QUOTATION BIAS IN REVIEWS OF THE DIET–HEART IDEA

UFFE RAVNSKOV

Råbygatan 2, S-223 61 Lund, Sweden

Abstract—Criticism of the diet–heart idea is often met with the argument that consensus committees have settled the issue unanimously. To see how these committees have explained discordant results, quotations from papers with such findings were sought in three recent authoritative reviews. Only two of twelve groups of controversial papers were quoted correctly, and only in one of the reviews. About half of the papers were ignored. The rest were quoted irrelevantly; or insignificant findings in favour of the hypothesis were inflated; or unsupportive results were quoted as if they were supportive. Only one of six randomized cholesterol-lowering trials with a negative outcome were cited and only in one of the reviews. In contrast, each review cited two, four, and six non-randomized trials with a positive outcome, respectively. It appears as if fundamental parts of the diet–heart idea are based on biased quotation.

Coronary heart disease   Atherosclerosis   Cholesterol   Bias   Citation   Quotation

INTRODUCTION

Authoritative committees have declared that a diet rich in cholesterol and saturated fatty acids and poor in polyunsaturated fatty acids, the so-called atherogenic diet, leads to a high cholesterol level in the blood which in turn leads to atheroma growth and coronary heart disease [1–3]. This hypothesis, the diet–heart idea, is said to be based on "strong, scientific data" [1], the evidence is "overwhelming" [3] or "extremely powerful" [2] and "controversy is unjustified" [1]. These wordings seem strange in view of the many contradictory papers which have been published. I found it therefore of interest to see how such papers were quoted in reviews.

MATERIAL

The Reviews

Three reviews [1–3] of the diet–heart idea were selected because of their magnitude and as-sumed authoritativeness being consensus statements from prestigious researchers.

The Contradictory Papers

Supportive evidence has allegedly been obtained along five different pathways (Fig. 1), but contradictory evidence has been presented along the same pathways. Below, the papers which I found have presented the most conflicting findings are presented, path by path, together with the proposals they contradict.

Path 1

Proposal. Dietary fat and cholesterol are the main determinants of the serum cholesterol level [1–3].

Evidence. Laboratory short-time experiments; and comparisons of food consumption data and blood cholesterol levels between populations [1–3].

Counter-evidence. The serum cholesterol of Polynesian [4] and African [5–7] tribes, is lower
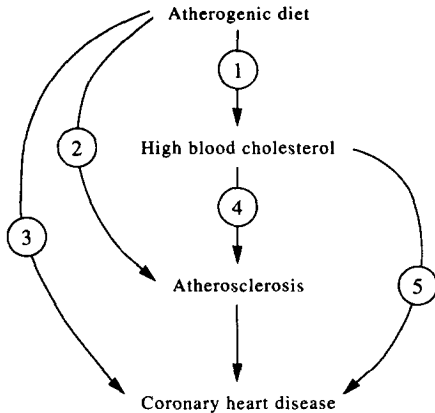
Fig. 1. The diet-heart idea. Arrows symbolize the different pathways along which evidence and counter-evidence have been produced. Figures correspond to those used in Materials.

[4] or much lower [5–7] than in most affluent countries although they eat twice the amount of saturated fat. This is no genetic trait; after migration to urban life their serum cholesterol rose [8] although their intake of saturated fatty acids decreased considerably [9].

Path 2

Proposal. The atherogenic diet is an important cause of atherosclerosis [1–3].

Evidence. Association between consumption data and autopsy findings between countries [1–3].

Counter-evidence. (a) Degree of atherosclerosis in the International Atherosclerosis Project

[10] was correlated to intake of total fat ($r = 0.67$), not to saturated fat ($r = 0.07$). In a more elaborate study of its New Orleans branch [11] no association was found either; if anything, coronary patients had eaten more polyunsaturated fatty acids than had the others. (b) No correlation was found in the Honolulu Heart Program either [12].

Path 3

Proposal. The atherogenic diet is an important cause of coronary heart disease [1–3].

Evidence. Associations between the availability of animal fat and mortality from coronary heart disease in various countries; and differences between the diet of coronary patients and healthy individuals [1–3].

Counter-evidence. (a) The association is neither specific for animal fat nor for coronary heart disease [13–16] and secular trends of coronary mortality do not follow trends of animal fat consumption [16]. (b) Overall, longitudinal studies within populations [17–27] have found no differences between the diet of coronary patients and others (Table 1).

Path 4

Proposal. A high cholesterol level in the blood is causally related to atherosclerosis (raised lesions).

Evidence. Association between individual serum cholesterol levels and degree of

Table 1. Intake of saturated and polyunsaturated fatty acids, and cholesterol (% of energy, and milligram per 1000 calories, respectively, if nothing else is stated) in coronary patients and non-coronary controls

Studies		Saturated fatty acids		Polyunsaturated fatty acids		Cholesterol	
		CHD	Controls	CHD	Controls	CHD	Controls
Zukel et al. [17]	r	18.7	18.9	3.6	3.6	—	—
Paul et al. [18]	p	59[a]	59[a]	13.2[a]	14[a]	721[b]	754[b]
Finegan et al. [19]	r	19	18	4	4	235	234
Bassett et al. [20]	r						
Hawaiian men		13.3	13.2	5.4	5.9	264	272
Japanese men		10.7	11.1	6.3	6.3	254	247
Yano et al. [21]	p	13	12	7	6	245	240
Kannel & Gordon [22]	p	28.9[c]	27.9[c]	10.8[d]	10.6[d]	225	229
Garcia-Palmieri et al. [23]	p						
Urban		13.6	13.5	6.7**	5.9	194	187
Rural		13.1	12.6	3.9	3.9	142	154
Gordon et al. [24]	p						
Framingham		15.3	14.9	5.8	5.4	215	202
Puerto Rico		13.5	13.3	6.0**	5.3	183	174
Honolulu		12.7	12.3	6.7**	6.0	248	239
McGee et al. [25]	p	12.7*	12.3	6.3*	6.0	257*	242
Kromhout & Coulander [26]	p	17.7	17.6	5.9	5.9	165	142
Kushi et al. [27]	p	17.4	16.9	2.6	2.7	266*	248

p = prospective; r = retrospective; [a]gram per day; [b]milligram per day; [c]animal fat; [d]vegetable fat; *$p < 0.05$; **$p < 0.01$.

Table 2. Mean serum cholesterol and 5-year incidence of coronary heart disease (rates per 10,000, age standardized) in three of the "Seven Countries". From Keys [34]

	Serum cholesterol (mg%)	Coronary heart disease
West Finland	253	367
East Finland	265	942
Dalmatia	186	163
Slavonia	198	313
Crete	202	12
Corfu	198	201

atherosclerosis on coronary angiography and at autopsy [1–3].

Counter-evidence. (a) No correlation was found in autopsy studies of unselected individuals [28–30]. (b) In angiographic follow-up studies progress of coronary atherosclerosis was unrelated to secular changes of the serum cholesterol level [31–33].

Path 5

Proposal. A high cholesterol level in the blood is causally related to coronary heart disease [1–3].

Evidence. Associations between population means of serum cholesterol and incidence of coronary heart disease at follow-up; migration studies; and cholesterol lowering trials [1–3].

Counter-evidence. (a) Within three of the "Seven Countries" [34], generally considered as the major supportive evidence, serum cholesterol did not correlate with mortality from coronary heart disease (Table 2). (b) The increased coronary mortality in Japanese migrants in the U.S.A. seen after migration was not associated with their serum cholesterol

level (or their diet) [35], but depended on their cultural upbringing [36]. (c) In the 30-year follow-up study in Framingham [37] serum cholesterol was no risk factor for coronary mortality after the age of 47. In the whole Framingham population including both sexes and all age classes, a decreasing, not an increasing serum cholesterol level, was found to be a risk factor, both for coronary and total mortality. (d) The number of unsupportive trials equals the number of allegedly supportive trials [38]. (e) All trials which had analysed the relationship between degree of cholesterol lowering and outcome found either a non-systematic correlation, or, in most cases, no correlation at all [38].

METHODS

Quotations from the contradictory papers were sought in the reviews and classified. A quotation was classified as *correct* if it impartially referred to the controversial finding of the contradictory paper; a quotation was classified as *irrelevant* if it gave meaningless or misleading information from the cited paper without mentioning its controversial findings; a quotation was classified as *inflated* if statistically insignificant results in favour of the diet–heart idea were exaggerated; or *contrary* if the contradictory paper was quoted as if it was supportive.

For each review all references to cholesterol lowering trials were recorded [38].

RESULTS

Table 3 shows that only two of the twelve groups of contradictory papers were cited

Table 3. Characterization of the quotations of contradictory papers from three authoritative reviews

	Atherosclerosis study group [1]	Diet and health [2]	The cholesterol facts [3]
Path 1 [4–9]	Ignored or contrary	Ignored	Ignored
Path 2a [10, 11]	Contrary	Irrelevant	Ignored
b [12]	—	Correct	Ignored
Path 3a [13–16]	Irrelevant	Correct	Ignored
b [17–27]	Irrelevant	Irrelevant or inflated	Ignored, inflated or contrary
Path 4a [28–30]	Ignored	Ignored	Ignored
b [31–33]	Ignored	Ignored	Ignored
Path 5a [34]	Inflated	Contrary	Ignored
b [35, 36]	Irrelevant	Irrelevant	Ignored
c [37]	—	Ignored	Contrary
d [38]	Inflated	Inflated	Inflated or contrary
e [38]	Ignored	Ignored	Ignored

Paths and letters refer to Fig. 1. All irrelevant, inflated and contrary quotations are given in Results. For definitions of quotation errors, see Methods. Dash: Contradictory paper(s) published after the review.

correctly, and only in one of the reviews. About half of the papers or observations were ignored; the rest were incorrect in one or more ways. The following sections present each of the incorrect quotations in abbreviated form (italics). Path numbers and letters correspond to the classification used in the material section and Table 3.

Irrelevant Quotations

Path 2

(a) *The extent of raised lesions ... was positively associated with intake of fat and animal protein* [2, p. 193].
Correct, but misleading as it was unrelated to intake of animal fat; see Materials.

Path 3

(a) *Epidemiologic evidence supporting an association between dietary fat and cholesterol and CHD mortality continues to mount* [1, p. 167A].
A misleading statement because the main point of the authors of the cited papers [13–16] was that the association was not a causal one; see Material.

(b) *... rates for coronary heart disease are lower for Puerto Ricans and Hawaiians than for residents of Framingham* [1, p. 168A].
Incidence of first major CHD events among middle-aged men in Framingham was twice that in Puerto Rico and Hawaii [2, p. 164].
The quotations are correct but meaningless. The salient point is that *within* these populations food consumption patterns were opposite as was expected; see Table 1.

Path 5

(b) *Coronary heart disease ... (was more common) in Americans of Japanese ancestry (compared to Japanese men in Japan and Hawaii)* [1, p. 168A].
Relative risks of first major CHD event ... were 0.46, 1.00, and 1.54 ... in Japan, Hawaii, and California [2, p. 164].
Correct, but misleading. The statements suggest that CHD incidence after migration followed changes of diet and serum cholesterol, but the striking, contradictory finding, which was not mentioned in the reviews, was that CHD incidence did not correlate with cholesterol concentration or diet *within* the Japanese population in the U.S. See Material section.

Inflated Quotations

Path 3

(b) *Percentage of calories from SFAs was positively associated with risk of CHD in the rural sample of the Puerto Rican and the Ireland–Boston Studies* [2, p. 193].
... showing the link between diet and CHD particular impressive results (were produced in) the Western-Electric [39], the Honolulu Heart [25], the Zutphen [26], and the Ireland–Boston [27] studies [3, p. 1725].
The quotations are inflated because the dietary differences between coronary patients and controls were not significant; see Table 1. Furthermore, the other insignificant and the contradictory results [17–24] were ignored. In the Western Electric study [39] intake of cholesterol, but not of fatty acids, was related to coronary mortality. (The latter study is not represented in Table 1 because no absolute figures were given).

Path 5

(a) *There is a strong association between population means of total cholesterol and CHD incidence* [1, p. 164A].
In the quoted study [34] no association was found in three of the seven countries; see Table 2.

(d) *The long-awaited results of the Lipid Research Clinics trial [40] provide the most conclusive strong evidence* [1, p. 185A].
The Lipid Research Clinics trial [40] and the NHLBI type II study [41] gave statistically significant results [3, p. 1723–1724].
The results were not statistically significant according to conventional statistical methods (e.g. two-tailed p was greater than 0.05).
The Multiple Risk Factor Intervention Trial [42] ... has shown that serum cholesterol levels can be reduced by diet [1, p. 184A].
Net reduction was 2 mg%.
One of the reviews [1] only referred to one [43] of the five randomized cholesterol-lowering trials with negative results which had been published at the time of publication [43–48], whereas the authors included six non-randomized trials with positive results [49–54]. The two other reviews [2] did not refer to any of six randomized trials with negative results [43–48, 55], whereas the authors of one of these reviews [3] included two non-randomized trials with positive results [56, 57], and the authors of the other review included four such trials [56–59].

Contrary Quotations

Path 1

... isolated groups ... have lower serum cholesterol levels consistent with their lower intakes of saturated fat [1, p. 168A].

One of the two cited groups had a low cholesterol but a very high intake of saturated fat [4].

Path 2

(a) *The Geographic Pathology study [10] ... support(s) ... that diets low in saturated fat ... are associated with a low incidence of coronary heart disease [1, p. 166A].*

The mentioned study [10] found no association between the consumption of saturated fat and atherosclerosis, and the association between diet and CHD was not addressed at all.

Path 3

(b) *Studies showing a correlation between saturated fatty acids and CHD include the Ireland–Boston study [27], The Western-Electric study [39] (and) the Zutphen study [26] [3, p. 1726].*

No correlation was found; see Table 1 and above.

Path 2

(a) *Cholesterol levels above 200–220 mg/dl are positively associated with risk of CHD in ... Finland, Greece [2, p. 164].*

In east Finland, the incidence of CHD was almost three times higher than in west Finland although mean serum cholesterol levels were almost identical. On the Greek island Corfu incidence of CHD was twelve times higher than on the Greek island Crete although mean serum cholesterol was lower on Corfu. See Table 2.

(c) *The results from the Framingham Study [37] indicate that a 1% reduction ... of cholesterol (corresponds to a) 2% reduction in CHD risk [3, p. 1723].*

For each 1 mg/dl drop of cholesterol there was an 11% *increase* in coronary and total mortality [37].

(d) *Trials in humans have shown that lowering ... cholesterol ... decreases the incidence of fatal CHD [3, p. 1722].*

The incidence of fatal CHD was not lowered significantly in any cholesterol-lowering trial [38].

DISCUSSION

Many of the studies referred to in this paper indicate that the diet–heart idea is wrong or at least incomplete. However, the aim with my study was not to discuss the etiology of atherosclerosis and coronary heart disease; cholesterol may possibly play a pathogenetic role, although it may not be so simple as has been postulated in the past. The aim was to study the correctness of reviews written by distinguished scientific bodies, because faulty knowledge from such reviews may have the same disastrous effect in science as a data virus in a computer.

References in medical papers are often erroneous, but those who have studied this problem have mostly pointed to citation errors such as incorrect spelling, omission of names, and faulty journal data. Few have studied incorrect quotation, although this error probably is more widespread than citation errors [60–62]. My analysis has demonstrated that a large number of relevant studies are ignored or incorrectly quoted in authoritative reviews of the diet–heart idea, and a recent, comprehensive review [63] demonstrated that the misquotations are not a selected collection of rare mistakes, but rather the tip of an iceberg.

Authors may have relevant reasons for ignoring a study in an overview. However, studies published in international, peer-reviewed journals presenting results in obvious conflict with the diet–heart idea should at least have been mentioned and authors should explain why they mistrust the findings or disagree with the conclusions.

More serious are the many exaggerations and other misleading ways of quotation. Some of the quoted authors may be blamed, as they in conflict with their data claimed their findings supportive. If referees were asked, they could have improved the reviews, but their words may have carried little weight against committees of esteemed researchers. As for the authors of the reviews themselves, wishful thinking may have guided their writing because examinations of contradictory findings were rare, opposing views were mostly wiped aside as being unwarranted and few details were given as to the essence of their criticism, and when quotations were incorrect, they were always biased in favour of the diet–heart idea. It was also striking that almost all of the randomized, non-supportive trials were ignored, whereas a large number of supportive, but non-randomized trials were

included, because non-randomized trials are highly susceptible to bias [64].

Such a way of writing may give the uninitiated reader a false impression of consensus, large segments of science are diverted to fruitless fields, and public health may suffer from costly, ineffective, and even harmful measures.

National health authorities should consult original papers and not rely on committee writings only. There is much more contradictory evidence than presented above [63, 65–67] and the ultimate proof, a reduction of mortality after cholesterol lowering of human beings, has failed. The rationale behind the diet recommendations given nationwide in many countries is therefore questioned.

REFERENCES

1. Kannel WB, Doyle JT, Ostfeld AM, et al. Optimal resources for primary prevention of atherosclerotic diseases. Atherosclerosis study group. **Circulation** 1984; 70: 157A–205A.
2. National Research Council **Diet and Health. Implications for Reducing Chronic Disease Risk**. Washington, DC: National Academy Press; 1989.
3. Gotto AM, LaRosa JC, Hunninghake D, et al. The cholesterol facts. A summary of the evidence relating dietary fats, serum cholesterol, and coronary heart disease. A joint statement by the American Heart Association and the National Heart, Lung, and Blood Institute. **Circulation** 1990; 81: 1721–1733.
4. Prior IA, Davidson F, Salmond CE, Czochanska Z. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau Island studies. **Am J Clin Nutr** 1981; 34: 1552–1561.
5. Shaper AG. Cardiovascular studies in the Samburu tribe of Northern Kenya. **Am Heart J** 1962; 63: 437–442.
6. Mann GV, Shaffer RD, Anderson RS, Sandstead HH. Cardiovascular disease in the Masai. **J Atheroscl Res** 1964; 4: 289–312.
7. Lapiccirella V, Lapiccirella R, Abboni F, Liotta S. Enquête clinique, biologique et cardiographique parmi les tribus nomades de la Somalie qui se nourissent seulement de lait. **Bull Wld Health Org** 1962; 27: 681–697.
8. Day J, Carruthers M, Bailey A, Robinson D. Anthropometric, physiological and biochemical differences between urban and rural Masai. **Atherosclerosis** 1976; 23: 357–361.
9. Stanhope JM, Sampson VM, Prior IAM. The Tokelau Island migrant study: serum lipid concentrations in two environments. **J Chron Dis** 1981; 34: 45–55.
10. Scrimshaw NS, Guzmán MA. Diet and atherosclerosis. **Lab Invest** 1968; 18: 623–628.
11. Moore MC, Guzmán MA, Schilling PE, Strong JP. Relation of selected dietary components to raised coronary lesions. Dietary-atherosclerosis study on deceased persons. **J Am Diet Ass** 1976; 68: 216–223.
12. Reed DM, MacLean CJ, Hayashi T. Predictors of atherosclerosis in the Honolulu heart program. 1. Biologic, dietary, and lifestyle characteristics. **Am J Epidemiol** 1987; 126: 214–225.
13. Yerushalmy J, Hilleboe HE. Fat in the diet and mortality from heart diesease. A methodologic note. **NY State J Med** 1957; 57: 2343–2354.
14. Yudkin J. Diet and coronary thrombosis. Hypothesis and fact. **Lancet** 1957; 2: 155–162.
15. Jolliffe N, Archer M. Statistical associations between international coronary heart disease death rates and certain environmental factors. **J Chron Dis** 1959; 9: 636–652.
16. Masironi R. Dietary factors and coronary heart disease. **Bull Wld Health Org** 1970; 42: 103–114.
17. Zukel WJ, Lewis RH, Enterline PE, et al. A short-term community study of the epidemiology of coronary heart disease. A preliminary report on the North Dakota study. **Am J Publ Health** 1959; 49: 1630–1639.
18. Paul O, Lepper MH, Phelan WH, et al. A longitudinal study of coronary heart disease. **Circulation** 1963; 28: 20–31.
19. Finegan A, Hickey N, Maurer B, Mulcahy R. Diet and coronary heart disease: dietary analysis on 100 male patients. **Am J Clin Nutr** 1968; 21: 143–148.
20. Bassett DR, Abel M, Moellering RC, Rosenblatt G, Stokes J. Coronary Heart disease in Hawaii: dietary intake, depot fat, "stress", smoking, and energy balance in Hawaiian and Japanese men. **Am J Clin Nutr** 1969; 22: 1483–1503.
21. Yano K, Rhoads GG, Kagan A, Tillotson J. Dietary intake and the risk of coronary heart disease in Japanese men living in Hawaii. **Am J Clin Nutr** 1978; 31: 1270–1279.
22. Kannel WB, Gordon T. The Framingham diet study: diet and the regulation of serum cholesterol. **The Framingham Study**. An epidemiologic investigation of cardiovascular disease. Section 24, Washington DC; 1970.
23. Garcia-Palmieri MR, Sorlie P, Tillotson J, Costas R, Cordero E, Rodriguez M. Relationship of dietary intake to subsequent coronary heart disease incidence: the Puerto Rico heart health program. **Am J Clin Nutr** 1980; 33: 1818–1827.
24. Gordon T, Kagan A, Garcia-Palmieri M, et al. Diet and its relation to coronary heart disease and death in three populations. **Circulation** 1981; 63: 500–515.
25. McGee DL, Reed DM, Yano K, Kagan A, Tillotson J. Ten-year incidence of coronary heart disease in the Honolulu heart program. Relationship to nutrient intake. **Am J Epidemiol** 1984; 119: 667–676.
26. Kromhout D, Coulander CDL. Diet, prevalence and 10-year mortality from coronary heart disease in 871 middle-aged men. The Zutphen study. **Am J Epidemiol** 1984; 119: 733–741.
27. Kushi LH, Lew RA, Stare FJ, et al. Diet and 20-year mortality from coronary heart disease. The Ireland–Boston diet-heart study. **N Engl J Med** 1985; 312: 811–818.
28. Landé KE, Sperry WM. Human atherosclerosis in relation to the cholesterol content of the blood serum. **Arch Pathol** 1936; 22: 301–312.
29. Mathur KS, Patney NL, Kumar V, Sharma RD. Serum cholesterol and atherosclerosis in man. **Circulation** 1961; 23: 847–852.
30. Paterson JC, Armstrong R, Armstrong EC. Serum lipid levels and the severity of coronary and cerebral atherosclerosis in adequately nourished men, 60 to 69 years of age. **Circulation** 1963; 27: 229–236.
31. Bemis CE, Gorlin R, Kemp HG, Herman MV. Progression of coronary artery disease. A clinical arteriographic study. **Circulation** 1973; 47: 455–464.
32. Shub C, Vlietstra RE, Smith HC, Fulton RE, Elveback LR. The unpredictable progression of symptomatic coronary artery disease. A serial clinical-angiographic analysis. **Mayo Clin Proc** 1981; 56: 155–160.
33. Kramer JR, Kitazume H, Proudfit WL, Matsuda Y, Williams GW, Sones FM. Progression and regression of coronary atherosclerosis: relation to risk factors. **Am Heart J** 1983; 105: 134–144.

34. Keys A. Coronary heart disease in seven countries. **Circulation** 1970; 41 (Suppl. 1): 1–211.

35. Marmot MG, Syme SL, Kagan A, Kato H, Cohen JB, Belsky J. Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: prevalence of coronary and hypertensive heart disease and associated risk factors. **Am J Epidemiol** 1975; 102: 514–525.

36. Marmot MG, Syme SL. Acculturation and coronary heart disease in Japanese-Americans. **Am J Epidemiol** 1976; 104: 225–247.

37. Anderson KM, Castelli WP, Levy D. Cholesterol and mortality. 30 years of follow-up from the Framingham study. **JAMA** 1987; 257: 2176–2180.

38. Ravnskov U. Cholesterol lowering trials in coronary heart disease: frequency of citation and outcome. **Br Med J** 1992; 305: 15–19.

39. Shekelle RB, Shryock AM, Paul O, et al. Diet, serum cholesterol, and death from coronary heart disease. The Western-Electric study. **N Engl J Med** 1981; 304: 65–70.

40. Lipid Research Clinics Program. The lipid research clinics coronary primary prevention trial results. I. Reduction in incidence of coronary heart disease. **JAMA** 1984; 251: 351–364.

41. Brensike JF, Levy RI, Kelsey SF, et al. Effects of therapy with cholestyramine on progression of coronary arteriosclerosis: results of the NHLBI type II coronary intervention study. **Circulation** 1984; 69: 313–324.

42. Multiple Risk Factor Intervention Trial Research Group. Multiple risk factor intervention trial. Risk factor changes and mortality results. **JAMA** 1982; 248: 1465–1477.

43. Research Committee. Low-fat diet in myocardial infarction. A controlled trial. **Lancet** 1965; 2: 501–504.

44. Rose GA, Thomson WB, Williams RT. Corn oil in treatment of ischaemic heart disease. **Br Med J** 1965; 1: 1531–1533.

45. Research committee to the medical research council. Controlled trial of soya-bean oil in myocardial infarction. **Lancet** 1968; 2: 693–700.

46. Woodhill JM, Palmer AJ, Leelarthaepin B, McGilchrist C, Blacket RB. Low fat, low cholesterol diet in secondary prevention of coronary heart disease. **Adv Exp Med Biol** 1978; 109: 317–330.

47. Frantz ID, Dawson EA, Kuba K, et al. The Minnesota coronary survey: Effect of diet on cardiovascular events and deaths. **Circulation** 1975; 51–52 (Suppl. II): II-4.

48. Frantz ID, Dawson EA, Ashman PL, et al. Test of effect of lipid lowering by diet on cardiovascular risk. The Minnesota coronary survey. **Arteriosclerosis** 1989; 9: 129–135.

49. Hood B, Sanne H, Örndahl G, Ahlström M, Welin G. Long-term prognosis in essential hypercholesterolemia. The effect of strict diet. **Acta Med Scand** 1965; 178: 161–173.

50. Nelson AM, Douglas C. Der Einfluss einer fettkontrollierten Ernährung auf Patienten mit Konarerkrankungen. **Med Welt** 1965; 16: 2063–2068.

51. Rinzler SH. Primary prevention of coronary heart disease by diet. **Bull NY Acad Med** 1968; 44: 936–949.

52. Miettinen M, Turpeinen O, Karvonen MJ, Elosuo R, Paavilainen E. Effect of cholesterol-lowering diet on mortality from coronary heart-disease and other causes. **Lancet** 1972; 2: 835–838.

53. Bierenbaum ML, Fleischman AI, Raichelson RI, Hayton T, Watson PB. Ten-year experience of modified-fat diets on younger men with coronary heart-disease. **Lancet** 1973; 1: 1404–1407.

54. Barndt R, Blankenhorn DH, Crawford DW, Brooks SH. **Ann Intern Med** 1977; 86: 139–146.

55. Miettinen TA, Huttunen JK, Naukkarinen V, et al. Multifactorial primary prevention of cardiovascular diseases in middle-aged men. Risk factor changes, incidence, and mortality. **JAMA** 1985; 254: 2097–2102.

56. Turpeinen O, Karvonen MJ, Pekkarinen M, Miettinen M, Elosuo R, Paavilainen E. Dietary prevention of coronary heart disease: the Finnish mental hospital study. **Int J Epidemiol** 1979; 8: 99–117.

57. Arntzenius AC, Kromhout D, Barth JD, et al. Diet, lipoproteins, and the progression of coronary atherosclerosis: the Leiden intervention trial. **N Engl J Med** 1985; 312: 805–811.

58. Nash DT, Gensini G, Esente P. Effect of lipid-lowering therapy on the progression of coronary atherosclerosis assessed by scheduled repetitive coronary arteriography. **Int J Cardiol** 1982; 2: 43–55.

59. Nikkilä EA, Viikinkoski P, Valle M, Frick MH. Prevention of progression of coronary atherosclerosis by treatment of hyperlipidaemia: a seven year prospective angiographic study. **Br Med J** 1984; 289: 220–223.

60. Neihouse PF, Priske SC. Quotation accuracy in review articles. **DICP Ann Pharmacother** 1989; 23: 594–596.

61. Evans JT, Nadjari HI, Burchell SA. Quotational and reference accuracy in surgical journals. A continuing peer review problem. **JAMA** 1990; 263: 1353–1354.

62. Gøtzsche PC. Reference bias in reports of drug trials. **Br Med J** 1987; 295: 654–656.

63. Smith RL. Diet, Blood Cholesterol and Coronary Heart Disease: A critical Review of the Literature, Vol. 2. Santa Monica, CA: Vector; 1991.

64. Chalmers TC, Celano P, Sacks HS, Smith H. Bias in treatment assignment in controlled clinical trials. **N Engl J Med** 1983; 309: 1358–1361.

65. Stehbens WE. Flaws in the lipid hypothesis of atherogenesis. **Pathology** 1988; 20: 395–398.

66. Gurr MI. Dietary lipids and coronary heart disease: old evidence, new perspective. **Prog Lipid Res** 1992; 31: 195–243.

67. Rosenman RH. The questionable roles of the diet and serum cholesterol in the incidence of ischemic heart disease and its 20th century changes. **Homeostasis** 1993; 34: 1–44.