VU Research Portal

Preventing low back pain in industry - Reply

van Poppel, M.N.M.; Koes, B.W.; Bouter, L.M.

published in

JAMA

1998

DOI (link to publisher)

[10.1001/jama.280.23.1993](https://doi.org/10.1001/jama.280.23.1993)

document version

Publisher's PDF, also known as Version of record

[Link to publication in VU Research Portal](#)

citation for published version (APA)

van Poppel, M. N. M., Koes, B. W., & Bouter, L. M. (1998). Preventing low back pain in industry - Reply. *JAMA*, 280(23), 1993-1994. <https://doi.org/10.1001/jama.280.23.1993>

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal ?

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

E-mail address:

vuresearchportal.ub@vu.nl

Ethnic and Socioeconomic Factors as Determinants of Health Status

To the Editor.—To address the racial disparity in cardiovascular disease (CVD), Dr Winkleby and colleagues¹ recommend the need for targeted intervention programs, changes in health policy reforms, and changes in the health care industry. These are indeed important remedies to reduce the growing health status gaps among women and men of color. It is equally important to better understand the role of different life experience as a potential pathway contributing to CVD risk factors among racial and ethnic populations. Winkleby et al report that a higher proportion of African American and Mexican American women lived below the poverty line compared with white women. They also report that African American women (with similar years of age as white women) were more likely to be unmarried and that Mexican American and African American women were also more likely to live in urban areas. Indeed, a higher proportion of Hispanic and African American women do in fact reside in crowded, urbanized settings; a higher proportion of single mothers are also African American.² Economic, time, and residential constraints may therefore partially explain the higher constellation of interrelated primary risk factors observed among women of color in the study by Winkleby et al.

Research shows higher socioeconomic status (SES) provides economic protection and reduces risk factor prevalence.³ However, Winkleby et al show that African American and Mexican American women with the highest measure of SES (>12 years of education) had greater levels of risk factors compared with white women who have similar years of education. The rate of African American and Mexican American women with more than 12 years of education who were living in poverty was also double the rate among white women (18.9% and 18.6% vs 7.1%). Such information suggests that higher socioeconomic position may not provide equal protection among persons of color.

Emerging evidence is beginning to establish a link between overt and subtle exposures to racial discrimination and higher levels of blood pressure among African Americans.⁴ Racial discrimination as well as constraints on other aspects of the life experience may compete with positive lifestyle modification associated with the sequelae of less leisure-time exercise, overweight, hypertension, and diabetes among African American and Mexican American women. The president recently announced the Racial and Ethnic Health Disparities Initiative with the policy objective of eliminating health status disparities among racial and ethnic minorities by the year 2010.⁵ The seminal work by Winkleby and colleagues highlights the need for investigators to design studies that not only assess genetic and behavioral determinants but also assess a priori hypotheses that quantify the relative role different life constraints contribute as a pathway to CVD risk factors among men and women of color. The subsequent challenge is for policymakers to develop tools and instruments that will effectively intervene at both individual and societal levels.

Sharon K. Davis, MPA, PhD
Harvard School of Public Health
Boston, Mass

1. Winkleby MA, Kramer HC, Ahn DK, Varady AN. Ethnic and socioeconomic differences in cardiovascular disease risk factors. *JAMA*. 1998;280:356-362.
2. US Department of Health and Human Services. *Women of Color Health Data*

Book. Bethesda, Md: Office of Research on Women's Health; 1997. NIH publication 98-4247.

3. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88:1973-1998.

4. Krieger N, Sidney S, Coakley E. Racial discrimination and blood pressure: the CARDIA study of young black and white adults. *Am J Public Health*. 1998;86:1370-1378.

5. Georges AC. President Clinton's new Racial and Ethnic Health Disparities Initiative. *Ethnicity Dis*. 1998;8:257-258.

In Reply.—Dr Davis encourages investigators to take epidemiologic findings a step further to elucidate how different life experiences may contribute to CVD risk factor differences among women of color and of low SES. Her comments are especially germane to the current dialogue that challenges the field of epidemiology to make the transition from a science that concentrates on individual risk factors for disease to one that addresses the dynamic systems that generate patterns of health and disease in populations.^{1,2} This challenge is highly relevant given the increasing diversity of Americans and the continuing disparities in health status experienced by ethnic minority and low-SES groups.³

Davis proposes that different life experiences as well as economic, time, and residential constraints may compete with heart-healthy behaviors to increase risk of disease. Our findings, based on a national sample of 1762 black, 1481 Mexican American, and 2023 white women, support Davis' ideas. We found, for example, that black and Mexican American women had greater increases in blood pressure than white women across age groups, resulting in significantly larger ethnic differences among older than younger women (eg, the black-white difference of 4 mm Hg at ages 25-34 years increased to 11 mm Hg at ages 55-64 years). This finding is consistent with the weathering hypothesis proposed by Geronimus,⁴ who suggests that "the health of African-American women may begin to deteriorate in early adulthood as a physical consequence of cumulative socioeconomic disadvantage." We also found that black women's rates of smoking remained high across all age groups, whereas white women's rates, which initially were high, decreased in older age groups. This finding suggests that the tobacco industry's aggressive marketing campaign may have "succeeded" in targeting black women with advertising campaigns and marketing and promotion initiatives.⁵

As health professionals explore causal pathways that may explain the higher rates of CVD risk factors among ethnic minority and low-income groups, we can be guided by the wisdom of the late Jonathan Mann, MD, who, when writing about the core of public health, emphasized that a blend of individual and societal factors are involved in determining

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. Please provide 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 essential for publication. Letters will not be returned unless specifically requested. Also see Instructions for Authors (July 1, 1998). 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, and Phil B. Fontanarosa, MD, Senior Editors.

health and associated behaviors and that for many, if not most people, the societal context weighs heavily, if not overwhelmingly, as a determinant of health status.⁶

Marilyn A. Winkleby, PhD
Stanford University
Palo Alto, Calif

1. Koopman JS. Emerging objectives and methods in epidemiology. *Am J Public Health*. 1996;86:630-632.
2. Pearce N. Traditional epidemiology, modern epidemiology, and public health. *Am J Public Health*. 1996;86:678-683.
3. Winkleby MA. Accelerating cardiovascular risk factor change in ethnic minority and low socioeconomic groups. *Ann Epidemiol*. 1997;7(suppl):S96-S103.
4. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethnicity Dis*. 1992;2:207-221.
5. Ernste V. How tobacco companies target women. *World Smoking Health*. 1991;16:8-11.
6. Mann JM. Society and public health: crisis and rebirth. *West J Med*. 1998;269:118-121.

Triglycerides and Small, Dense Low-Density Lipoprotein

To the Editor.—In a recent meta-analysis of 17 prospective studies involving 47 000 subjects, elevated triglyceride levels were associated with an increase in coronary artery disease (CAD) risk of 30% in men and 75% in women.¹ In 1 study, the 8-year incidence of CAD was 14% in subjects with triglyceride levels of 1.60 to 2.50 mmol/L (142-221 mg/dL) vs 9.5% in those with cholesterol levels of about 8.00 mmol/L (310 mg/dL).² Miller et al³ found an odds ratio (OR) for CAD of 1.5 with a fasting triglyceride level of 1.13 mmol/L (100 mg/dL), whereas Stampfer et al⁴ found an OR of 1.4 per 1.13 mmol/L (100 mg/dL) increase in nonfasting triglycerides. An increase in triglycerides of 1.00 mmol/L (90 mg/dL) has the same effect on the extent of coronary atherosclerosis as would aging 10 years. Consequently, I was fascinated by the observations by Dr Lamarche and associates⁵ showing higher prevalence and OR for CAD with triglycerides than with other lipoproteins: triglyceride level about 1.52 mmol/L (135 mg/dL), prevalence, 77%, OR, 3.5; low-density lipoprotein (LDL) cholesterol level about 3.70 mmol/L (143 mg/dL), prevalence, 68%, OR, 2.4; small, dense LDL, prevalence, 69%, OR, 2.5; and apolipoprotein B level about 1.1 g/L, prevalence, 69%, OR, 2.7.

Increasing levels of triglycerides make LDL small, dense, and more atherogenic. Serum triglyceride levels therefore provide an indirect measurement of LDL particle size. The apolipoprotein B level is generally considered the best surrogate for the number of atherogenic particles. The elevated apolipoprotein B level acts synergistically with small, dense LDL (OR, 6.0, when both abnormalities are present).⁶

These data, along with my own experience, suggest that serum levels of triglycerides higher than 1.13 mmol/L (100 mg/dL) and an apolipoprotein B level higher than 1.0 g/L might identify a subgroup of individuals with very high risk of CAD, possibly due to small, dense LDL. From a therapeutic point of view, combination therapy may be necessary in such subjects with qualitative and quantitative abnormalities of LDL cholesterol. Statins are highly effective in lowering LDL particle numbers, with little impact on their size. Fibrates and niacin have the greater impact on the LDL particle size with less impact on the number.

In clinical practice, LDL cholesterol levels are usually calculated rather than measured, because of the high cost of this determination. Measurement of LDL particle size is even more expensive and not readily available. It might be informative if Lamarche et al could reanalyze their data and propose new, simplified triglyceride and apolipoprotein B cut points as clinical surrogates for small, dense LDL.

Enas A. Enas, MD
Rockford, Ill

1. Hokanson JE, Austin M. Plasma triglyceride level is a risk factor for cardiovascular disease independent of high density lipoprotein levels: a meta-analysis of population-based prospective studies. *J Cardiovasc Risk*. 1996;3:213-229.

2. Jeppesen J, Hein H, Suadicani O, Gyntelberg F. Triglyceride concentration and ischemic heart disease: an eight-year follow-up in the Copenhagen Male Study. *Circulation*. 1998;97:1029-1036.
3. Miller M, Seidler A, Moalemi A, Pearson T. Normal triglyceride levels and coronary artery disease events: the Baltimore Coronary Observational Long-Term Study. *J Am Coll Cardiol*. 1998;31:1252-1257.
4. Stampfer MJ, Krauss R, Ma J, et al. A prospective study of triglyceride level, low-density lipoprotein particle diameter, and risk of myocardial infarction. *JAMA*. 1996;276:822-828.
5. Lamarche B, Tchernof A, Mauriege P, et al. Fasting insulin and apolipoprotein B levels and low-density lipoprotein particle size as risk factors for ischemic heart disease. *JAMA*. 1998;279:1955-1961.
6. Lamarche B, Tchernof A, Moorjani S, et al. Small dense low-density lipoprotein particles as a predictor of the risk of ischemic heart disease in men: prospective results from the Quebec Cardiovascular Study. *Circulation*. 1997;95:69-75.

In Reply.—Dr Enas legitimately emphasizes the need to consider plasma triglyceride and apolipoprotein B concentrations as clinical surrogates for the presence of small, dense LDL particles in order to improve our ability to predict ischemic heart disease risk. Plasma triglyceride levels may indeed represent the most relevant crude surrogate for LDL particle size. We have previously reported that a plasma triglyceride cutoff point of 1.90 mmol/L (168 mg/dL) corresponded to the value at which both optimal sensitivity (84%) and specificity (83%) were obtained.¹ Similar results were obtained when we analyzed the data from the Quebec Cardiovascular Study (results available on request). This cutoff point is substantially higher than the value suggested by Enas (1.13 mmol/L [100 mg/dL]). However, the small, dense LDL phenotype remains an "arbitrary" phenotype because there is currently no agreement on the LDL particle size value above which CHD risk is increased. Thus, it may well be that deleterious changes in LDL particle size in terms of ischemic heart disease risk may occur at plasma triglyceride values of 1.40 to 1.60 mmol/L (124-142 mg/dL).

On the other hand, the correlation between apolipoprotein B concentrations and LDL particle size, although significant from a statistical viewpoint, has been reported to be weak ($r = -0.25$) in 2 different cohorts of men.^{1,2} We have also previously reported, as noted by Enas, that apolipoprotein B and small, dense LDL appeared to have synergistic effects on risk of ischemic heart disease. Indeed, individuals with small, dense LDL but with reduced plasma apolipoprotein B concentrations (indicating a reduced number of LDL particles) were not at increased risk for ischemic heart disease.² On the other hand, the most significant increase in ischemic heart disease risk (OR, 6.0) was observed when these 2 abnormalities were present concurrently. These observations suggest that (1) elevated plasma apolipoprotein B is not invariably associated with small, dense LDL particles, and (2) a significant proportion of ischemic heart disease risk attributed to small, dense LDL may be modulated by the presence (or absence) of elevated apolipoprotein B levels.

For these reasons and until we have sound evidence that measuring LDL particle size is a cost-effective approach in the management of CAD risk, we also share the view that particular attention should be given to plasma triglyceride levels, particularly in the presence of elevated plasma apolipoprotein B concentrations.³ Plasma triglyceride levels higher than 1.70 to 1.90 mmol/L (151-168 mg/dL) will most frequently identify individuals with small, dense LDL. When observed in combination with elevated apolipoprotein B concentrations, it may represent a condition that may be referred to as "atherogenic hypertriglyceridemia."⁴

Benoît Lamarche, PhD
Gilles R. Dagenais, MD
Jean-Pierre Després, PhD
Laval University
Ste-Foy, Quebec

1. Tchernof A, Lamarche B, Nadeau A, et al. The dense LDL phenotype: association with plasma lipoprotein levels, visceral obesity and hyperinsulinemia in men. *Diabetes Care*. 1996;19:629-637.

- Lamarche B, Tchernof A, Dagenais GR, Cantin B, Lupien PJ, Després JP. Small, dense LDL particles and the risk of ischemic heart disease: prospective results from the Quebec Cardiovascular Study. *Circulation*. 1997;95:69-75.
- Lamarche B, Lewis GF. Atherosclerosis prevention for the next decade: risk assessment beyond LDL cholesterol. *Can J Cardiol*. 1998;14:S41-S51.
- Lamarche B, Després JP, Moorjani S, Cantin B, Dagenais GR, Lupien PJ. Prevalence of dyslipidemic phenotypes in ischemic heart disease: prospective results from the Quebec Cardiovascular Study. *Am J Cardiol*. 1995;75:1189-1195.

Excessive Weight Gain and Effects on Lipids With Intensive Therapy of Type 1 Diabetes

To the Editor.—Dr Purnell and colleagues¹ found that weight gain is associated with increased cardiovascular risk in the group treated with intensive insulin therapy. However, weight gain was associated with a greater increase in total cholesterol and low-density lipoprotein (LDL) cholesterol in the conventional therapy group than in the intensive therapy group, suggesting that treatment and weight gain may have an interactive effect on cholesterol. For example, in the highest quartiles of weight gain, a 13% increase in body mass index (BMI) was associated with a 12% increase in LDL cholesterol in the conventional therapy group. In comparison, a 29% increase in BMI was associated with only a 6.8% increase in LDL cholesterol in the intensive therapy group. However, the authors stated, “Weight gain with conventional therapy resulted in smaller increases in BMI, lipids, and systolic blood pressure.”

In the original analysis of the Diabetes Control and Complications Trial (DCCT), the intensive therapy group had greater improvements in total cholesterol, LDL cholesterol, and triglycerides ($P < .01$) and had a 43% lower rate of macrovascular complications ($P = .08$) than the conventional therapy group, despite the greater amount of weight gain in the intensive therapy group.^{2,3} The interaction of weight gain and cholesterol levels may result from different intensities of glycemia controls between conventional and intensive therapy groups.

Ming Wei, MD
The Cooper Institute for Aerobics Research
Dallas, Tex

- Purnell JQ, Hokanson JE, Marcovina SM, Steffes MW, Cleary PA, Brunzell JD. Effect of excessive weight gain with intensive therapy of type 1 diabetes on lipid levels and blood pressure: results from the DCCT. *JAMA*. 1998;280:140-146.
- The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med*. 1993;329:977-986.
- The Diabetes Control and Complications Trial Research Group. Effect of intensive diabetes management on macrovascular events and risk factors in the Diabetes Control and Complications Trial. *Am J Cardiol*. 1995;75:894-903.

To the Editor.—The study by Dr Purnell and colleagues¹ also has important implications for rethinking the role of intensive glucose control with insulin in type 2 diabetes. Previous clinical trials^{2,3} and now the study by Purnell et al¹ suggest that treatments that raise insulin levels, increase weight and worsen cardiovascular risk factors despite improving glycemia. In 3 European cohorts totaling 17 000 nondiabetic working men, the all-cause mortality was significantly greater for those who had the highest glucose intolerance but not high enough for a diagnosis of diabetes.⁴ Duration rather than severity of glycemia is a greater risk factor for coronary artery disease (CAD), suggesting a shared underlying pathophysiologic process, endothelial cell dysfunction. Enderle et al⁵ suggest that a longer period of undetected diabetes rather than poor glucose control impairs endothelial-dependent vasodilation in type 2 diabetes.

Excess glucose directly exerts many adverse effects on endothelial cells leading to macrovascular disease. Clearly it is desirable to lower the blood glucose level in diabetes, but this should not be at the expense of raising insulin and worsening endothelial function. Rather than considering glycemic control the bottom line, perhaps we should consider glucose levels

in type 2 diabetes more of a marker or result of generalized endothelial dysfunction along with insulin resistance, hypertension, microalbuminuria, and dyslipidemia. We must rethink our focus on intensive treatment of glycemia with insulin-raising therapies and consider dietary modalities to primarily improve endothelial dysfunction.

Eric S. Freedland, MD
Eicotech Corporation
Marblehead, Mass

Eicotech corporation distributes nutrition products that serve as adjuncts to its dietary program for diabetes.

- Purnell JQ, Hokanson JE, Marcovina SM, Steffes MW, Cleary PA, Brunzell JD. Effect of excessive weight gain with intensive therapy of type 1 diabetes on lipid levels and blood pressure: results from the DCCT. *JAMA*. 1998;280:140-146.
- Abaira CC, Colwell J, Nuttall F, et al. Cardiovascular events and correlates in the Veterans Affairs Diabetes Feasibility Trial. *Arch Intern Med*. 1997;157:181-188.
- Turner R, Cull C, Holman R. United Kingdom Prospective Diabetes Study 17: a 9-year update of a randomized, controlled trial on the effect of improved metabolic control on complications in non-insulin dependent diabetes mellitus. *Ann Intern Med*. 1996;124:136-145.
- Balkau B, Shipley M, Jarrett J, et al. High blood glucose concentration is a risk factor for mortality in middle-aged nondiabetic men. *Diabetes Care*. 1998;21:360-367.
- Enderle M-D, Benda N, Schuelling R-M, et al. Preserved endothelial function in IDDM patients, but not in NIDDM patients, compared with healthy subjects. *Diabetes Care*. 1998;21:271-277.

In Reply.—Dr Wei suggests that final lipid levels in each treatment group in the DCCT represented an interaction between improved glycemic control and increased body weight. An important point we had hoped to make with our article was that 75% of the intensively treated group who did not gain an excessive amount of weight retained the benefits of improved glycemic control on lipids. Therefore, in the majority of subjects enrolled in the intensive treatment group of the DCCT, intensive therapy would be expected to improve risks for both microvascular and macrovascular complications. However, in the top quartile of subjects who gained excessive weight and became obese, the benefits of intensive therapy on lipid levels appeared to be lost. At follow-up, the top quartile for weight gain with intensive treatment had nearly identical lipid levels compared with the conventional therapy group and significantly higher levels than all 3 of the lower quartiles in the intensively treated group. Specifically, the top quartile of intensive therapy had higher levels of triglycerides and total and LDL cholesterol; increased cholesterol in intermediate-density lipoprotein and dense LDL fractions; and lower high-density lipoprotein (HDL) cholesterol compared with the lowest quartile of weight gain (who did not change weight on average over the duration of the study). The findings of higher triglycerides, an increase in dense LDL particles, lower HDL cholesterol, greater waist-to-hip ratio, and higher insulin requirements with excessive weight gain in the fourth quartile compared with the first quartile suggest the emergence of the insulin resistance syndrome in the former, which is associated with a greater risk for CAD in subjects without diabetes.^{1,2} Although we did not seek to minimize the risk of weight gain with conventional treatment on CAD risk factors, the constellation of lipid and blood pressure abnormalities in the subjects who experienced excessive weight gain with intensive therapy is particularly worrisome.

We believe the data from our study support the current recommendations by the American Diabetes Association that prevention of obesity in someone gaining an excessive amount of weight with intensive therapy is a reason to loosen glycemic targets.³

We agree with Dr Freedland that, unlike subjects with type 1 diabetes, those with type 2 diabetes are typically obese at the time of diagnosis, and further weight gain with intensive therapy may aggravate lipid risk factors for CAD in a number of subjects. Results from the United Kingdom Prospective Diabetes Study⁴ (UKPDS) demonstrated that significant reductions in myocardial infarction and all-cause mortality oc-

curred in subjects treated with metformin. This treatment group had less weight gain than those initially treated with sulphonylurea or insulin. These results might argue, therefore, in favor of the benefit of minimizing weight gain on CAD risk in subjects with type 2 diabetes. Since the UKPDS was not designed to study the effect of weight change on CAD risk, and because of the complex treatment algorithm used by the UKPDS to achieve target glycemic goals, debate on this subject will likely continue.

Jonathan Q. Purnell, MD
John D. Brunzell, MD
University of Washington
Seattle

1. Gardner CD, Fortmann SP, Krauss RM. Association of small low-density lipoprotein particles with the incidence of coronary artery disease in men and women. *JAMA*. 1996;276:875-881.
2. Stampfer MJ, Krauss RM, Ma J, et al. A prospective study of triglyceride level, low-density lipoprotein particle diameter, and risk of myocardial infarction. *JAMA*. 1996;276:882-888.
3. American Diabetes Association. Patient selection and goals of therapy. In: Banks P, ed. *Intensive Diabetes Management*. Alexandria, Va: American Diabetes Association; 1995:48.
4. UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). *Lancet*. 1998;352:854-865.

Temporal Artery Biopsy to Diagnose Temporal Arteritis

To the Editor.—In their Contempo article, Drs Gordon and Levin¹ published data for the target artery length for temporal artery biopsy specimens. I assume that they are recommending that a surgeon obtain a temporal artery segment that is at least 20 mm in length in situ. Smooth muscle retraction followed by formalin fixation may shorten such a surgical specimen. Do the authors have data on the degree of shrinkage in a processed temporal artery biopsy? (Most pathologists assume that formalin fixation that will cause a 10% reduction in the length of a nonbony structure, but documentation is hard to come by.) Alternatively, if a 2-cm-long portion of formalin-fixed artery is recommended, what is the recommended artery length that should be obtained? Without clarification, I fear that some surgeons will be called to task because of a lack of understanding of tissue-processing artifacts.

Alan Caroe, MD
York Hospital
York, Pa

1. Gordon LK, Levin LA. Visual loss in giant cell arteritis. *JAMA*. 1998;280:385-386.

In Reply.—As most articles that correlated temporal artery biopsy length with sensitivity studied fixed tissue at gross examination, we agree with Dr Caroe that our recommended length of artery before fixation should be increased slightly. However, even more important than those few extra millimeters is the division of the artery into several segments at gross. This way the entire length of the artery can be examined microscopically. At the University of Wisconsin Hospital and Clinics, approximately 20 glass slides containing 3 to 4 sections are examined, each containing 6 to 8 segments of artery at different levels, making it easier to detect "skip" lesions. Thus, it's not just the length, but how you slice it.

Lynn K. Gordon, MD, PhD
University of California, Los Angeles
Leonard A. Levin, MD, PhD
University of Wisconsin Medical School
Madison

Cost-effectiveness Analyses of Statistically Ineffective Treatments

To the Editor.—Although the publication of guidelines¹ has provided an important contribution to standardizing cost-

effectiveness (CE) research, some methodological points still need clarification. In comparing treatment A (innovative treatment) with treatment B (reference treatment), a CE analysis is appropriate when A is more effective, ie, when the difference in clinical effectiveness between A and B is significant.

When the difference in clinical effectiveness is expressed in terms of a significant difference in survival, the CE analysis can be undertaken without difficulty. But when the difference is expressed as the difference between quality-adjusted life-years (QALYs) per patient treated with A minus QALYs per patient treated with B, the problem arises of whether a significant *P* value continues to be needed for conducting a CE analysis.

When available, primary data of quality-adjusted survival (ie, information on survival length and health-related quality of life collected from individual patients) do not pose any problems because statistical significance can be based on individual data expressed as QALYs. In contrast, when dealing with secondary data of utility that provide no information on statistical variability (ie, when the QALYs are not collected in the ratio of 1:1 with the number of patients), the choice of a CE model becomes questionable.

The publication of CE studies wherein primary data show no statistical survival difference between A vs B, but secondary data show a difference in the QALYs not supported by statistical testing, is becoming more frequent,²⁻⁵ but their rationale remains unclear. Dr Freedberg and associates,² in comparing the CE of rifabutin vs no prophylaxis in patients with acquired immunodeficiency syndrome (AIDS), found no statistical difference in unadjusted survival (primary data) but constructed a CE ratio in which the difference in effectiveness was 0.0108 QALY per patient (QALYs per patient, 3.2675 for rifabutin vs 3.2567 for controls). Likewise, Hayman et al³ in comparing radiation therapy vs no radiation therapy in breast cancer found no statistical difference in unadjusted survival (primary data) but constructed a CE ratio in which the difference in effectiveness was 0.35 QALY per patient. A similar problem applies to the study by Hlatky et al,⁵ which has been criticized for the lack of a significant survival difference.⁶

Given that a CE analysis based on the cost per QALY gained assumes that there is a clinical difference between A and B, the following question arises: When primary data show no difference in unadjusted survival, but secondary data reveal a small difference in the QALYs not supported by statistical testing, are CE analyses appropriate?

Sabrina Trippoli, PhD
Andrea Messori, PhD
Policlinico Careggi Hospital
Florence, Italy

1. Weinstein MC, Siegel JE, Gold MR, et al, for the Panel on Cost-effectiveness in Health and Medicine. Recommendations of the Panel on Cost-effectiveness in Health and Medicine. *JAMA*. 1996;276:1253-1258.
2. Freedberg KA, Scharfstein JA, Seage GR, et al. The cost-effectiveness of preventing AIDS-related opportunistic infections. *JAMA*. 1998;279:130-136.
3. Hayman JA, Hillner BE, Harris JR, Weeks JC. Cost-effectiveness of routine radiation therapy following conservative surgery for early-stage breast cancer. *J Clin Oncol*. 1998;16:1022-1029.
4. Liljegren G, Karlsson G, Bergh J, Holmberg L. The cost-effectiveness of routine postoperative radiotherapy after sector resection and axillary dissection for breast cancer stage I: results from a randomized trial. *Ann Oncol*. 1997;8:757-763.
5. Hlatky MA, Rogers WJ, Johnstone I, et al. Medical care costs and quality of life after randomization to coronary angioplasty or coronary bypass surgery. *N Engl J Med*. 1997;336:92-99.
6. Jacobson MW. Cost-effectiveness of coronary bypass surgery versus angioplasty. *N Engl J Med*. 1997;336:1840-1841.

In Reply.—Our response to Drs Trippoli and Messori is premised on the inevitability of choices in the health sector. More often than not, clinicians, managed care organizations, and AIDS drug assistance program managers must make urgent decisions based on the best available information. They do not have the luxury of demanding proof beyond a reasonable scientific doubt or of suspending judgment in anticipation of

better experimental data. Inaction itself is a decision with consequences for patients and society.

Recognizing the imperatives of decision making, physicians and others with a decision-making perspective typically advocate an inclusive, rather than exclusive, attitude toward the data admissible to decision analytic¹ and CE² studies. First, they argue, clinical decisions will be made, and resources will be allocated, with or without conclusive evidence that these decisions are optimal. Second, decision analyses and CE analyses should be regarded as aids to decision making, not as statements of scientific truth. While these authors advocate synthesizing evidence from diverse sources, they also stress the importance of disclosing the uncertainty around estimates of cost-effectiveness, either through sensitivity analysis, as we have provided in our study, or through more sophisticated statistical or simulation methods.^{2,3}

Our analysis of rifabutin, for which no direct evidence of a survival advantage over placebo has been proven (although it has been suggested⁴), is a case in point. We used CE analysis to show that even in the best case, in which the effect implied by clinical studies could be conclusively established, use of this drug would still not be very cost-effective. In fact, rifabutin is more expensive and less effective than azithromycin in preventing *Mycobacterium avium* complex infection. This has 2 policy implications: First, clinical decision makers can be secure that they are not withholding cost-effective therapy if they recommend less expensive (and in this case more effective) regimens to prevent *M avium* complex infection in human immunodeficiency virus-infected patients, and second, performance of bigger and better clinical trials of rifabutin is probably not a good investment of research dollars.

Kenneth A. Freedberg, MD, MSc
George R. Seage III, ScD, MPH
Elena Losina, PhD
Donald E. Craven, MD
Boston Medical Center and
Boston University Schools of Medicine and Public Health
Boston, Mass
Julie A. Scharfstein, ScD
Johns Hopkins School of Hygiene and Public Health
Baltimore, Md
A. David Paltiel, PhD
Yale University School of Medicine
New Haven, Conn
Milton C. Weinstein, PhD
Harvard School of Public Health
Cambridge, Mass

1. Kassirer JP, Moskowitz AJ, Lau J, Pauker SG. Decision analysis: a progress report. *Ann Intern Med*. 1987;106:275-291.
2. Weinstein MC, Siegel JE, Gold MR, et al, for the Panel on Cost-effectiveness in Health and Medicine. Recommendations of the Panel on Cost-effectiveness in Health and Medicine. *JAMA*. 1996;276:1253-1258.
3. Drummond MF, O'Brien B, Stoddart GL, Torrance GW. *Methods for the Economic Evaluation of Health Care Programmes*. 2nd ed. Oxford, England: Oxford University Press; 1997.
4. Moore RD, Chaisson RE. Survival analysis of two controlled trials of rifabutin prophylaxis against *Mycobacterium avium* complex in AIDS. *AIDS*. 1995;9:1337-1342.

Preventing Low Back Pain in Industry

To the Editor.—We find numerous severe faults with the article by Ms van Poppel and colleagues¹ that sharply limit its contribution to the scientific understanding of the efficacy of lumbar supports. Complaint of back pain was not separated from the critical questions of whether the back was injured while the subject was at work and how to prevent such injuries. As fewer than half the persons assigned to wear the support actually wore it, the findings are necessarily suspect. Both compliance and days lost due to back pain were self-reported rather than being assessed objectively, whether back pain occurred while wearing or not wearing a support was not

shown, and whether supports when worn were worn properly was not indicated. Statistical power was compromised by the large and unequal numbers of participants who lost interest or withdrew and the likelihood that this occurred nonrandomly. Moreover, it seems that the initial randomization of work groups did not remove nonrandom features among persons: for instance, median sick leave in the past year reported by the lumbar support intervention group was 27% higher than that reported by the next highest group. Thus, from this evidence, there is no way to know whether the effect (or lack of effect) can be attributed to correct full-time use of a lumbar support.

We strongly disagree with the authors' criticism of our findings² on the basis of changes in exposure or workers' compensation laws: the former was tightly controlled and the latter did not change during the study period. Dismissing historical cohort studies on the basis of nonrandomization demonstrates flawed reasoning. Such studies, when premised on evidence gathered from objective sources, can provide much stronger indications of effect than small, partially randomized trials like this one that rely heavily on each participant's personal recall of pain.

Jess F. Kraus, MPH, PhD
David L. McArthur, PhD, MPH
University of California, Los Angeles
School of Public Health

1. van Poppel MNM, Koes BW, van der Ploeg T, Smid T, Bouter LM. Lumbar supports and education for the prevention of low back pain in industry: a randomized controlled trial. *JAMA*. 1998;279:1789-1794.
2. Kraus JF, Brown KA, McArthur DL, et al. Reduction of acute low back injuries by use of back supports. *Int J Occup Environ Health*. 1996;2:264-273.

To the Editor.—The conclusions of the study by Ms van Poppel and colleagues¹ are not unexpected. In 1972, Beals and Hickman² reported the importance of psychological evaluation in rehabilitation efforts concerning industrial injuries to the back and extremities. The authors found psychological ratings by psychologists to be superior to ratings made by physicians in predicting return to work. In 1975, Wiltse and Rocchio³ reported the Minnesota Multiphasic Personality Inventory hysteria and hypochondriasis scales to be predictive of the result of lumbar chemonucleolysis and lumbar laminectomy. The surgeon's rating of the psychogenic component of the patient's pain was also predictive. In 1991, Bigos et al⁴ reported factors associated with increased likelihood of back pain or low back claims included job satisfaction, quality of interaction between the worker and his or her supervisor, and poor employee appraisal rating. I had a similar experience at the Department of Labor and Industries Rehabilitation Center in Seattle, Wash.

I believe it is abundantly clear that industrial low back pain is primarily a psychosocial problem with huge economic impact. However, the problem should not be deferred only to psychologists, sociologists, and economists, but it is imperative that medical professionals dealing with this problem have knowledge in the psychosocial realm. To attempt to solve the problem on a purely medical model is to ensure continued frustration at best.

Loy E. Cramer, MD
Tacoma, Wash

1. van Poppel MNM, Koes BW, van der Ploeg T, Smid T, Bouter LM. Lumbar supports and education for the prevention of low back pain in industry: a randomized controlled trial. *JAMA*. 1998;279:1789-1794.
2. Beals RK, Hickman NW. Industrial injuries of the back and extremities. *J Bone Joint Surg Am*. 1972;54:1593-1611.
3. Wiltse LL, Rocchio PD. Preoperative psychological tests as predictors of success of chemonucleolysis in the treatment of the low-back syndrome. *J Bone Joint Surg Am*. 1975;57:478-483.
4. Bigos SJ, Battie MC, Spengler DM, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. *Spine*. 1991;16:1-6.

In Reply.—Although any study has its limitations, we believe that the results of our randomized controlled trial are valid and contribute to the existing evidence on the (lack of) efficacy of lumbar supports in the prevention of back pain in industry.¹

One potential limitation of our study is the relatively low compliance rate. It is possible that, on account of possible selection of noncompliant subjects, an existing effect of the lumbar supports was missed that would have been found had the compliance rate been higher. Higher compliance rates could, for example, be obtained, by making the use of the supports mandatory. However, not every employer will choose or be able to do this. The results reported in our article are results that can be expected if an employer decides to introduce lumbar supports for workers involved in manual material handling, without making the use mandatory (and thus accepting partial noncompliance). It is important, however, to realize that the analysis of the results in the subgroup of workers with a high compliance rate did not show any indication of beneficial effects of lumbar supports.

The main outcome measures used in our study were back pain and sick leave because of back pain. We did not distinguish between back pain occurring at work or at home. Because of the multifactorial origin in most cases it is not possible to determine the exact cause of an episode of back pain. This implies, for instance, that back pain occurring at home can be at least in part work related.

Although Drs Kraus and McArthur criticize our study because no objective data sources were used, back pain can only be measured subjectively—who other than the worker can determine whether the worker has back pain? Sick leave because of back pain was measured both as self-reported sick leave and sick leave in the company medical records. Although our article included only self-reported sick leave data, we also analyzed the data on sick leave using the company medical records, which showed similar results: no differences in sick leave could be demonstrated between intervention groups. Since medical records were not available for temporary workers (almost 30% of the total study group), these findings were not presented in the article.

We disagree that the intervention groups were not similar at baseline. Furthermore, in most analyses the 2 groups randomized “with” and “without” lumbar supports were combined. The differences in baseline characteristics between the groups with and without lumbar supports were even smaller.

In conclusion, our study was a pragmatic, randomized controlled trial that reflected what may occur in the workplace, without an artificially imposed increase in compliance rate. We believe it provides strong evidence on the lack of effect of lumbar supports in the workplace that can be expected in daily practice.

Mireille N. M. van Poppel, MSc
Bart W. Koes, PhD
Lex M. Bouter, PhD
Institute for Research in Extramural Medicine
Vrije Universiteit
Amsterdam, the Netherlands

1. van Poppel MNM, Koes BW, Smid T, Bouter LM. A systematic review of controlled clinical trials on the prevention of back pain in industry. *Occup Environ Med.* 1997;54:841-847.

Hot Butter and Cheese Dispensers at the Movie Theater: A Real Sizzler?

To the Editor.—I encountered a new public health hazard in New York, NY, and Miami, Fla, when I took my 6-year-old son to movie theaters owned by different national chains. I noted a new customer “convenience”—self-service hot butter-flavored and cheese-flavored toppings in electrically heated metal dispensers. Using these dispensers anyone can pump hot liquid onto their popcorn, nachos, or person.

As a plastic surgeon who treats burn patients, I am intimately familiar with the potential injury that can occur to children, shorter persons, or people in wheelchairs from highly viscous fluids and hot metal containers. Because of their viscosity, the hot toppings remain in contact with skin and clothing for a longer time than plain hot water would. Water heated to 70°C (158°F) causes a deep burn in less than 1 second of contact with human forearm skin.¹

My inspection of 4 dispensers revealed that they had 3 types of temperature dials. Three of 4 temperature dials had a maximum setting of 212°F and the fourth had a maximum setting of 185°F. None of the temperature dials could be locked, and all the dials were in plain view so anyone could raise the temperature to a potentially unsafe level. Although 1 of the temperature dials was set to “only” 150°F, the other 3 were turned to the maximum heat settings of 185°F and 212°F. I could not inspect the dial on another type of dispenser, but by bringing along my own thermometer, I was able to measure the temperature of the butter flavoring from 4 dispensers where the temperature had been set by theater employees. The temperatures measured 140°F, 150°F, 175°F, and 190°F. Three of the 4 machines had small labels that read “Caution Hot,” and there was no label on the fourth machine.

The metal dispensers are not thermally insulated, thus exposing the public to contact burns. The tops of the dispensers are not locked, and the dispensers are not bolted to counter-tops; therefore, the public is also at risk from accidental spillage. Curious, unsupervised children who cannot read or do not heed the warning label on the side of the dispenser could be injured playing with the dispensing pumps.

Physicians, caregivers to children, and adults who use wheelchairs should be aware of the potentially hazardous food dispensers found in some movie theaters.

M. Felix Freshwater, MD
Miami, Fla

1. Moritz AR, Henriques FC. The relative importance of time and surface temperatures in the causation of cutaneous burns. *Am J Pathol.* 1947;23:695.

In Reply.—On reading Dr Freshwater’s letter, I checked with our concessions, operations, and insurance departments to determine whether we had had any mishaps with our self-serve butter or cheese topping systems. I learned this has not been a problem for General Cinema. We have few, if any, self-serve cheese stations, and even in those theaters with self-serve buttering equipment, I was unable to discover any incidents in which a customer had received a burn while using the equipment. (Our self-serve buttering units can melt the popcorn topping at temperatures as low as 80°F to 90°F.)

Freshwater’s concerns, however, are indeed valid and will be given serious consideration when we examine our concessions operations. I believe my colleagues at AMC, Regal, Loews, United Artists, National Amusements, Hoyts, and Carmike would join me in saying that we try to make the movie-going experience as enjoyable and safe for our patrons as we possibly can. It is in our best interest as well as that of our patrons to prevent any sources of potential injury in all aspects of our operations.

Brian Callaghan
General Cinema Theatres
Chestnut Hill, Mass

The letter by Dr Freshwater was shown to AMC and Sony & Loews Theaters, who declined to reply.—ED.