

Table 1. (Continued)

No.	Year	Authors (reference No.)	Subjects			Follow-up (y)	Cases [†] (n)	Dietary method (times)	Dietary factor examined	Range or median Multivariate relative risk 95% confidence interval					P for trend	Factors used for adjustment	Association
			#	Sex	Age (y)					—(Low)	—	—	—	—(High)			
9	2004	Song et al. (30)	38,025	W	≥45	5.9	918	VFPQ (1)	*Starch (g/d)	1.00	0.95	1.05	1.06	0.88	0.61	Age, SM, BMI, vigorous PA, AL, HRT, SP, history of HT/PTS, history of high CL, FH	N
									Ref	0.77-1.17	0.85-1.28	0.86-1.30	0.71-1.09				
									255	296	328	365	433				
								*Magnesium (diet & SP) (mg/d)	1.00	1.06	0.81	0.86	0.89	0.05	Age, SM, BMI, PA, AL, FH, IT (E)	N	
								Ref	0.88-1.28	0.66-1.00	0.70-1.06	0.71-1.10					
								*Magnesium (diet only) (mg/d)	1.00	291	319	349	399	0.09	Age, SM, BMI, PA, AL, FH, IT (E)	N	
								Ref	0.83-1.22	0.79-1.18	0.68-1.04	0.71-1.10					
10	2003	Montonen et al. (32)	4,316	M/W	40-69	10	156	DH (1)	Total grain (g/d)	10-181	182-248	249-339	340-1535		0.001	Age, sex, residential area, SM, BMI, IT (E, FT & BR, VG)	Y
									1.00	0.80	0.48	0.38					
									Ref	0.54-1.20	0.28-0.81	0.19-0.77					
									Whole grain (g/d)	0-109	110-162	163-237	238-1321		0.02	Age, sex, residential area, SM, BMI, IT (E, FT & BR, VG)	N
									1.00	1.05	0.52	0.65					
									Ref	0.71-1.55	0.31-0.88	0.36-1.18					
									Rye (g/d)	0-58	59-112	113-181	182-1026		0.30	Age, sex, residential area, SM, BMI, IT (E, FT & BR, VG)	N
									1.00	0.99	1.00	0.65					
									Ref	0.66-1.48	0.65-1.54	0.36-1.18					
									Other whole grain (g/d)	0-5	6-23	24-75	76-632		0.69	Age, sex, residential area, SM, BMI, IT (E, FT & BR, VG)	N
									1.00	1.20	1.12	1.14					
									Ref	0.77-1.88	0.71-1.79	0.69-1.87					
									Refined grain (g/d)	0-45	46-73	74-110	111-567		0.05	Age, sex, residential area, SM, BMI, IT (E, FT & BR, VG)	N
									1.00	0.70	0.68	0.62					
									Ref	0.47-1.07	0.43-1.06	0.36-1.06					
									Refined grain from wheat (g/d)	0-33	34-58	59-90	91-389		0.11	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	N
									1.00	0.81	0.70	0.69					
									Ref	0.53-1.22	0.45-1.10	0.41-1.14					
									Dietary fiber (g/d)	2.6-19.2	19.3-25.3	25.4-33.1	33.2-118		0.04	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	N
									1.00	0.70	0.67	0.51					
Ref	0.46-1.07	0.40-1.11	0.26-1.00														
Soluble fiber (g/d)	0.53-4.5	4.6-5.8	5.9-7.3	7.4-22.7		0.21	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	N									
1.00	0.50	0.74	0.57														
Ref	0.31-0.81	0.44-1.25	0.29-1.12														
INCP (g/d)	1.1-8.7	8.8-12.0	12.1-16.5	16.6-69.3		0.03	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	Y									
1.00	0.75	0.72	0.47														
Ref	0.50-1.12	0.45-1.17	0.25-0.91														
Cellulose (g/d)	0.48-3.2	3.3-4.2	4.3-5.3	5.4-15.2		0.19	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	N									
1.00	0.53	0.67	0.60														
Ref	0.32-0.85	0.39-1.14	0.29-1.21														
Lignin (g/d)	0.48-2.3	2.4-3.1	3.2-4.1	4.2-14.5		0.16	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	N									
1.00	0.79	0.69	0.68														
Ref	0.52-1.20	0.42-1.15	0.36-1.30														
Cereal fiber (g/d)	0.47-12.0	12.1-17.3	17.4-24.4	24.5-111		0.01	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	Y									
1.00	0.81	0.74	0.39														
Ref	0.54-1.21	0.46-1.18	0.20-0.77														
Fruit fiber (g/d)	0-0.99	1.0-2.0	2.1-3.3	3.4-36.8		0.87	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	N									
1.00	0.68	0.79	0.92														
Ref	0.39-1.20	0.39-1.60	0.40-2.13														
Vegetable fiber (g/d)	0.11-3.7	3.8-5.0	5.1-6.7	6.8-26.5		0.86	Age, sex, geographic area, SM, BMI, IT (E, FT & BR, VG)	N									
1.00	1.02	0.89	1.19														
Ref	0.58-1.79	0.43-1.85	0.46-3.04														
10	2004	Montonen et al. (32)	4,304	M/W	40-69	23	383	DH (1)	*Vitamin E (diet only) (mg/d)	<5.51	5.51-6.26	6.27-7.31	>7.31		0.02	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	Y
									1.00	0.79	0.78	0.69					
									Ref	0.60-1.04	0.58-1.06	0.51-0.94					
									*α-Tocopherol (diet only) (mg/d)	<4.66	4.66-5.30	5.31-6.20	>6.20		0.01	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	Y
									1.00	0.72	0.80	0.66					
									Ref	0.34-0.96	0.59-1.08	0.49-0.90					
									*β-Tocopherol (diet only) (mg/d)	<0.38	0.38-0.49	0.50-0.64	>0.64		0.18	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N
									1.00	0.97	1.09	0.76					
									Ref	0.73-1.29	0.82-1.43	0.56-1.03					
									*γ-Tocopherol (diet only) (mg/d)	<0.79	0.79-1.23	1.24-2.06	>2.06		0.04	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N
									1.00	0.91	0.75	0.77					
									Ref	0.69-1.20	0.56-1.01	0.57-1.03					
									*δ-Tocopherol (diet only) (mg/d)	<0.09	0.09-0.15	0.16-0.28	>0.28		0.02	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	Y
									1.00	0.85	0.81	0.69					
									Ref	0.64-1.13	0.60-1.08	0.51-0.93					
									*α-Tocotrienol (diet only) (mg/d)	<1.07	1.07-1.54	1.55-2.05	>2.05		0.47	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N
									1.00	0.95	0.91	0.91					
									Ref	0.72-1.25	0.68-1.21	0.67-1.23					
									*β-Tocotrienol (diet only) (mg/d)	<1.69	1.69-2.15	2.16-2.67	>2.67		0.03	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N
									1.00	0.97	0.77	0.76					
Ref	0.74-1.28	0.58-1.03	0.56-1.03														
*γ-Tocotrienol (diet only) (mg/d)	<0.04	0.04-0.07	0.08-0.12	>0.12		0.11	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N									
1.00	0.95	0.88	0.79														
Ref	0.72-1.26	0.65-1.18	0.59-1.07														
*δ-Tocotrienol (diet only) (mg/d)	<0.002	0.002-0.006	0.007-0.01	>0.01		0.06	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N									
1.00	1.01	0.69	0.84														
Ref	0.77-1.32	0.51-0.93	0.53-1.13														
*Vitamin C (diet only) (mg/d)	<49.7	49.7-66.2	66.3-87.9	>87.9		0.77	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N									
1.00	0.97	0.91	0.97														
Ref	0.73-1.29	0.68-1.22	0.72-1.32														
*Total carotenoids (diet only) (μg/d)	<186.2	186.3-286.5	286.7-451.9	>451.9		0.07	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	Y									
1.00	0.71	0.97	0.71														
Ref	0.53-0.96	0.71-1.27	0.52-0.96														
*α-Carotene (diet only) (μg/d)	<9.4	9.4-34.4	34.5-103	>103		0.55	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N									
1.00	1.11	1.02	0.94														
Ref	0.83-1.49	0.75-1.38	0.69-1.28														

Table 1. (Continued)

No. Year	Authors (reference No.)	Subjects n Sex Age (y)	Follow-up (y) (n)	Cases (n)	Dietary method (times)	Dietary factor examined	Range or median Multivariate relative risk 95% confidence interval	P for trend	Factors used for adjustment	Association				
						*β-Carotene (diet only) (μg/d)	<698 1.00 Ref 0.62-1.11	698-1104 0.83 1.14 0.87-1.51	1105-2121 0.74 0.82 0.54-1.01	>2121	0.25	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N	
						*γ-Carotene (diet only) (μg/d)	<6.3 1.00 Ref 0.59-1.04	6.4-28.5 0.78 0.67-1.18	28.6-60.3 0.88 0.61-1.10	>60.3	0.29	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N	
						*Lycopene (diet only) (μg/d)	<112 1.00 Ref 0.59-1.05	113-494 0.79 0.65-1.14	495-1064 0.86 0.60-1.09	>1064	0.23	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N	
						*β-Cryptoxanthin (diet only) (μg/d)	<0.24 1.00 Ref 0.40-0.73	0.24-1.36 0.54 0.43-0.76	1.37-4.18 0.57 0.44-0.78	>4.18	<0.001	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	Y	
						*Lutein & zeaxanthin (diet only) (mg/d)	<749 1.00 Ref 0.69-1.21	749-935 0.91 0.71-1.26	936-1156 0.95 0.55-1.01	>1156	0.09	Age, sex, geographic area, occupation, SM, BMI, FH, IT (E)	N	
11	2003 Reunanen et al. (33)	19,518 M/W	20-98	15.6	855	NVQ (1)	≤2 1.00 Ref 0.81-1.27	3-4 1.01 0.79-1.21	5-6 0.98 1.21	≥7 0.92 0.73-1.16	—	Age, sex, BMI, SM, leisure time PA	N	
12	2003 Saremi et al. (34)	2,680 M/W	≥15	11	824	NVQ (1)	NV 1.00 Ref 0.69-1.34	<1 1.09 0.74-1.13	1-2 0.92 1.13	≥3 1.01 0.82-1.26	0.01 0.81	0.60	Age, sex, BMI	N
13	2003 Schulze et al. (35)	91,246 W	26-46	7.8	741	VFFQ (2)	<1 1.00 Ref 0.90-1.32	1 1.09 1.02-1.68	2-4 1.31 1.26-2.36	≥5 1.72	<0.001	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	Y	
						Bacon (servings/wk)	<1 1.00 Ref 1.03-1.59	1 1.28 1.20-2.27	≥2 1.65	>2 1.65	<0.001	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	Y	
						Hot dogs (servings/wk)	<1 1.00 Ref 0.83-1.23	1 1.01 1.04-2.11	≥2 1.48	>2 1.48	0.051	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	Y	
						Sausage, salami, bologna, other processed meats	<1/wk 1.00 Ref 0.89-1.30	1/wk 1.08 1.04-1.62	≥2/wk 1.30	>2/wk 1.30	0.019	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	Y	
						Total red meat (servings/wk)	<1 1.00 Ref 0.72-1.70	1 1.11 0.76-1.86	2-4 1.19 0.78-2.04	≥5 1.26	0.259	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	N	
						Beef or lamb as MD (servings/wk)	<1 1.00 Ref 0.86-1.24	1 1.03 0.87-1.42	≥2 1.11	>2 1.11	0.41	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	N	
						Pork as MD (servings/wk)	<1 1.00 Ref 0.80-1.13	1 0.95 0.69-1.48	≥2 1.01	>2 1.01	0.91	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	N	
						Hamburgers (servings/wk)	<1 1.00 Ref 0.94-1.34	1 1.12 1.05-1.70	≥2 1.34	>2 1.34	0.026	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	Y	
						Beef, pork, or lamb as SW or XD (servings/wk)	<1 1.00 Ref 0.81-1.15	1 0.97 0.71-1.11	≥2 0.89	>2 0.89	0.31	Age, BMI, AL, PA, FH, SM, history of high blood pressure, history of high blood CL, HRT, OC, GI, IT (E, Mg, CF, CDF, CL, SEA, MUEA, PUEA, TFE)	N	
14	2004 Rosengren et al. (36)	1,361 W	39-65	18	74	NVQ (1)	≤2 1.00 Ref 0.32-0.98	3-4 0.56 0.23-0.90	5-6 0.45 0.26-1.29	≥7 0.57	—	Age, SM, PA, ED, BMI, serum CL, serum triglycerides	N	
15	2004 Tuomilehto et al. (37)	14,629 M/W 6,974 M	35-64	12	381 203	NVQ	≤2 1.00 Ref 0.47-1.13	3-4 0.73 0.45-1.05	5-6 0.70 0.40-1.12	7-9 0.67 0.25-0.81	≥10 0.45	0.12	Age, study year, BMI, SBP, ED, occupational PA, commuting PA, leisure-time PA, SM, AL, tea drinking	Y
		7,655 W			178		≤2 1.00 Ref 0.48-1.05	3-4 0.71 0.25-0.60	5-6 0.39 0.20-0.74	7-9 0.21 0.06-0.69	>10 0.21	<0.001	Age, study year, BMI, SBP, ED, occupational PA, commuting PA, leisure-time PA, SM, AL, tea drinking	Y

¹ Ascertainment of cases was carried out using the following procedures:

Study 1: death certificates which mentioned diabetes as a contributing or underlying cause of death reviewed by a medically trained clerk;

Studies 2, 3, and 13: the definition of diabetes by the National Diabetes Data Group (38) and/or the World Health Organization (WHO) (39) [1] 1 or more classic symptoms (thirst, polyuria, weight loss, pruritus), plus a fasting plasma glucose concentration of ≥ 7.8 mmol/L (140 mg/dL) or a random plasma glucose concentration of ≥ 11.1 mmol/L (200 mg/dL); 2) at least 2 elevated plasma glucose concentrations on different occasions (fasting ≥ 7.8 mmol/L (140 mg/dL) and/or random ≥ 11.1 mmol/L (200 mg/dL) and/or ≥ 11.1 mmol/L (200 mg/dL) after 2 h or more on oral glucose tolerance testing) in the absence of symptoms; or 3) treatment with hypoglycemic medication (insulin or oral hypoglycemic agents), which was confirmed by a validated questionnaire that was mailed every 2 y. In a study by Lopez-Ridaura et al. (14), however, the new guideline from the American Diabetes Association (ADA) (40) (fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL) was incorporated in 1997;

Study 4: presence of any of the following: 1) fasting glucose level of ≥ 7.0 mmol/L (126 mg/dL); 2) nonfasting glucose level of ≥ 11.1 mmol/L (200 mg/dL); 3) current use of diabetic medication (self-report); or 4) a positive response to the question: Has a doctor ever told you that you had diabetes (sugar in the blood)?

Study 5: affirmative response to the following question on one of the follow-up surveys: Since baseline (or respective follow-up), were you diagnosed for the first time by a doctor as having sugar diabetes?

Study 6: at least one or more following criteria met: 1) he or she confirmed that he or she had been told by a doctor that he or she had diabetes during any of the four follow-up contacts; 2) a hospitalization record listed the International Classification of Diseases-9-Clinical modification (ICD-9-CM) code 250 on any one of 10 diagnoses on the hospital discharge sheet; or 3) death certificate included the ICD-9 code 250;

Studies 7 and 12: 2-h 75 g oral glucose tolerance test using the WHO criteria (≥ 11.1 mmol/L (200 mg/dL)) (39);

Study 8: presence of diabetes, use of drugs for diabetes, or use of insulin injections assessed using a self-administered questionnaire;

Study 9: the definition of diabetes by ADA (40) confirmed by a validated questionnaire that was mailed annually;

Studies 10, 11, and 15: a nationwide register of patients receiving drug reimbursement (in Finland, all diabetic persons needing drug therapy are entitled to reimbursement of drug costs);

Study 14: at least one or more following criteria met: 1) a doctor's diagnosis of diabetes according to a questionnaire; 2) fasting plasma glucose level ≥ 7.0 mmol/L (126 mg/dL); or 3) having been hospitalized at least once with a principal or secondary diagnosis of diabetes.

² The intakes of trans fat and cereal fiber, the glycemic load, and the ratio of polyunsaturated fat intake to saturated fat intake were categorized in quintiles. Each woman was assigned a score for each variable on the basis of her quintile of intake (a higher score represented a lower risk), then the four scores were summed, and the total score was categorized into quintiles.

³ A dietary pattern characterized by high consumption of vegetables, legumes, fruit, whole grains, fish, and poultry.

⁴ A dietary pattern characterized by high consumption of red meat, processed meat, refined grains, French fries, high-fat dairy products, sweets and desserts, high-sugar drinks, and eggs.

Abbreviations:

[Study No.] 1, Study among White Seventh-Day Adventists (1960–, USA); 2, Nurses' Health Study (1976–, USA); 3, Health Professional Follow-up Study (1986–, USA); 4, Atherosclerosis Risk in Communities Study (1987–, USA); 5, Iowa Women's Health Study (1986–, USA); 6, First National Health and Nutrition Examination Survey (1971–, USA); 7, Insulin Resistance and Atherosclerosis Study (1992–, USA); 8, Study among randomly selected adults from the civil register (1987–, Netherlands); 9, Women's Health Study (1993–, USA); 10, Finnish Mobile Clinic Health Examination Survey (1966–); 11, Mobile Clinic Health Examination Survey (1973–, Finland); 12, Study among the Pima Indians (1978–, USA); 13, Nurse's Health Study II (1989–, USA); 14, Study among randomly selected women in Göteborg (1979–, Sweden); 15, Study in 2 eastern provinces and a southwestern region in Finland (1982–).

[Subjects] M, men; W, women; BMI, body mass index; VitE, vitamin E; SP, dietary supplement.

[Dietary method] NVQ, not-validated questionnaire; VFFQ, validated food frequency questionnaire; VFFI, validated food frequency interview; 24HR, a single 24-h dietary recall; VQ, validated questionnaire; DH, diet history interview.

[Dietary factor examined] *, energy-adjusted intake; SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; P/S, ratio of polyunsaturated to saturated fatty acid; CHO, carbohydrate; %E, percentage of energy intake; SP, dietary supplement; INCP, insoluble noncellulose polysaccharides; MD, main dish; SW, sandwich; XD, mixed dish.

[Range or median]—, not available; NV, never; AN, almost never; OH, occasionally heavy.

[Multivariate relative risk]—, not available; #, value estimated from figure.

[95% confidence interval] Ref, referent; —, not available; #, value estimated from figure.

[p for trend]—, not available.

[Factors used for adjustment] PA, physical activity; FT, fruit; BMI, body mass index; AL, alcohol consumption; FH, family history of diabetes; IT, intake; VE, vegetable fat; AF, animal fat; EBF, fruit fiber; VDF, vegetable fiber; CDF, cereal fiber; Mg, magnesium; Ca, calcium; K, potassium; E, energy; SM, smoking; SP, dietary supplement; P/S, ratio of polyunsaturated to saturated fatty acid; TF, trans fatty acid; GL, glycemic load; MS, menstrual status; HRT, hormone replacement therapy; P, protein; CL, cholesterol; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid; VG, vegetable; WG, whole grain; HPTS, hypertension; HPC/L, hypercholesterolemia; FFQ, food frequency questionnaire; ED, education; W/H, ratio of waist to hip; FPL, fasting insulin level; FGL, fasting glucose level; DF, dietary fiber; Fe, iron; SBP, systolic blood pressure; GT, glucose tolerance; F, fat; VitC, vitamin C; FT & BR, fruit and berry; OC, oral contraceptive use; GI, glycemic index; CR, caffeine.

[Association] Y, yes; N, no.

study. Another study investigating the effects of antioxidant vitamins indicated the decreased risk of type 2 diabetes by vitamin E, α -tocopherol, γ -tocopherol, δ -tocopherol, β -tocotrienol, carotenoid, and β -cryptoxanthin. The decreased risk of type 2 diabetes was also observed in both studies examining the effect of caffeine.

Table 3 summarizes the results of association of intakes of foods and food groups with the risk of type 2 diabetes. Three studies assessed the effect of grain on the incidence of type 2 diabetes. All three indicated a favorable effect of grain consumption. A beneficial effect of whole grain was also observed in several studies. Furthermore, the risk of type 2 diabetes was positively associated with the ratio of refined to whole grain and inversely associated with several types of whole grain products such as dark bread, whole-grain breakfast cereal, brown rice, wheat germ, bran, and other whole grains in one study. Although the association between meat and type 2 diabetes was investigated in only a few studies, the results appeared to be consistent. Not only did several studies show an increased risk of type 2 diabetes by higher consumption of processed meat, bacon, and hot dogs, but one study showed an increased risk from higher consumption of meat, hamburger, sausage, salami, bologna, and other processed meats. The effects of vegetables, fruits, and beans were also investigated in only a small number of studies, but one study indicated an inverse association of vegetable and fruit consumption to the risk of type 2 diabetes. Additionally, one study investigated the association of nut and peanut butter consumption with the incidence of type 2 diabetes. The beneficial effects of both nuts and peanut butter were observed.

An inverse relation of coffee to the incidence of type 2 diabetes was shown in five out of eight analyses. A beneficial effect of decaffeinated coffee was also observed in several studies. On the other hand, no significant asso-

ciation between tea consumption and type 2 diabetes was indicated in any study. Finally, for dietary pattern, an inverse association between the risk of type 2 diabetes and dietary score (characterized by low intake of trans fatty acid, high intake of cereal fiber, high ratio of polyunsaturated to saturated fatty acid, and low glycemic load) was observed in one study, whereas another indicated a positive association between the risk of type 2 diabetes and the Western pattern score (characterized by high consumption of red meat, processed meat, French fries, high-fat dairy products, refined grains, sweets and desserts, high-sugar drinks, and eggs).

Discussion

In the present paper, we systematically reviewed 15 individual cohort studies (31 articles) on the association between dietary factors and the risk of type 2 diabetes. Because there is no publication of systematic review of cohort studies on diet and type 2 diabetes, this paper may be useful for future research on this area.

It is important to note that the interpretation and application of the findings should be conducted with great caution not only because the results are not necessarily consistent, but also because only a relatively small number of cohort studies are available at present. However, the following findings may be considered as low-to-medium level evidence: for nutrients, decreased risk by vegetable fat, polyunsaturated fatty acid, dietary fiber (particularly cereal fiber), magnesium and caffeine, and increased risk by trans fatty acid, heme iron, glycemic index, and glycemic load; for foods and food groups, decreased risk by grain (particularly whole grain) and coffee, and increased risk by processed meat. In addition, antioxidant vitamins and nuts might reduce the risk of type 2 diabetes. However, all 15 studies reviewed in the present paper were conducted in Western countries; thus, given the large differences of dietary habits among countries and/or societies, it may

Table 2. Summary of association between dietary factors and incidence of type 2 diabetes: energy and nutrients.

Dietary factors examined	Study (n)	Significant positive association (n)		Significant inverse association (n)	
		Highest vs. lowest categories	Trend	Highest vs. lowest categories	Trend
Energy	4				
Protein	2				
Fat	3				
Vegetable fat	6			3	2
Animal fat	6				
Saturated fatty acid	7				
Monounsaturated fatty acid	6				
Polyunsaturated fatty acid	6			1	1
P/S	3			1	1
Trans fatty acid	4	2	2		
Linoleic acid	3				
Oleic acid	1				
α -Linolenic acid	1				
Long-chain n-3 fatty acid	2				
Carbohydrate	5				
Sugar	1				
Starch	2				
Glucose	2	1	1		
Sucrose	3			1	1
Fructose	2	1	1		
Lactose	2				
Maltose	1				
Glycemic index	3	2	2		
Glycemic load	4	2	2		
Dietary fiber	6			2	3
Fruit fiber	5				
Vegetable fiber	5				
Cereal fiber	8			6	5
Legume fiber	1				
Soluble fiber	2				
Insoluble fiber	1			1	1
INCP	1			1	1
Cellulose	1				
Lignin	1				
Cholesterol	2	1	1		
Magnesium (diet only)	8			3	4
Magnesium (diet and supplement)	3			2	2
Potassium	2				
Calcium	2				
Iron	1				
Heme iron	2	2	2		
Heme iron from red meat	1	1	1		
Heme iron from other sources	1				
Non-heme iron	1				
Iron from supplement	1				
Vitamin E	3			1	1
Tocopherol					
α -Tocopherol	1			1	1
β -Tocopherol	1				
γ -Tocopherol	1				1
δ -Tocopherol	1			1	1
Tocotrienol					
α -Tocotrienol	1				
β -Tocotrienol	1				1
γ -Tocotrienol	1				
δ -Tocotrienol	1				
Vitamin C	1				
Carotenoid	1			1	
α -Carotene	1				
β -Carotene	1				
γ -Carotene	1				
Lycopene	1				
β -Cryptoxanthin	1			1	1
Lutein and zeaxanthin	1				
Caffeine	2			2	2

Abbreviations: P/S, ratio of polyunsaturated to saturated fatty acid; INCP, insoluble noncellulose polysaccharides.

Table 3. Summary of association between dietary factors and incidence of type 2 diabetes: food and food group.

Dietary factors examined	Study (n)	Significant positive association (n)		Significant inverse association (n)	
		Highest vs. lowest categories	Trend	Highest vs. lowest categories	Trend
Grain	3			3	3
Whole grain	4			2	3
Rye	1				
Dark bread	1			1	1
Whole-grain breakfast cereal	1			1	1
Popcorn	1				
Cooked oatmeal	1				
Brown rice	1				1
Wheat germ	1				1
Bran	1			1	1
Other whole grain	2			1	1
Refined grain	4				
Refined grain from wheat	1				
Refined/whole grain	1	1	1		
Meat	2	1			
Processed meat	2	2	2		
Bacon	2	2	2		
Hot dogs	2	1	1		
Sausage etc. ¹	2	1	2		
Red meat	1				
Beef or lamb as MD	1				
Pork as MD	1				
Hamburgers	1	1	1		
Beef, pork, or lamb as SW or XD	1				
Fruit and vegetables	3			1	
Vegetables	2				
Fruit	2				
Mature beans	1				
Nuts	1			1	1
Peanut butter	1			1	1
Coffee	8			5	4
Decaffeinated coffee	2				2
Tea	2				
Dietary score ²	1			1	
Prudent pattern score ³	1				
Western pattern score ⁴	1	1	1		

¹ Sausage, salami, bologna, other processed meats.

² The intakes of trans fat and cereal fiber, the glycemic load, and the ratio of polyunsaturated fat intake to saturated fat intake were categorized into quintiles. Each woman was assigned a score for each variable on the basis of her quintile of intake (a higher score represented a lower risk), then the four scores were summed, and the total score was categorized into quintiles.

³ A dietary pattern characterized by high consumption of vegetables, legumes, fruit, whole grains, fish, and poultry.

⁴ A dietary pattern characterized by high consumption of red meat, processed meat, refined grains, French fries, high-fat dairy products, sweets and desserts, high-sugar drinks, and eggs.

Abbreviations: MD, main dish; SW, sandwich; XD, mixed dish.

be inappropriate to extrapolate these findings to people in non-Western countries.

Specific types of dietary fat rather than total fat appeared to play an important role in the development of type 2 diabetes. While no appreciable association between total fat and the risk of type 2 diabetes was observed in any study, several studies found decreased risk with vegetable fat and polyunsaturated fatty acid and increased risk with trans fatty acid. As vegetable fat consists of fats commonly found in non-animal foods

including fruit, vegetable, grains, and nuts, it can be considered that vegetable fat represents a combination of several potentially healthful fat subtypes including polyunsaturated and monounsaturated fatty acids (24). An inverse association between the ratio of polyunsaturated to saturated fatty acid and the risk of type 2 diabetes was also found in one study. Thus, consuming a diet high in unsaturated fats (such as those in natural vegetable oils, nuts, and seeds) and low in saturated fat (such as those in animal products) and trans fat (such

as those in vegetable shortening and hard margarine) could have substantial benefits for type 2 diabetes (4). Because there are some studies neither supporting nor denying this possibility, however, more research must be done on the relation between fat and type 2 diabetes.

Soluble fiber causes the slow absorption and digestion of carbohydrates that leads to a reduced demand for insulin (41), and insoluble fiber shortens the intestinal transit, allowing less time for carbohydrates to be absorbed (42); thus, dietary fiber is one nutrient that may provide protection against type 2 diabetes. A beneficial effect of dietary fiber was found in several studies. In particular, cereal fiber appeared to be more effective for the prevention of type 2 diabetes than fibers derived from other foods. Therefore, it may be recommended to increase the intake of dietary fiber, especially that derived from cereal products, for the prevention of the disease.

Magnesium is an important component of many unprocessed foods including whole grains and nuts (43), and is a cofactor for several enzymes critical for glucose metabolism (44). Several studies found an inverse association between magnesium and type 2 diabetes, while no report indicated an increased risk from magnesium intake. A particularly beneficial effect of magnesium was observed in two large cohort studies where diet was assessed multiple times during follow-up (14); therefore, further research should be undertaken to gather stronger evidence.

The relation between caffeine, which is abundant in coffee, and the risk of type 2 diabetes was investigated in only two studies where a beneficial effect of caffeine was indicated. Caffeine is a strong stimulant of pancreatic beta cells (45), which may be beneficial for people at risk of type 2 diabetes who usually have impaired insulin secretion (46). Additionally, the thermogenic effect of caffeine may increase energy expenditure, which may facilitate weight reduction and maintenance (47). As only a limited number of studies are available at present, further investigation is needed before drawing a firm conclusion.

Type 2 diabetes is a common manifestation of hemochromatosis, a disease of massive iron overload (48); it can be speculated that excessive iron stores may promote insulin resistance and lead to the development of type 2 diabetes (20). Only two studies examined the association between iron and risk of type 2 diabetes, both of which showed that heme iron increased the risk (no association was found between intakes of total iron, non-heme iron, or iron from supplements and the incidence of type 2 diabetes). In one study, however, heme iron intake from red meat was positively associated with the risk of type 2 diabetes while heme iron intake from sources other than red meat was not associated with the risk of the disease; therefore, the association between heme iron and type 2 diabetes may have been confounded by other components of red meat intake (20). Thus, more research is needed to identify heme iron as a risk factor for type 2 diabetes. Moreover, even after establishing heme iron as a risk factor for the dis-

ease, application of this finding to the prevention of type 2 diabetes should be carried out with caution, because insufficient intake of iron (particularly heme iron) is a risk factor for iron deficiency anemia (49).

Oxidative stress may contribute to the pathogenesis of type 2 diabetes by increasing insulin resistance or impairing insulin secretion (50); thus, dietary antioxidants may have a protective effect against the development of the disease by inhibiting the peroxidation chain reactions (32). The relation between antioxidant vitamins and the risk of type 2 diabetes was investigated in only two studies where beneficial effects of several nutrients examined (vitamin E, α -tocopherol, γ -tocopherol, β -tocotrienol, total carotenoid, and β -cryptoxanthin) were found. Unfortunately, the number of subjects examined in these two studies was relatively small ($n=895, 4,304$); therefore, more research is required before drawing a firm conclusion.

Dietary carbohydrate may be associated with the development of type 2 diabetes by affecting blood glucose and insulin concentration (23). However, no study investigating the association between total carbohydrate and the risk of type 2 diabetes found a clear association, while both glycemic index and glycemic load were positively related to the risk of type 2 diabetes in several studies. Additionally, while beliefs that added sugar, primarily sucrose, should be avoided and that naturally occurring sugars should be restricted in the diabetic diet are prevalent (29), the results of two studies on sugar and type 2 diabetes were not consistent, and in fact, only one of the two studies showed that glucose and fructose were positively related and sucrose was inversely related to the risk of type 2 diabetes. Although a firm conclusion should be drawn after the accumulation of more findings, it is suggested that dietary sugar, if consumed within a normal range of quantity, may not be associated with the risk of type 2 diabetes. However, it should be emphasized that only moderate sugar intake should be incorporated within the boundaries of acceptable energy intake in a well-balanced diet because excessive sugar may not only cause an excessive energy intake, but also adversely affect the entire dietary balance (for instance, by reducing fruit consumption and main meals) (29).

Although several studies assessed the effect of energy intake on the risk of type 2 diabetes, no study indicated a clear association. These results may be partly accounted for by the fact that accurate assessment of energy intake is quite challenging in dietary surveys (51). In fact, many studies have indicated that overweight and obesity are the most important risk factor for type 2 diabetes (52, 53); therefore, an excess of energy intake, which may cause a positive imbalance of energy metabolism, can be considered a risk factor of type 2 diabetes.

An inverse association between consumption of grain (particularly whole grain) and the development of type 2 diabetes was observed in several studies (no study found a positive association). Whole-grain products are a good source of several vitamins, minerals, and fiber,

which may contribute to the inverse association between whole-grain intake and the disease (17). Furthermore, one study indicated a positive correlation between the ratio of refined grain to whole grain and the risk of type 2 diabetes. Thus, it can be considered that substituting whole grain for refined grain may reduce the risk of type 2 diabetes.

Five out of eight analyses supported a beneficial effect of coffee on the development of type 2 diabetes, while no analysis found an adverse effect. However, higher consumption of coffee may adversely affect health in other aspects. For instance, some cohort studies have shown an increased risk of coronary heart disease in heavy coffee drinkers (54–56), although in others no increase in risk has been found (57–59). Additionally, the biological mechanism behind the inverse association between coffee and the risk of the disease is unknown (37). Thus, even if a beneficial effect of coffee against type 2 diabetes is established, increased coffee intake should be recommended only with caution.

While findings regarding total meat and red meat were not consistent, higher consumption of processed meat consistently increased the risk of type 2 diabetes in two studies that explored this issue. This positive association was largely independent of not only the intake of nutrients associated with processed meat, but also the Western pattern, which was characterized by high intakes of red and processed meat and which has been associated with diabetes risk. The increased risk by processed meat, therefore, may be due to the components of processed meat such as nitrites and advanced glycation end-products typically administered or developed in processing and preparation (35). However, as only a few studies have investigated this issue, confirmation from other studies is needed.

Nuts are high in nutrients that may decrease the risk of type 2 diabetes such as polyunsaturated fatty acid, dietary fiber, and magnesium, and may be beneficial for glucose and insulin homeostasis (13). Only one study investigated the association between nut consumption and the risk of type 2 diabetes where a beneficial effect of nuts was found. Higher nut consumption may lead to more weight gain because of high fat content, but no association between nut consumption and weight change was found in this study (13). Additionally, although one may speculate that frequent nut consumption results in increased risk of coronary heart disease because of its high fat content, several cohort studies have consistently indicated an inverse association between nuts and the risk of the disease (60–62). The establishment of the beneficial effect of nuts may make it possible to recommend regular nut consumption as a replacement for refined grain products or red or processed meats that may increase the risk of chronic disease (13). Thus, further investigation is warranted.

Fruits, vegetables, and beans are good sources of dietary fiber, minerals such as magnesium, and antioxidant nutrients such as carotenoids, which may lower the risk of developing diabetes (26). Only a small number of studies examined the relation of these foods to

type 2 diabetes, and only one of eight analyses showed a beneficial effect (fruits and vegetables). Since an adequate intake of fruits and vegetables appears to lower the risks of mortality from cancer (63) and cardiovascular disease (64), the relation of fruits and vegetables with type 2 diabetes should be further investigated.

Although many studies have focused on the role of single nutrients, foods, or food groups in disease prevention or promotion, recent evidence suggests that there are health benefits from food patterns that include mixtures of food containing multiple nutrients and nonnutrients (65). While this approach makes it difficult to elucidate the mechanisms through which the diet composition affects a particular health outcome, it does represent a practical approach to making realistic nutrition recommendations for improving health (65). For type 2 diabetes, a diet low in trans fatty acid intake and glycemic load and high in cereal fiber intake and the ratio of polyunsaturated to saturated fatty acid appeared to decrease the risk of type 2 diabetes (in one study), while a diet high in consumption of red meat, processed meat, refined grain, French fries, high-fat dairy products, sweets and desserts, high-sugar drinks, and eggs appeared to increase the risk (in one study).

In the present paper, as the literature search was limited to only one database MEDLINE (PubMed) with a manual check of the reference lists of included papers, articles appearing in the journals that were not included in the MEDLINE and/or written in languages other than English have not been identified. Additionally, research with a negative result may likely be unpublished (publication bias). Furthermore, we examined only 15 individual cohort studies since we considered as research appropriate for the present study only highly reliable studies that included, for example, quantitative dietary assessment, clear definition of the incidence of diabetes, and adjustment using known factors associated with diabetes. Thus, one may speculate that it would have been better to examine a greater number of studies by broadening the inclusion criteria. Given the difficulties of dietary assessment, however, it may not be worth referring to studies in which an insufficiently accurate dietary assessment method is used. From this point of view, the findings from the studies examined here may be as reliable as possible.

It may be interesting to conduct a meta-analysis. However, our review showed that there are relatively a limited number of cohort studies (one to eight studies for each dietary factor). Additionally, a reliable meta-analysis requires the homogeneity of subjects examined and methods used, which has not necessarily been met in the existing research. Thus, we consider that it is too early to conduct a meta-analysis of cohort studies on diet and diabetes.

When comparing and/or summarizing the results of several studies, the differences among studies of subjects examined and methods used should be taken into account. As diabetes is a disease the incidence of which is difficult to define, various methods were used to define the incidence of type 2 diabetes in the 15 studies

examined in the present paper. While oral glucose tolerance testing (≥ 11.1 mmol/L (200 mg/dL)) (study 7) or fasting blood glucose level (≥ 7.0 mmol/L (126 mg/dL)) (studies 4 and 14) was used in the several relatively small studies, three studies in Finland (studies 10, 11, and 15) used a nationwide register because in Finland all diabetic persons needing drug therapy are entitled to reimbursement of drug costs. Other studies used documents such as death certificate (studies 1 and 6) or questionnaire (the definition of NDDG (38) and WHO (39) (≥ 7.8 mmol/L (140 mg/dL)) (studies 2, 3, and 13), the definition of ADA (40) (≥ 7.0 mmol/L (126 mg/dL)) (study 9), or self-reports of symptom and use of drugs (studies 5 and 8)). The questionnaire used in studies 2, 3, and 13 and that used in study 9 showed good validity (97–98% of the self-reported diabetes cases using a questionnaire were confirmed by medical record review), while diabetes tended to be overreported by the questionnaire used in study 5 (only 64% of subjects who reported diabetes in the questionnaire were confirmed as being diabetic by their physician) and the validity of the questionnaire for diabetes was not indicated in study 8. Such differences in the definition and the method used to identify a case of diabetes may have influenced the results on the association between dietary factors and the risk of type 2 diabetes.

Dietary assessment methods also varied among studies. In most of the studies examined, relatively accurate methods such as validated self-administered questionnaire (studies 2, 3, 5, 8, 9, and 13), validated food frequency interview (studies 4 and 7), and diet history interview (study 10) were applied. However, several studies used less accurate methods such as non-validated questionnaire (studies 1, 11, 12, 14, and 15) and one 24-h recall (study 6). The difference in the dietary assessment method used may also have some impact on the results of diet and diabetes association.

As mentioned previously, relatively accurate dietary assessment instruments were applied in many studies examined in the present paper. Unfortunately, the underestimation of energy intake is a serious problem in dietary surveys. In particular, evidence suggests that obese subjects are likely to underestimate energy intake to a large extent (66). If all foods and nutrients were underestimated to the same degree when energy intake was underestimated, however, the solution would be relatively simple because techniques such as energy adjustment should improve food and nutrition intake estimates (67–69). If, on the other hand, certain types of foods and nutrients are selectively underestimated, the methods of energy adjustment cannot solve all the problems (67–69). Actually, several studies where the accuracy of reported energy intake was assessed against the total energy expenditure measured by the doubly labeled water method (the gold standard for measuring energy intake) have found that the percentage of energy intake from fat decreased and the percentage of energy intake from protein increased as energy intake underestimation increased, suggesting selective underestima-

tion of fat intake and selective overestimation of protein intake (70, 71). Additionally, in some studies using not only the total energy expenditure measured by the doubly labeled water method but also using the 24-h urinary nitrogen excretion (the gold standard for protein intake), the magnitude of underestimation of energy intake was greater than that of protein intake, indicating selective underestimation of other energy-yielding nutrients (fat, carbohydrate, and alcohol) (72, 73). Moreover, a similar finding was observed in a study using total energy expenditure estimated by body composition and self-reported physical activity and 24-h urinary nitrogen excretion (74). Although this problem has not been given sufficient attention in nutritional epidemiology (69), selective underestimation of dietary intake may generate misleading conclusions with regard to diet and health relationships. Therefore, although differential underestimation of specific foods and nutrients is quite difficult to investigate, further research on this issue is needed.

Very recently, the associations of diet and nutrition to the risk of type 2 diabetes have been reviewed and summarized (75). In that review, non-starch polysaccharide (which is identical with dietary fiber in practice) was considered to 'probably' decrease the risk of the disease, which was consistent with the finding obtained in the present paper. An apparently different conclusion from ours, however, was drawn regarding saturated fats, which were regarded as a 'probable' risk factor in that review. In the present paper, however, several studies found an increased risk from processed meat, which was not only high in saturated fat but also frequently consumed in normal life. Thus, although it may be difficult to draw a firm conclusion as to whether saturated fat directly influences the development of type 2 diabetes, our finding is similar to the summary of the review in terms of an increased risk from foods high in saturated fat. Although beneficial or adverse effects of several other nutrients/foods are indicated at lower levels of evidence ('possible' or 'insufficient'), some of which are consistent with the findings in the present paper, further research is needed to corroborate the data.

As a result of systematically reviewing relatively reliable cohort studies on the association between dietary factors and the risk of type 2 diabetes, the following findings may be considered as low-to-medium level evidence: for nutrients, decreased risk with vegetable fat, polyunsaturated fatty acid, dietary fiber (particularly cereal fiber), magnesium, and caffeine, and increased risk with trans fatty acid and heme iron; for foods and food groups, decreased risk with grain (particularly whole grain) and coffee, and increased risk with processed meat. However, these conclusions depend on the findings of studies conducted in Western countries, suggesting that the application of the findings to people in other countries may not be appropriate. Additionally, as findings from only a limited number of cohort studies are available, more research with a prospective design is needed particularly from other than Western countries.

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適正摂取量(AI)・許容上限摂取量(UL)の算定に関する
栄養疫学的研究**

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