

Grain Processing and Technology Trends to Be Driven by Health Needs

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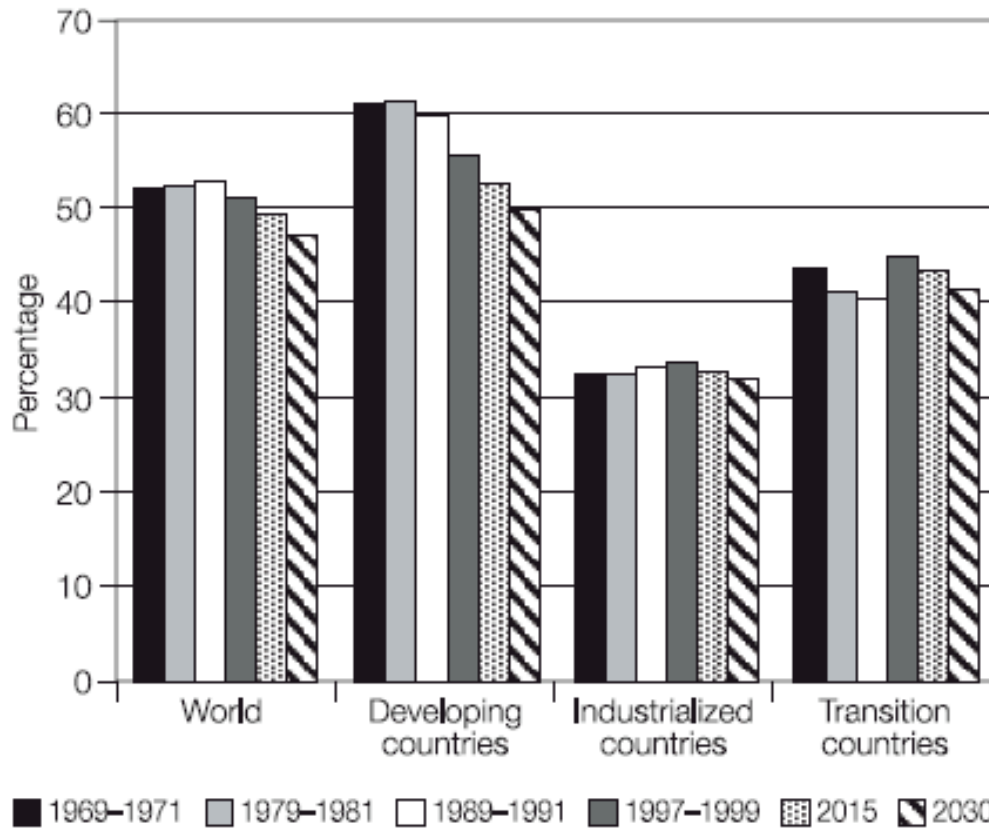


Nutritionally we must eat certain fats and certain amino acids, but our bodies can restructure carbohydrates fairly well.

Why eat grains at all?

Plants are in the first tier of the food web, grains can be made into an enormous variety of good tasting food, and humans can remain primary consumers in the trophosphere.

Figure 1
The share of dietary energy derived from cereals



Source: adapted from reference 4 with the permission of the publisher.

WHO 03.18



We BELIEVE Whole Grains are Good for Us to Eat

“Believe” isn’t enough for scientists.

We need data, even “proof” if possible. We observe populations to see how they have lived. But this is just one time point, we need to see what happens with people over time, for years, how much and what they eat, how much exercise they get, etc. This is why we do longitudinal studies, with large populations, over many years, with multiple detailed observations.



- Whole grains are not magical foods created by someone like the Monkey King to accomplish great tasks and become immortal.
- Whole grains evolved on earth along with humans, we were not satisfied with the taste and texture so we “improved” them.
- We made them into “refined grains”, easy to eat and drink, sweet, and appealing to look at.
- But:
- At the same time, we are getting fat, lazy, and formerly rare diseases are becoming common and chronic.
- Perhaps these things are related.



Obesity and “adult” diabetes are becoming epidemic in children

More than one thing contributes to this problem:

- Not enough exercise
- Too many simple sugars and starches
- We consume more energy than we use - what would happen to your car if you put twice as much gas in it as it could use and most of it could not simply run out? Your car would have to get bigger, with gasoline bulging out all over.



- WHO has voiced strong statements of the problem and what they believe to be answers.
- WHO Technical Report 916 from 2003. Diet, Nutrition and the Prevention of Chronic Diseases

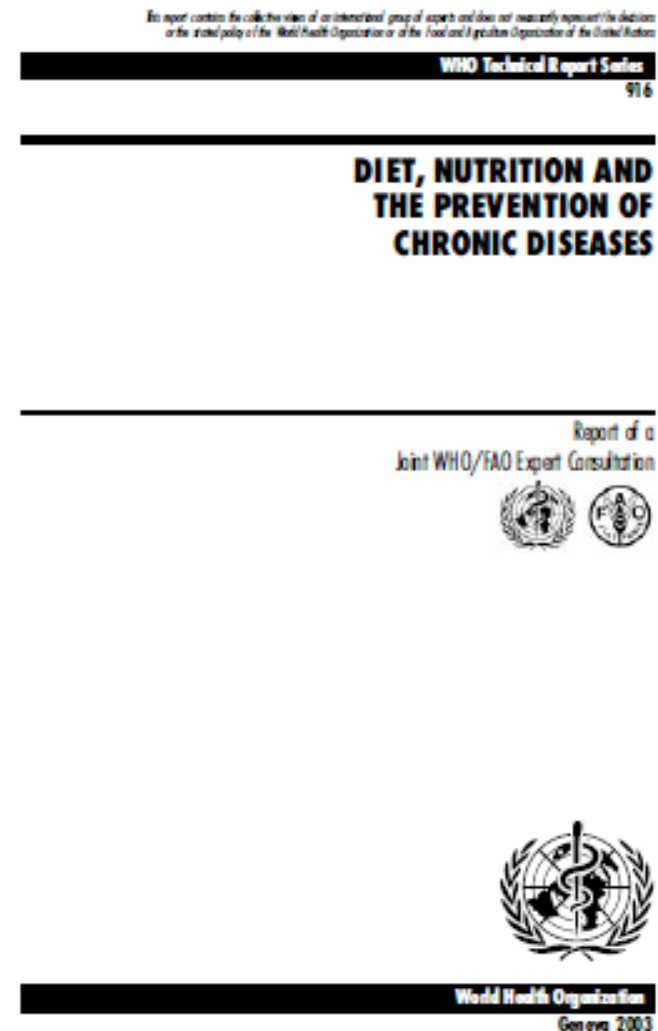


Table 10
Summary of strength of evidence on lifestyle factors and risk of developing cardiovascular diseases

Evidence	Decreased risk	No relationship	Increased risk
Convincing	Regular physical activity	Vitamin E supplements	Myristic and palmitic acids
	Linoleic acid		Trans fatty acids
Probable	Fish and fish oils (EHA and DHA)	Stearic acid	High sodium intake
	Vegetables and fruits (including berries)		Overweight
	Potassium		High alcohol intake (for stroke)
	Low to moderate alcohol intake (for coronary heart disease)		
	α -Linolenic acid		
	Oleic acid		
	NSP		
	Wholegrain cereals		
	Nuts (unsalted)		
	Plant sterols/stanols		
Possible	Folate		Fats rich in lauric acid
	Flavonoids		Impaired fetal nutrition
Inufficient	Soy products		Beta-carotene supplements
	Calcium		Carbohydrates
	Magnesium		Iron
	Vitamin C		

EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; NSP, non-starch polysaccharides.



Table 9

Summary of strength of evidence on lifestyle factors and risk of developing type 2 diabetes

Evidence	Decreased risk	No relationship	Increased risk
Convincing	Voluntary weight loss in overweight and obese people Physical activity		Overweight and obesity Abdominal obesity Physical inactivity Maternal diabetes ^a
Probable	NSP		Saturated fats Intrauterine growth retardation
Possible	n-3 fatty acids Low glycaemic index foods Exclusive breastfeeding ^b		Total fat intake Trans fatty acids
Insufficient	Vitamin E Chromium Magnesium Moderate alcohol		Excess alcohol

¹ NSP, non-starch polysaccharides.

^a Includes gestational diabetes.

^b As a global public health recommendation, infants should be exclusively breastfed for the first six months of life to achieve optimal growth, development and health (59).

Many epidemiological studies have shown the relationship between whole grain and cardiovascular disease.

Table 1. Prospective epidemiologic studies of whole grain-food consumption and atherosclerotic cardiovascular disease

Study	Grain*	Population sample	Gender	Subjects at risk, n	Events, n	Follow-up, y	Findings
Fraser et al [15]	Usually consumed whole wheat [†]	US Seventh Day Adventists	Men and women	31,208	134 nonfatal MI	8	RR = 0.56 for usual consumption of whole wheat rather than white bread, inverse but not significant for CHD death
Pietinen et al [16]	Various grains, including rye bread (eaten whole in Finland)	Finnish smokers in a clinical trial	Men	21,930	635 CHD deaths	6	RR = 0.75 for CHD death for the highest vs lowest quintile of rye bread consumption; not significant for other grain foods.
Rimm et al [17]	Breakfast cereal	US health professionals	Men	43,757	734 incident MI	6	RR = 0.83 for CHD incidence in those who ate any breakfast cereal 2-4 vs little or no times per week
Key et al [18]	Daily wholemeal bread [†]	British health-conscious people	Men and women	10,771	598 CVD deaths	17	RR = 0.86 for CHD death (similar and significant for total mortality); 62% were daily whole grain eaters; added bran did not trend towards reduced risk; sample included vegetarians whose risk is about half the general population
Jacobs et al [19]	Whole grain	low sample from drivers licenses	Women	34,492	438 CHD deaths	9	RR = 0.70 in the highest vs lowest quintile of whole grain intake
Jacobs et al [20]	Whole grain	low sample from drivers licenses	Women	38,740 (includes CHD at entry)	1097 CVD deaths	9	RR = 0.82 for total CVD death for those in the highest vs lowest quintile of whole grain intake; similar reductions were seen for CHD, stroke, and other CVD deaths, although the finding was weakest for stroke
Liu et al [21]	Whole grain	US nurses	Women	75,521	761 incident CHD	10	RR = 0.75 for CHD for the highest vs lowest quintile of whole grain intake
Liu et al [22]	Whole grain	US nurses	Women	75,521	352 cases of incident ischemic stroke	12	RR = 0.69 for ischemic stroke in the highest vs lowest quintile of whole grain intake
Jacobs et al [23]	Whole grain bread	Norwegians sampled in 3 countries	Men and women	33,848	733 CVD deaths	14	RR = 0.77 between high and low whole grain-bread intake scores for both CVD and CHD deaths
Liu et al [24]	Whole grain breakfast cereal	US physicians	Men	86,190	1381 CVD deaths (488 MI and 146 stroke)	6	RR = 0.80 for men who ate whole grain breakfast cereals several times per week or more for total and CVD death, with reductions in CHD death but not stroke.
Mozaffarian et al [25]	Dark bread	US residents aged > 65y without CVD at baseline	Men and women	3688	811 incident CVD	9	RR = 0.76 for dark bread (specific comparison not specified); breakfast cereals unrelated

CHD—coronary heart disease, CVD—cardiovascular disease, MI—myocardial infarction.
 *Dietary information was obtained from various food frequency questionnaires, except as noted.
[†]Based on a single question.
[‡]Based on a single 24-hour recall.



In North America “Diseases of Affluence” are epidemic

In a recent blog posting, Dr. Kevin Patterson discusses his experience as a field surgeon in Afganistan, and his surprise that the patients’ internal organs were not coated in a layer of fat as were those of the citizens of Canada that he treated regularly.

<http://maisonneuve.org/pressroom/article/2010/nov/15/the-diseases-affluence/>



In a recently published longitudinal epidemiological study the issue of internal gut fatty tissue has been studied with respect to whole grains:

AJCN 2010; 92: 1165-71



Whole- and refined-grain intakes are differentially associated with abdominal visceral and subcutaneous adiposity in healthy adults: the Framingham Heart Study^{1–5}
Nicola M McKeown, Lisa M Troy, Paul F Jacques, Udo Hoffmann, Christopher J O'Donnell, and Caroline S Fox



The conclusions included:
consumption of whole grains was correlated with **lower** visceral adipose tissue, but consumption of refined grain was correlated **higher** amounts of adipose tissue. There was a suggestion that refined grain consumption added to a whole grain diet was associated with a reduction in the 'protective' effect of whole grains.

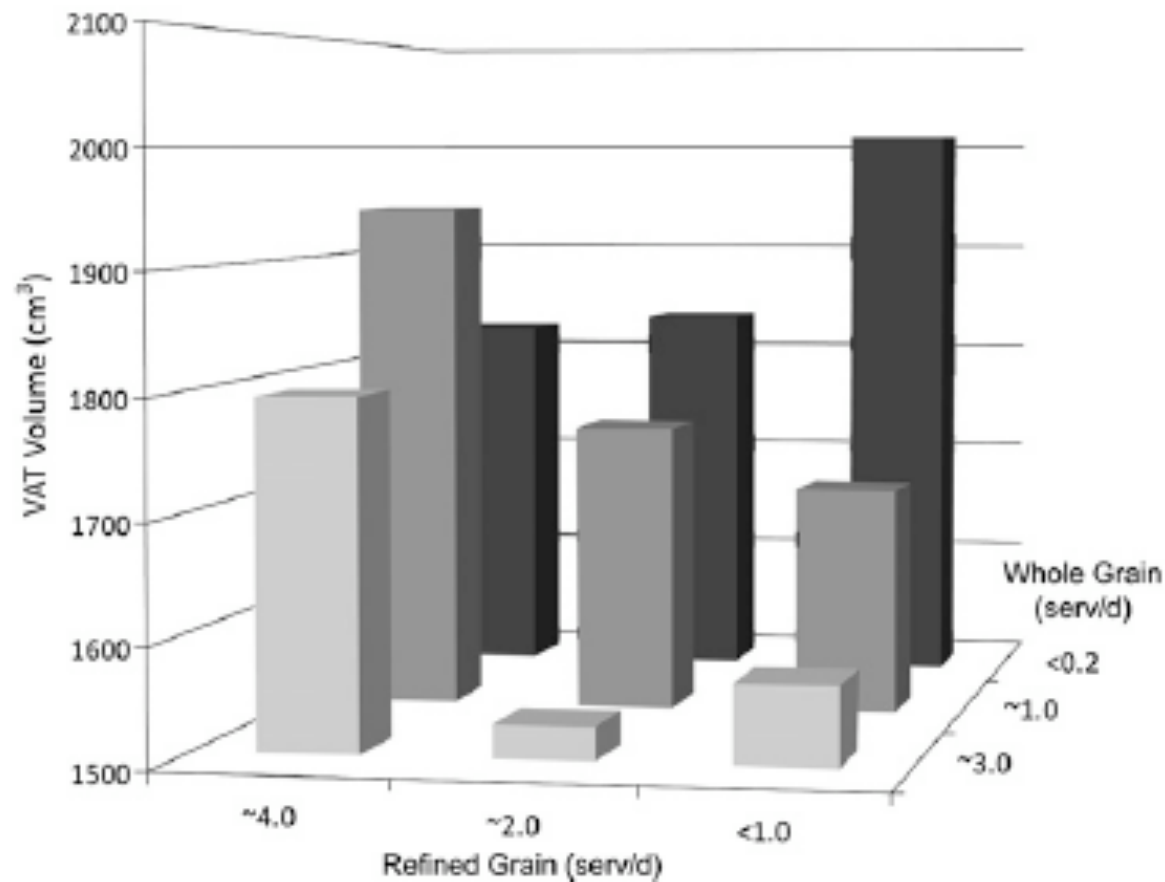


FIGURE 1. Joint classification of whole- and refined-grain intake on visceral adipose tissue (VAT) volume. Multivariate-adjusted VAT was determined according to joint classifications of whole-grain and refined-grain intake. Analyses were adjusted for age, sex, current smoking status, alcohol intake, and total energy intake. (Supplemental Table 3 under “Supplemental data” in the online issue presents the multivariate-adjusted mean \pm SE VAT volumes according to the joint classification of whole- and refined-grain intake.)

TABLE 3

Mean (95% CI) multivariate-adjusted waist circumference measures and abdominal measures of subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) models by whole- and refined-grain intake quintile (Q) categories

	Grain-intake categories					<i>P</i> for trend
	Q1	Q2	Q3	Q4	Q5	
Whole grain						
Waist circumference (cm) ¹	97.0 (95.8, 98.2)	97.6 (96.5, 98.8)	97.2 (96.0, 98.4)	94.2 (93.0, 95.5)	93.7 (92.4, 94.9)	<0.001
SAT (cm ³) ¹	2895 (2772, 3017)	2941 (2819, 3063)	2917 (2793, 3041)	2616 (2490, 2743)	2552 (2422, 2682)	<0.001
SAT (cm ³) ²	2756 (2661, 2852)	2825 (2731, 2920)	2855 (2759, 2952)	2724 (2625, 2822)	2739 (2638, 2841)	0.28
VAT (cm ³) ¹	1883 (1807, 1959)	1861 (1785, 1936)	1808 (1731, 1885)	1642 (1563, 1720)	1563 (1482, 1643)	<0.001
VAT (cm ³) ³	1864 (1805, 1923)	1823 (1765, 1882)	1780 (1720, 1840)	1730 (1669, 1791)	1676 (1614, 1739)	<0.001
Refined grain						
Waist circumference (cm) ¹	95.9 (94.7, 97.2)	95.8 (94.6, 97.0)	95.2 (94.0, 96.4)	96.4 (95.2, 97.6)	97.3 (96.0, 98.6)	0.06
SAT (cm ³) ¹	2748 (2620, 2876)	2743 (2618, 2868)	2728 (2603, 2853)	2862 (2737, 2987)	2934 (2800, 3068)	0.01
SAT (cm ³) ²	2769 (2670, 2868)	2812 (2715, 2909)	2793 (2696, 2890)	2789 (2692, 2886)	2748 (2644, 2852)	0.60
VAT (cm ³) ¹	1727 (1648, 1806)	1680 (1602, 1757)	1684 (1606, 1761)	1818 (1741, 1895)	1928 (1846, 2011)	<0.001
VAT (cm ³) ³	1765 (1703, 1826)	1720 (1660, 1780)	1729 (1669, 1789)	1811 (1752, 1871)	1894 (1830, 1958)	<0.001

¹ Adjusted for age, sex, smoking status, total energy, and alcohol intake.

² Adjusted for covariates listed in footnote 1 with further adjustment for VAT.

³ Adjusted for covariates listed in footnotes 1 and 2 with further adjustment for SAT.



Epidemiological studies have the advantage of being able to be carried out on free living populations, but since they are observational and not experimental, one must be very careful at determining cause and effect.



The English Epidemiologist, Sir Austin Bradford Hill (1897-1991) suggested criteria to consider before concluding that “A” was a cause of “B”



Bradford Hill's Criteria for Causation

- 1. Strength of the association
- 2. Consistency – repeatedly observed, different observers
- 3. Specificity of the association
- 4. Temporality – e.g. cause must precede outcome
- 5. Biological Gradient – dose/response
- 6. Plausibility -



Criteria Continued:

- 7. Coherence – data must fit with existing theory and knowledge
- 8. Experimental evidence
- 9. Analogy – comparison to other, known, relationships.



“About one fifth of the overweight and obese people in the world are Chinese. China was once considered to have one of the leanest populations, but it is fast catching up with the West in terms of the incidence of overweight and obesity; disturbingly this transition has happened in a remarkably short time.”



“Although the prevalence of obesity in China is relatively low compared with Western countries like the United States, where over half of the adults are either overweight or obese, it is the rapid increase of the condition, especially among children that is particularly alarming”



British Medical Journal 2006: 333:
363-4 Dr. Yangfeng Wu, professor and
Chair of the Department of
Epidemiology, Cardiovascular Institute
and Fu Wai Hospital, Chinese Academy
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