menarche, parity, age at menopause, family history) is necessary, it is crucial not to adjust for mediating factors, which represent possible pathways by which diet may reduce cancer incidence. For instance, persons who consume a high-fat diet may be more likely to be overweight than persons who satisfy their energy needs primarily with carbohydrates or proteins. Eliminating all such factors systematically underestimates the overall effect of a high-fat diet, as did mutually eliminating the effect of different types of fat from Tables 3 and 4. Multicollinearity may nullify even strong associations.

GUIDELINES FOR LETTERS. Letters discussing a recent JAMA article should be received within 4 weeks of the article’s publication and should not exceed 400 words of text and 5 references. Letters reporting original research should not exceed 500 words and 6 references. All letters should include a word count. Letters must not duplicate other material published or submitted for publication. Letters will be published at the discretion of the editors as space permits and are subject to editing and abridgment. A signed statement for authorship criteria and responsibility, financial disclosure, copyright transfer, and acknowledgment is required for publication. Letters not meeting these specifications are generally not considered. Letters will not be returned unless specifically requested. Also see Instructions for Authors (July 7, 1999). Letters may be submitted by surface mail: Letters Editor, JAMA, 515 N State St, Chicago, IL 60610; e-mail: JAMA-letters@ama-assn.org; or fax (please also send a hard copy via surface mail): (312) 464-5824.

Edited by Margaret A. Winker, MD, Deputy Editor, and Phil B. Fontanarosa, MD, Interim Coeditor.

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The authors have provided interesting data that should be studied further. However, by adjusting uniformly for different types of variables, they essentially shifted the focus onto the direct effect of fat and away from the effect of dietary patterns on cancer incidence. Similar considerations apply to the reported lack of association between fiber intake and colorectal cancer in the same cohort. Findings that individual dietary ingredients do not directly affect carcinogenesis cannot invalidate the important public health message that dietary behaviors may prevent cancer.

Knut M. Wittkowski, PhD, DSc
Steven J. Shiff, MD
Jules Hirsch, MD
Rockefeller University
New York, NY


In Reply: Dr Newmark suggests that we did not find an association between fat intake and breast cancer risk because vitamin D may protect against a high-fat diet, and average vitamin D intake in our population is higher than the US average of 60 IU/d.

We performed separate analyses of fat intake and breast cancer risk among women with low (≤60 IU/d) and high (>60 IU/d) intake of vitamin D. For a 5% energy increase in fat intake among low consumers of vitamin D, the relative risk (RR) of breast cancer was 0.95 (95% confidence interval [CI], 0.86-1.04) vs 0.97 (95% CI, 0.94-1.01) among high consumers. These findings do not support Newmark’s hypothesis.

Newmark notes that results of prospective studies are inconsistent with other evidence on diet and breast cancer. However, the likelihood of bias in case-control studies is great. Also, international comparisons are severely confounded by reproductive history, physical activity, and food availability. Although animal studies consistently show effects of total energy restriction, the effects of dietary fat are inconsistent. This is why large, long-term, prospective studies are needed.

Dr Wittkowski and colleagues suggest that collinearity could explain our results. Collinearity could explain the observation that the significant protective effects seen for some types of fats in Table 3 become nonsignificant when mutually adjusted in Table 4. Analyses of types of fat do need to control for each other because of intercorrelations. However, these correlations are not so high as to obscure important biological relationships. In a similar analysis with fewer cases, specific types of fat strongly predicted risk of coronary heart disease as expected by metabolic studies. Also, collinearity cannot explain our major finding, that total fat intake was not associated with breast cancer risk, as total fat was not modeled with other fat types. If the results for total fat were caused by collinearity, then fat intake would have to be strongly associated with other covariates. Yet, the RR for a 5% energy increase in fat intake adjusted only for age was 0.96 (95% CI, 0.93-0.98), virtually identical to the multivariate result given in Table 2 (0.97 [95% CI, 0.94-1.00]).

Wittkowski and colleagues also suggest that persons consuming a high-fat diet may be more overweight, thus adjusting for obesity would be inappropriate as it would be an intermediary between a high-fat diet and breast cancer. However, we found that BMI, calculated as weight in kilograms divided by the square of height in meters, did not vary much across fat intake:

<table>
<thead>
<tr>
<th>% of Energy From Fat</th>
<th>Median BMI, kg/m²</th>
</tr>
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<tbody>
<tr>
<td>≤20</td>
<td>23.0</td>
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<tr>
<td>20.1-25</td>
<td>23.6</td>
</tr>
<tr>
<td>25.1-30</td>
<td>24.1</td>
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<tr>
<td>30.1-35</td>
<td>24.3</td>
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<tr>
<td>35.1-40</td>
<td>24.3</td>
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<tr>
<td>40.1-45</td>
<td>24.2</td>
</tr>
<tr>
<td>45.1-50</td>
<td>24.1</td>
</tr>
<tr>
<td>&gt;50</td>
<td>23.9</td>
</tr>
</tbody>
</table>

This is consistent with the finding that dietary fat is not an important determinant of body weight. Anticipating this criticism, we performed an analysis without adjusting for obesity, shown in Table 5, Model H, of our article. The results were exactly the same as those shown in Table 2, which did adjust for obesity: for a 5% of energy increase in fat intake, the RR was 0.97 (95% CI, 0.94-1.00).

Michelle D. Holmes, MD, DrPH
Bernard Rosner, PhD
Walter C. Willett, MD, DrPH
Harvard Medical School
Boston, Mass


Left Bundle-Branch Block and the ECG in Diagnosis of Acute Myocardial Infarction

To the Editor: Dr Shlipak and colleagues assessed the value of our electrocardiographic (ECG) criteria to diagnose acute myocardial infarction (AMI) in patients with left bundle-branch block (BBB). The conclusion, that our algorithm is worse than a “thrombolysis to all” approach, seems unsubstantiated. Idiosyncratic characteristics of the studied population may explain the low sensitivity of our ECG criteria, whereas assumptions in the decision analysis may have amplified the benefits of thrombolysis. The criterion standard selected for a study like this is critical. Elevations in creatine kinase-MB isoenzyme and troponin I (sensitive and specific markers of myocardial damage) cannot be equated to benefit from thrombolysis. The unusually high prevalence of AMI (56%) among patients with cardiac arrest in this series is unlikely to reflect acute coronary thrombosis. Unfortunately, the distribution of clinical presentations was not reported. Although the sensitivity remained low when only patients with chest pain were considered, the smaller sample size may have compromised the precision of the estimate.

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The extrapolation of benefit and risk estimates from the Fibrinolytic Therapy Trialists (FTT) analysis seems biased. In FTT, patients with right BBB and left BBB were pooled. Patients with right BBB are more likely to be included in thrombolysis trials, probably because of the straightforward ECG diagnosis. Their higher baseline mortality may explain the marked benefit of thrombolysis found in the FTT subanalysis. Furthermore, patients with left BBB enrolled in thrombolysis trials may have been specially selected. The pathogenesis of our ECG signs (transmural myocardial injury due to acute occlusion of an epicardial coronary artery) should not be overlooked. Thrombolysis does not benefit patients without ST segment elevation, and it cannot be safely assumed that it does benefit patients with left BBB without our proposed signs. Patients with AMI and left BBB are elderly (mean age of 76 years in a national registry), and thus their risk of thrombolysis-induced stroke was underestimated in the model. The incidence of stroke in thrombolysis-treated patients with BBB in FTT was 2.1% (above the range tested in the sensitivity analysis).

As indicated elsewhere by the authors, only highly specific diagnostic tests are clinically useful in low-prevalence conditions. Thus, a prospective validation of our ECG algorithm would be valuable. In the meantime, the high predictive value of the proposed signs should trigger thrombolysis. In their absence, patients should undergo further testing (serum markers, perfusion imaging, coronary angiography) to identify the minority in need of reperfusion.

Elena B. Sgarbossa, MD
Sergio L. Pinski, MD
Galen S. Wagner, MD
Rush-Presbyterian-St Luke’s Medical Center
Chicago, Ill

In Reply: The 103 patients in our study included 65 with chest pain, 27 with acute pulmonary edema, and 11 with cardiac arrest. The sensitivity of the ECG algorithm was 6% in patients with chest pain, with a 95% confidence interval (CI) of 1% to 27%. The accuracy of this estimate is supported by the work of Kontos et al who found a similar estimate for the low sensitivity of the algorithm of Dr Sgarbossa and colleagues.

The poor prognosis for patients with right BBB who experience myocardial infarction has been demonstrated by many prior investigators, and was confirmed by recent work in which the in-hospital mortality for these patients was 22.6%, nearly identical to that in patients with left BBB (23%). In designing our decision analysis, we were aware that the FTT analysis included some patients with right BBB within the designation of “bundle-branch block.” That this category has a predominance of patients with right BBB is speculation on the part of Sgarbossa and colleagues. We chose the estimate of thrombolytic-related stroke from the entire sample of the trials because the relatively small sample of the BBB subgroup made that stroke estimate imprecise. However, we did perform our analysis using the stroke value mentioned by Sgarbossa and colleagues (2.1%). Under these conditions, the threshold for the likelihood of AMI such that “thrombolysis for all” is superior to “apply ECG algorithm” increased to 16%, but was still less than the rate of AMI we actually found of 28% (95% CI, 17%-40%).

Finally, we are surprised that Sgarbossa et al appear to believe that 28% — the prevalence of MI in patients with left BBB and ischemic symptoms — represents “low prevalence.” Because myocardial infarction is, in fact, rather common in these patients, we reaffirm our support of the American College of Cardiology/American Heart Association guidelines, which recommend acute reperfusion therapy for patients with left BBB and clinical presentations indicative of AMI.

Michael G. Shlipak, MD, MPH
Alan S. Go, MD
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Screening Mammography for Women Younger Than 50 Years

To the Editor: Drs Antman and Shea add to the confusion concerning screening women aged 40 to 49 years for breast cancer. The Canadian trial the authors cite did not have sufficient statistical power to evaluate women in this age group in

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the early years of follow-up. Furthermore, the 19 Canadian women cited in the article already had advanced breast cancer at the time they were randomized to have mammography, while only 5 with breast cancer were assigned to the control group.

The authors fail to point out that the screening trials were never intended to permit the analysis of women between 40 and 49 years as a separate subgroup and lacked statistical power to do so. The cutpoint between 49 and 50 years is a contrivance that resulted from data grouping. Many of the parameters of screening (recall rates and recommendations for biopsy rates) are the same, regardless of age, while others (positive predictive value and cancer detection rates) change steadily with increasing age along with the prior probability of breast cancer.6 None of the parameters of screening changed abruptly at age 50.

The assertion that “the number screened to save 1 life is substantially lower for women 50 years and older” may also apply to women aged 60 years or older compared with those younger than 60 years, and for women aged 70 years or older compared with those younger than 70 years.

The statement that “only 2% of 40- to 49-year-old US women develop breast cancer . . .” is misleading and trivializes the risk by not providing any context. In fact, “only” 3% of women aged 50 to 59 years develop breast cancer during that decade of life, and “only” 4% develop breast cancer during their 60s.

Besides the Gothenburg trial, the Malmo study7 also demonstrated a significant benefit of screening (33% reduction in breast cancer deaths).

Antman and Shea have used outdated information in their Table 1. The data that were provided to the consensus development conference in 1997 revealed that 5 (not 4) Swedish trials show a 29% (not 24%) statistically significant mortality reduction for women aged 40 to 49 years.5 The analysis that includes all of the trials, including the Canadian one, shows that the mortality reduction of 16% is statistically significant, not “nonsignificant” as Antman and Shea state.

In its January 23, 1997, statement, the panel never mentioned the new data that showed statistically significant mortality reduction from screening women in their 40s.6

Recommendations for screening should be evidence based. There are no data supporting the authors’ suggestions that screening should start at age 45 years, or that only women at high risk should be encouraged to be screened.

Daniel B. Kopans, MD
Massachusetts General Hospital
Boston

In Reply: Most of the points made in Dr Kopans’ letter were also made in our article. We pointed out that the imbalance of the 19 women in the mammography group vs 5 in the control group suggested a problem with randomization.

We pointed out that the initial trials were never intended to permit subset analysis of women aged 40 to 49 years, and thus the 12 policy groups appropriately initially recommended screening in the age groups covered by the clinical trials.

We specifically pointed out that age and breast cancer risk are continuums and that the cut points of 40 and 50 years are arbitrary. We deliberately used quotation marks in the summation of other authors’ opinions “that ‘only’ 2% of 40- to 49-year-old US women develop breast cancer” to specifically indicate our discomfort with minimizing the 2% risk (which represents thousands of women), and pointed out that despite this 2% risk, breast cancer is the major cause of mortality in this age group.

It is quite clear from our table that the Malmo trial found a 49% reduced breast cancer mortality in the 40- to 49-year-old group with an observed risk of 0.51 compared with the expected value. Although more recent data may have been available to the consensus development conference, we chose to cite the most recently published data to enable readers to refer to these studies.

We chose to reference a different meta-analysis. But most importantly we did not recommend that screening should start at age 45 years. Based on the continuum of risk and age (as also pointed out by Kopans), we recommended initiating screening based on risk.

We pointed out that despite the lack of study data, the convention was to screen women younger than 40 years old who have strong family histories or known genetic risk.

Karen Antman, MD
Steven Shea, MD
Columbia University College of Physicians and Surgeons
New York, NY

Direct-to-Consumer Advertising: Education or Anathema?

To the Editor: Mr Holmer1 makes a case in support of direct-to-consumer (DTC) advertising that puts critics on the defensive. After all, who can be against free speech and educating and empowering patients?

Holmer plays down the inflationary effect of DTC advertising, with which the costliest drugs are pitched with all the skill that the advertising budgets of pharmaceutical companies can buy. There is no corresponding lobby for less expensive drugs with lower profit potential. Instead, spin doctors take over from

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medical doctors and profitability supersedes the presentation of balanced information.

Holmer neglects the role of managed care. Because benefit managers are able to negotiate for lower drug prices, pharmaceutical manufacturers seek to offset managed care “losses.” What could be simpler than doing an end run around the cost-containment efforts of health managers and physicians by going directly to patients to create demand that will be difficult to refuse?

Nor does Holmer discuss the effect on physicians who become involuntary appendages of manufacturers’ public relations departments as they field questions inspired by print and television drug ads. He argues that physicians are not unduly influenced by the DTC ad campaigns because only 51% of patients who inquire actually get a prescription (according to a unpublished manufacturer’s survey Holmer cites), but this statistic may also be interpreted as a costly waste of physician time in the other half of the cases.

This kind of cost-shifting is eroding the close and collaborative relationship that physicians once had with the pharmaceutical industry. Our relationship is becoming competitive and, at times, hostile. Thus far, industry may be considered the winner. Some health insurers, including Medicaid, are now spending more on pharmaceuticals than on physicians’ fees.1,2

Holmer hints that physicians should not complain because they share the wealth created by DTC advertising. But such collusion would ultimately erode the barriers to cost-effective prescribing. This is hardly preferable, even if the only other choice is for physicians to be ill-treated.

Higher costs, friction between professional ethics and business, and the inevitable abuses of unbridled competition may, in the long run, cause the pharmaceutical industry to look back to the gentler pre-DTC times with nostalgia.

Philip R. Alper, MD
University of California at San Francisco

In Reply: Dr Alper implies that DTC advertising has changed the traditional patient-physician relationship. I believe that DTC advertising is a product of the same force that is changing the patient-physician relationship, the information revolution. As Lloyd M. Krieger wrote in a recent op-ed piece in the New York Times, “Health care is undergoing an information revolution. Not only are hospitals and doctors learning to track and compare the cost and effectiveness of treatments, but patients are also savvier about their ailments and their doctors. Patients who see me are quoting from medical articles they found on the Internet as often as they are citing commercials for a wonder drug they saw during Frasier.”

I did not hint that physicians “share in the wealth created by DTC advertising” except in the sense of gaining satisfaction from enhanced communication with patients and better health outcomes. We in the pharmaceutical industry do not feel we are competing with physicians. While it is true that the share of the health care dollar allocated to outpatient pharmaceuticals grew from 6% in 1996 to 7.2% in 1997,1 this should be attributed not to DTC advertising but to the fact that health care professionals are turning increasingly to cost-effective drugs to improve outcomes while controlling costs.

To the Editor: As members of the Committee on Bioethical Issues of the Medical Society of the State of New York, we agree with Dr Hollon1 that DTC advertising creates consumer demand, but disagree with Mr Holmer’s2 claim that such advertising builds bridges between patients and physicians.

Direct drug advertising provides no real benefit to patients, is potentially harmful, and is costly. We therefore urge the US Food and Drug Administration (FDA) to review and strengthen its policies concerning this practice. We believe that direct promotion to consumers should be limited to over-the-counter drugs. We urge the FDA to request and designate additional resources for the policing of its regulations by preclearing submissions from advertisers, monitoring advertisements, and investigating complaints from consumers and health care professionals. False, misleading, or deceptive drug promotions and advertisements that cannot be supported by clinically valid and statistically reliable data or that contain confusing or misleading words and phrases or give an overall inaccurate impression3 should be forcefully dealt with and, if necessary, penalties imposed and those responsible prosecuted.

We urge the FDA to establish a commission to oversee the marketing activities of the pharmaceutical industry and to monitor its adherence to the FDA rules and regulations. Such a commission in Sweden functions as a “watchdog” in monitoring written information such as advertisements.4 The FDA should empower such a commission to monitor drug promotions on radio, television, and the World Wide Web. We believe that Congress should strengthen the FDA by providing adequate funds to permit the FDA to fulfill its oversight responsibilities of prescription drug advertising and to establish an oversight commission.

Fred Rosner, MD
Pieter Kark, MD
Samuel Packer, MD
Allen Bennett, MD
Jeffrey Berger, MD
Medical Society of the State of New York
Lake Success

Physicians will always—and should always—wield the prescribing pen. For this, and many other reasons, the pharmaceutical industry will work to minimize any friction caused by DTC advertising.

Dr Rosner and colleagues fail to acknowledge the value of DTC advertising in addressing the problem of underdiagnosis and undertreatment of disease, which I documented in my article. For example, only 1 depressed person in 10 receives adequate medical treatment, and one third of people with major depression do not seek treatment. Nor do Rosner et al document the alleged potential harm of DTC advertising. Instead, they call for a ban on promoting prescription medicines directly to consumers and additional “policing” by the FDA, which already regulates DTC advertising and does not need additional authority.

Alan F. Holmer, JD
Pharmaceutical Research and Manufacturers of America
Washington, DC


These letters were shown to Dr Hollon, who declined to reply.—Ed.

Tobacco and Alcohol Use in Children’s Animated Films: Pecos Bill Kicks the Habit

To the Editor: As a lover of children’s animated films, I was intrigued by the results of Dr Goldstein and colleagues’ study, of the use of tobacco and alcohol in children’s animated films. Following the use of the Joe Camel character in advertising by the RJ Reynolds Tobacco Company, DiFranza et al showed that sales of Camel cigarettes to the underage market dramatically increased. In that study, children recognized the cartoon character more readily than did adults, associated it more easily with Camel cigarettes, and were more likely to think that Joe Camel was “cool.” In another study children as young as 3 years old were not only able to recognize the character but to associate it with cigarettes.

Hollywood and parents need to be aware that children may interpret the use of alcohol and tobacco in animated films as a nod of approval. After reading the study by Goldstein et al, I asked my 5-year-old nephew if he recognized the characters in Disney’s 101 Dalmatians (a film he has seen approximately 6 times) smoked. He immediately responded that Roger smoked a pipe. I cannot think that the addition of a pipe greatly enhanced this character’s persona, yet, in my limited experience, children do remember such seemingly trivial features.

The November 1998 tobacco settlement has now banned the use of cartoon characters such as Joe Camel from tobacco company advertising. One can only hope that the film industry will not wait for legislation banning the portrayal of alcohol and tobacco use in children’s animated films to make these changes on its own.

Lauri M. Kusseim, MD
University of Pennsylvania Medical Center
Philadelphia

To the Editor: Dr Goldstein and colleagues clearly show the prevalence of tobacco and alcohol use in G-rated children’s animated films. Of interest is the recent Disney release of Melody Time, a 1948 animated production advertised as “fully restored.” However, it is actually an abridged version, as the final cartoon segment “Pecos Bill” has been altered to remove all tobacco references. In the original version, the title character chain-smokes cigarettes, sings about sucking tobacco, and even lights a cigarette butt with a lightning bolt. All of these scenes have been removed from the current release. Although this should be viewed as a positive action by physicians, there is an underlying concern among cineastes that older films will continue to be digitally sanitized to make them more palatable for children.

Richard A. Drachman, MD
University of Medicine and Dentistry of New Jersey
New Brunswick

country. Her call for awareness to the film industry is also right on target, positing that the portrayal of alcohol and tobacco use and abuse in children’s animated films may affect the moral development of our children.

Dr Drachtman raises an important question not directly addressed by our research: is it acceptable to alter earlier children’s films to make them “more palatable for children”? As researchers, we do not advocate changing older films to remove pictures of characters using tobacco or alcohol. Yet, as parents (as well as cineastes), we do not object if film producers who rerelease films make changes out of their own concern for our children’s health. By rereleasing an old film, they are in fact marketing the product to new generations of children. Thus, when Hollywood producers decide that a depiction of unhealthy products is more damaging than any potential financial loss, we wholeheartedly support them.

In the original 1948 Melody Time, Pecos Bill was a “rootin’ tootin’ cowboy,” who “becomes so tough that he can roll a cigarette while riding atop a cyclone.” 1 By showing this former chain-smoking cowboy, Disney clearly demonstrates that Hollywood can create children’s characters that are tough but tobacco-free. As we all now pay more attention to the portrayals of tobacco and alcohol use in children’s films, we applaud the efforts by animated film producers to eliminate such portrayals.

Adam O. Goldstein, MD
Rachel A. Bearman
Glen R. Newman, PT
University of North Carolina at Chapel Hill


Sexual Dysfunction in the United States

To the Editor: Dr Laumann and colleagues1 indicate that sexual dysfunction, in both men and women, is a major health problem. In men they reported a 21% prevalence of premature ejaculation, whereas others2 have noted that the incidence may be as high as 36%.

A small study3 found premature ejaculation to be associated with hypogonadotropic hypogonadism. Extrapolating the finding of a 21% prevalence of premature ejaculation with the occurrence of hypogonadotropic hypogonadism leads to the possibility that a large number of men may have chronic hormonal inadequacy. More study is needed to determine the magnitude and the nature of the risks involved. Prompt confirmation of these possible relationships is necessary as hypogonadism may be common in the US male population.

Paul G. Cohen, MD
Atlanta, Ga

In Reply: Premature ejaculation is a common complaint among men of all ages. Despite the prevalence of the problem, few studies have investigated etiological factors associated with the condition. Small-scale studies4 have suggested a neurophysiological basis in a subgroup of men with lifelong or primary premature ejaculation. Others have argued that endocrine4 or psychological factors5 may be important. In our analysis of data from the National Health and Social Life Survey,6 a history of urinary tract symptoms, general health problems, and the presence of emotional stress were all positively associated with self-report of premature ejaculation. Of note, this was the only sexual dysfunction in men that was not associated with significant quality-of-life effects.

Our study did not address the role of diminished testosterone levels, since endocrine measures were not included. However, our findings are consistent with other literature suggesting a heterogeneity of causal factors. Although we question whether hypogonadism, either primary or secondary, is the major etiological factor, we concur with the need for additional studies in this area. Serotonergic agents are used increasingly in the treatment of premature ejaculation, and we could not find published reports of hormonal therapy for this condition.

Raymond C. Rosen, PhD
Edward O. Laumann, PhD
Anthony Paik, MA
University of Medicine & Dentistry of New Jersey
Piscataway

Dr Rosen has received research and consulting support from Pfizer, Inc, and several other pharmaceutical companies, and Dr Laumann has served as an advisor to Pfizer, Inc.


CORRECTION

Incorrect Wording: In the Brief Report entitled “Andrew Jackson’s Exposure to Mercury and Lead,” published in the August 11, 1999, issue of The JOURNAL (1999;282:569-571), there was an incorrect number in the Table. The value given for the 1839 sample for lead, 68 ppm, should have been 18 ppm. This incorrect value does not affect the published conclusions.

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