

Research **Institute**

Thought leadership from Credit Suisse Research
and the world's foremost experts



Fat: The New
Health Paradigm

Contents

- 03 Introduction
- 04 Summary
- 07 What is fat?
- 09 Fat in our diets
- 12 The price of fat
- 14 How much fat should we eat?
- 24 Medical research: The debate
- 42 The voice of medical professionals
- 44 Consumers' behavior and perceptions
- 50 Regulators and healthcare costs
- 52 The fat trade
- 58 The evolution of fat
- 60 The new paradigm: Fat on the rise

Appendices

- 72 Glossary

Guidelines

References and Further Readings

For more information, please contact:

Richard Kersley, Head Global Thematic and
ESG Research, Credit Suisse
Investment Banking,
richard.kersley@credit-suisse.com

Michael O'Sullivan, Chief Investment
Officer, UK & EMEA, Credit Suisse
Private Banking & Wealth Management,
michael.o'sullivan@credit-suisse.com



Introduction

Healthy living and changing lifestyles have proved powerful investment themes in recent years spanning a wide range of both companies and industries in their reach. Research from the Credit Suisse Research Institute has sought to both enhance the debate and provide our clients with guidance in their investment process.

This study follows on from our 2013 report “Sugar: Consumption at a Crossroads” in examining another key component of nutrition and dietary make-up. We analyze in depth the ecosystem of “fat”, looking at the five types of fats, the main fat-rich foods and who produces them, the medical research on fat and the perception of doctors, consumers and health officials.

Fat is one of the three macronutrients of any diet; protein and carbohydrates are the other two. Over the last fifty years, general nutritional wisdom has been to recommend moderate consumption of fat, lower the intake of saturated fats (butter, lard, milk, red meat, coconut oil) and cholesterol (eggs, poultry, beef) and increase the consumption of polyunsaturated fats (soybean, sunflower, corn, cottonseed oils) and carbohydrates (pasta, bread, sugar, etc).

Fat is a complex topic and these recommendations have been an area of significant debate over the past thirty years. Some believe that these dietary recommendations—closely followed by the U.S. population—are the leading cause of the country’s high obesity levels and the fast growing number of people suffering from metabolic syndrome. Others support maintaining the current “generally accepted principles” with a limit of 10% of daily energy intake from saturated fats and no limits on monounsaturated fats (olive oil, canola oil, palm oil, nuts), polyunsaturated fats or carbohydrates.

Our market surveys show that most consumers’ and doctors’ perception on fat are aligned with the official nutritional recommendations. Yet, some consumers are clearly making new choices. Consumption of butter is growing globally at a rate of 2-4% a year, and in the first half of this year volume sales volume of whole milk in the U.S. grew 11%, while skim milk shrank by 14%. Egg consumption in the U.S. has grown by 2% and organic eggs consumption by 21% in the last twelve months.

We believe that we are at a turning point. Our own analysis and the most recent medical research support these new trends. Medical research has shown that eating cholesterol has basically no influence on the level of cholesterol in the blood or on potential heart diseases. Neither has the link between saturated fat intake and cardiovascular risk ever been proven. On the other hand, a high intake of omega-6 polyunsaturated fats (vegetable oils) has not been proven as beneficial for our health and trans-fats have been shown to have negative health effects. The higher intake of vegetable oils and the increase in carbohydrate consumption in the last 30-40 years are the two leading factors behind the high rates of obesity and metabolic syndrome in the U.S. Saturated and monounsaturated fats are not.

The conclusion of this report is simple. Natural unprocessed fats are healthy and key to the evolution of a society that focuses on developing healthy individuals, not just on treating those who are sick. Natural foods high in monounsaturated and saturated fats are one of the preferred sources of energy for our bodies to use and store. Omega-3 has strong protective properties for our heart and brain. Welcome to the new world of fat.

Stefano Natella

Global Head of Equity Research, Investment Banking

Giles Keating

Vice Chairman of IS&R and Deputy Global Chief Investment Officer, Private Banking & Wealth Management

Summary

We have spent almost a year looking into this complex topic: fat as a nutrient and as an important component of any diet. We leveraged our internal resources, we reviewed over 400 medical research papers and books, accessed global databases on food consumption, and consulted several academics and industry experts. What are main conclusions of our analysis?

- Fat consumption has increased globally over the past fifty years from 19.5% of total energy intake in 1961 to 26% in 2011. We expect this trend not only to continue but to accelerate over the next fifteen years.
- Triangulating several topics such as anthropology, breast feeding, evolution of primates, height trends in the human population, or energy needs of our various vital organs, we have concluded that natural fat consumption is lower than “ideal” and if anything could increase safely well beyond current levels.
- The 1960s brought a major change in the perception of fat in the world and particularly in the U.S., where saturated fat was blamed for being the main cause behind an epidemic of heart attacks. We will see that it was not saturated fat that caused the epidemic as its consumption declined between 1930 and 1960. Smoking and alcohol were far more likely factors behind the heart attack epidemic.
- Saturated fat has not been a driver of obesity: fat does not make you fat. At current levels of consumption the most likely culprit behind growing obesity level of the world population is carbohydrates. A second potential factor is solvent-extracted vegetable oils (canola, corn oil, soybean oil, sunflower oil, cottonseed oil). Globally consumption per capita of these oils increased by 214% between 1961 and 2011 and 169% in the U.S. Increased calories intake—if we use the U.S. as an example—played a role, but please note that carbohydrates and vegetable oils accounted for over 90% of the increase in calorie intake in this period.
- A proper review of the so called “fat paradoxes” (France, Israel and Japan) suggests that saturated fats are actually healthy and omega-6 fats, at current levels of consumption in the developed world, are not necessarily so.
- The big concern regarding eating cholesterol-rich foods (e.g. eggs) is completely without foundation.

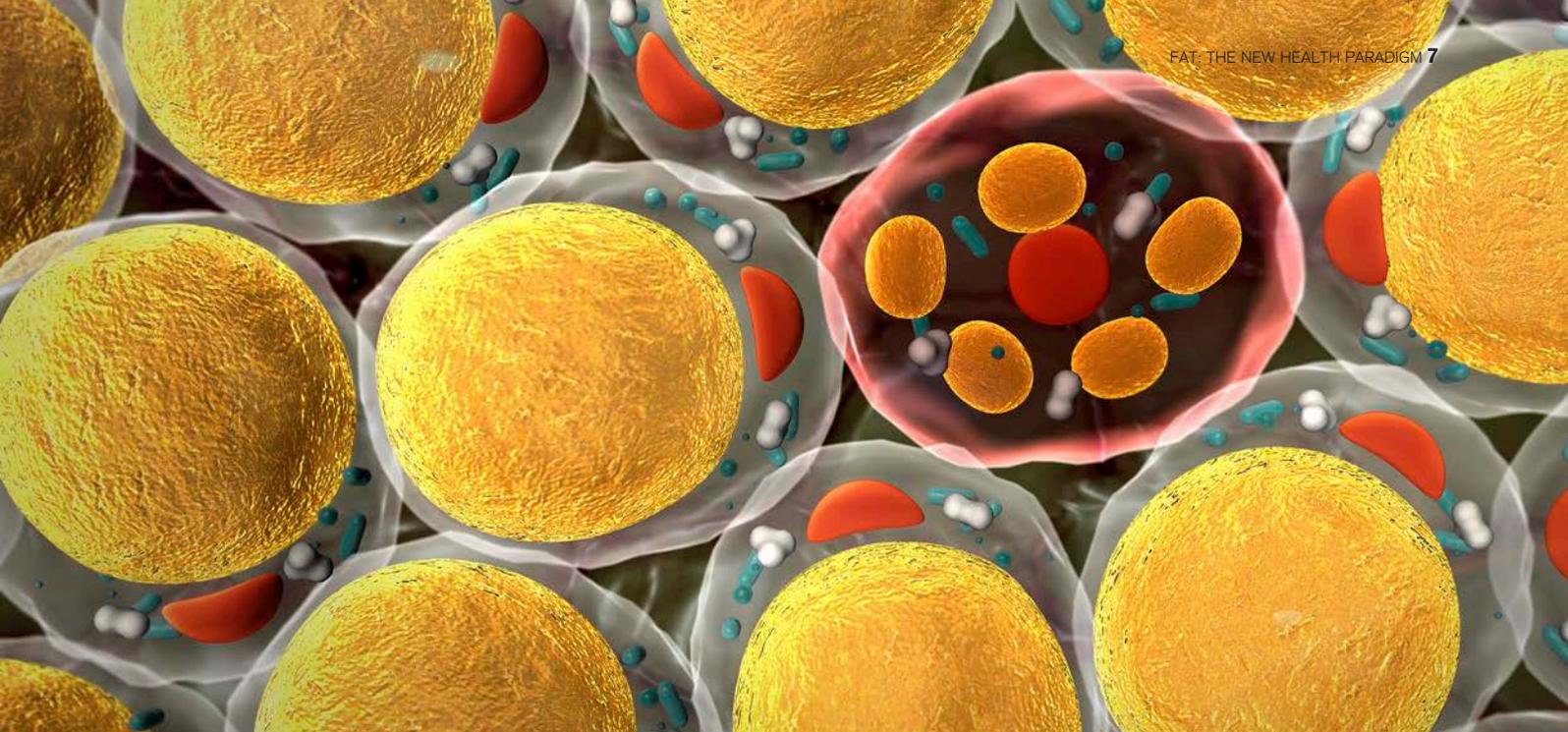
There is basically no link between the cholesterol we eat and the level of cholesterol in our blood. This was already known thirty years ago and has been confirmed time and time again. Eating cholesterol rich foods has no negative effect on health in general or on risk of cardiovascular diseases (CVDs), in particular.

- Doctors and patients’ focus on “bad” and “good” cholesterol is superficial at best and most likely misleading. The most mentioned factors that doctors use to assess the risk of CVDs—total blood cholesterol (TC) and LDL cholesterol (the “bad” cholesterol)—are poor indicators of CVD risk. In women in particular, TC has zero predictive value if we look at all causes of death. Low blood cholesterol in men could be as bad as very high cholesterol. The best indicators are the size of LDL particles (pattern A or B) and the ratio of TG (triglycerides) to HDL (the “good” cholesterol). A VAP test to check your pattern A/B costs less than \$100 in the U.S., yet few know of its existence.
- Based on medical and our own research we can conclude that the intake of saturated fat (butter, palm and coconut oil and lard) poses no risk to our health and particularly to the heart. In the words of probably the most important epidemiological study published on the subject by Siri-Tarino et al: “There is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD.” Saturated fat is actually a healthy source of energy and it has a positive effect on the pattern A/B.
- The main factor behind a high level of saturated fats in our blood is actually carbohydrates, not the amount of saturated fat we eat. Clinical tri-



als show that a low carbohydrate diet is much more effective in lowering the level of saturated fat in our blood than a low-fat diet.

- The same cannot be said about transfats. Since 1933, research has consistently shown that transfats have negative health effects. After the U.S. ban on transfats, we believe more countries will follow suit. Main substitutes of transfats in the food chain could be palm oil and the oleic versions of some of the solvent-extracted vegetable oils.
- Among the “good” fats we find omega-3 (flaxseed, salmon, fish oil) and monounsaturated fat (olive oil, meat and nuts). Most research on omega-3 consistently shows benefits from additional intake of this fatty acid. Additional intake of 1 gram per day of omega-3 reduces the risk of CVD death by 5-30%. It has been shown to be beneficial also in lowering the risk of mental illnesses such as Alzheimer or dementia.
- The research on monounsaturated fat has in most cases been carried out as research on the so called “Mediterranean diet” which for years has been touted as one of the reasons for lower CVD death rates in Southern Europe. It is, however, pretty difficult to define what a “Mediterranean diet” actually is. Our view based also on our own research is that monounsaturated fatty acids are neutral from a health point of view. There is little to support that nuts and olive oil help reduce the risk of CVD deaths.
- In our view, the most controversial area in the research on the health implications of fat intake is polyunsaturated omega-6 fatty acids. Per capita consumption of omega-6 fatty acids has increased by 89% over the past fifty years driven by the wider adoption of solvent-extracted vegetable oils and transfats by the food industry. On the positive side, intake of omega-6 (replacing carbohydrates or saturated fat) lowers both total cholesterol and the “bad” LDL cholesterol. As stated previously, neither is a good indicator of CVD risk. Among fifteen studies looking directly at the effects on actual CVD deaths only a couple show that there is a small statistically relevant positive benefit—20% reduction in CVD death risk—linked to an increase of omega-6 consumption replacing carbohydrates or saturated fat. Most studies though were carried out increasing both omega-6 and omega-3, so it is not clear which was responsible for the positive effects.
- Clinical studies that increased only omega-6 are old, but they show that a high intake (of 10-15% of total energy) could increase CVD events and deaths by similar percentages. Our own analysis carried out on a group of 22 European countries shows that there is actually a positive correlation between the increase in omega-6 intake and the level of CVD deaths. More research on the topic is therefore needed before anybody can assess the full impact of a high level of omega-6 intake on health.
- We conducted two proprietary surveys of doctors, nutritionist and consumers to understand better their perception of the issues we mentioned previously. All three groups showed superficial knowledge on the potential benefits or risks of increased fat consumption. Their views are influenced significantly more by public health bodies or by WHO and AHA rather than by medical research. Even on the “easy” topic of cholesterol, 40% of nutritionist and 70% of the general practitioners we surveyed still believe that eating cholesterol-rich foods is bad for your heart.
- Consumers show a positive perception of fish, nuts, chicken, eggs, yogurt and milk as sources of fat and a negative view of beef, pork, cheese, margarine and butter. They are neutral to positive on vegetable oils in general, and olive oil commands a very positive “healthy” image.
- Health care officials and government bodies have been consistently behind developments on the research front. Research showed that transfats were quite unhealthy as early as 1993, yet a full ban of transfats in the U.S. will only happen in 2018, while in Europe only Switzerland and Denmark have so far banned them. The stance of most officials and influential organizations such as WHO or AHA is now well behind research in two main areas: saturated fats and polyunsaturated omega-6 fats. Our view is that saturated fats intake is at worst neutral for CVD risks and the current 10% upper limit should be lifted. We would also expect a review at some point of the neutral stance on carbohydrates; carbohydrates are one if not the major cause behind the fast growth of metabolic syndrome cases in the U.S.—4% a year—which includes type 2 diabetes and obesity.
- What is the outlook? Globally, we expect fat to grow from the current 26% of calorie intake to 31% by 2030, with saturated fat growing the fastest and going from 9.4% of total energy intake to 13%. This implies that fat consumption per capita will grow 1.3% a year over the next fifteen years versus a rate of 0.9% over the last fifty years. We expect saturated fat to grow at 2% a year versus a historical rate of 0.6% a year; monounsaturated at 1.3% a year versus 1.0%; polyunsaturated omega-6 to decline 0.2% a year versus a 1.3% past growth rate and polyunsaturated omega-3 to grow at 0.7% a year versus 1.6% a year over the last 50 years.
- Among foods, the main winners are likely to be eggs, milk and dairy products (cheese, yogurt and butter) and nuts with annual rates of growth around 2.5-4%. The losers are likely to be wheat and maize and to a lesser extent solvent-extracted vegetable oils. Meat consumption per capita should grow at 1.4% a year and fish at 1.6% supported by a fast expanding aquaculture industry.



What is fat?

Macronutrients are the fuel that allows our bodies to produce energy. Fat is one of three macronutrients in our diet, together with carbohydrates and protein, and is widely present in many of the foods we eat. It is the most efficient macronutrient we can eat and is easily absorbed and stored in our body for future energy needs.

Technically, fats are compounds of long chain organic acids made of carbon and hydrogen atoms and called fatty acids. Those with no double bonds between carbons are called saturated and tend to have a solid state; those with one or more double bonds between carbons are called unsaturated and tend to be liquid.

Unfortunately, over the last century the word 'fat' has developed a negative connotation, particularly in the Western world. It can be used to qualify some of the food we eat, to point to excessive weight in some parts of our body (i.e., having a "fat belly") and to identify overweight people. The same has not happened with the word 'protein' or the word 'carbohydrate'. We do not call a person "protein" and we do not qualify food as "this looks too proteinic." Yet we do not refer to someone who is muscular as very "protein" or "proteinic."

Figure 1

Most representative foods by main type of fat

Saturated		Monounsaturated	Polyunsaturated		Transfats*
			Omega-6	Omega-3	
Butter	Bread	Nuts	Soybean oil		Shortening
Coconut Oil	Lard	Olive Oil	Cottonseed oil		Margarine
Palm Kernel	Beef	Canola Oil			
	Pork	Sunflower oil			
	Sheep	Peanuts			
Cheese	Palm Oil	Fish		Flaxseed	
Milk	Chicken			Salmon	
Yogurt	Eggs				
	Fish				

Source: Credit Suisse

* Containing some level of trans fats.

If we look at fat purely as food, the main perception of the western world is that “fat makes you fat.” Yet the fat content in our body does not depend on just eating fat. We turn dietary carbohydrates into body fat and occasionally we do the same with dietary protein. But as fat has become a negative attribute of physical appearance (in a way that it was not at, say, the time of Rubens...), fat-rich food has come to be perceived as unhealthy.

For the purposes of this paper, we divide dietary fats into five main groups (see Figure 1): saturated fats (dairy, eggs, meat, etc.); monounsaturated fats (olive oil, lards, palm oil, duck fat); polyunsaturated omega-6s (sunflower oil, soybean oil, cottonseed oil, etc.); polyunsaturated omega-3s (fish oil, flaxseed) and hydrogenated oils or transfats (margarine, shortenings). In nature, no food contains only one type of fat. In Figure 2, we list the fat content of the main foods we eat and we break down the fat content into the contribution from each of the five groups above.

In addition, determining the precise fat content of beef, for example, is not an easy exercise, as the fat content varies according to the breed of cow we choose (Aberdeen, Jersey, etc.) and to what part of the animal we are considering. Finally, the content of omega-3 or omega-6 in beef meat is a function of what the cow has been eating. Meat from grass-fed cows contains 88mg of omega-3 per 100g compared with less than 60mg for grain fed cows.

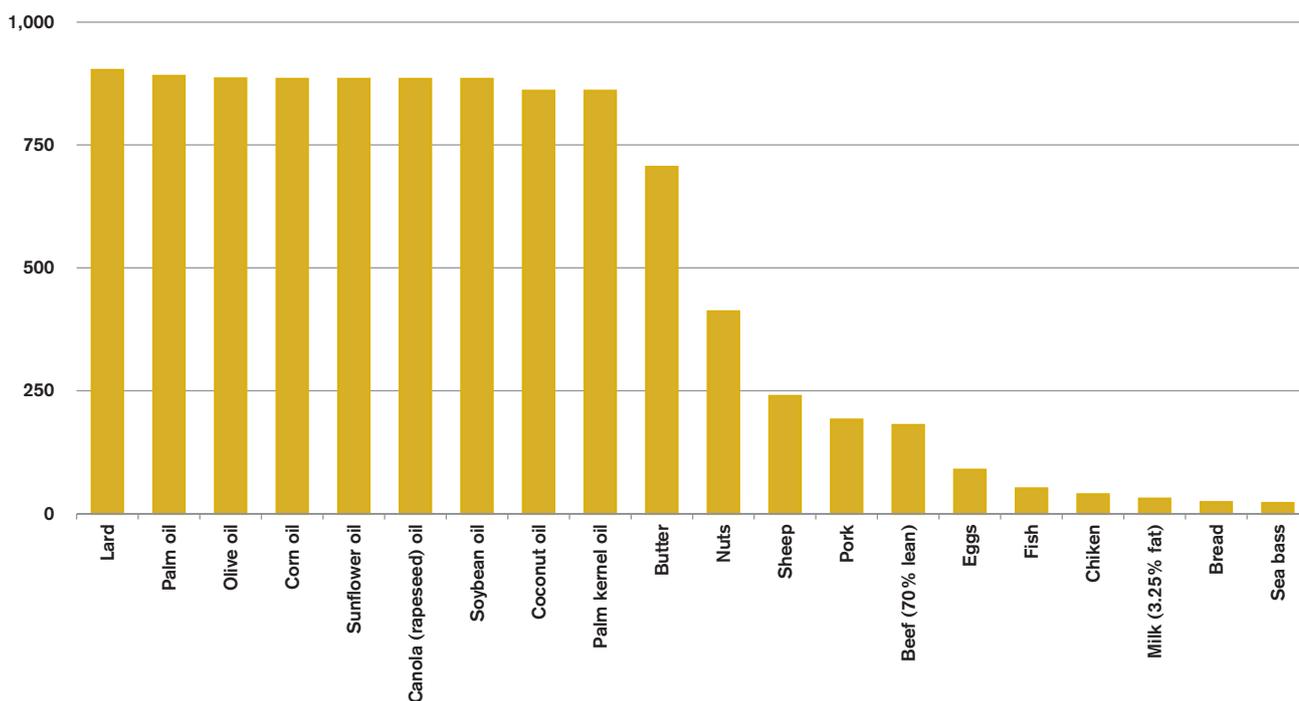
In our research, we will also focus on a particular dietary fat that has captured the attention of doctors, heart patients, consumers and food manufacturers: cholesterol. Cholesterol is a lipid molecule that is biosynthesized by all animal cells. In other words, our body can produce cholesterol. It is a key structural component of the cell membrane of any animal and is required to maintain the membrane’s structural integrity and fluidity. A person with an average weight carries 35 grams of cholesterol in the body, or 0.05% of body mass.

If we try to apply this to a population or a country, the exercise is even more complex. In the field of nutrition, one of the most difficult things is measuring accurately a population’s daily calorie intake or what percentage of total energy intake comes from fat, protein or carbohydrates. Surveys like the U.S. NHANES or NDNS in the U.K. that extrapolate data from interviews with a few thousand individuals to the rest of a population tend to underestimate calorie consumption—people’s perception of how much they eat tends to underestimate how much they really eat.



Figure 2

Caloric fat content – basic foods



Source: Nutrition data.



Fat in our diets

Fat is an essential part of our diet, but it is not easy to determine precisely how much fat or protein a person ingests daily. Nobody eats foods that are made purely of fat or of protein. Most foods contain fat, protein and carbohydrates in varying proportions. To quantify the proportion of fat, protein and carbohydrates we eat, we have to take each food we eat and break it up into these three components. Below you can see the typical composition of several of the main foods we eat.

Figure 3

Common foods – percent of calories from each macronutrient

	From carbohydrates	From fat	From protein
Apples (raw, with skin)	96%	3%	2%
Bananas (raw)	93%	3%	4%
Beef (short loin, t-bone steak, USDA choice, broiled)	0%	60%	40%
Bread (white)	78%	11%	11%
Butter	0%	99%	1%
Cheese (cheddar)	1%	72%	26%
Chicken (breast, meat and skin cooked roasted)	0%	36%	64%
Milk (whole)	30%	49%	21%
Salad (vegetable, tossed, without dressing)	77%	4%	19%
Sea bass (cooked dry heat)	0%	19%	81%
Spaghetti, cooked, un-enriched, with added salt	81%	5%	14%
Walnuts	8%	83%	8%

Source: Nutrition Data

On the other hand, measuring calorie or fat intake from food availability data like in the FOA-STAT database tends to overestimate actual consumption as invariably some food gets wasted or is used for secondary applications (e.g. biofuel or meal to feed animals). We compare numbers under the two methods in Figure 4.

However, either method can be extremely helpful in understanding nutritional trends provided we use each on a consistent basis. We cannot compare them, and probably the “true” numbers are somewhere between what the two methods sug-

gest. As we will see later on though, both do however point to the same trend. We will use mostly FAOSTAT data as that allows us to move across regions and countries; occasionally we will focus on national surveys. The only two caveats using FAOSTAT data to compare what happened over the last fifty years is that the most recent numbers (2011) on total calories and fat consumption are probably overstated relative to those collected fifty years ago. Two main reasons:

- Food waste in developed markets has increased over time as families have had access to “excess” food; in developing markets the opposite might be true as the

Figure 4

Comparison of FAOSTAT and National Survey interviews

Country	Year	National Survey/ interviews fat/energy %	FAOSTAT fat/energy %	Difference %	National Survey/ interviews Total calories	FAOSTAT Total calories	Difference calories
Australia	1998	32.5%	37.2%	4.7%	2,207.7	3,007.0	799.3
China	2003	20.0	25.2	5.2	2,051.4	2,833.0	781.6
France	2004	37.2	41.0	3.8	2,176.9	3,555.0	1,378.1
Germany	2004	35.9	36.6	0.7	2,459.8	3,411.0	951.2
India	2006	22.5	19.0	(3.5)	2,605.9	2,334.0	(271.9)
Italy	2000	35.0	37.7	2.7	2,459.8	3,670.0	1,210.2
Japan	2004	25.3	28.5	3.2	NA	2,843.0	NA
Spain	2005	38.5	40.7	2.2	2,185.0	3,303.0	1,118.0
Sweden	2005	34.0	35.9	1.9	2,125.0	3,129.0	1,004.0
U.K.*	2012	32.9	36.4	3.5	1,802.8	3,414.0	1,611.2
U.S.	2010	34.0	39.6	5.6	2,127.9	3,659.0	1,531.1
Average (ex India)		32.5	35.9	3.4	2,177.4	3,282.4	1,153.9

* Latest UK Faostat data available is 2011

Source: FAOSTAT, Harika, et al. A Systematic Review of Data from 40 Countries. Ann Nutr Metab 2013

wider adoption of refrigerators has reduced food spoilage.

- Several fatty foods like meat, milk or yogurt had a higher fat content fifty years ago. For example, a 100 gr cut of meat now has just 10-15 gr of fat compared to 20-25 gr fifty years ago. At current consumption levels in the U.S., for example, that translates into a difference of 90 calories on a daily basis.

So let us look at the data. FAOSTAT shows that global fat consumption as a percentage of energy stood at 26% in 2011, protein consumption stood at 11% and carbohydrates consumption stood at 60% (alcohol was at 2%). The regional dispersion was quite high: at the low end was Africa with 20% of energy sourced from fat; at the high end North America with 40%. However, NHANES, which is probably a more accurate source of food intake (as FAOSTAT data are not fully adjusted for waste) states that fat consumption in the U.S. was 33% in 2010. This shows how different data collection and processing methods can influence estimates.

A detailed study “Global, regional, and national consumption levels of dietary fats and oils in 1990 and 2010” by Micha et al. run globally but leveraging local data and published in 2014 shows saturated fat consumption at 9.4% globally, omega-6 at 5.9% and omega-3 at 0.5%. If we assume that these data are compatible with FAOSTAT data, we consume 10.2% of our energy in the form of monounsaturated fats.

As fat has been identified for years as a culprit behind the obesity and metabolic syndrome epidemic as well as a significant contributory factor in cardiovascular related deaths, it is worth understanding not only how much fat and what type of fats we consume today, but also how this picture has evolved over the past fifty years.

The bottom 20 countries in fat consumption—as a percentage of total daily caloric intake—tend to be poor countries in Africa and Asia (see Figures 6 and 7), with a range of 11-18%. The top 20 are European countries, as well as the U.S., Canada, Australia and New Zealand, with a range of 39-45%. If you look at just saturated fat, Bangladesh has the lowest intake at 2% and Samoa the highest at 27.5% (mainly due to the consumption of coconut oil); in Europe Romania has the highest at 20%. However, a different database places France at the top with 15.5% and Romania with barely above 10%.

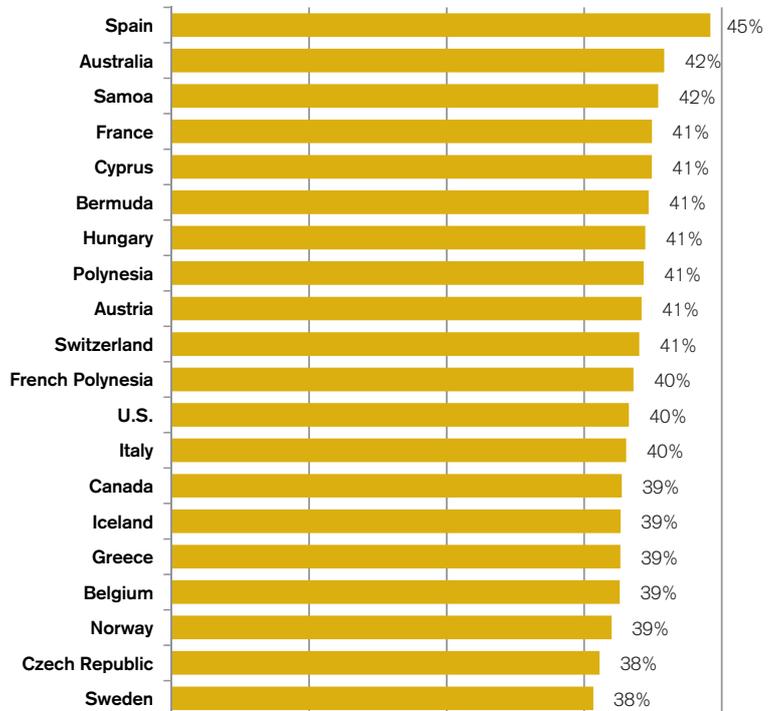
What influences these geographical differences?

1. **Income and changes in income:** The availability and consumption of animal and vegetable fats is closely linked to income. The higher the income, the higher the percentage of fat in the diet. The percentage tends to rise quickly as incomes grow from \$500 to \$10,000 a year. Beyond this, growth flattens, and at incomes above \$25,000 a year the percentage tends to stabilize.
2. **Urbanization and urban lifestyle:** Urbanization drives a higher level of fats in our diet. In Bangladesh for example the consumption of fat in urban areas is eight times higher than in rural areas. Availability and income explain most of this trend.
3. **Climate:** As fat is a very efficient source of heat for our bodies, colder climates tend to show higher levels of fat consumption. For eskimos, fat accounts for over 60% of food intake, for Egyptians just 16%. Hot climates induce lower percentages of energy intake derived from fat.
4. **Local availability and cultural factors:** Butter is a main staple in France as olive oil is in Italy and Greece, coconut in many islands in the Pacific (Samoa, Vanuatu and Sao Tome for example), and palm oil in Malaysia and Indonesia. In the U.S., Brazil and Argentina the availability of vegetable seed that could be processed into oils has certainly been a factor.
5. **Nutrition guidelines and official restrictions.** Since the 1960s, the U.S. health authorities and influential organizations such as the AHA (American Heart Association) have been instrumental in driving down the consumption of fats, particularly saturated fat, on the hypothesis that the levels of fat and saturated fat consumed fifty years ago were bad for our health, particularly for our hearts. We will analyze this connection in detail later, but clearly this drove the consumption of meat, eggs and milk down and that of vegetable oils up. In some countries such as Hungary and Denmark the perceived negative health effects of fat consumption drove the health authorities to impose taxes on foods with saturated fat content above a certain level. In the U.S., food label disclosures require companies to show the level of saturated fat, and foods that contain saturated fats cannot be marketed as "healthy."

Figure 5

Top 20 countries by fat consumption

Proportion of dietary energy supply derived from fat

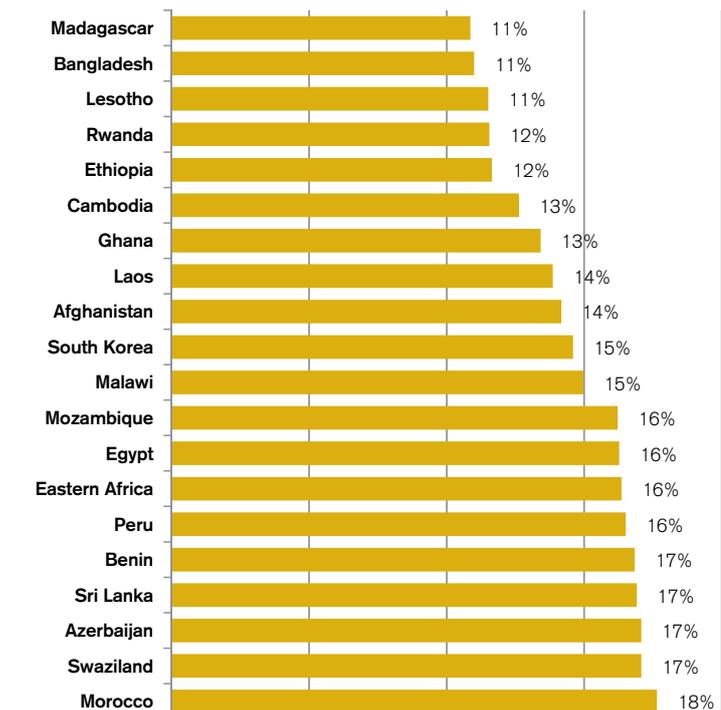


Source: FAOSTAT

Figure 6

Bottom 20 countries by fat consumption

Proportion of dietary energy supply derived from fat



Source: FAOSTAT



The price of fat

Price has been always an important driver of consumers' decisions. This is the case for fat-rich products as well.

As we discussed earlier, what drove this change was a huge ideological crusade—as opposed to scientific—against fat and saturated fat, led by health officials and a small group of U.S. medical academics, probably combined with the desire to provide cheaper fat sources to food manufacturers and a fast growing world population. In the new low fat world, the taste and texture provided by saturated fat was in most cases replaced by sugar (e.g., in yogurts, desserts, ice-cream, etc) and vegetable oils (e.g., in biscuits, crackers, etc).

Lowering “fat” prices in the food chain while keeping food “tasty” with less fat or a different fat was a powerful combination. Medical research linking fat and particularly saturated fat to cardiovascular deaths added fuel to this change. Let us now analyze the “price driver” behind it.

We took a sample of some of the foods that contribute the most to fat intake in the U.S., grouped them under the “prevalent” type of fat and built a historical price trend per gram of fat dating back to 1985. There are four interesting observations.

1. The price of a gram of fat in the U.S. has increased by 89% since 1985—or 2% a year in nominal terms (compared to a CPI of 3.2% a year). Polyunsaturated fat

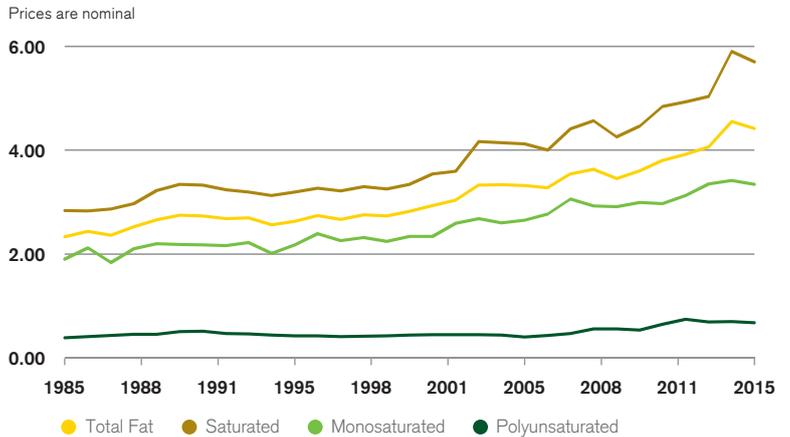
prices rose by 74% or 1.9% a year while saturated fats experienced a 100% increase or 2.4% a year.

2. Polyunsaturated fats—mostly omega-6—have consistently been the cheapest source of fat. In 1985, the price of a gram of polyunsaturated fat was 86% less than that of a gram of saturated fat and 90% less than a gram of monounsaturated fat. At the end of April 2015, the same percentages were 74% less and 89% less. So there is no doubt that this was a major driver of the switch from saturated to polyunsaturated fat.
3. At US\$18 cents per gram of fat at the end of April 2015 red meat was the most expensive source of fat; margarine at US\$0.45 cents was the cheapest. Butter, which is mostly saturated fat, was twice as expensive as margarine.
4. The price-demand elasticity for fat-rich foods is the highest for pork at -0.7% (a 1% increase in price triggers a 0.7% fall in demand), followed by beef (-0.6%) and chicken (-0.4%). Milk is quite inelastic (-0.04%), but butter and cheese are not (-0.25% for both). Finally, eggs show a good profile with a price elasticity of just -0.1%.



Figure 7

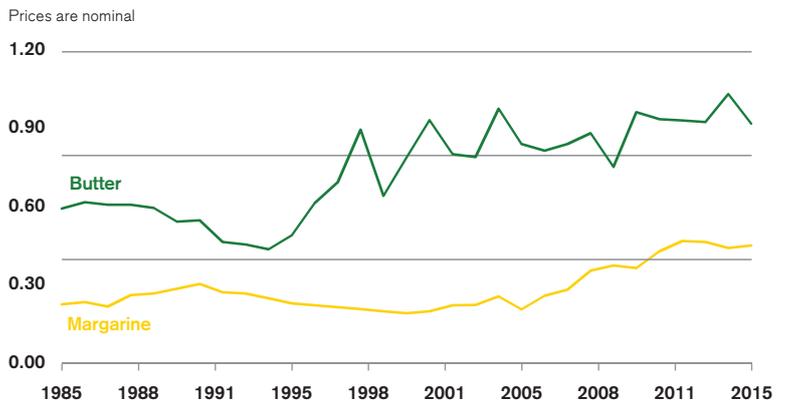
U.S. retail price per gram of fat



Source: U.S. Bureau of Labor Statistics, Nutrition Data, Credit Suisse Research

Figure 8

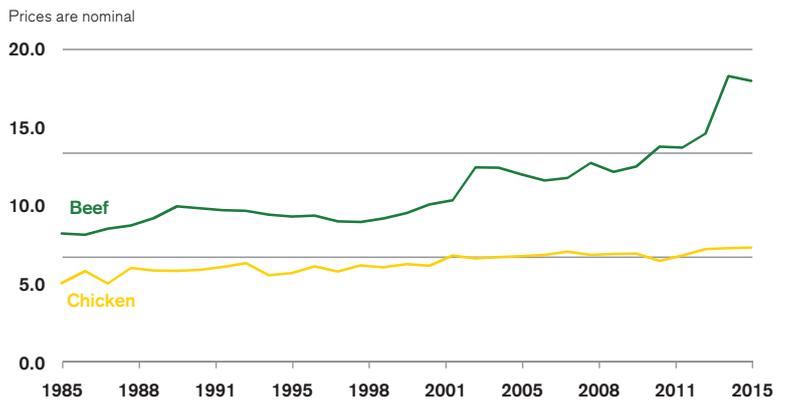
U.S. retail price per gram of fat: butter versus margarine



Source: U.S. Bureau of Labor Statistics, Nutrition Data, Credit Suisse Research

Figure 9

U.S. retail price per gram of fat: beef versus chicken



Source: U.S. Bureau of Labor Statistics, Nutrition Data, Credit Suisse Research

Comparing red meat with chicken and butter with margarine over the past 30 years is interesting. Was it price that drove chicken consumption up massively versus red meat? Was price the driver of the switch between butter and margarine until the trans fats scare hit margarine at the end of the 2000s?

The answer to both questions is “yes.” A combination of price and fear of red meat, as well as of saturated fats. The price of a pound of chicken was 26% lower than that of red meat in 1980; today it is still 43% lower. The price of a pound of margarine was 53% lower than butter in 1985; today it is still 33% lower.

If we look at the last five years—to move beyond the impact of the trans fats scare—we can see that the price of butter has increased 26% in this period—almost 5% a year—while margarine has gone up in price by 19%. Butter is now “trading” at a 50% premium to margarine and sales volumes are up 24% for butter and down 24% for margarine. This is probably due to the trans fats issue rather than a reassessment of saturated fats, but it shows that perceptions can change quickly.

How much fat should we eat?

So far we have analyzed how much and what type of fats we eat. The key question is: how much fat should we consume? Does it vary with age, gender or genetics? Do people in different regions benefit from eating different levels of fat?

We have looked at several different areas of investigative research—anthropology, nutrition, clinical trials, epidemiological research, etc.—to address this question. Contrary to what might be expected, our analysis suggests we now probably consume not enough fat, not enough saturated fat and too much carbohydrates. This is particularly true in the western world. Here are several points to consider:

Evolution

Consumption of fat has changed over the past few years and has certainly changed over the last 3.8 million years since Lucy first walked the earth. Historical data on fat consumption over such a long time frame, as you can imagine, are not easily available. The farther back we go, the less precise we can be. But stable isotope analysis allows us to understand better the diets of a variety of ancient hominid species by looking at their fossilized teeth.

This analysis indicates that the human diet evolved from purely tubers and roots to adding meat around two million years ago and making it the main food staple up to 10-12,000 years ago. Research shows that for the hunter-gatherers of the upper Paleolithic period, fat was likely to account for at least 40-60% of daily calories, followed by protein at say 20-30% and carbohydrates from tubers and plants at 10-20%. This changed 8-10,000 years ago in the Neolithic period with the introduction of cereals, which over time replaced fat and protein to become the main energy source in several populations.

Part of this change was driven by the beginning of a more stable agricultural culture; more recently—in the past 50 years—the increase in carbohydrate consumption has been driven further by medical research, new food guidelines introduced in the 1960s and new product lines developed by food manufacturers.

Anthropologists (Holt, Formicola and Anagnostis among others) have concluded that the skeletons and teeth of humans in the period up to 15-20,000 years ago were much healthier—taller and with less tooth decay—than those following the onset of the Neolithic period. This has led some to conclude that the diet of the Paleo-

lithic period with 40-60% of energy from fat was much healthier—at least in terms of teeth, bone health and height—than that of the Neolithic period that relied much more on cereals.

The tall man concept

We could apply the same idea to more recent times. During the 20th century, many European parents held up the U.S. as an example of what their children should aspire to on the nutrition front. The notion was that milk and meat made Americans grow tall and healthy.

There are a few excellent research papers on the topic (one of the most interesting is by Komlos, Lauderdale in 2007). At the end of the 19th century, Americans were the tallest population in the world. By the 21st century, northern Europeans (the Netherlands, Denmark and Norway) had become the tallest, and Americans were left with being the most obese and least healthy among the most affluent populations in the world (see the 2013 report commissioned by the NIH to the National Research Council and the Institute of Medicine: “U.S. Health in an International Perspective: Shorter Lives, Poorer Health”) Data from NHANES show that the average heights of white males and females in the U.S. have stayed basically the same since 1935. Part of this might reflect the Depression and World War II, but it also appears inconsistent with the sharp increase in income and medical progress in the period. Diet might help explain this. Interestingly, butter, milk, and meat consumption in the U.S began to decline in the early 1900s and were replaced first by more vegetable oils and trans-fats and later by more carbohydrates. Note that the northern European countries have a diet that is more fat-rich with fewer carbohydrates than the current U.S. diet, with a strong emphasis on fish, which is rich in omega-3.

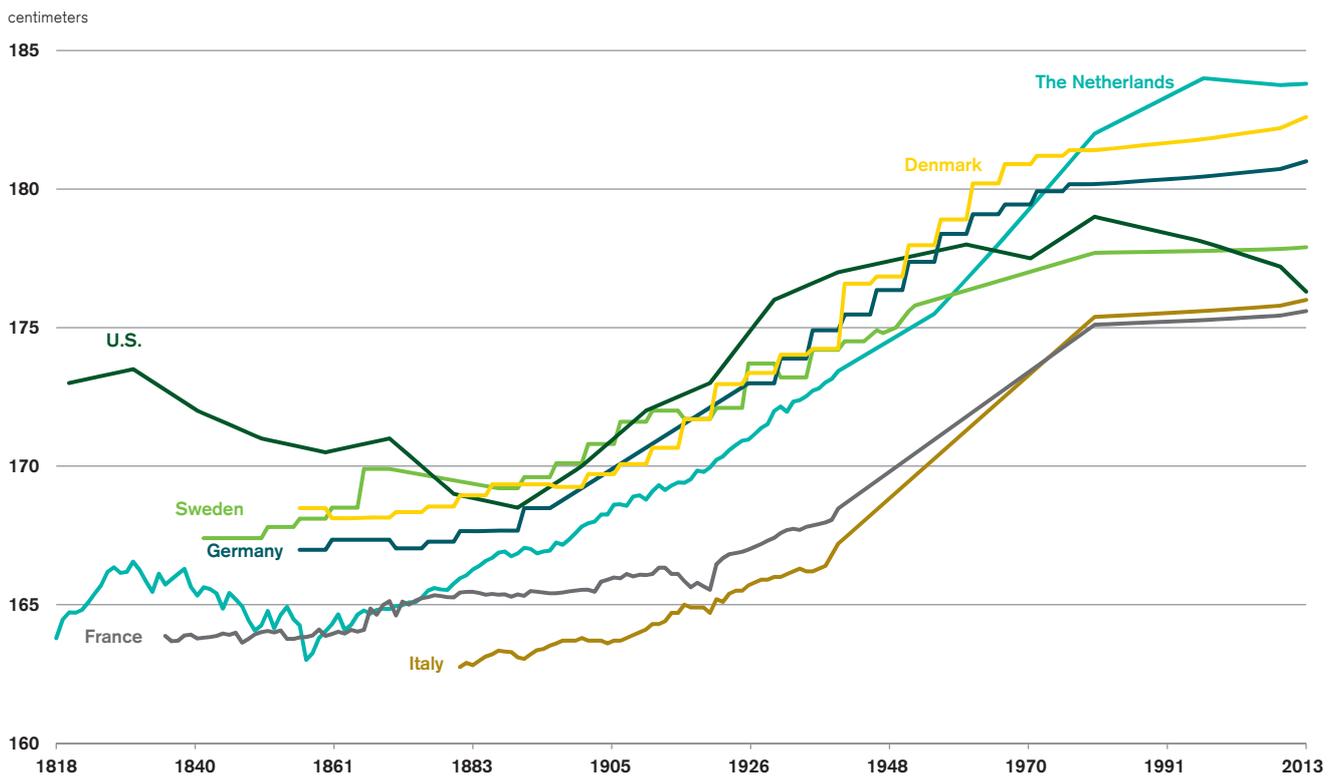




PHOTO: ISTOCKPHOTO.COM/NEHOPELON

Figure 10

Median male height in various countries from 1820-2013



Source: Olson, Randy (2014): Historical median heights for various countries, 1818-2013.

<http://dx.doi.org/10.6084/m9.figshare.1066523>

Animal lessons

One advantage of looking at animals to explore what is the right amount of fat we should eat is that animals are not influenced by ads or books on what are the best diets or the best food. This is not necessarily true for pets, so we will focus on wild animals. One disadvantage of looking at animals is that they are not 100% like us, so we need to be careful with parallels. Focusing on mammals, the Jaminets in their book, *The Perfect Healthy Diet*, analyze differences and similarities in detail.

Different mammals have different diets: cows and sheep eat grass, lions and wolves just meat. So, what do they have in common? Their ability to convert what they eat into energy their bodies can use. Vegetarian species tend to be ruminants like cows —fermenting the plants they eat, early in their digestive track—or gorillas, which

ferment fiber in the colon. Fibers and carbohydrate-rich plants get converted by both mostly into short chain fatty acids—saturated and monounsaturated fat—through the fermentation process promoted by bacteria. The food is fiber, but the resulting nutrient is mostly fat and some carbohydrates: 60-70% fat, 5-20% carbohydrates.

What about carnivores like wolves? Don't they just eat protein? Meat contains both fat and protein. 100 grams of deer meat contains 24 grams of protein and 1.4 grams of fat (90/10 protein/fat), but the liver of the deer is 42%/58% protein/fat in terms of calories. Alpha wolves, which eat first in the pack, eat the offal. Lean meat is for omega wolves.

For humans it is difficult to generalize, as we each have very different diets. But our colon is 20% of total gut volume versus 53% for primates. So we are not equipped to process large amounts of fiber and convert it into fat. We are actually more efficient. Aiello and Wheeler in 1995 suggested that humans were able to fuel their larger brains with a relatively small gut because increased dietary quality reduced the need for gut mass. With less need to chew foliage, our teeth and jaws became smaller and our brain doubled in size. The hypothesis was that the main driver of this increased dietary quality was the increased use of animal products or fat which could be directly absorbed in the small intestine.

So if gorillas or cows get 60-70% of their energy from fat and we are genetically equipped to process fat (rather than convert fibers to fat through fermentation), why should we not eat 60-70% of food in a “fat” form? And should it be animal fat or vegetable fat?

Research suggests that our species traditionally sourced most of the fat intake from animals. Several studies of some of the few remaining hunter-gatherer cultures point to two important facts. First, no single hunter-gatherer culture has been found to be just vegetarian. Second, animals provide at least 50% of all their calories. As fat accounts for 60-80% of calories in meat (the balance is protein), these cultures derive at least 30-40% of energy from animal fat.

Breast milk

There is also another interesting parallel across mammals. Babies are fed breast milk for up to 3 years depending on the species. How similar is the nutrient content of breast milk for different mammals? Figure 19 shows that the fat content of breast milk varies from 52% for monkeys to 90% for the gray seal. Humans stand at 56%, with the balance made up of carbohydrates at 38% (mostly lactose) and protein at 6%.

Figure 11

Mammals breast milk content (%)

Species	Fat	Protein	Lactose
Antelope	21%	50%	29%
Bear, polar	87%	13%	1%
Bison	27%	34%	40%
Buffalo, Philippine	70%	18%	13%
Camel	56%	19%	26%
Cat	63%	28%	9%
Cow:			
Ayrshire	53%	21%	27%
Guernsey	56%	19%	25%
Jersey	58%	18%	23%
Deer	77%	18%	5%
Dog	59%	30%	12%
Dolphin	66%	22%	12%
Elephant	80%	12%	8%
Goat	51%	20%	30%
Horse	29%	22%	49%
Human	56%	6%	38%
Kangaroo	43%	57%	0%
Monkey	52%	13%	35%
Pig	64%	20%	17%
Rabbit	69%	26%	5%
Seal, gray	90%	8%	2%
Sheep	54%	25%	21%
Whale	84%	15%	2%

Source: Robert D. Bremel, University of Wisconsin and from Handbook of Milk Composition, by R. G. Jensen, Academic Press, 1995.

Also, if we break down the fat part of human breast milk by its components, we get 45% saturated fat, 38% monounsaturated, 15% omega-6 and 3% omega-3. Note also that breast milk contains a high level of cholesterol: up to 160 mg per liter versus just 100mg per liter for cow milk. This leads to two interesting conclusions. First, in humans 25% of the solids in breast milk is saturated fat. If saturated fat is as bad for you as the current recommended level below 10% would suggest, surely evolution would have taken care of lowering the saturated fat level of breast milk.

Medical research on dairy products support this conclusion. Out of 18 studies published between 2010 and 2013, 8 show that full-fat dairy lowers CVD risk, 9 suggest no effect and only 1 points to a slight increase in CVD risk linked to dairy consumption.

Bottom-up energy needs

Another way to assess what should be the “right” amount of fat in our diets is to derive it from the energy needs of each of our organs.

It has long been conventional medical wisdom that the preferred fuel of the body is carbohydrates. However, advances in biochemistry have demonstrated that the various organs use different fuels, both at rest and during physical activity.

As a measure of resting energy requirement (REE), the brain uses roughly 22% of the body's total energy. Because it is fastidiously protected against pathogens, only small molecules can cross the blood-brain barrier. This means that the brain mostly burns glucose but, as we mentioned before, it also can run happily on ketone bodies (broken down from fat in the liver). Indeed, ketones may be an essential fuel. Breast milk provides babies with a considerable proportion of ketones for the brain, and the use of ketogenic diets has long been clinically successful for treating epilepsy.

Of the other major organs, the liver (21% of REE) and the heart (9% of REE) predominantly burn fatty acids. The heart burns only fat, but the liver is metabolically flexible, often utilizing excess protein.

Of the minor organs, the kidneys require mostly glucose, as do the eyes and red blood cells. The intestines burn a mix of fuel. The small intestine prefers protein, specifically glutamine, which provides the nitrogen necessary for rapid cell turnover. The large intestine prefers short chain fatty acids, which are a byproduct of the bacterial digestion of fiber. Indeed, butyric acid (also found in butter) has been shown to be protective against colon cancer.

Muscle utilizes a mix of fuels. At rest, muscle cells oxidize almost 100% fatty acids, which account for 22% of the body's REE. When exercising, muscle requires faster access to energy and begins to burn carbohydrates, stored as glycogen within the tissue. It also burns some proteins

Figure 12

Energy needs of the major organs

Organ or tissue	Metabolic rate* (kcal/kg/day)	Percent resting energy expenditure	Fuel source (in order of preference)
Adipose	4.5	4%	Fatty Acids
Other including intestine, skin, blood, etc.	12	16	Fatty Acids Glucose Protein
Muscle	13	22	Fatty Acids Glucose Protein
Liver	200	21	Fatty Acids Protein
Brain	240	22	Glucose Ketones
Heart	400	9	Fatty Acids
Kidneys	400	8	Glucose

Source: McClave S, Snider H. Dissecting the energy needs of the body. *Curr Opin Clin Nutr Metab Care* 4:143-147. # 2001)

* Metabolic rate is calculation of body energy expenditure per unit of time

(branch chain amino acids) and in very short bursts phosphocreatine (a molecule that serves as a rapidly mobilizable reserve of high-energy phosphates in skeletal muscle and the brain).

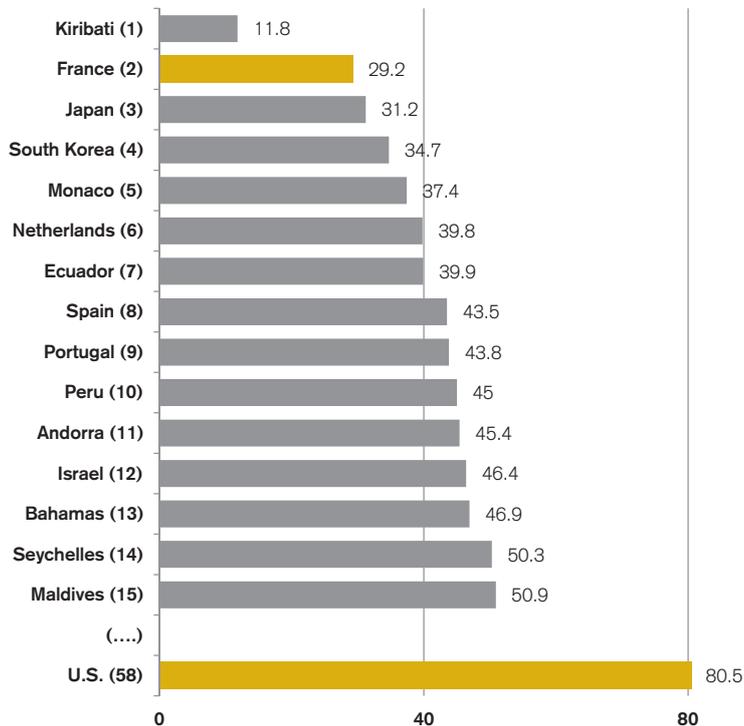
As the intensity of activity rises, so does the need for glucose and amino acid. For example, at a slow jog, muscles still burn 60% fat, but in a 100-meter sprint, carbohydrate and protein will make almost all the fuel. Counter-intuitively, athletic training increases the cellular machinery to oxidize fatty acids, thus increasing the fat/glucose ratio in exercise.

When adding up the composite fuel requirements, it is clear that glucose is not the predominant fuel the body uses. At rest (and even during moderate exercise) fat may account for at least 50-60% of fuel burned by tissue and much of the remainder is protein. This also means that according to current dietary guidelines (which imply more than 50% carbohydrates consumption) our liver must turn carbohydrates into fat to give our tissues the fuel they require.

Figure 13

2014 coronary heart disease by country

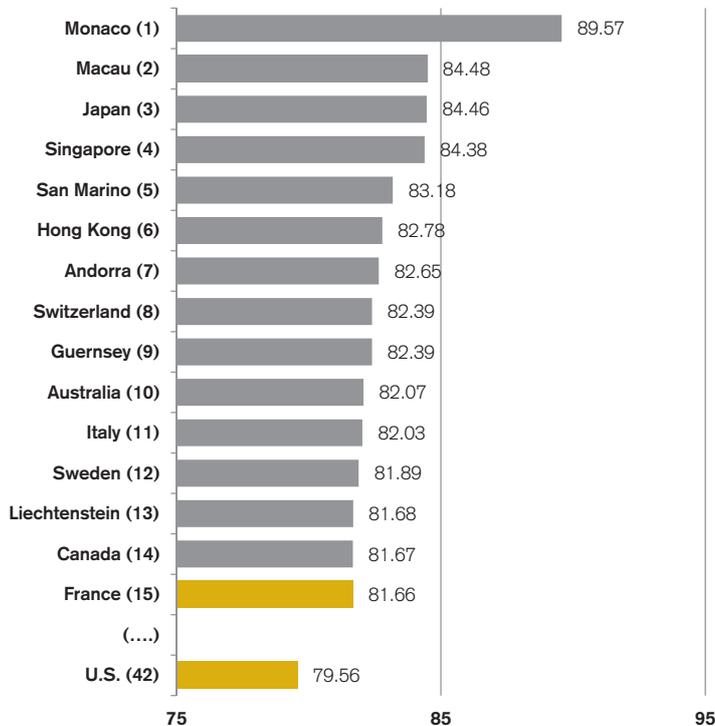
Death rate per 100,000



Source: WHO 2011

Figure 14

2014 life expectancy by country



Source: Infoplease



The paradoxes: France, Israel and Japan

In France, the average consumption of saturated fats is 15.5% and total fats account for 41% of the diet. So according to the WHO, AHA and most national food guidelines which suggest 10% as the upper limit for saturated fats, and 30% for total fat intake, we might conclude that France has a relatively unhealthy population. Yet when we compare the French health-care data to those for other developed countries like the U.S. and the U.K.—where saturated fats are lower on a relatively basis—we find just the opposite. For those with a curious mind, in Kiribati—a small island in the Pacific showing the lowest CVD mortality—saturated fat accounts for 27% of total energy intake (mostly coconuts).

Is this a paradox? Only if you believe that saturated fats are not good for you. Looking beyond France and Kiribati, if we plot saturated fat consumption versus deaths by coronary heart disease (CHD) for 22 European countries (using 2005-2010 data), we can see that France is not an exception, but part of a wider conclusion supported by solid statistical data: the level of saturated fat consumption has no correlation with the percentage of CHD deaths. Those who think that saturated fats are good for you will point to the slope of the linear regression fitting these data. However, the R-square is very small and this conclusion is not statistically relevant.

The Israeli paradox is also an interesting one. It refers to a study at the end of the 1990s that showed that Israeli Jews had a relatively high level of coronary heart issues and cardiovascular deaths despite a prevalent diet low in saturated fats. Note that this population also consumed a higher proportion of vegetable oils: in 1996 the average Israeli consumed 24 kg per year of vegetable oils (ex-olive oil) versus 23 kg for the U.S., 13 kg for Europe and 9kg globally.



The study's authors estimated that Israeli Jews' calorie intake of omega-6 fatty acids was close to 11% versus a global average just below 5% at that time. Similarly, research from Susan Allport in 2006 showed that Israelis had a higher percentage of omega-6 in their adipose tissue: 24% versus 16% for Americans and 10% for Europeans. As our body does not synthesize omega-6, diet must be a key reason for the difference.

If you were to assume that saturated fats are good for you and omega-6 fats are "not so good", the Israeli example would not be a paradox and neither would the French one. They would simply be data points supporting a pretty logical hypothesis.

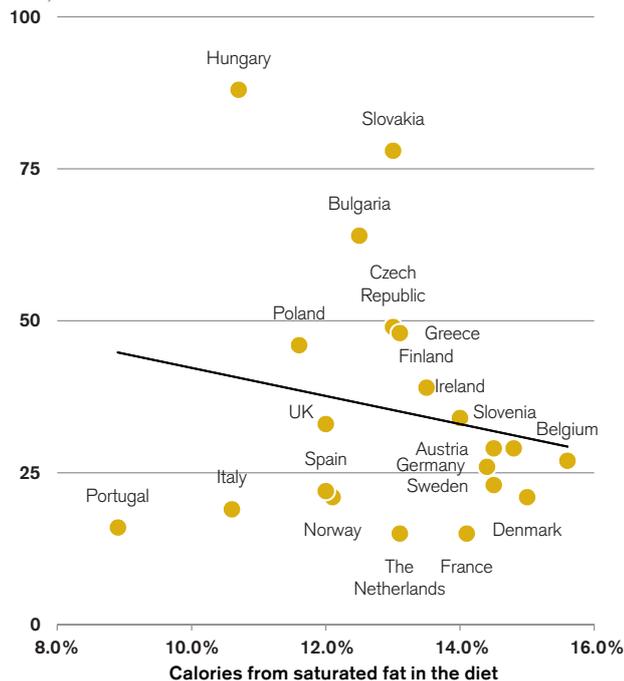
In epidemiological analysis on this topic, Japan—with its low incidence of CVD and CHD—is often cited as supporting evidence by those that believe saturated fats are unhealthy. Japanese consume low levels of saturated fats and have an excellent track record on heart diseases. However, it should also be pointed out that they eat more than twice the amount of fish per person consumed globally—54 kg per year versus 19 kg per year globally and significantly higher than the 22kg for the U.S. and Europe. Fish tends to be rich in omega-3 fatty acids—particularly salmon, sardines, mackerel and anchovies.

Figure 15

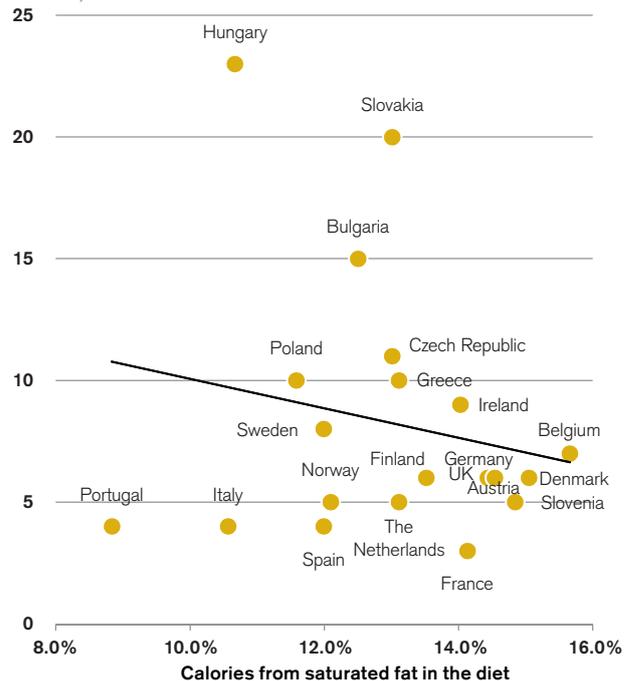
Saturated fat consumption and CVD deaths by country

Deaths from CVD per 100,000

Men, $R^2 = 0.0435$



Women, $R^2 = 0.0469$



Source: European Cardiovascular Disease Statistics 2008 and 2012 editions; National Nutritional and Dietary Surveys. Credit Suisse Research



So, taken together, these three examples appear to fit very well with the idea that saturated fat and omega-3 are generally good for you, while solvent-extracted vegetable oils are not necessarily as beneficial as has been touted.

Were the carbohydrates to blame?

In the case of France, the reason behind better health data may not be a higher consumption of saturated fat, but a correspondingly lower consumption of carbohydrates. Let us compare the U.S. data from NHANES for 1971-75 to those for 2009-10 for fat/protein/carbohydrate consumption and obesity levels.

If we look at the U.S. male population 20 years and above (see Figure 24), average energy intake was 2,453 calories in 1971-75 and 2,564 calories, or 4.5% higher, in 2009-10. Meanwhile the percentage of obese males rose from 12.1% to 35.5%. In the same period, fat consumption increased just 2%, saturated fats declined by 7% and protein increased just 7% (again based on both NHANES and FAOSTAT data saturated fat consumption declined). Carbohydrates consumption, on the other hand, increased 30%.

Looking at the changes in actual foods, rather than in macronutrients, is also telling. Red meat consumption declined by 24%, butter and lard by 39% and eggs by 21%, and dairy rose by just 5%. Conversely, the consumption of vegetable oils (rich in omega-6) soared by 89%, chicken by 139% (which contains saturated fat and omega-6 in almost equal quantities), maize by 100% (rich in omega-6) and sugar by 25%. It seems clear that saturated and monounsaturated fats have very little to do with the soaring levels of obesity among the U.S. population but that carbohydrates and/or polyunsaturated fats (mostly omega-6) have a lot to account for and are also the two main factors behind the overall increase in calorie intake. Excessive consumption of carbohydrates and omega-6 have been shown to trigger insulin resistance through an inflammatory response.

Note that obesity is not just an isolated “illness.” Most obese people have a higher probability of experiencing car-

diovascular problems, diabetes, and other metabolic illnesses. In retrospect, it would be easy to conclude that in the U.S. the stance of many health officials and medical researchers against saturated fat—and the concomitant switch into carbohydrates and potentially omega-6—created a health disaster of major proportions.

From macronutrients to foods: are meat and milk bad?

Some might consider a discussion on fat, protein and carbohydrates too theoretical. Few foods contain only one of these three macronutrients. So let us look at “real” food and see if the trend mentioned above is matched by the available data on food consumption.

In our minds, the word “fat” is directly associated with two foods: dairy and meat. Not surprisingly most milk or yogurt sold nowadays is low fat and the preference of consumers is for lean, not fatty, and, in most cases, white meat. We should, thus, expect to see a marked decline in “fat” dairy and “fatty” meat over the past 30-40 years.

More specifically looking just at the U.S. between 1975 and 2013, milk consumption per capita declined 28%. The mix change is also interesting: whole milk declined 79%—from 25 gallons per person a year to 5—and low fat milk increased 200%—from 5 gallons per capita to 15 gallons per capita. This trend is now reversing. Butter consumption is now 10% higher than 40 years ago, but it declined year after year from 18 pounds per person in 1910 to 4 pounds per person in 1997 before experiencing a resurgence when medical research highlighted the potential negative effects of trans fats, which led to a slump in margarine sales. Meat went through a similar process, and per person consumption is 3% below where it was 40 years ago. Red meat (pork and beef) declined 25% to 97 pounds a year per person, while white meat

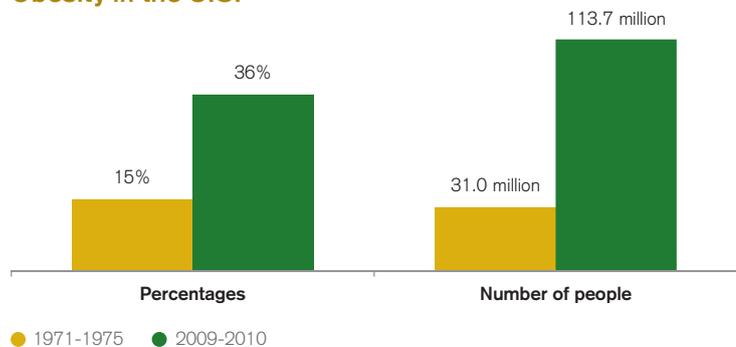


Sugar consumption per person rose from around 80 pounds a year per person to 111 pounds a year. Consumption of vegetable oils increased from basically zero in 1910 to 8 pounds per person in the same period, and consumption of margarine rose 4 times, or by 350%, to 9 pounds per person a year and overtook butter.

As a result, consumption of omega-6 fats increased 169% in the same period, while—it is worth reiterating—saturated fats remained basically flat. Logic would suggest that if the American diet had to be blamed for the heart attack epidemic of the 1950-60s, polyunsaturated omega-6 fats, refined carbohydrates and trans-fats were much more likely to be the culprits than saturated fat. But as Nina Teicholz clearly articulates in her well researched book *The Big Fat Surprise* in the 1960s logic took a walk on the wild side.

Figure 16

Obesity in the U.S.



Source: NHANES

Figure 17

Potential drivers

	1971-1975	2009-2010	% change 1971-2009
Potential drivers			
Calorie Intake (NHANES)	1,955	2,195	12%
Macronutrients (NHANES)			
Carbohydrates Grams per day	215	280	+30
% of daily energy intake	44	51	
Protein Grams per day	82	88	+7
% of daily energy intake	17	16	
Fat Grams per day	79	80.5	+2
% of daily energy intake	37	36	
Saturated fat Grams per day	29	27	-7
% of daily energy intake	14	11	
Food (FAOSTAT) (grams per day)			
Eggs	48	38	-21
Butter + Lard	23	14	-39
Corn	17	34	+100
Wheat	180	218	+21
Vegetable Oils	44	83	+89
Red Meat	235	179	-24
Chicken	59	141	+139
Dairy	669	703	-5
Sugar	133	166	+25

Sources: NHANES, FAOSTAT

(chicken and turkey) increased 98% to 57 pounds a year per person.

Given what has happened to the American population, with obesity going from 14.5% of the male population in 1971-75 to 36.1% in 2009-10, we should question the benefits of low fat meat and dairy in keeping the population lean and healthy. The longer term data, though, present a very interesting picture.

Saturated fat and the 1950-60s U.S. heart attack epidemic

Low fat foods, particularly foods low in saturated fats, were officially promoted in the 1960-70s as a way of tackling the “epidemic” of heart attacks in the U.S. that began in the 1950s. Yet, if you look at butter consumption—butter is 68% saturated fat in calories—it had already declined 56% from 1910 to 1960. In 1960, Americans consumed only 8 pounds of butter per person, less than half the 18 pounds of butter they consumed in 1910.

Lard, which is 45% monounsaturated and 39% saturated fat, probably had little to do with it as well. Between 1910 and 1960, consumption declined 10%, so it is unlikely to be the culprit. Consumption of pork meat—which derives 41% of calories from saturated fat—doubled from 1910 to 1960. Beef consumption—44% saturated fats—increased 19% in the same period.

So, if we add them all up, we can estimate the change in saturated fat intake for an average American in the period 1910-1960 (see Figure 27). Based on our calculations saturated fat consumption did not increase! Red meat might have been behind the heart attack epidemic; saturated fat certainly not. What else did Americans consume in larger and larger quantities between 1910 and 1960? Sugar, vegetable oils and margarine.



Figure 18

Saturated fat and omega-6 – growth 1910-1960

Food	Calories/100g		Consumption (pounds/ capita /day)		Change in calories	
	Saturated fat	Omega-6	1910	1960	Saturated fat	Omega-6
Coconut oil	793.0	17.0	0.1	0.7	13.0	0.3
Palm kernel	741.0	17.0	-	0.1	2.0	0.0
Butter	485.0	25.0	8.0	3.5	(59.8)	(3.1)
Palm	451.0	71.0	-	0.2	2.5	0.4
Ground nut oil	159.0	292.0	-	0.2	0.9	1.6
Olive oil	124.0	88.0	0.1	0.2	0.3	0.2
Sheep	112.0	17.0	1.9	1.4	(1.5)	(0.2)
Sunflower	97.0	362.0	-	-	-	-
Beef	81.0	5.0	24.2	28.7	10.0	0.6
Pork	78.0	16.5	17.7	22.0	9.2	1.9
Nut	45.0	114.0	1.3	1.7	0.5	1.2
Egg	33.0	14.0	17.6	19.2	1.4	0.6
Milk	19.0	1.2	335.8	294.9	(21.3)	(1.3)
Cottonseed	239.0	477.0	0.5	2.9	15.7	31.4
Soybean	141.0	468.0	1.0	6.5	21.2	70.5
Fish	14.3	12.2	5.1	4.7	(0.1)	(0.1)
Margarine	11.9	36.5	0.7	4.2	1.1	3.5
Lard	41.0	10.5	3.7	3.4	(0.4)	(0.1)
Shortening	23.2	7.0	3.6	5.7	1.3	0.4
Poultry	12.0	10.5	5.4	10.9	1.8	1.6
Change 1910-1960 in absolute calories					-2.1	109.5
Change 1910-1960 in % calories					-0.5%	169%

Source: FAOSTAT, USDA, Credit Suisse Research analysis



PHOTO: ISTOCKPHOTO.COM/MAXIM PETRICHUK

Smoking gun or drinking binge?

Medical academia, led by Ancel Keys, focused on nutrition as the area that would reveal the leading cause of the stroke and heart attack epidemic in the U.S. in the 1950-60s. Death due to CVDs—age adjusted—rose by 57% between 1930 and 1960. True, Americans were eating more at the time, but they also smoked a lot more: 10.7 cigarettes per day per adult in 1960 versus 4.3 in 1930, a 149% increase. In the same period, cigarette smoking rose 125% in France and 95% in the U.K.. Alcohol consumption also rose sharply in the U.S. from 1.2 gallons per person a year in the 1930s to 2.1 gallons in the 1960s, a 75% increase.

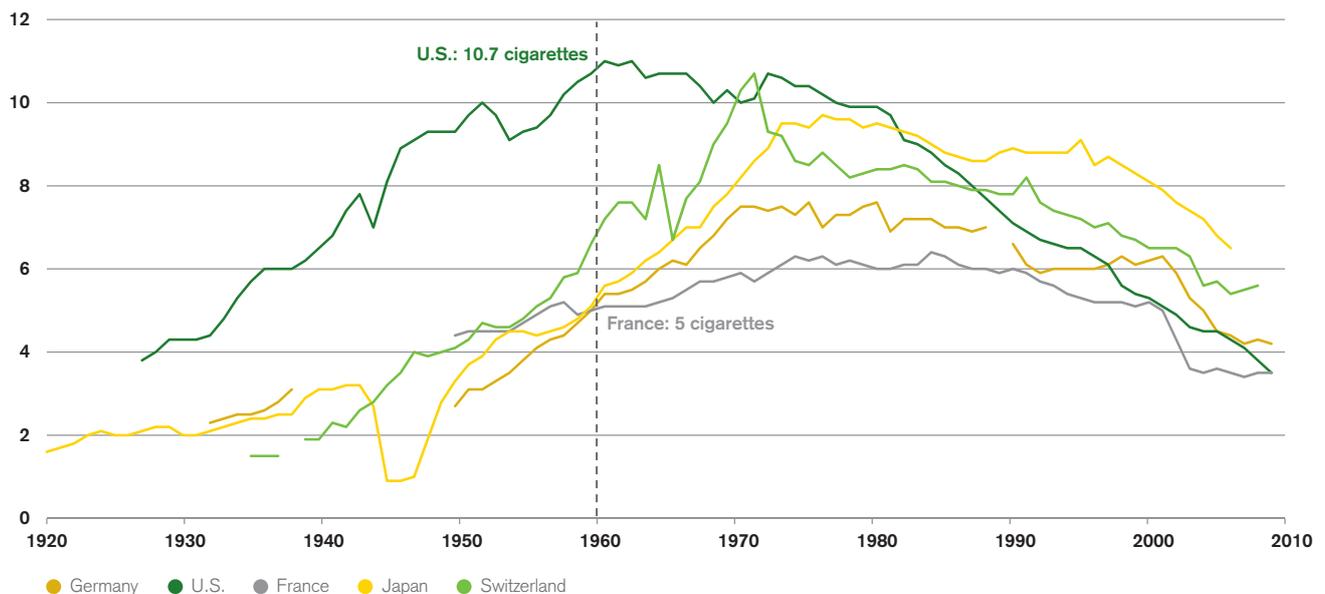
Research post 1960 shows that smoking increases cardiovascular death (CVD) risk by over 3 times and that heavy alcohol consumption increases doubles the CVD risk.

Was it just the growth in consumption in the U.S. or also the level of consumption? Both. Relative to the rest of the world Americans in the 1960s smoked a lot more. The French smoked 5 cigarettes per day in 1960 and the British 7.6. U.S. adults in 1960 smoked 10.8 cigarettes per day, which was 4 times more than the world average! Rather than framing guidance entirely around saturated fats, it could be argued that a 50% reduction in cigarette smoking or alcohol would have probably achieved a lot more than any dietary recommendation.

Figure 19

Cigarette consumption — selected countries (1920-2010)

Number of cigarettes/adult/day



Sales of cigarettes total cigarettes (including estimated number of hand-rolled cigarettes) for (West) Germany, France, and USA. For Japan and Switzerland only manufactured cigarettes. Adults are over 15 years old. Germany is West Germany only from 1950-89.

Source: Max Roser. 'International Smoking Statistics'. <http://www.pnlee.co.uk/ISS.htm>

Medical research: The debate

Medical research on fat intake and its “health” implications has focused for years mostly on its potential impact on cardiovascular diseases. In the 1960s the U.S. experienced a rapid rise in the number of heart attacks among its adult population. Cardiovascular diseases became the #1 cause of death in the U.S. Also, autopsies of people who died of heart attacks found that in most cases the coronary arteries presented advanced levels of atherosclerosis, a hardening of artery walls due to plaque build-up. Plaques are made of lipids (particularly saturated fats), calcium, cholesterol and cellular waste.

The medical establishment translated this for the general public into a simplistic but effective message: saturated fat is clogging your arteries. As explained earlier, carbohydrates and particularly sugar are more likely to make people fat than fat itself. We will see later on that carbohydrates are also far more likely to create fat “deposits” in arteries.

In this section, we will analyze the most recent medical research on the relationship between the different types of fat and several illnesses—not just heart disease, but also cancer, fatty liver disease, metabolic syndrome and mental illnesses.

We will focus only on the five basic types of fat introduced before. However, each of these main types of fat—saturated or monounsaturated fat for example—can be broken down into different “expressions” depending on the number of carbon atoms (short chain or long chain). This can be important.

A major consideration when understanding the impact of saturated fats on health biomarkers is that different types of saturated fats have very different effects on the body. Short chain saturated fat such as butyrate (found in butter or metabolized by bacteria in the colon from fiber) is absorbed easily by the intestine and burned rapidly in surrounding tissues as fuel. Very long chain saturated fat (more than 18 carbon atoms) are not absorbed well and pass through the digestive system untouched. Stearic acid, for example, has 18 carbon atoms and comprises 30-40% of the saturated fat in red meat and 55% in dark chocolate. It has been repeatedly studied and found to have no effect on cholesterol levels. Indeed, researchers have argued for stearic acid (which accounts for 20-25% of U.S. saturated fat intake) to be counted separately when calculating saturated fat levels, although this has not been done.

We will look at both epidemiological research, (the study of health and disease conditions in certain segments of the population), cohort studies (the analysis of a

group of people who do not have a particular disease to see who develops that disease over a certain time period) and clinical randomized trials (studies in which participants are assigned randomly to separate groups to compare different treatments).

We consider increased risk of 10% or less to be practically insignificant given the inability to measure precisely daily intakes of any food.

The cholesterol myth

As is the case for fat, consumers have been confused by the use of the same word for dietary cholesterol (eggs, liver, butter etc.) and blood plasma cholesterol. Under the much-touted assumption that high plasma cholesterol is bad for you, people make a “logical” connection that eating more cholesterol will raise blood cholesterol. Add to this that until 2010 American Dietary Guidelines recommended dietary intake of less than 200mg per day of cholesterol (equivalent to one large egg), and the result has been a common fear of eating cholesterol-rich foods—the “egg whites” strategy. Yet the truth is that eating cholesterol does not significantly change the level of cholesterol in your blood.

A study published in 1991 by Kern et al. detailed the effects on plasma cholesterol of an 88-year-old man’s diet of 25 eggs per day: there was basically no effect. Recent medical research—including a meta-analysis that reviewed 40 cohort studies and clinical randomized trials—has shown that dietary cholesterol is statistically not significantly associated with coronary artery disease or ischemic or hemorrhagic stroke. Those who suffer from a genetic disease called familial hypercholesterolemia represent the only exception.

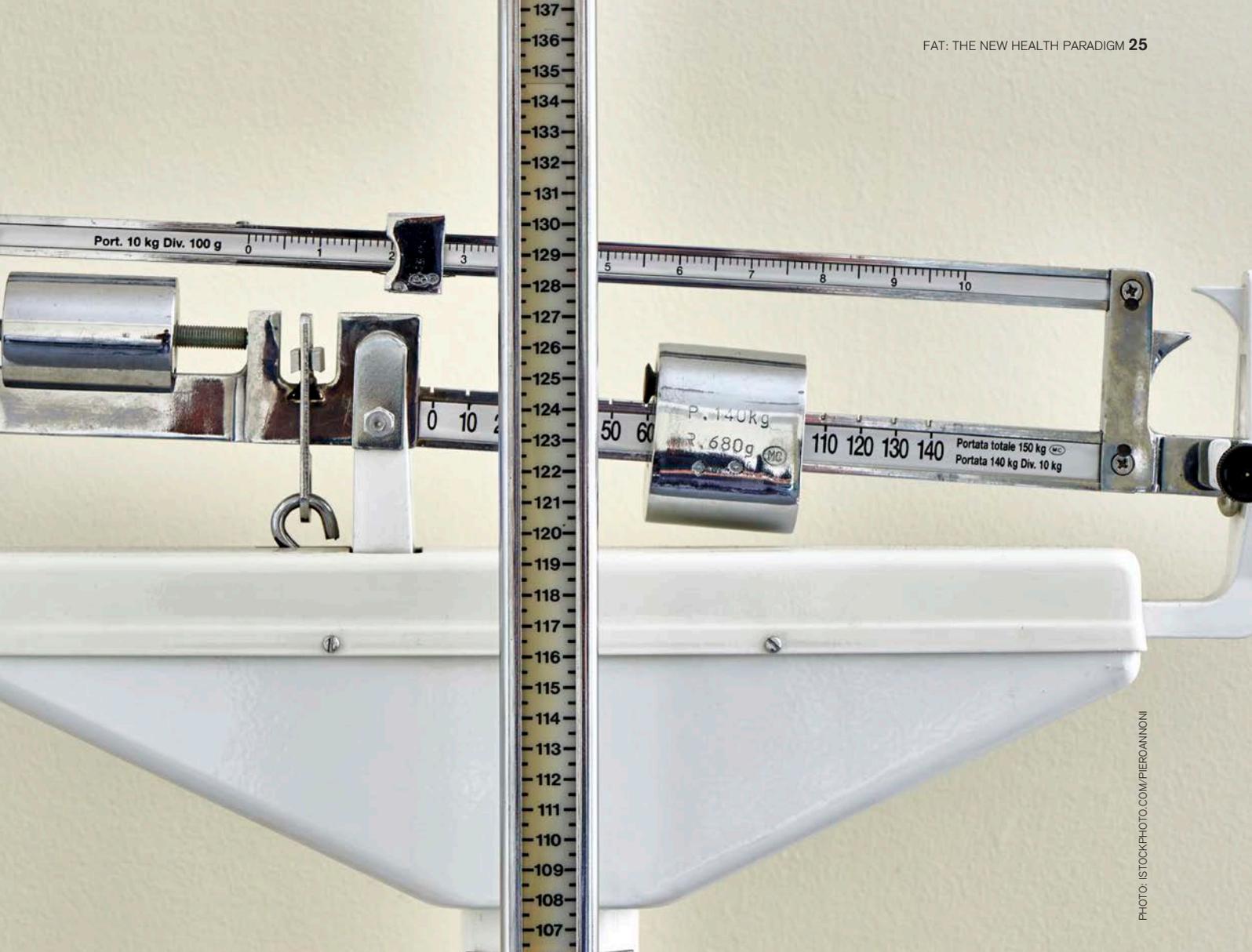


PHOTO: ISTOCKPHOTO.COM/PIEROANNONI

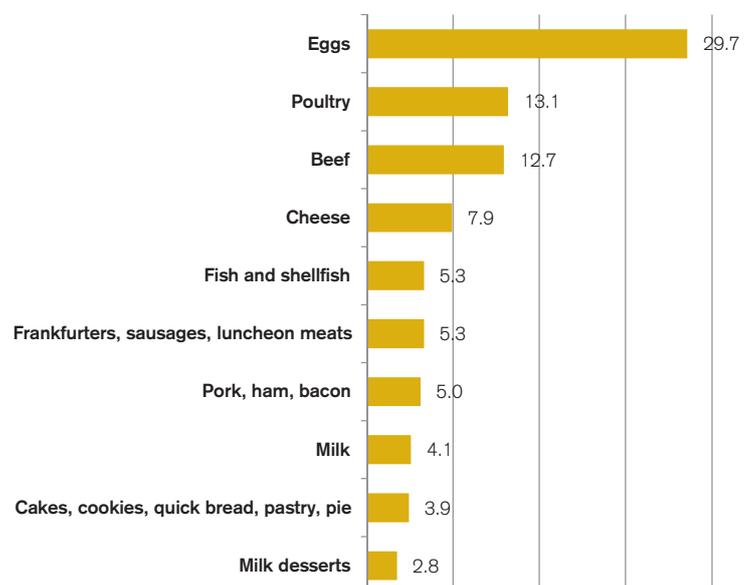
Some research shows that dietary cholesterol is associated with a 10% increased CVD risk in older adults suffering from type 2 diabetes. As we mentioned before, smoking, for example, increases the CVD risk by 300%. Compared to that 10% is not very significant.

This is compelling evidence, but why does cholesterol intake not significantly affect total blood cholesterol levels? Cholesterol is a complex organic molecule made up mostly of lipids and protein. It is biosynthesized by all animals—the body produces it—and plays several important roles in our body. It plays a part in producing hormones such as estrogen, testosterone, progesterone, aldosterone and cortisone, helps the production of vitamin D, produces bile acids which aid in digestion and vitamin absorption, and is critical in both the creation and maintenance of human cell membranes.

The liver controls the level of cholesterol in our body. It produces up to 1,000-2,000 mg of cholesterol per day and is able to remove cholesterol by converting it into bile salts and expelling it via the feces. If we eat more cholesterol, the liver tends to reduce the biosynthesis of cholesterol (not exactly on a 1-to-1 basis, as the process is more complicated): cholesterol biosynthesis and cholesterol absorption are negatively correlated.

Figure 20

Main food sources of cholesterol: US adults



Source: O'Neil CE, Keast DR, Fulgoni VL III et al. Food sources of energy and nutrients among adults in the U.S.: NHANES 2003–2006. Nutrition 2012



That is why there is a strong case for going back to eating full eggs, leaving behind the egg-white omelets and for eating more of the foods listed below that have been shunned because of their high cholesterol content.

Is high cholesterol bad?

A second myth that has lasted for over fifty years is that high plasma cholesterol is synonymous with high CVD risk. Among the different markers of CVD risk cholesterol is by far the weakest and quite unreliable. But we should not limit ourselves just to heart attacks. Let us also look at cancer and other causes of death and their relation to plasma cholesterol levels.

If cholesterol levels in the blood were a good predictor of potential heart failure it should work well with both sexes and at all ages. Also, lower levels of cholesterol should correlate well with low levels of cardiovascular deaths. But this is not the case. Only for cholesterol levels above 240 mg/dl and only in the case of men, the probability of cardiovascular death is higher. But this is not recent research. At a conference held in 1990 by the U.S. National Heart, Lung and Blood Institute researchers reviewed 19 cohort studies linking total cholesterol levels and mortality rates. There are more recent studies that reach the same conclusion, but this is probably one of the most interesting ones.

The combined studies covered 48,837 deaths over a five-year period: 45% of all deaths were cardiovascular related, 33% cancer related, 4% respiratory and 5% digestive. The pooled study divided the population into four groups: total cholesterol lower than 160 mg/dl (TC-low), TC between 160 and 200 mg/dl (TC-reference), TC between 200 and 240 mg/dl (TC-medium), and TC higher than 240 mg/dl (TC-high). Here are a few results of this analysis:

- CVD death for men showed a 4% higher risk among TC-low versus the TC-reference (TC 160-200), 16% higher among TC-medium and 48% among TC-high. Note again that smoking is a 300% factor.
- CVD death for women was 10% higher among TC-low, 6% lower among TC-medium and 3% lower among TC-high.
- Cancer deaths for men were 18% higher among TC-low and 5% lower among TC-high. For women, cancer deaths were 5% higher among TC-low and 3% lower among TC-high.
- Total deaths for men were 17% higher among TC-low and 14% higher among TC-high; for women 10% higher among TC-low and 3% lower among TC-high.

We can draw the following conclusions:

1. High cholesterol (above 240mg/dl) is only a marker of higher cardiovascular death for men. Please note that high cholesterol does not cause heart attacks, it is just a marker.
2. For all other illnesses, higher cholesterol levels pointed to lower death levels. Why? Because cholesterol helps support, or is a marker of, a better immune system.

A few examples will illustrate this last important point:

- A 1998 study of a group of 2,446 unmarried men of 15-49 years of age analyzed the relationship between total cholesterol and the probability of contracting HIV. Those with TC-low had a 66% higher probability of contracting HIV eight years after the first check than the TC-reference, probably due to a weaker immune system.
- In 2001, a study of 3,572 Japanese-American men born between 1900 and 1919 in Oahu, published by the Lancet, showed that the group with the lowest levels of cholesterol at the blood examinations in years 1984-87 and years 1991-93 had a 64% higher mortality risk.
- Finally, several studies show that higher cholesterol in older patients is negatively correlated with the probability of developing dementia. An interesting study led by Dr. Michael Melkie of Johns Hopkins University in 2005 showed that increasing levels of

cholesterol between ages 70 and 79 led to a 23-27% reduction in the risk of developing dementia between ages 79 and 88. This might not be surprising, when considering that the brain contains roughly 25% of the cholesterol in our body.

Finally, while statins are not a focus of this report, there is a lot of literature that suggests that statins are effective in dealing with heart related illnesses because they lower cholesterol. This is not necessarily the case. Several studies show that statins are effective because they have strong anti-inflammatory effects in atherogenesis in addition to lowering cholesterol levels. Probably the key is likely to be the first, not the second effect.

The old ways: “good” and “bad” cholesterol; the new ways: pattern A and B

Once you reach a certain age, it is typical to have a regular medical checkup. Around fifty years ago, doctors focused on total cholesterol as a marker of potential heart diseases—anything above 200 mg/dl was considered a marker of a potential heart attack. Fast-forward to 2015 and patients are now hearing that the conversation has moved now to discuss their “good” cholesterol (HDL or high density lipoprotein) and their “bad” cholesterol (LDL or low density lipoprotein).

Neither is the best marker available to identify CVD risk: if anybody is truly concerned they should ask their doctor to order a vap test (vertical auto profile lipid panel). This is particularly true if the LDL “bad” cholesterol is low and under the “old” prevailing assumptions deemed to be “safe.” Let us see why.

Figure 21

VAP cholesterol test example

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
VAP Cholesterol Profile					
Lipids					
LDL Cholesterol	114		mg/dL	<130	01
HDL Cholesterol	65		mg/dL	>=40	01
VLDL Cholesterol	14		mg/dL	<30	01
Cholesterol, Total	192		mg/dL	<200	01
Triglycerides	67		mg/dL	<150	01
Non HDL Chol. (LDL+VLDL)	127		mg/dL	<160	01
apoB100-calc	84		mg/dL	<109	01
LDL-R (Real)-C	92		mg/dL	<100	01
Lp(a) Cholesterol	13.0	High	mg/dL	<10	01
IDL Cholesterol	9		mg/dL	<20	01
Remnant Lipo. (IDL+VLDL3)	18		mg/dL	<30	01
Clinical Consideration					
Probable Metabolic Syndrome	No			No	01
Sub-Class Information					
HDL-2 (Most Protective)	16		mg/dL	>10	01
HDL-3 (Less Protective)	49		mg/dL	>30	01
VLDL-3 (Small Remnant)	9		mg/dL	<10	01
LDL1 Pattern A	13.9		mg/dL		01
LDL2 Pattern A	35.6		mg/dL		01
LDL3 Pattern B	36.8		mg/dL		01
LDL4 Pattern B	5.8		mg/dL		01
LDL Density Pattern	A			A	01
<div style="display: flex; justify-content: space-around; border: 1px solid black; padding: 5px;"> Pattern B Small, Dense LDL Pattern A/B Pattern A Large Buoyant LDL </div>					
Heavy Metals Profile II, Blood					
Lead, Blood	2		ug/dL	0 - 19	02

Source: Credit Suisse Research

For years, LDL and HDL have been identified as the bad and the good cholesterol. Both are lipoproteins, particles that contain triacylglycerol, phospholipids and cholesterol and amphipathic proteins called apolipoproteins (Apo B and Apo A 1), which facilitate the movement of LDL and HDL cholesterol through the blood. In Classical Greek, Apo-lipo-protein means protein deposited on/ attached to the fat.

Both LDL and HDL are produced in the liver and have a key function, carrying energy in the form of triacylglycerol or cholesterol around the blood.

LDL delivers cholesterol to cells in the body, where it is used in the membranes. It is called low density because lipids (mostly cholesterol) make up around 50% of its mass and protein 25% (lipid are lighter than protein). LDL is considered “bad” because in the presence of excess LDL more cholesterol tends to deposit in the walls of arteries.

HDL is involved in reverse cholesterol transport. It is called high density because the larger part of the particle is made up of protein (20% cholesterol and 50% protein). HDL is considered “good” because it takes the “excess” cholesterol in the blood and delivers it back to the liver, therefore removing lipid material that might contribute to the formation of plaques.

Most doctors tend to measure LDL, HDL, Triglycerides and Total Cholesterol. A high LDL suggests that the concentration of LDL in the blood is high. However, more recently, medical research has shown that a better marker of potential CVD risk is the size of the LDL particles in your blood. The larger the size the better, as smaller particles tend to accumulate and stick to the endothelial wall causing the formation of the plaque. The higher the number of particles for a given concentration, the more likely they are smaller on a relative basis. But how can we measure the number of LDL particles?

Each LDL particle has one specific apolipoprotein that facilitates the transport of the lipoprotein across the blood. There is one apolipoprotein Apo B 100 (called simply Apo B) for each LDL particle; so one protein per particle. We can measure the number of Apo B proteins and therefore the number of LDL particles.

It is possible to have normal or even low cholesterol or a low LDL, but a high number of LDL particles. High levels of triglycerides and low levels of HDL tend to be closely correlated with small LDL particles. Among the patients most likely to show this pattern are those with metabolic syndrome. Research shows that the more individual components of the metabolic syndrome are present in a person—such as high BMI, abdominal obesity, hypertension, insulin resistance, high triglycerides and low HDL—the more likely it is that LDL particles will be small (or that their number will be elevated).

Patients with high LDL and large particles (or low numbers of LDL particles) are not necessarily at risk of a heart attack. Giving statins to this group might achieve very little. Conversely, patients with low total cholesterol and low LDL but large numbers of (small) LDL particles carry a higher risk.

How big is this risk? Back in 1988 a study led by Austin and others analyzed plasma samples from 109 cases and 121 controls. Quoting from the study: “The LDL subclass pattern characterized by a preponderance of small, dense LDL particles was significantly associated with a **threefold** increased risk of myocardial infarction, independent of age, sex, and relative weight. Multivariate logistic regression analyses showed that both high-density lipoprotein cholesterol and triglyceride levels contributed to the risk associated with the small, dense LDL subclass pattern.”

One way to measure which pattern each one of us has is by a vap test to assess the level of Apo B to Apo A1—the apolipoprotein associated with HDL—in other words, to compare pattern A and pattern B. For those interested, in the U.S. it costs \$75–85, but is not covered by most insurance plans.

Subsequent studies to the one above in most cases reached the same conclusion in highlighting the ratio of Apo B to Apo A1 as the best marker of CVD risk, particularly for individuals with low total cholesterol and LDL. One worth quoting, because it was one of the most extensive, is the AMORIS study run in Sweden that followed 175,553 Swedish men and women in two periods, 1985–89 and 1990–1996. Levels of LDL, HDL, Apo B, Apo A1, etc. were taken at the beginning of each period and compared with incidences of myocardial infarction approximately five years later.

The research team divided individuals into quartiles and compared outcomes for the highest and lowest quartiles of each marker: LDL, HDL, TC, TG, Apo B, Apo A1 and Apo b/Apo A1. The ratio of Apo B to Apo A 1 was the best predictor, with a risk ratio of 4 between top and bottom quartile for men and 3 for women. The same risk ratios for TC, TG, and LDL were respectively 1.9, 2.3 and 2.8 for men and 1.2, 3.1 and 1.6 for women. The study also found that total cholesterol loses its predictive power beyond the age of 70, which is not the case for Apo B/ Apo A 1.

The consensus call: trans fat

Rarely in medical sciences is there such a productive connection between the findings of medical research and the reaction of health authorities and companies as in the case of trans fats. Trans fats—hydrogenated vegetable oils—became a kitchen staple in the form of, for example, margarine and shortening, and a key ingredient in the food industry in the 1950s. In the U.S., the FDA estimated that, at the end of the 1990s, 95% of prepared cookies, 100% of crackers and 80% of frozen breakfast products contained trans fats. Current estimates by NHANES suggest that Americans still get close to 3% of their calories from trans fats, versus 1% in Europe.

Dr. Fred Kummerow wrote the first medical research paper on the topic in 1957, and in the early 1970s, Mary Enig—a student at the University of Maryland—added further pressure with several negative articles. But it was two papers published in the early 1990s that caught everybody's attention: Intake of trans fatty acids and risk of coronary heart disease among women by William Willett of Harvard Medical School in the *Lancet* 1993, and Trans-fatty acids intake and risk of myocardial infarction, *Circulation* 1994. The first paper, which was based on a Nurses' Health Study covering 85,095 healthy women, concluded that the "intake of trans fat was directly related to a 50% increased risk of coronary heart disease." For those women whose margarine consumption over the previous ten years had been stable, the risk was higher: 67%. The second paper focused on 239 patients admitted to one of six hospitals in the Boston area. Intake of transfat was directly related to a 1.4 times higher risk of myocardial infarction (top quintile versus lower quintile). In 2003, Denmark became the first country to ban transfats. The FDA and other organizations followed relatively quickly with tough limits as more and more papers concurred on this topic. By January 1, 2006 the FDA mandated that all foods containing transfats in excess of 0.5 g per serving had to show the level of transfats in the nutrition label. At the same time, the U.S. Dietary Guidelines Advisory Committee recommended that consumption of transfats be kept below 1% of total energy (20-25 calories or 5-6 grams per day).

The food industry realized that having that label was not a great selling point and a couple of years later almost all products that originally contained transfats had been reformulated with zero transfats or smaller amounts. Not quite. The label criteria effectively allowed companies to reduce the portion sizes shown on the labels so that they did not have to declare the presence of transfats in the product. Several other organizations around the world took similar steps and the transfats debate was basically over by the end of 2010. The U.S. finally banned transfats only in 2015, twelve years after Denmark, but the USDA is still allowing companies three years to fully implement the ban. We see little logic in this. If it is bad, it should be stopped immediately; nobody will die of hunger... there are plenty of other things you can eat.

Transfats: the alternatives

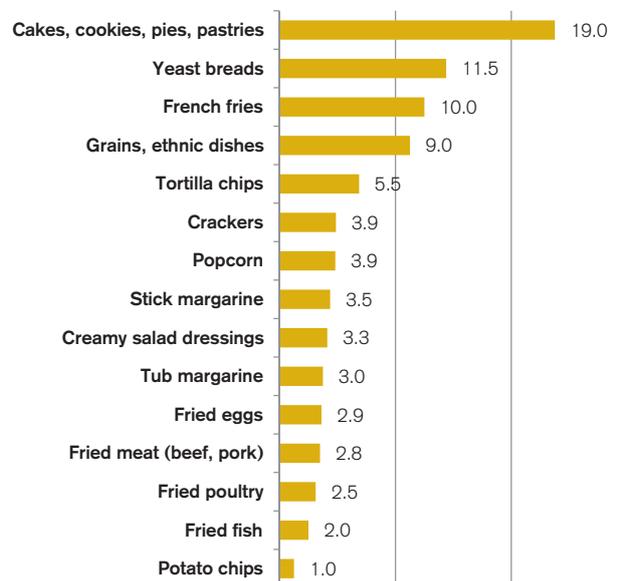
The food industry in the U.S.—and we believe soon in Europe—faces the issue of replacing transfats in the manufacturing process. Price matters and the ingredients industry has been working for years on modified versions of solvent-extracted oils through plant breeding or genetic modification of the seeds. These oleic version mimic the fat structure of olive oil but allow for "stable" frying at higher temperatures than olive oil. Through interesterification or fractioning of fats some of these oils can be converted in the plasticity (read: consistency) demanded by the food manufacturers. The only problem is that fractioning requires sophisticated and expensive technology. Similarly interesterification is either expensive and slow when using an enzymatic process or 30% of fats is lost when using a chemical process.

If the industry acts rationally and saturated fats lose their negative perception—as we believe it will gradually happen—the best option for the industry particularly for baked products would be to use palm oil, palm kernel oil or coconut oil. Why go into further genetically modified seed oils, when we have ideally-suited natural ones? These so called "tropical oils" have been for years the subject of extremely negative campaigns and dubious research papers. If saturated and monounsaturated fats are not negative for our health—as we will show later—none if these oils should be negative for our health. Several multinational food companies already use them; we expect most companies in the food business to increase the proportions of these oils in the manufacturing process.

Figure 22

Main food sources of transfats: US adults

Percentage of total TFA contributed by the respective food category



A good example is provided by a recent paper by Sun et al. published in May 2015 on palm oil. The paper focuses on the fact that palm oil increases LDL cholesterol and minimizes the fact that also increases HDL cholesterol. But as we seen before, LDL is a poor marker of cardiac risk; so even ignoring the positive HDL effect, the conclusion is pretty superficial. For those in search of reassurance, we point to a paper published in 2006 by Tarrago-Trani et al. under a USDA and National Institutes of Health sponsorship. This detailed paper analyses the potential alternatives the industry has to replace trans fats. Two quotes are worth mentioning, the first on palm oil and the second on coconut oil and palm kernel oil:

- “Several controlled feeding studies have shown that palm oil, that contains 50% saturated fat (45% palmitic, 5% stearic, 40% monounsaturated, and 10% polyunsaturated fatty acids) has no detrimental effects on blood lipid profiles. In fact, in some studies, a slight positive effect on HDL cholesterol and Apo A1 was noted.”
- “Lauric acid (a saturated fat) has been shown to decrease the TC/HDL ratio, due to a larger increase in HDL cholesterol. Mensink et al. have commented that even though consumption of saturated fat should not be promoted (*sic!*), lauric acid-rich palm kernel oil and coconut oil (approximately 50% lauric acid) are still a better alternative for the food industry, in terms of effect on TC/HDL cholesterol ratio, than partially hydrogenated oil in products that require solid fats for texture.”

Omega-3

There is a general consensus that omega-3 oils have beneficial health effects on humans. Both clinical studies and epidemiological studies support this conclusion. The benefits are tangible and not only in 5-30% lower overall mortality risk from CVD events.

On the cardiac front, there are several studies detailing the beneficial effects of omega-3. We mentioned before the Japanese paradox that ties the higher consumption of fish with lower CVD risk. Meta-analysis by Skeaff and others in 2009 covered 22 cohort studies representing 230,000 individuals with follow-ups of 5-40 years. In these studies, fish intake ranged between 0 and 23 grams per day to 23-180 grams per day. Those with higher fish consumption showed an 18% lower risk of death by coronary heart attack.

Clinical trials have shown similar results, with a few exceptions. The most extensive clinical trial was run in Italy and published in 1999. In the GISSI study, 11,324 patients who experienced a myocardial infarction were given 882 mg/day supplements of EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid), both omega-3s. No patients were on statins. After 3.5 years the patients taking the supplements experienced 15% fewer cardiac events (fatal and non-fatal); they also experienced a 20% reduction in overall mortality. A similar experiment ten years later, which included patients using statins, showed just a 9% risk reduction.



A meta-analysis of 14 clinical trials published in 2005 showed that omega-3 intake around or above 1 gram per day lowered the risk of coronary heart failure by a combined 23%. A meta-analysis of 35 clinical trials on the effects of statins showed that statins reduced the risk of coronary heart failure by a combined 13%, or 22% if we focus only on the 20 studies that dealt with secondary prevention of CHD. So, there is some growing interest in the possibility of replacing or supplementing statin therapy with an omega-3 therapy given the efficacy and the lower cost.

Other benefits of omega-3 center around the anti-inflammatory effects and beneficial impacts on the immune system. Some benefits have been shown in treating rheumatoid arthritis, inflammatory bowel disease (e.g. Crohn's disease) and improvements in brain development and functioning.

The latter is probably the most interesting area, as our nervous system contains a large amount of DHA. A double-blind randomized clinical trial published in 2003 showed that children aged 4, whose mothers took 1.18 grams of DHA and 0.8 of EPA from week 18 of their pregnancy until delivery, scored much better in tests of mental processing. Other epidemiological studies showed a significant correlation between depression and low fish consumption. Clinical trials providing supplements of omega-3 showed improvements in patients suffering from bipolar and unipolar disorders.

Finally, we found several studies showing that omega-3 intake improves the cognitive ability of elderly people. A study published in 2003 by Morris et al. showed that people aged 70 or older who ate fish at least once a week had a 60% lower probability of developing Alzheimer's disease over a four year period.



Another interesting study was done in France by Heude et al. This study measured the composition of fatty acids in the erythrocyte (red blood cell) membrane (where a critical part of nutrients are transported and absorbed) and the cognitive function of 246 elderly people at the beginning of the study and four years later. Those with higher omega-6 and low omega-3 in their erythrocyte membrane showed sharper declines in cognitive functions.

Based on all this evidence, it is not surprising that most health organizations recommend an intake of omega-3 (EPA+ DHA) between 250-1,000 mg per day.

Monounsaturated: a good source of energy, but not much more

Much of the medical research on monounsaturated fats has been carried out as “diet” research focusing on the Mediterranean diet. There are very few “fat-rich” foods in nature where monounsaturated fats account for more than 50% of total fats: olive, avocado and hazelnut oil, and macadamia nuts. Olive oil is certainly the best known and has been the central focus of the Mediterranean diet.

In her book *The Big Fat Surprise*, Nina Teicholz provides a rare analysis of how the term “Mediterranean diet” came about. Marketing was clearly key, if not the key point, as nobody agreed—or has yet agreed—on what is a “Mediterranean” diet. Greeks eat quite differently from Southern Italians, Spanish, Southern French and coastal North-Africans. One common denominator of these “food cultures,” however, is olive oil. Olive oil is 73% monounsaturated fat.

The most in-depth study on the Mediterranean diet is the Lyon Diet Heart Study, which compared in a clinical randomized trial two diets— a Mediterranean diet (30.4% calories from fat, 12.9% monounsatu-

rated, 3.6% omega-6, 0.8% omega-3, 34% carbohydrates) and a “prudent” Western diet (34% of calories from fat; 10.8% monounsaturated, 5.3% omega-6, 0.3% omega-3, 35% carbohydrates)—in terms of their ability to help prevent additional issues after the first myocardial infarction. The bottom line: Mediterranean diet won 4 to 1. There were only 1.2% cardiac events in those on the diet in the 46 months follow-up compared with 4.1% for the control group.

Does it prove anything? Unfortunately not. Was it the olive oil, the lower omega-6, the higher omega-3? How sure are we that the patients followed the diet? Was the sample large enough?

Another interesting cohort study was published by Estruch et al. in 2013 in the prestigious *New England Journal of Medicine*. The study followed 7,447 Spaniards divided into three groups: one on a Mediterranean diet rich in fish, fresh fruit and vegetables, white meat and olive oil with no sodas, pastries or red meat; another group on the same diet but replacing olive with tree nuts, and a third (control) on a low fat/high carbohydrate diet (relative to the

Figure 23

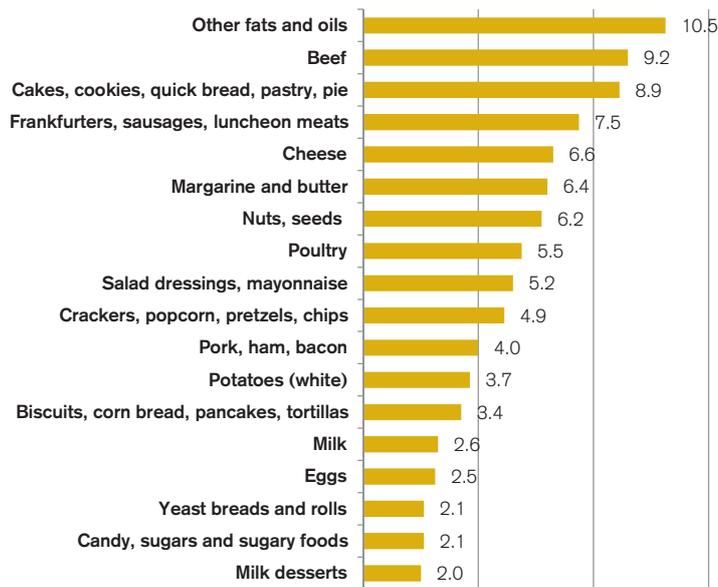
Top 12 foods in Omega-3 per 100 grams

#	Food	Milligrams of omega-3
1	Flaxseed oil	53,304
2	Fish oil	35,311-11,860
3	Flaxseed seeds	22,813
4	Walnuts	9,079
5	Butternuts (nuts)	8,719
6	Caviar	6,820
7	Smoked salmon	3,010
8	Mackerel (raw)	2,670
9	Atlantic salmon farmed (raw)	2,506
10	Atlantic salmon wild (raw)	2,018
11	Oysters	1,648
12	Sardines / anchovies	1,479

Source: Nutritiondata

Figure 24

Main food sources of monounsaturated fat: US adults



Source: O'Neil CE, Keast DR, Fulgoni VL III et al. Food sources of energy and nutrients among adults in the U.S.: NHANES 2003–2006. Nutrition 2012

other two) based on low-fat dairy products, bread, pasta, rice, potatoes, fresh fruits and vegetables, lean fish, but with no vegetable oils, no nuts, and no red meat.

Results: over a five-year period the two groups on a Mediterranean diet showed a 30% greater reduction in cardiovascular disease (CVD) than the control group. However, the

absolute numbers look less impressive: 3.8% of the olive oil group recorded cardiac events in the following five years, 3.4% for the nuts group and 4.4% for the control group. If we focus on death from all causes: 4.6%, 4.7% and 4.7% respectively. We believe this was more a test of a low fat versus a high fat diet, rather a true test of the “Mediterranean” diet.

Also, if we check for the characteristics of the control group, we find that only 6.7% of individuals in this group had a BMI below 25 versus 7.7% of the oil group and 8.3% for the “nuts” group. In addition, the control group had 49% of participants with a BMI over 30 versus 47% for the oil group and 44% for the “nuts” group. Looking just at the BMI segmentation it appears that the control group was significantly less healthy than the other two, particularly when compared to the “nuts” group. The same data were leveraged to show the effectiveness of these diets in breast cancer. Here the results were far more convincing. Note that those who did best were given extra virgin olive oil, but we do not know if this was because olive oil is 75% monounsaturated or it is rich in phenols which have anti-oxidant properties.

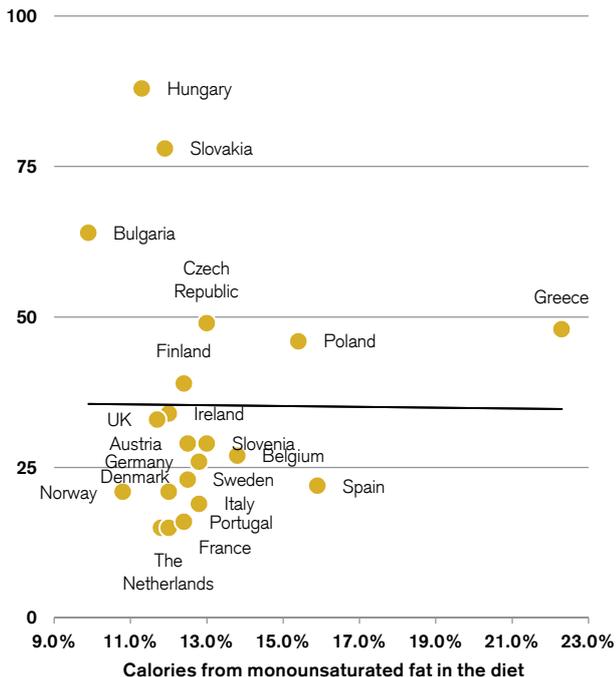
Another way is to look at the epidemiological data and replicate the analysis that Hoenselaar ran a few years ago in Europe assessing the relationship between fat consumption and CVD events on a country-by-country basis. We extended this study by looking at the fat subcomponents (saturated fats, monounsaturated fats and omega-6, not just total fat), getting more recent data (2006-2010) and including more countries (22 in total). The results show no cor-

Figure 25

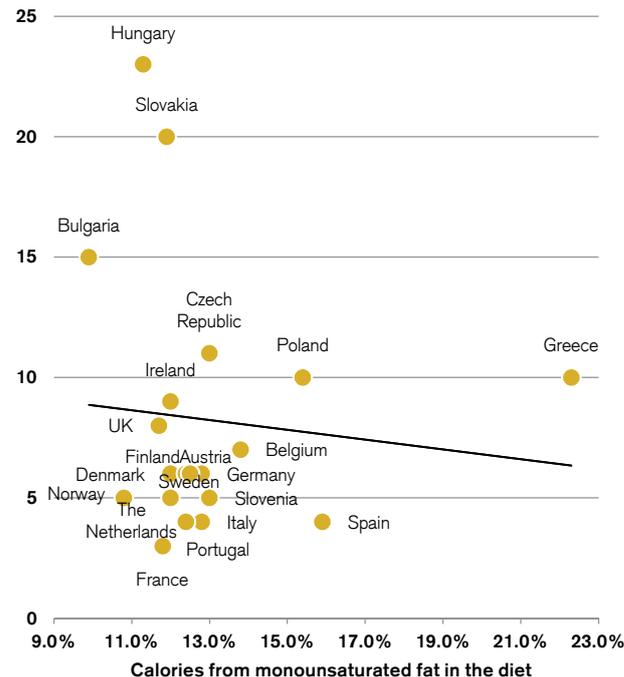
Monounsaturated fat consumption and CVD deaths by country

Deaths from CVD per 100,000

Men, R² = 0.0019



Women, R² = 0.0007



Source: European Cardiovascular Disease Statistics 2008 and 2012 editions; National Nutritional and Dietary Surveys. Credit Suisse Research

relation whatsoever between monounsaturated fat consumption and CVD occurrence.

We believe there is a very good case to make that monounsaturated fats are a good fuel source, but as expected they do not have massive beneficial health aspects. Most clinical trials show that monounsaturated fats at the levels consumed currently have no negative implications for CVD risk or death by other causes. Some studies show that monounsaturated fat-rich diets tend to increase HDL-c and Apo A1, lower TGs and Apo B. Also, on the positive side, substituting carbohydrates or omega-6 with monounsaturated fats tends to have a positive effect on CVD risk. But is it because carbohydrates or omega-6 are bad, or because monounsaturated fats are good, or both?

Being just a “good fuel source” in the area of nutrition is quite positive, as there are several foods that have plenty of negative implications for our health. Note that saturated fat is converted in the liver into a monounsaturated fatty acid. So it would be surprising if our bodies transformed one source of energy into a less efficient one or one with negative effects on our well-being.

Saturated fats: the wrong target

One of the biggest myths in nutrition is that saturated fat intake above a certain level—say 10% based on most dietary guidelines—significantly increases your risk of heart attack. This conclusion that has held for almost half a century is inconsistent with the wealth of epidemiological data or scientific evidence in the form of clinical randomized trials. Plenty of research funding has been earmarked to study and back this hypothesis, yet we cannot find a single research paper written in the last ten years that supports this conclusion. On the contrary, we can find at least 20 studies that dismiss this hypothesis.

We logged calls to both the World Health Organization (WHO) and American Heart Association (AHA) asking for evidence behind the rather negative statements on saturated fats on their websites without much success. As research analysts, we believe that rational analysis based on solid data should be the backbone of any conclusion. Based on our analysis, here are our main conclusions:

Saturated fats are a healthy source of energy and have NO negative implications for your heart (or other organs).

Saturated fat intake increases HDL-c and makes LDL-c particles (measured by Apo B) larger, reducing CVD risk.

A high level of saturated fats in plasma blood—not a good thing—is driven by the amount of carbohydrates we eat, not by the amount of saturated fat we eat.

As was the case for cholesterol, there has been questionable logic used to assert that saturated fats increase the risk of a heart attack. **A very high level of saturated fat in the blood is indeed associated with a very high risk of heart attack. But saturated fat in the blood is quite unrelated to the level of saturated fat consumed. We will cover this shortly.**

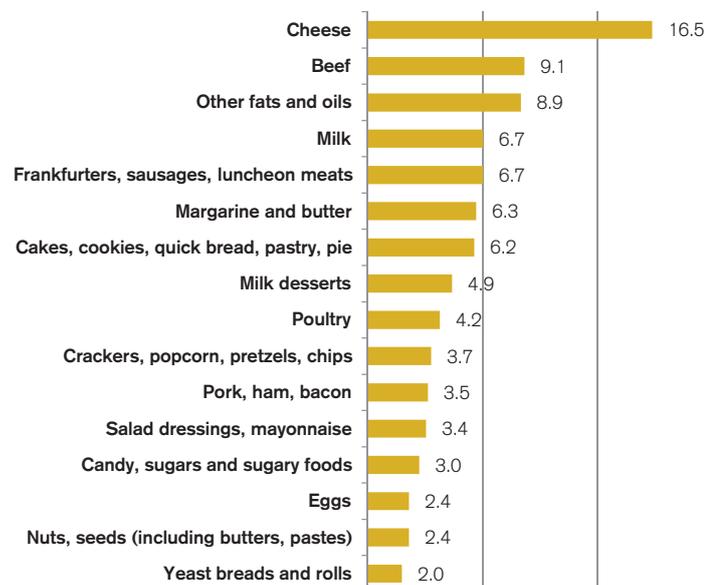
Epidemiological research

Most of the interesting epidemiological research on the potential link between CVD risk and saturated fat has been published in the last ten years, with a heavy concentration in the last five years.

1. We showed earlier (Figure 15) the analysis we carried out to assess the relationship between saturated fat consumption and CVD events on a country-by-country basis. This analysis suggests that saturated fat is neither positively nor negatively correlated with CVD events.
2. A very interesting study was published in 2004 by a team of researchers at Harvard Medical School and the School of Public Health. It focused on the data from the Nurse Health Study that looked at the CHD death rates over 20 years for 78,778 nurses in the Boston area. One important conclusion: “Intakes of saturated fat were not statistically significant predictors of CHD events when adjusted for non-dietary (e.g. age, physical activity) and dietary risk factors (e.g. smoking, wine, etc.)”
3. Another interesting study was published in the American Society for Nutrition in 2009 by Jakobsen et al. It focused on replacing 5% of energy from saturated fats with an equivalent amount of monounsaturated fat, polyunsatu-

Figure 26

Main food sources of saturated fat: US adults



Source: O’Neil CE, Keast DR, Fulgoni VL III et al. Food sources of energy and nutrients among adults in the U.S.: NHANES 2003–2006. Nutrition 2012



rated fat or carbohydrates. The results of the analysis of 11 cohort studies dated between 1963 and 1973 showed that when saturated fat was replaced by monounsaturated fat the risk of coronary events was 19% higher, 13% lower for polyunsaturated fats and 7% higher for carbohydrates. In other words, saturated fat intake produced slightly better outcomes in terms of CVD risk than monounsaturated and carbohydrates, and slightly worse than polyunsaturated (both omega-3 and omega-6, with a slightly higher percentage of omega-6).

4. Finally, the most important epidemiological study on the topic was published in 2010 in the American Society for Nutrition by Siri-Tarino et al. The "et al" include two of the most influential research fellows in the "fat" area: Frank Hu and Ronald Krauss. They analyzed 21 studies focusing on strokes and CHD events published between 1981 and 2007 (Figure 27)¹. Their conclusion: "there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD" and also "saturated fat consumption is associated with a lower risk of stroke."

Clinical research

In this area of research, there have been few controlled randomized trials. There are two key findings we will focus on. First, individuals who eat a diet rich in saturated fat do

not present a higher CVD risk. Second, although eating saturated fats is not associated with a higher CVD, a higher level of saturated fat in the blood is a marker of higher CVD risk. But it is not dietary saturated fat that causes an increase in saturated fat in the blood. It is the carbohydrates, through a process called "de novo lipogenesis", in which the liver converts carbohydrate into fat.

1. The first trial was run in in the early 1970s but was published only in 1989. It involved 9,057 institutionalized men and women in six Minnesota state mental hospitals. The study lasted four and a half years and was run on a double-blind basis. It focused on the effects of two "fat" diets on myocardial infarctions, cardiac deaths and all-cause deaths: the control diet was 39% fat (18% saturated, 16% monounsaturated and 5% polyunsaturated); the treatment diet was 38% fat (9% saturated, 14% monounsaturated and 15% polyunsaturated). The results showed no difference in cardiac events or all-cause deaths between the two diets: halving saturated fat had no effect.

2. In the early 2000s, the topic was revisited. Ronald Krauss ran several research projects. A key one focused on 278 men who were fed a controlled diet over a period of 8 weeks. In the first week, all men ate the same diet, after which they were divided into four groups: A. high carbohydrate diet (54% carbohydrates/16% pro-

¹ For those with just basic statistics, a risk ratio of say (0.8-1.2) implies there is not "statistical" risk; a risk ratio of say (1.1-1.2) implies that there is a statistical risk at a certain confidence level

tein/7-8% saturated fat); B. medium carbohydrate diet (39% carbohydrates/29% protein/7-8% saturated fat); C. low carbohydrate and low saturated fat (26% carbohydrates/29% protein/7-8% saturated fat); and D. low carbohydrate and high saturated fat (26% carbohydrates/29% protein/15% saturated fats).

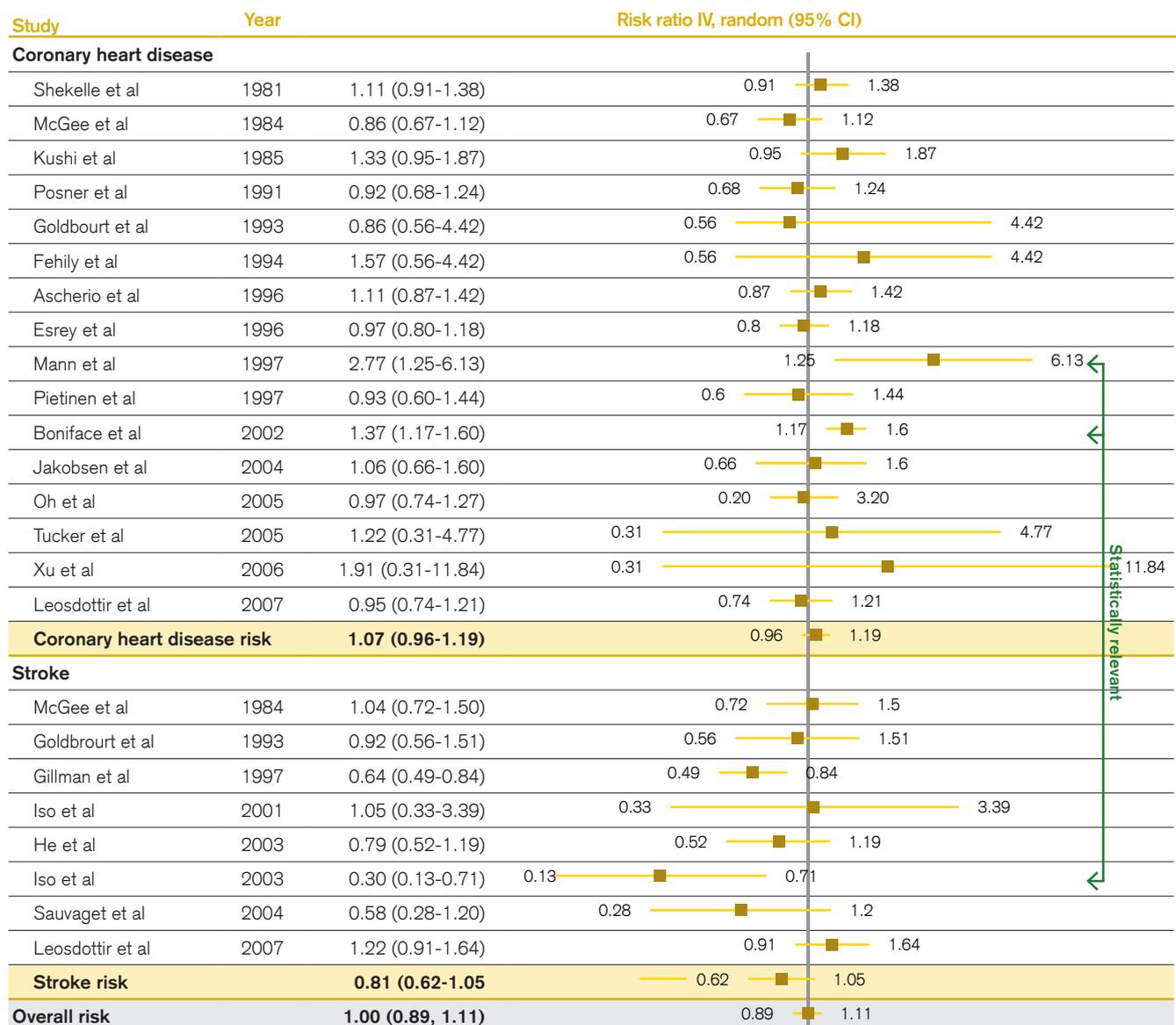
The most interesting conclusions of this study were as follows. 1. The number of small and very small LDL particles was significantly reduced in the low carbohydrate diets (C. and D.) even on higher intake versus lower saturated fat intake. 2. Higher saturated fat intake increased HDL and increased large LDLs particles (see Figure 26), which was clearly positive. 3. Of the low carbohydrate diets, the one with higher saturated fat led

to higher LDL (as we have seen before LDL is not a great marker of CVD risk, but worth mentioning for completeness).

3. A study by Puaschitz et al. published at the end of 2014, which followed 2,412 Norwegian patients with an established coronary artery disease over almost five years concluded that “there was no significant association between saturated fat intake and risk of coronary events”. But what was very interesting about this study was that the patients with the highest level of saturated fat intake were 20% less likely to have a history of myocardial infarction, hypertension or a bypass graft on the coronary artery.
4. A recent study by Yamagishi et al. published in 2015 that reviewed six studies focused on the Japanese pop-

Figure 27

Meta-analysis of 21 prospective cohort trials



Source: Krauss et al. AJCN 83:1025, 2015. Presentation by Ronald M. Krauss for ConAgra Foods Science Institute. Siri-Tarino et al, 2010.

ulation found that the level of intraparenchymal hemorrhage (brain hemorrhage which accounts for 8-13% of all strokes) and ischemic strokes was the lowest among those who consumed the highest relative level of saturated fat (17-25 gr/day). The same studies were inconclusive in establishing a correlation between saturated fat intake and myocardial infarction.

5. The link—or better the lack of a link—between dietary saturated fat and saturated fat in plasma blood was demonstrated by two studies by Volek, Phinney and Forsythe. In the first study, published in 2008, 40 overweight men and women were randomly assigned to two diets (directly provided by the research center): 1. A very low carbohydrate diet with 12% carbohydrates, 59% fat (29% saturated fat) and 28% protein; 2. A low fat diet with 56% carbohydrates, 24% fat (11% saturated) and 20% protein. The first diet was a lot more effective in getting people to lose weight. But the most interesting aspect was that saturated fat in plasma blood decreased 57% in the low-carbohydrate diet versus a 24% decline in the low-fat diet. Note that the dietary intake of saturated fat in the first diet was 3 times higher than in the low fat diet (36 grams versus 12 grams per day).

6. In the second study, 8 normal men were given food at a caloric level required to maintain body weight. Two diets: 1. A low-carbohydrate diet rich in saturated fat (13% carbohydrates, 59% fat, of which 31% saturated and 5% polyunsaturated fats, and 29% protein); and 2. A low carbohydrate diet rich in polyunsaturated fats (12% carbohydrates, 58% fat with 17% saturated and 15% polyunsaturated fat, and 30% protein). Both diets significantly decreased the levels of saturated fat in plasma blood relative to a reference starting point (a diet with 34% carbohydrates and 41% fat, with 17% saturated fat). The first diet high in saturated fats led to a 39% decline in saturated fat in plasma blood and the second rich in polyunsaturated fats to a 47% decline. It did not matter what fat was eaten; lower carbohydrates ensured a decrease of saturated fats in the blood.

How can we explain this “strange” result in the last two clinical randomized trials? Because the liver converts carbohydrates into saturated fats and releases them into the blood. **These two trials show that it is eating an excess of carbohydrates that increases the level of saturated fat in the blood, not saturated fat.**

Polyunsaturated omega-6: the big debate

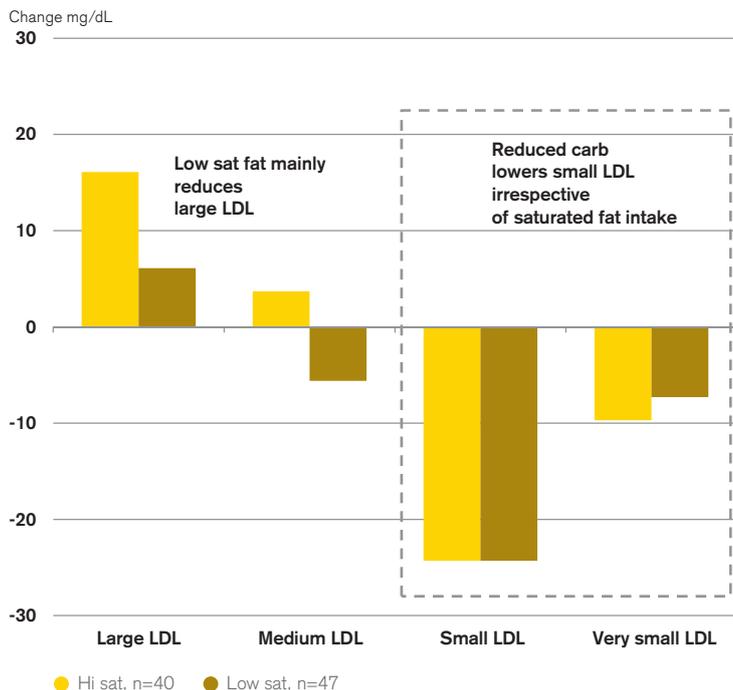
The most controversial area in the field of medical research in blood lipids is the effect of polyunsaturated omega-6 fats intake on cardiovascular risk markers, CVD risk, and other illnesses. As we have seen earlier, omega-6 consumption per capita has increased 89% globally and 95% in the U.S. in the last fifty years due the introduction of solvent extracted vegetable oils (soybean oil, corn oil, cottonseed oil, etc.) and the increased reliance on soy and corn-based feeds for animal husbandry. Numerous health authorities and health organizations recommend a daily intake of omega-6 in the 5-10% range.

Most of the positive research we reviewed on omega-6 fatty acids focuses on the effect that omega-6 intake has on blood plasma lipids. It lowers total cholesterol and LDL cholesterol; but it also lowers HDL cholesterol. Several other trials show the benefits of replacing saturated fat or carbohydrates with omega-6 fats.

On the negative side, there are numerous research studies that show how omega-6, at the doses recommended above, has a pro-inflammatory effect on several key areas of our body: liver, heart, bowel, joints—in the form of arthritis—and the brain. Nevertheless omega-6 fatty acids play an important role in promoting and regulating the detection and resolution of the inflammation. Some experiments link several of the symptoms of metabolic syndrome to the inflammatory response triggered by high doses of omega-6. It is less certain that omega-6 is the only cause of inflammation, but it certainly plays a role.

Figure 28

Effect of diet on LDL particles



Source: Krauss et al. AJCN 83:1025, 2006

Epidemiological studies

1. Jakobsen et al conducted a thorough meta-analysis of the prospective cohort studies in this area. They concluded that replacing 5% of saturated fats with polyunsaturated fats lowered coronary events by 13%. However, looking at the male and female populations separately, the effect was statistically neutral and only one of the eleven studies showed benefits at a 95% confidence interval.

Also—and most importantly for our analysis—they did not show the composition of polyunsaturated fats used in each study and overall. Without a breakdown of omega-3 and omega-6 it is difficult to draw conclusions on omega-6 alone. If anything, considering that the polyunsaturated fats measured in this meta-study included omega-3, the effect of the omega-6 replacement of saturated fats might have been actually negative (as omega-3 has an anti-inflammatory effect). We also do not know if the saturated fat—measured by surveys of the patients—included some trans fats; if this were the case the analysis might indicate the opposite of the official conclusion: replacing saturated fats with polyunsaturated omega-6 fats might not be such a good idea. A definitive conclusion is therefore impossible.

2. De Goede et al published an interesting study focusing on 20,069 Dutch men and women with data on diet and plasma blood markers collected between 1993 and 1997 with a 10-year average follow-up. In this case, omega-6 and omega-3 were measured separately. The lowest quintile had mean omega-6 and omega-3 intakes as a percentage of total daily calories of 3.6% and 0.4% respectively; the highest quintile 8% and 0.7% respectively. The increase in omega-3 and 6 was at the expense of carbohydrates (47.6% in the lowest quintile and 42.5% in the highest).

In men, a higher intake of omega-6 correlated with lower HDL-c, no change in total cholesterol and statistically no effect on the ratio of TC to HDL. For women, a higher intake of omega-6 led to lower total cholesterol and lower HDL, but statistically no change in the ratio of TC to HDL. Regarding CHD events, a 5% absolute increase in omega-6 replacing carbohydrates led to no statistically relevant effect. The authors concluded that increasing omega-6 from 3.6% to 8% of total calorie intake (a 21% increase in grams per day) had neither a positive nor a negative effect on cardiac events. Unfortunately, the study did not keep omega-3 intake flat (a 55% increase from lowest quintile to highest one in grams per day). Considering the positive effects of omega-3 on CHD events, the neutral conclusion of this study on omega-6 should be taken with a grain of salt.

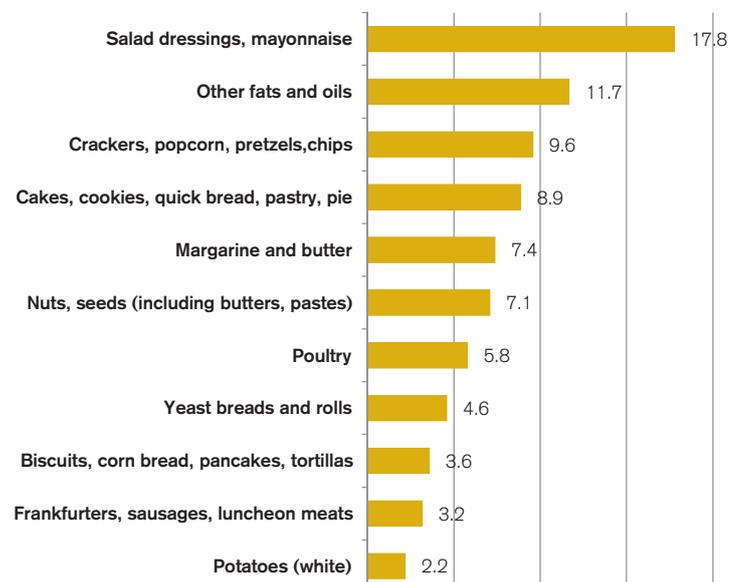
3. Finally, we replicated again the analysis that Hoenselaar carried out a few years ago in Europe; this time assessing the relationship between omega-6 consumption and CVD events on a country-by-country basis (see Figure 40). This analysis—which showed no correlation between saturated or monounsaturated fat consumption and CVD levels—here tells a different story. There is a significant correlation between omega-6 consumption in Europe and CVD events. The r-square of this correlation is 0.53 for men and 0.55 for women. The three countries with the highest omega-6 consumption (Hungary, Bulgaria and Slovakia, all between 8.7% and 11.3%) are also the three countries with the highest level of CVD events: more than 60 per year per 100,000 inhabitants. It worth also noticing that in Hungary and Bulgaria the source of most omega-6 intake is sunflower-seed oil and in Slovakia it is canola oil.

Clinical studies

1. The first relevant clinical study on omega-6 intake dates back to 1965. The Rose study focused on “Corn oil in the treatment of ischemic heart disease.” Patients were divided in three groups: control, olive oil and corn oil. Those in the last two groups replaced saturated fats like butter, milk and eggs with oils. Patients in the oil groups consumed around 60 grams of oil per day, 26–28% of daily calories. After two years, the cholesterol levels in the control group and the olive group were basically unchanged; for the corn oil group the cholesterol level had declined almost 8%. Yet it was of little benefit. The percentage of patients that died or had a re-infarction (fatal or non-fatal) was 48% in the corn oil group and 43% in the olive oil group versus 25% for the control group. The sample was small, 26 people per group, but the results were significant.

Figure 29

Main food sources of polyunsaturated fat: US adults



Source: O'Neil CE, Keast DR, Fulgoni VL III et al. Food sources of energy and nutrients among adults in the U.S.: NHANES 2003–2006. Nutrition 2012

2. Three years later, a team of doctors in London ran a similar experiment with a control group of 194 men eating a normal diet and an “oil group” of 199 men eating less saturated fat and 85 grams of soybean daily, which represented over 30% of daily calories. Again, cholesterol levels fell in the oil group by a substantial 22% in the first six months. In this case, the difference in the level of re-infarction between these two groups was not significant.
3. More recently, in 2009, Mozaffarian et al reviewed eight clinical trials that had been published between 1968 and 1992 in a very well presented meta-analysis. The team’s conclusion was that replacing 5% of saturated fats with polyunsaturated fats reduced the risk of CVD by 19%. It is worth noticing, though, that six out of the eight studies are inconclusive from a statistical point of view—at a 95% confidence interval. The pooling of these studies—common practice in meta-analysis—moves the needle in favor of polyunsaturated. Also, it is worth noting that the two separate studies mentioned above were not included in this meta-analysis.
4. Several academics raised issues with the conclusions of the prior meta-analysis. The most authoritative and detailed came from Ramsden et al in 2010. Ramsden brought up several compelling issues with the meta-analysis we just reviewed based on thorough research on how these studies were conducted and investigating what the patients actually ate. A couple of points bear highlighting: 1. At least four of the studies considered used a mix of Omega-3 and Omega-6 to replace carbohydrates or saturated fats. 2. In several situations, because of the period of most of these

trials, the “substituted” saturated fat was not just that but more likely a mix of saturated fats and transfats. This would make comparisons more favorable to omega-6.

In reviewing the scope of the meta-analysis, Ramsden and team excluded 2 of the studies selected by Mozaffarian and added 2 more: the Rose study and the Sidney Diet Heart study. They separated the 5 studies that used a mix of omega-6 and omega-3 from the 3 that used only omega-6. They also identified which oils were used in each study: mostly soybean oil in the first group and corn and safflower in the second. Their conclusion was that omega-6 alone from corn and safflower oil replacing carbohydrates or saturated fat increased the risk of CVD or cardiac event by 13% and the combined effect of omega-3 and omega-6 decreased it by 22%. This was not the case for soybean oil. Looking at all-cause mortality, the same percentages were a 16% increase and an 8% decrease. These results albeit they were statistically not significant cast a shadow of doubt on the perceived benefits of omega-6.

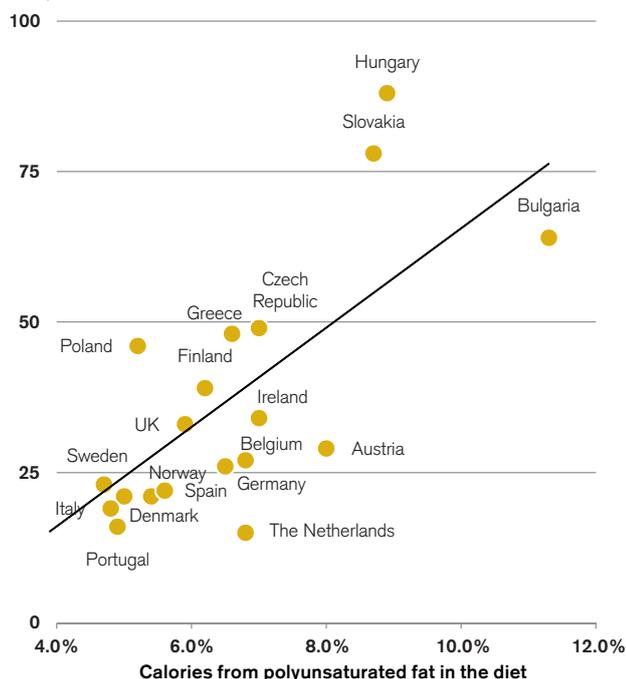
5. A major analysis in 2013 of the Sidney Diet Heart study mentioned above—which involved 458 men who had suffered a myocardial infarction in 1966—showed a significant negative effect when increasing the intake of omega-6 linoleic acid. The experimental group of this randomized control trial involving 221 men increased intake of omega-6 to

Figure 30

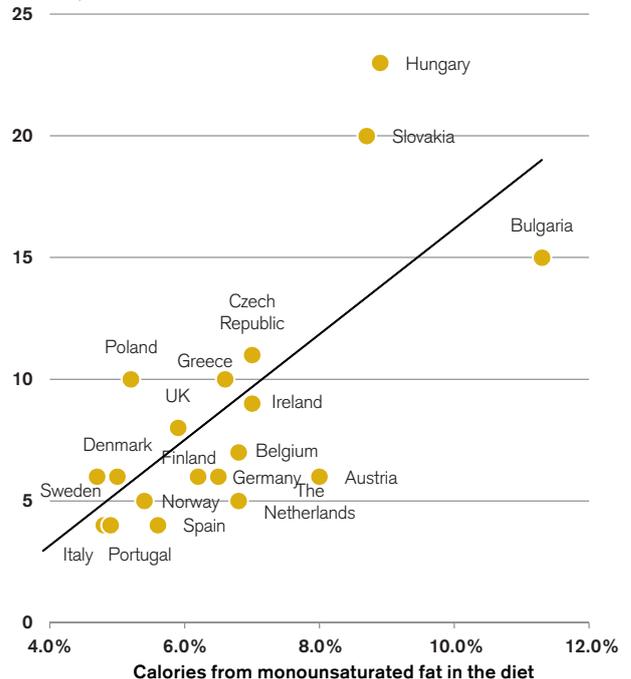
Polyunsaturated fat consumption and CVD deaths by country

Deaths from CVD per 100,000

Men, R² = 0.5106



Women, R² = 0.5359



Source: European Cardiovascular Disease Statistics 2008 and 2012 editions; National Nutritional and Dietary Surveys. Credit Suisse Research

15% of total energy using safflower oil and safflower margarine and reducing saturated fat intake to less than 9.3%. The control group of 237 men had an average omega-6 intake of 8% and saturated fat of 14%. What happened over the following 39 months? The experimental group—that with a high intake of omega-6—showed a higher number of all-cause deaths—18% versus 12% for the control group—and a higher number of deaths from cardiovascular disease—17% versus 11% for the control group.

These results were statistically relevant and showed a 29% higher risk of dying of any cause and a 35% higher risk of dying of cardiovascular disease for those with an average omega-6 intake of 15%. Note that the total cholesterol (TC) of the experimental group was 8% lower than that of the control group and had declined by 13% since the beginning of the study. This further confirms that omega-6 indeed lowers TC; but this does not necessarily mean it lowers CVD risk as well.

What is the right level?

It is difficult to determine the right level of omega-6 intake. But logic might help. As we have seen before, the 5-10% recommended level for omega-6s was more the result of a decision to lower saturated fat. Since 1961 per-capita consumption of carbohydrates in the U.S. has risen 18% and that of solvent extracted vegetable oils by 169%. If you share our view that saturated fats are at worst neutral for our well-being—and more likely, a good source of energy—than there is no reason to maintain the 10% target in omega-6.

- Another way to assess the “right” level of omega-6 intake is to focus on what the ideal ratio between omega-6 and omega-3 intake should be. If we look at our “recent” past—from 100 years ago back to 20,000 years ago—it is very clear that the intake of omega-6 was a lot lower than it is now. The ratio of omega-6 to omega-3 is now 7 to 1 globally; it was probably 3-4 to 1 before vegetable oils became so popular. If we take 1961 consumption data in our model and zero vegetable oil consumption and adjust for a lower—turn of the twentieth century—consumption of maize and wheat which are both high in omega-6, we get a 4 to 1 ratio. So the efforts to justify a much higher ratio in our view get little support from either medical research or from looking at our evolution.
- It is also well known that omega-3 and omega-6 are metabolized by the same enzyme; excess omega-6 leads our bodies to prefer oxidizing omega-6 ahead of other fats. This crowds out the ability of our bodies to transform omega-3 into useful EPA and DHA. Excess metabolization of omega-6 leads to excess production of arachidonic acid which is a pro-inflammatory fatty acid. So should we not just focus on increasing omega-3 and lower the target for omega-6?
- The human race has never been exposed to these levels of omega-6 intake, so genetically we are probably not well equipped to handle them. The current strategy of 10% of energy from omega-6 is not without risk.
- There is research linking “excess omega-6 consumption” to the epidemic of obesity and metabolic syndrome. Massiera et al. published an interesting report in 2010 showing that rats fed excess omega-6 (18% of energy) became fatter than controls at 5-6% omega-6 consumption. Po-Shiuan et al. in 2009 showed that insulin resistance, leptin resistance, fatty liver and high blood pressure are all prompted by excessive inflammatory signaling triggered by high doses of omega-6 in rats. Experiments linking obesity and omega-6 intake on humans have been limited for ethical reasons.
- A recent meta-analysis by Farvid et al., published in August 2014 which reviewed 14 cohort studies, concluded that dietary linoleic acid (an omega-6 fatty acid) intake reduces by 15% the risk of CHD events. However, only 3 out of the 14 studies showed that there was a statistically significant reduction in risk, the other 11 were inconclusive. Also one of the 3 mentioned above compared levels of omega-6 (in the form of linoleic acid) consumption of between 1.1% (lowest) and 2.6% (highest) of total energy. The straight average of all the 14 studies showed a range of omega-6 consumption between 2.7% and 5.6% of total calorie intake. The top average level is clearly well below the 10% recommended by some organizations.
- In addition, there is plenty of literature on the dangers that solvent extracted oils pose to our health. The books of Nina Tetcholz and the Jaminets have a full chapter on them. The focus is mostly on the lack of stability of these oils as they are subject to lipid peroxidation. This is a process which involves the formation and propagation of lipid radicals and leads to the destruction of membrane lipids and at the same time the production of a variety of breakdown products such as alcohols, ketones, alkanes, aldehydes and ethers. Some of these by-products could be extremely toxic if taken in high doses.
- Some European countries have imposed a rule that requires frying oils to be discarded once polar compounds—basically free radicals—represent more than 25% of the frying oil. In experiments run by the USDA in 1997 using frying oils for 8 hours a day and at temperatures of 190 and 204 C, different cooking oils deteriorated at different rates. At 204 C, cottonseed oil reached the 25% TPM (total polar material) limit only in 2 days; for soybean, corn and canola oils, the limit was reached after 3, 4 and 5 days respectively. Beef tallow reached the limit after 13 days. The higher

the content of saturated and monounsaturated fats, the longer it took for the frying oil to get to the 25% TPM limit.

- Companies in the vegetable oil business appear to be feeling some change in consumer perception, while the noise on the effects of high omega-6 intake is gathering some momentum. Recently, the DailyMail in the U.K. published the results of a test on several frying ingredients (olive, corn, sunflower, and canola oils, plus butter, goose fat and lard). In the samples taken from several restaurants, corn and sunflower oil showed aldehydes at 20 times the maximum levels recommended by the WHO. Olive oil, canola oil, butter, lard and goose fat were better.

Food companies that manufacture products requiring a frying process, would love to be able to use more stable fats— with less volatile by-products— and fats that can be re-used multiple times. Two of the largest players in solvent-extracted vegetable oils have for years been developing and expanding the market for an oleic version of soybean oil. There are already “oleic” versions of canola, sunflower, soybean and safflower oils. All are obtained by genetically modifying the seeds, and aim at increasing the monounsaturated component and decreasing the omega-6 component of the oil.

This allows manufactures to produce a solvent-extracted oil more similar to olive oil in its fat structure and which is therefore more stable. For example, the oleic version of sunflower oil has 86% in monounsaturated, 4% in omega-6; in the original version the same percentages are 30% and 59%. Note however that these are still “special” oils, and therefore much more expensive. Production is gradually ramping up and we believe that the general substitution of the omega-6 rich version is on its way.

The recently announced full ban on transfats in the U.S. has renewed the interest in these new oleic versions. As we mentioned before, transfats in the food industry are most likely to be replaced by saturated fats—palm oil for example—which are very stable or modified versions of these oleic oils created by inter-esterification. High oleic solvent-extracted oils have oxidative stability indexes (the time required for the oil to lose its properties when cooking at a specific temperature) of 15-25 hours—normal soybean oil goes up to 6-8 hours—allowing for multiple re-uses of the same oil. In comparison, palm oil has an oxidative stability index of 20-30 hours.

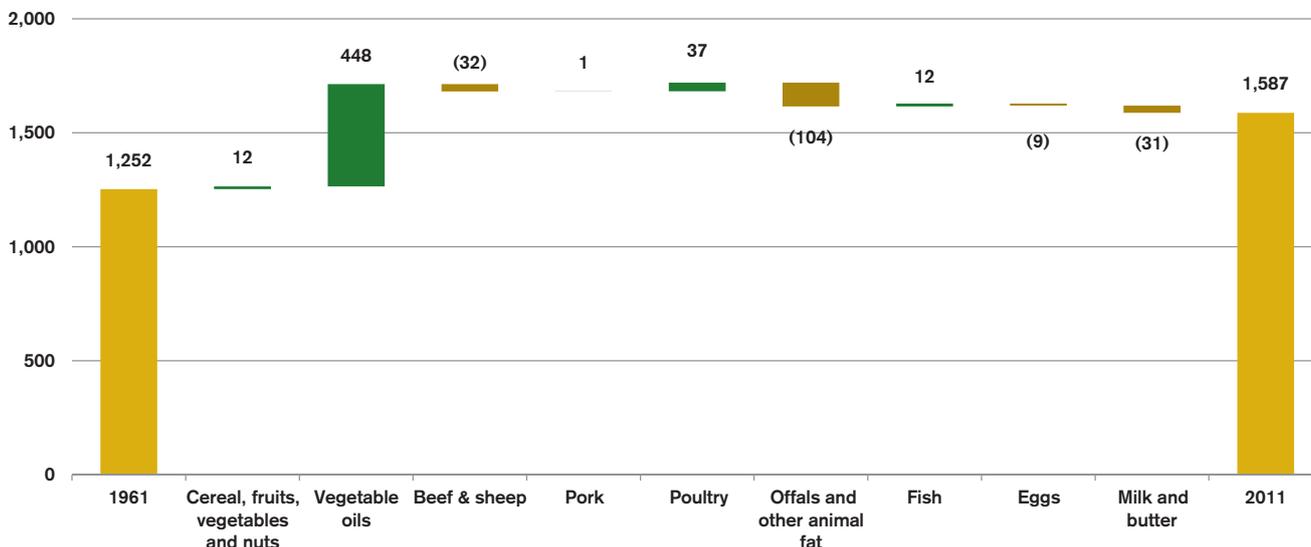
Red meat: the medical research view

We focused this chapter on the medical research on the five main types of fat. We feel red meat also deserves the proper hearing based on sound medical research. Red meat is a controversial food that has traditionally been associated with heart disease due to its relatively high content of saturated fat. In the case of ground beef—75% lean for example— saturated counts for 47% of total fats, but then only 10% of the total weight. Red meat has also been linked to colorectal cancer and to a higher mortality rate. We discussed saturated fat as a factor in CVD risk, but what about meat? Red meat is a nutrient-dense food that contains proteins, significant amounts of important minerals (like iron, magnesium and zinc) and key vitamins (like B12).

As we mentioned previously, humans have been eating red meat throughout evolution and have digestive systems that are well equipped to handle it. Some traditional societies like the Masai depend almost exclusively on meat and other products of animal origin (mostly milk).

Figure 31

U.S. fat consumption – calories 1961-2011



Source: FAOSTAT, Nutrition Data, Credit Suisse Research

However, due to the “evolution” of cattle raising techniques and the processing of the end-products, the meat consumed by most modern urban societies is vastly different from the meat consumed by traditional populations and by our ancestors. Therefore, it is important to distinguish between different types of red meat:

- **Conventional Red Meat:** relatively unprocessed but the livestock are factory farmed. Traditionally grain-fed.
- **Processed Meat:** Products usually from conventionally raised cows which then go through processing methods, for example frankfurters and bacon.
- **Grass-Fed, Organic Meat:** This meat comes from animals that have been born and raised on certified organic pastures (free of persistent pesticides or chemical fertilizers), never receive antibiotics or growth-promoting hormones and are fed only certified organic grasses.

Dietary guidelines around the world recommend limiting consumption of red meat and/or replacing it with white meat or fish. The main concern is that some red meats are high in saturated fat, which raises total cholesterol and LDL blood cholesterol, which in turn has been widely considered to increase the risk of heart disease. We have already discussed the subject of saturated fat and heart disease extensively.

Red meat has also been associated with a greater risk of colorectal cancer. Two cohort studies conducted in the 1990's (Willet et al and Giovannucci et al) found a higher risk of colon cancer among those who eat red meat. However such studies pool together processed and unprocessed red meat. On the other hand, two other meta studies, one that looked at data from 35 studies (Alexander and Cushing) and the other from 25 studies (Alexander, Weed, Cushing and Lowe), found that the effect of unprocessed red meat was very weak for men and nonexistent for women.

The men that consumed higher levels of red meat had a 21% higher risk than those who eat little meat; for women just a 1% higher risk. In addition, the authors concluded that the available epidemiologic data were not sufficient to support an independent and unequivocally positive association between red meat intake and CRC due to heterogeneity across studies, inconsistent patterns of associations across the subgroup analyses, and the likely influence of confounding by other dietary and lifestyle factors.

Other studies show that it may not be the meat itself that is contributing to the increased risk of cancer, but harmful compounds that form when the meat is cooked (Cross, Sinha in 2002). When meat is cooked at high temperatures, it can form compounds such as Heterocyclic Amines (HAs), Polycyclic Aromatic Hydrocarbons (PAHs) and Advanced Glycation End-Products (AGEs). This doesn't just apply to meat; other foods can also form harmful compounds when heated excessively. Gentler cooking methods, like stewing and

steaming instead of grilling and frying, can help avoid the issue.

In a more recent study conducted by Harvard School of Public Health researchers in 2012 found that red meat consumption was associated with an increased risk of total mortality (Pan, Sun, Bernstein et al). They found that a single serving of unprocessed red meat daily is associated with a 13% increased risk of death from all causes, while a single serving of processed red meat increased total mortality by 20%. Four servings of red meat daily raise the risk of mortality to 70%.

An earlier epidemiological study of over half a million people found similar results (Sinha, Cross, et al in 2009). Critics of these studies claim that while they can prove correlation that does not prove causation and that they lack controls of confounding variables (smoking, exercising, body mass index, with a bias of meat eaters in general having a “less healthy” profile than non-meat eaters).

It is hard to dismiss the nutritional value of red meat. In addition to 20 grams of protein, a 100 gram portion of raw ground beef (10% fat) contains vitamin B3 (25% of the recommended dietary allowance or RDA), vitamin B12 (37% of the RDA), vitamin B6 (18% of the RDA), iron (12% of the RDA), zinc (32% of the RDA) and selenium (24% of the RDA), as well as other vitamins and minerals in smaller amounts. Red meat is also rich in nutrients like Creatine and Carnosine, which are important for muscle and brain function. Grass-fed beef is even more nutritious than grain-fed, containing Omega-3s, and more A and E vitamins. Several studies shows that grass-fed beef contains between 2 and 5 times the level of omega-3 relative to grain-fed beef and a more favorable ratio of omega-6 to omega-3—2-3 for grass-fed beef versus 4 for grain-fed beef.

As with many other foods and nutrients there is a great deal of consumer confusion and contradictory recommendations, as scientific studies find it challenging to isolate the impact of consumption from lifestyle factors and substitution for other foods. In addition, given the current industrial treatment of meat products, it is also difficult to separate the impact of red meat itself from the cattle raising/feeding practices and additives put into the final product. The probability of suffering potential adverse effects can probably be lessened by following safe cooking practices and trying, to the extent possible, to avoid industrially raised and overly processed meat.

The voice of medical professionals

As with our report “Sugar: Consumption at the crossroads” we decided to survey a group of doctors to see how much their views were aligned with most recent medical research and the recommendations of health authorities. We surveyed 151 doctors; 44% based in North America, 33% in Europe and 23% in Asia. In terms of specialization, 33% are general practitioners, 50% specialized in nutrition and 17% in lipidology and obesity. Collectively this group of doctors sees 37,000 patients a month, a relevant sample.

In general, most doctors’ views in the area of nutrition are closer to the recommendations of the health authorities. They understand the role of the three main macronutrients, but they are often unaware of the most recent research on the topic. The negative perception of saturated fats is still prevalent among many doctors and beef, pork and butter are widely considered a source of “bad” fats. Here are the most interesting findings:

General nutrition

In term of macro nutrients, 45% of the doctors surveyed said that their perception of protein has improved, versus only 5% saying it has worsened; 29% of the doctors said that their perception of fat has improved versus only 7% saying it has worsened; and 15% only said that their perception of carbohydrates has improved versus 26% saying it has worsened.

Answering what makes you fat if eaten in large quantities, the doctors correctly pointed to sugar and carbohydrates (32% and 26%); fat and saturated fats are not as bad (23% and 16%) and protein collected only 2% of the responses.

However, the doctors believed that the best diet for weight loss is a low calorie one (65%), followed by low carbohydrate (36%) and low fat (7%). Among nutritionists, 42% prefer the low carbohydrate diet, against 30% for the general practice group.

Fat and saturated fat

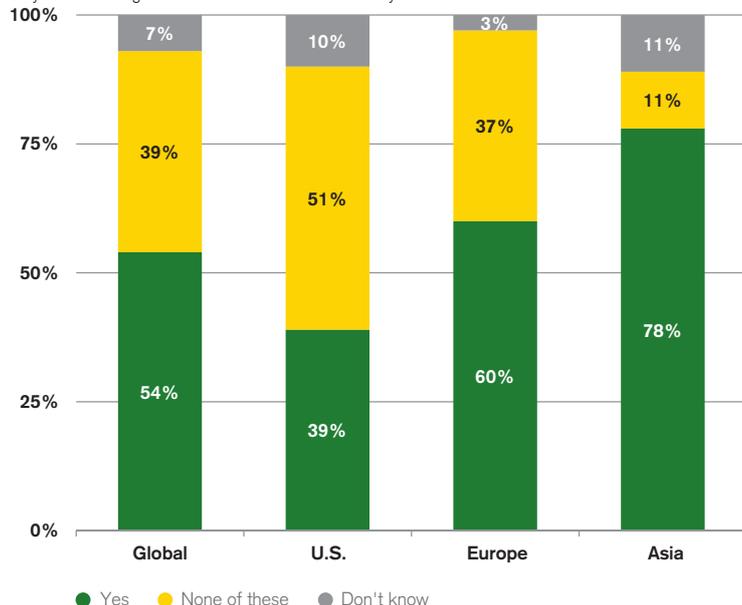
Among the doctors we surveyed, 69% believed that the right percentage of fat in the diet should be in the 20-35% range; 19% said that it should be in the 10-20% range and 13% that it should be above 35%.

For 92% of the doctors excess fat consumption could lead to cardiovascular issues, followed by 87% suggesting obesity as a consequence, 74% type 2 diabetes and 64% high blood pressure. These figures were broadly the same for general practice doctors as well as nutritionists.

Figure 32

Perception of diet components

Do you think eating foods rich in cholesterol is “bad” for your heart?



Source: Credit Suisse, Doctors’ Survey

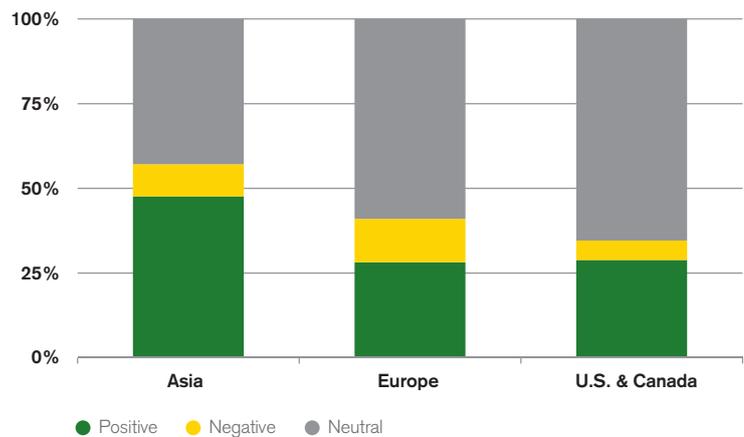


Let us go a little deeper into vegetable oils, which 66% of the doctors surveyed believed are beneficial to our health. Olive oil was considered the healthiest by far, with 64% of the doctors ranking it as the best. Olive oil was followed by canola oil (22% of the doctors selecting it as the best and 33% as second best), and sunflower oil (7% selected it as the best and 19% as second best).

At the other end we find palm oil and coconut oil, which 40% of the doctors placed in “least healthy” group, followed by soybean oil with 11%. Surprisingly, even in Asia where palm oil and coconut oil are more commonly used, local doctors’ perceptions of these two oils does not change.

Figure 33

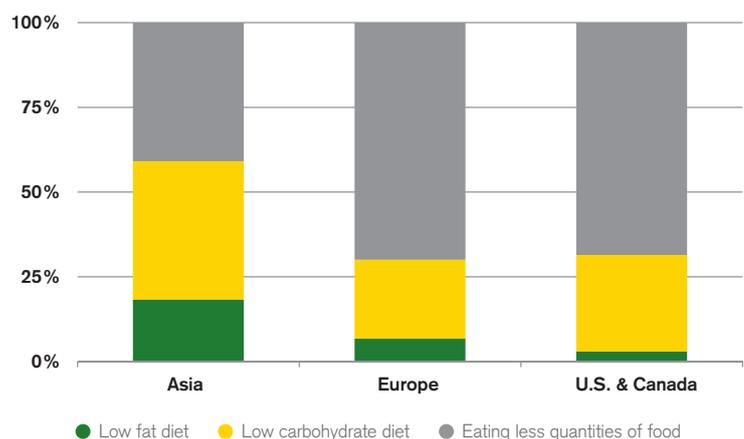
Changes in attitude towards fats



Source: Credit Suisse, Doctors’ Survey

Figure 34

Diet suited to losing more weight



Source: Credit Suisse, Doctors’ Survey

The large majority—78% of all doctors—felt that omega-3 fats were beneficial; for omega-6 the same percentage was 41% and for saturated fats just 9%.

Among fats, more than 80% of the doctors believed that saturated fats and trans fats are linked to obesity and more than 89% stated that both are linked to cardiovascular issues.

54% think that eating cholesterol rich foods raises blood cholesterol and again 54% believe that eating cholesterol-rich food is not good for our heart; 39% feel that this is not the case. Nutritionists score slightly better, with only 40% believing that dietary cholesterol raises blood cholesterol, versus 68% for the general practice sample. This is a clear example of the level of misinformation that exists among doctors.

Food and ingredients

We asked the doctors to identify among “fat” foods which ones contain good fats and which ones contain bad fats. Beef, pork and butter topped the list of the foods containing “bad” fats, as suggested by 80% of the doctors (see Figure 42 to Figure 44). Shockingly to us, butter was considered worse than margarine; 83% of the doctors thought butter was a “bad” fat versus 73% for margarine. Among the “good” fats, fish and nuts collected more than 90% of the votes, followed by vegetable oils with 81% and chicken with 67% of the votes. Eggs and milk were in a neutral territory.

Consumers' behavior and perceptions

In the previous sections, we touched indirectly on consumer behavior. We saw how saturated fat consumption in the developed world has declined steadily as a percentage of total calories since the 1960s, replaced mainly by carbohydrates and vegetable oils rich in omega-6. What has driven this change? What do people think now, fifty years later?

The failure of the low fat diet promoted in the 1960s to control obesity and the trans fats issue have led some people to rethink their views on fat and saturated fats. How big a change? We conducted a proprietary survey of consumers' perceptions on fat as well as their purchasing intentions to see if a new trend is emerging in the consumption of fat. We focused on 2,846 individuals (51% male) in six countries: the U.S., U.K., China, India, Japan, and Indonesia (the survey was run by Luc. id Consumer Data).

In general, consumers' views are similar to those of the doctors and clearly influenced by the recommendations of health authorities. The negative perception of saturated fats is still prevalent among the public, but they have a much better perception of milk and eggs and a much worse view of margarine. But this is changing quickly. As we will see later on, sales of whole milk in the U.S. for the first half of 2015 were up 11% in volume terms, and skim milk down 14%. Butter and eggs showed similar trends. Here are the most interesting findings from our customer survey:

Macronutrients

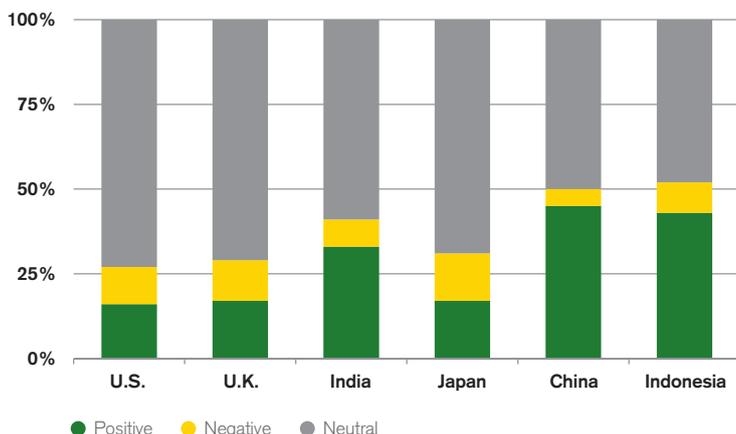
In terms of macro nutrients, 90% of consumers believe that protein is good for their health and 60% are more likely to increase protein intake. Regarding fat, 29% think that it is good for their health, but only 15% are likely to increase its consumption. Surprisingly, 60% think that carbohydrates are healthy and 32% are likely to consume more; only 23% though think that sugar is healthy.

Answering what causes you to gain weight, consumers pointed basically equally to saturated fat (29%), sugar (26%) and fat (24%). Carbohydrates are "blamed" by only 18% of those surveyed. Here doctors are clearly better informed. Protein collects only 4% of the responses.

Consumers believe that the best diet for weight loss is a low calorie one (53%), followed by a low-fat diet (24%) and low carbohydrate diet (16%). Geographically, Asia is the big outlier with 42% of people interviewed considering the low-fat diet the "right" one, 31% pointing to the low-calorie one and only 20% to the low-carbohydrate one.

Figure 35

Perceived effects of cholesterol intake on heart



Source: Credit Suisse, Luc. id Consumers Data

Fat and cholesterol

Those who felt fat was a healthy nutrient highlighted it as a good source of energy and a key part of a balanced diet. Those who were negative about it pointed to the fact that it causes weight gain as the main reason to limit or reduce consumption.

Among consumers, 66% believe that eating foods rich in cholesterol negatively affects our hearts and 78% felt that eating cholesterol-rich foods raises blood cholesterol. This is not surprising given that most health authorities have misinformed the public on this topic for over fifty years. Plenty of "not-very-healthy" foods still carry the "No-Cholesterol" heart symbol on their package.

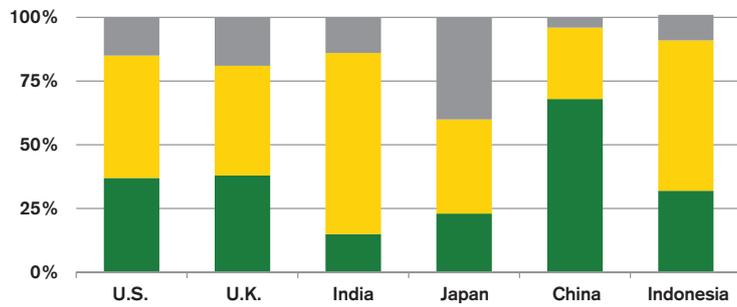


Figure 39

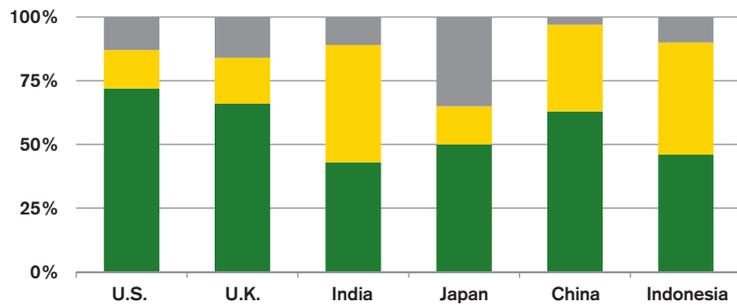
Perception of component fats

Beef

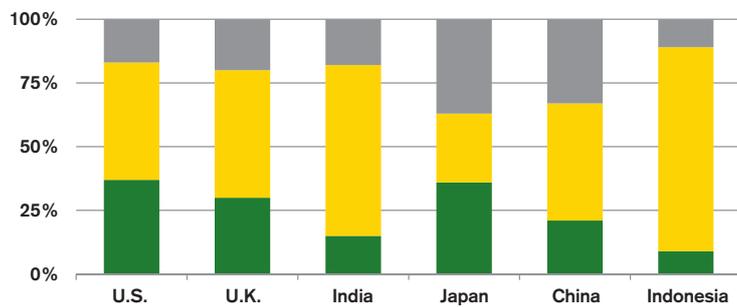
● Contains more good fats ● Contains more bad fats ● Don't know



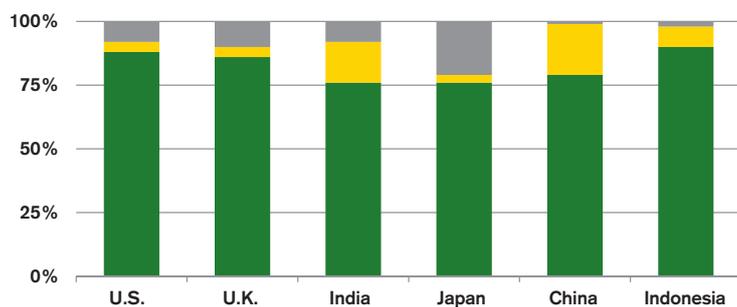
Chicken



Pork



Fish



Source: Credit Suisse, Luc. id Consumer Data

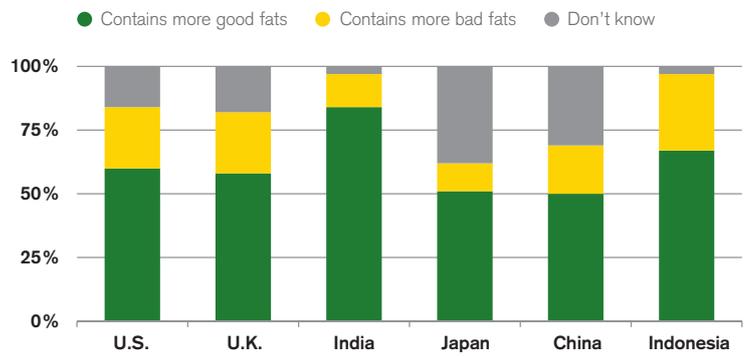




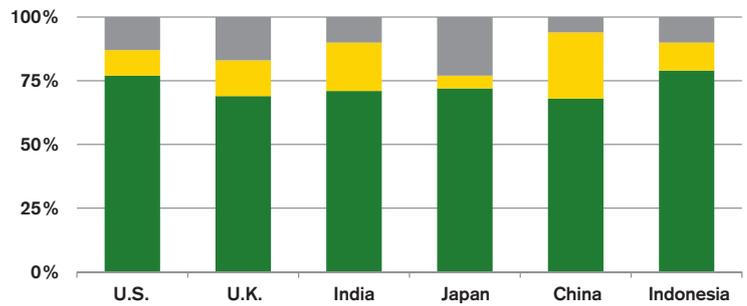
Figure 40

Perception of component fats

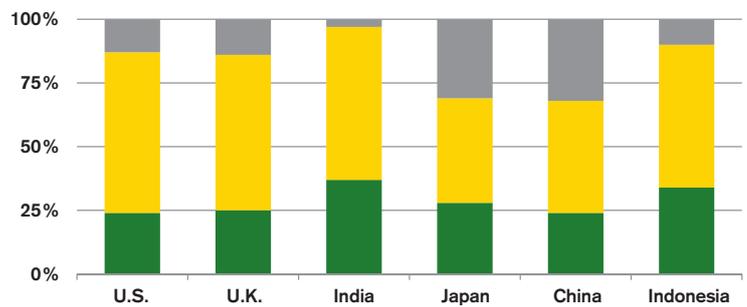
Milk



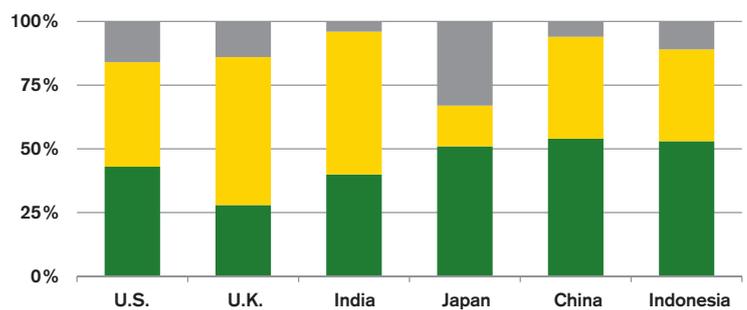
Yogurt



Butter



Cheese



Source: Credit Suisse, Luc. id Consumer Data

Figure 41

Perception of component fats



Source: Credit Suisse, Luc. id Consumer Data

Food and ingredients

We asked consumers to identify which “fat-rich” foods contain more “good” fats and which ones contain more “bad” fats. Among the “good” fats, fish ranked the highest, with 84% of the consumers interviewed saying that it contains “good” fats and only 7% saying that it contains “bad” fats. Nuts were the next “best”, with 76% of consumers placing them among “good” fats and only 12% among “bad” fats.

On the dairy front, not surprisingly 72% of consumers believed yogurt contains more “good” fats and only 13% suggested the opposite. But over 60% of consumers said that they will purchase more low-fat yogurt compared to 22% in favor of the full-fat version. On milk consumers are more positive than doctors: 62% believe it contains more “good” fats and 22% more “bad” fats. On whole versus skim milk, 39% of consumers suggested they will buy more whole milk and 49% more skim milk.

On cheese, 38% believed it contains more “good” fats versus 47% in the more “bad” fats camp. Finally on butter, 27% believed it contains more “good” fats and 59% more “bad” fats. This is slightly better than margarine where the split is 22% “good” fats and 60% “bad” fats.

Regarding meat, chicken is a clear winner, with 63% of consumers suggesting it contains more “good” fats and 22% pointing to more “bad” fats. Beef counted 35% of consumers in the “good” camp and 47% in the “bad” camp. Pork was close, as 31% of those surveyed felt it has more “good” fats, versus 51% suggesting the opposite. Eggs scored pretty well against a negative opinion on cholesterol: 65% thought they contain more “good” fats and 18% believe the opposite.

On vegetable oils, 50% of consumers thought they contain more “good” fats and 34% thought they contain more “bad” fats. Doctors have a slightly better opinion. But different oils score differently and we see also geographical factors influencing consumers’ views.

Olive oil is considered the healthiest oil by far, by 76% of consumers. Olive oil is followed by sunflower oil (65%), coconut oil (60%), and soybean oil (56%). Canola oil and palm oil both have less than 50% of consumers considering them healthy choices: 44% for canola and 40% for palm oil. This varies a lot across regions: for example in India 70% of consumers consider soybean oil healthy, versus 54% in the U.S.



Regulators and healthcare costs

As we have seen, nutritional science is not a simple area. The separation of nutritional and medical science and the difficulty of conducting randomized trials with large groups of people for long periods of time makes conclusions far from definitive. The relative risks or benefits of one option versus the other—say saturated fats versus omega-6—is measured in a few percentages and not multiples.

This contrasts with a community of health organizations—global and national—which shows no shadow of a doubt when recommending what type of fats or carbohydrates we should take and in what quantities. See below the list of recommendations from WHO, USDA, AHA, EFSA, and many more organizations. Particularly noteworthy in this context is the parallel that the AHA draws between transfats and saturated fats, the bad guys. Based on what we have seen, does this make sense?

Such a level of certainty and high conviction is not matched by the medical research on this topic and one would expect recommendations to be sprinkled with prudence and several caveats (e.g., based on most recent research; other research points to... etc.).

We look initially at two hypotheses that might explain such high levels of conviction, and then consider a third—and in our view more likely—hypothesis at the end:

1. Health authorities know a lot more than the medical research community.
2. Health authorities have always been right in the area of nutrition (so we should simply follow their advice).

On the first point we defer to a detailed paper by Robert Hoenselaar published in 2011, which focused on one hot topic: the mismatch between saturated fat recommendations by health authorities and medical research on the topic. He uncovers systematically how health authorities cited the papers that supported their previous stance and ignored those that did not. This is termed selective bias, and entails a great deal of risk when applied to medicine and nutrition.

Here are further examples. The USDA/USD-HHS draft report published in 2015, recommends saturated fat consumption below 10% and suggests that saturated fat should be replaced with unsaturated fat, particularly polyunsaturated fatty acids. On a positive note, the draft paper proposed to drop the recommendation to limit daily cholesterol consumption to less than 300 mg and to lift the existing recommendation on total fat content (20-35%). This is important, as lifting the upper limit on fat will allow manufacturers to develop products with higher fat content and less sugar or refined carbohydrates (any product with more than 35% fat cannot use the word “healthy” on the label, even if the fat comes all from nuts or some other acknowledged healthy source).

The report quotes 220 papers as the basis for the recommendations. There are 18 papers quoted that refer to saturated fat and CVD risks: nine focus on the effects of the Mediterranean diet, five on the effects of vegetarian diets, and only four on general research on the topic. Research by Krauss, Volek and others is not even mentioned and obviously not taken into account.

The EFSA (European Food Safety Authority) scores a little better. Its latest report dates back to 2010, so obviously the most recent research has not been considered. Its recommendation on saturated fat is largely consistent with the recommendation of the health authorities of its member countries and suggests an intake of less than 10%. The research quoted to support this conclusion in the text is mostly epidemiological.

What is interesting is the recommendation that the adequate omega-6 intake should be 4% of total energy, but does not set an upper limit. This is the result of a compromise: DACH-2008 (Germany, Austria, Switzerland) sets it at 7-10% for both omegas; The Nordic Nutrition Recommendations-2004 suggests 4-9% omega-6; UK COMA-1991, 6%; France's AFSSA-2001, 4%, the Netherlands GR-2001, 2%.

So the spread is 2% to 9%, which indicates a great deal of uncertainty on what the target should be. Are they accessing different research from the USDA/USDHHS board? Also, it is interesting to see that the highest omega-6 recommendation in the Nordic Countries of 4-9% has a caveat alongside it "Higher levels of omega are not recommended because of potentially harmful effects of very high intakes."

Finally, it is worth reviewing the dates of the contribution recommendation from the member countries: from 1991 to 2008. This highlights another problem: in a field in which research—particularly clinical randomized trials—is progressing rapidly, the public health bodies should meet more often and review their recommendations with higher frequency. Caveats should be acceptable and make the public understand that this is not a precise science.

Let us focus briefly on the second hypothesis: "recommendations by health authorities should be accepted at face value because they have been always right."

We have already covered the mistakes made by the health authorities in the 1960s and 1970s. Saturated fat was blamed for the "epidemic" of heart attacks in the U.S. and the general public was persuaded to reduce saturated fat and get more carbohydrates—up 18% per capita since 1961—and more omega-6 rich vegetable oils—up 169% per capita. In the meantime, saturated fat consumption went down 17% for men and 2% for women.²

The results of these recommendations are a population in which obesity has risen from 12% to 34% and metabolic syndrome has risen gradually to affect 35% of the U.S. adult population. Leaving aside the number of lost lives, the current cost to the system from obesity and metabolic syndrome in the U.S. is now estimated at over US\$150 billion a year in medical costs alone, a costly mistake.

Here is our final hypothesis on why health authorities have remained so certain of their position and unwilling to change their view on saturated fats, omega-6 or carbohydrates:

3. Health authorities advance very slowly and are afraid to change the market's status quo (not a wise medical posture).

Figure 42

Regulators versus medical research

Nutrient	Regulators' View / Recommendations	Medical Research View
Fat	○	●
Saturated Fat	●	●
Monounsaturated Fat	●	●
Omega-3	●	●
Omega-6	●	○
Trans-Fats	●	●
Cholesterol	●	●
Carbohydrates	●	●
Proteins	●	●

● Recommended intake/ viewed positively
 ○ Neutral view
 ● Recommended to limit/ viewed negatively

Highlighted are nutrients where there is a difference between regulators recommendation and medical research view

Source: Credit Suisse, Dietary guidelines for Americans. <http://www.health.gov/dietaryguidelines> and other regulators around the world (see regulation section)

We have known since the 1960-70s that dietary cholesterol has no influence on blood cholesterol. Yet it took more than fifty years for the USDA/USDHHS to lift recommended upper limits of fat consumption. It took close to 20 years in the U.S.—that was quick—to ban trans-fats. So we should not look at public health authorities as leading indicators of potential health hazards, but rather as lagging behind.

Bureaucracy tends to move slowly, but when the health risks tied to "incorrect" information are so high, one would hope for swift action and the courage to reverse past mistakes. There was no fundamental reason to move from butter to solvent extracted vegetable oils. If we assume that research was the main reason—as it was claimed at that time—the health authorities now have enough information to change their recommendations, or if still in doubt issue no recommendations.

2 Based on FAOSTAT data



The fat trade

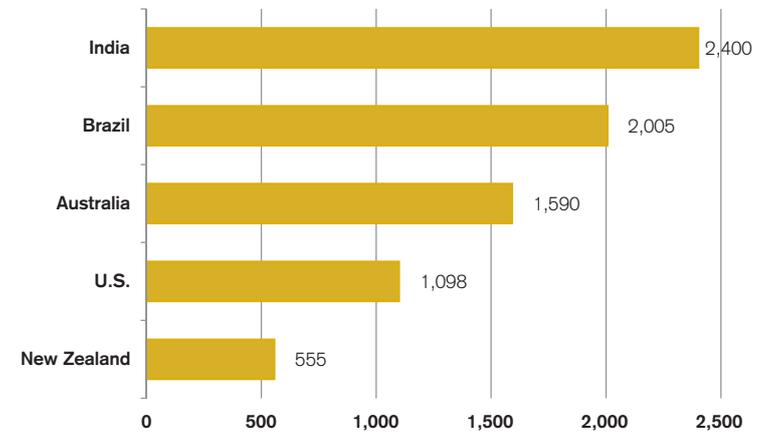
Which are the countries that dominate the 'fat trade'? Depends on the product. As we can see in the following tables, most countries' production closely matches demand. This is pretty logical as transport represents a significant part of the cost of most of the products we eat to get our fat calories. This is common to most agricultural products where the value added in the production process is limited. Vegetable oils are obviously the easiest to move around, as they do not require refrigeration and can be transported in bulk. Processed meat like hams or salami is next. Butter, cheese and to a certain extent milk are easy to handle but in some cases need refrigeration if not treated (e.g. by UHT). Fish and meat are more complicated and require specialized transport. Eggs, well not so easy...



Figure 43

Main beef exporters by country

Thousands of metric tons

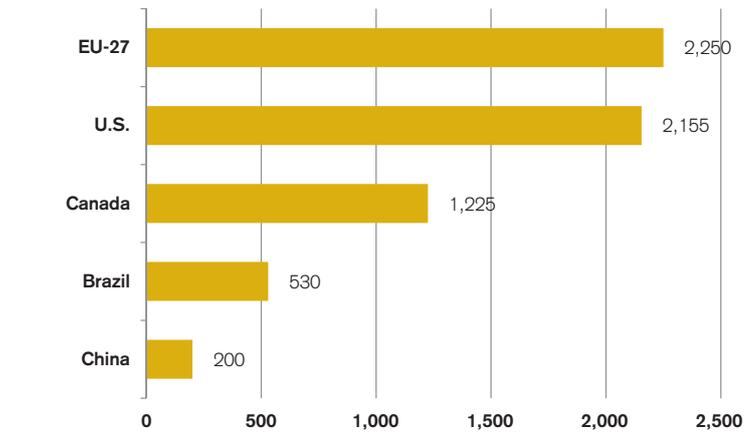


Source: Indexmundi

Figure 44

Main pork exporters by country

Thousands of metric tons

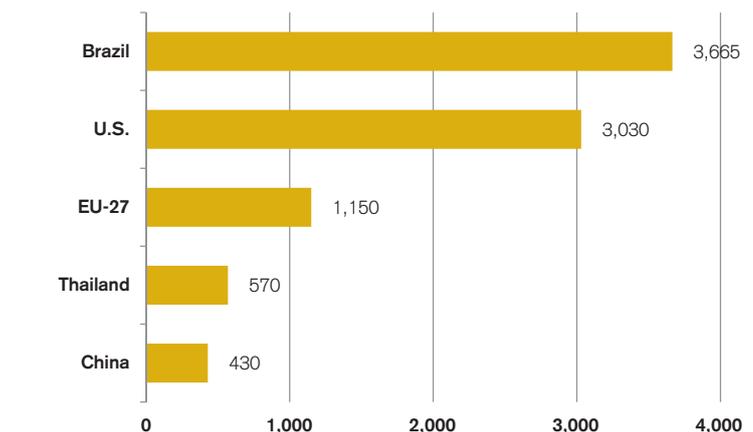


Source: Indexmundi

Figure 45

Main poultry exporters by country

Thousands of metric tons



Source: Indexmundi

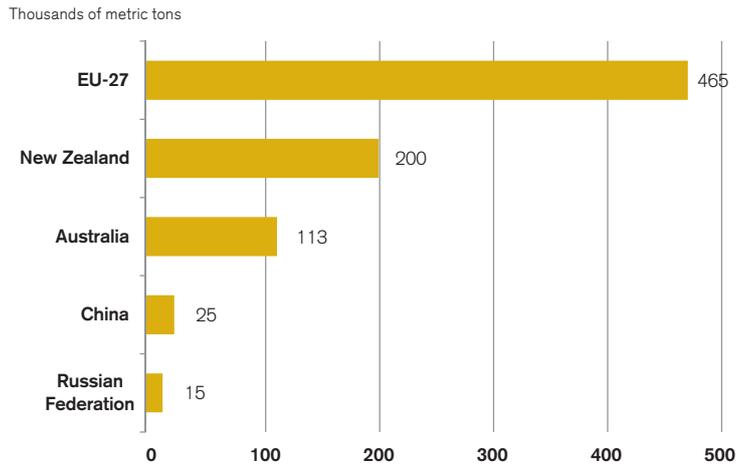
In terms of meat, the largest exporters of beef³ are India, Brazil, Australia and the U.S. These are the only countries with over 1,000MT in exports. Together represent over 70% of beef exports. The largest producer of beef is the U.S., although most of the beef is grain fed, which is not ideal, as we will see later, due to the imbalance between omega-6 and omega-3 fats. Most of the grass-fed beef is produced in Australia, Brazil and Argentina. As we will see later, grass-fed beef contains on average a ratio of omega-6 to omega-3 fats of 3 to 1, while for grain-fed beef the same ratio is on average 6 to 1. The main reason behind this is that grass—fresh green grass—is extremely rich in omega-3s, while grains—particularly corn—are rich in omega-6s.

For pork³, Europe, the U.S. and Canada account for over 80% of exports, but China accounts for over 50% of global production and consumption. Production in China is more than double that of the European Union. For poultry, Brazil, the U.S. and Europe account for 75% of total exports, but China again tops domestic production and consumption. Chicken³ production has gone through a major transformation over the past 50 years. It now takes only 35-40 days to get a broiler chicken in special feeding pens to get to a weight of 2kg, compared to 68 days in the 1960s. The difference between “caged” and free range chicken is not just the time to slaughter (35 days versus 80 days). The fat composition is also different: the meat of free range (not caged) chicken has higher levels of saturated, monounsaturated and omega-3 fats and much less omega-6s.

3 For a detailed industry review of the meat industry, please refer to our report “Latam Food: What about a Protein Diet?” published in July 2015 by Vincenzo Paternostro.

Figure 46

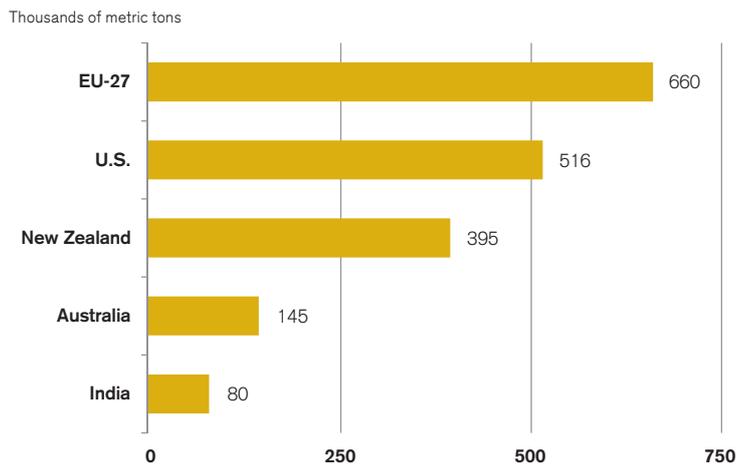
Main fluid milk exporters by country



Source: Indexmundi

Figure 47

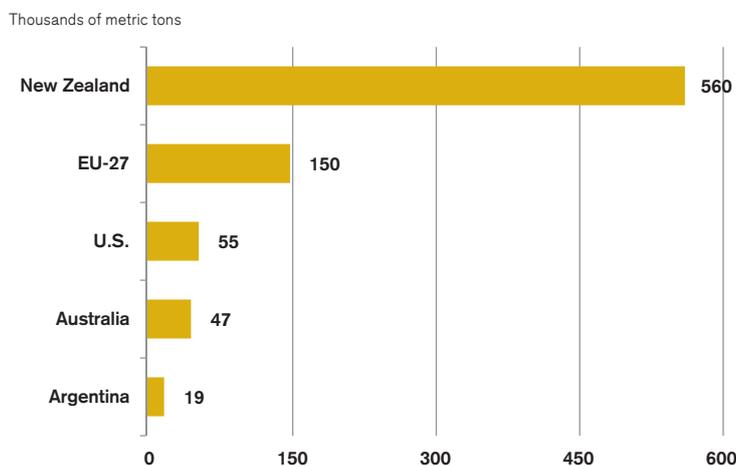
Main dry milk exporters by country



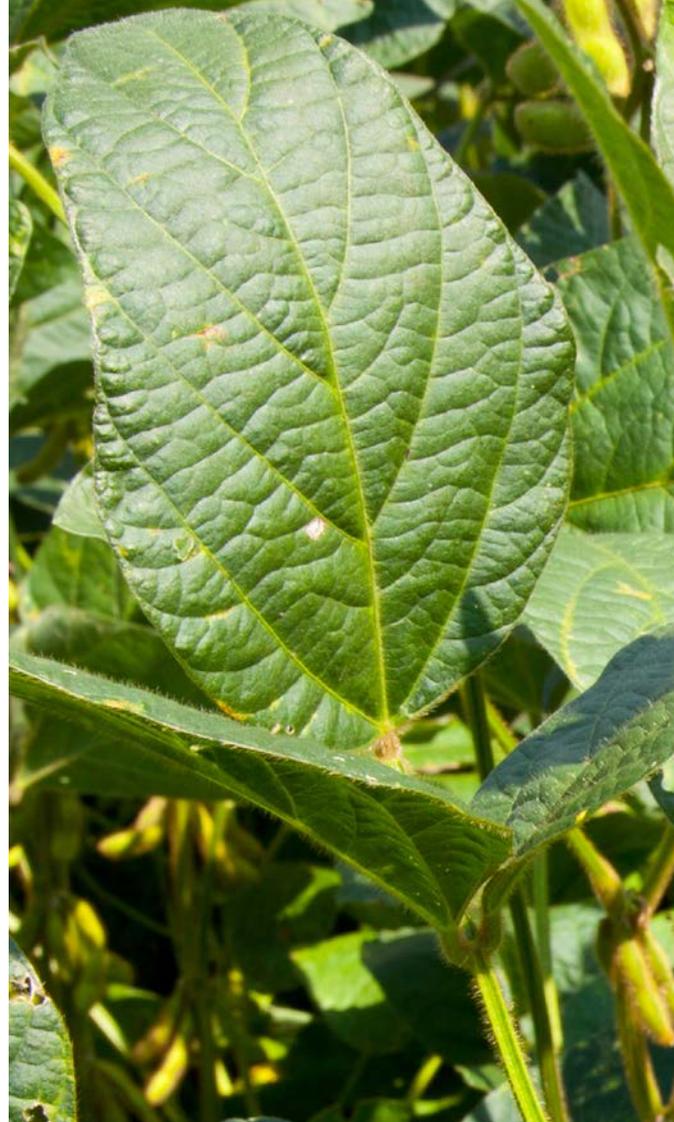
Source: Indexmundi

Figure 48

Butter exporters by country



Source: Indexmundi



Eggs tend to be mostly domestically produced and consumed. The largest producer of eggs is clearly China (490 billion eggs a year), followed by the U.S., India and Mexico. Here, too, there is a significant difference between cage-free eggs and pen-fed eggs. Cage-free eggs show omega-6 accounting for 16% of total fat and omega-3 for 1.2%; for pen-fed eggs, the same percentages are 18% and 0.7%. The focus on the level of omega-3 and omega-6 in eggs has led to a new egg “product” with a much higher omega-3 content made by feeding the chicken fish oil or flaxseed, although the resulting eggs have been criticized as having a “fishy” taste.

Fish capture has been quite stable over the past 10 years, declining slightly from 96 million metric tons in 2000 to 93 million metric tons in 2010, a 3% decline. But aquaculture has grown steadily from 35.5 million metric tons a year to 53 million tons a year in the same period, a 49% increase—36% of the fish, crustaceans and mollusks we consume now are farmed. More than 80% of the world aquaculture production is located in Asia, and China alone accounts for over 60% of world production, followed by Indonesia at 8% and India at 6%.

On the dairy front, New Zealand alone accounts for 66% of dry powder whole milk and EEUU and New Zealand together account for 78% of liquid milk exports. Note, though, that powder exports account for 83% of all milk exports. Europe con-



trols 30% share of all milk production (50% more than the U.S.), the bulk of which—almost 75%—contributes to Europe’s unmatched leadership in cheese and yogurt production. New Zealand and Europe (mostly Ireland) account for 83% of all butter exports, and four countries alone—India, Ireland, New Zealand and the U.S.—are responsible for 70% of the world’s butter/ghee production.

As is the case with beef, the fat profile of milk and other dairy products depends on what the cows eat. Milk from grass-fed cows contains between 2.5 and 5 times the levels of omega-3 of the milk of grain-fed cows and presents a better omega-6 to omega-3 ratio: 1.5-2 for grass-fed cows versus 4-13 for grain-fed cows. Grass-fed milk also contains 1.5 to 2.5 times higher levels of conjugated linoleic acid (CLA), which helps reduce insulin sensitivity. In a few studies, CLA has also been shown to increase HDL with no impact on LDL and reduce inflammation markers. Not surprisingly, in the U.S. organic grass-fed milk sells at a 150% plus premium to “normal” milk (\$3.99 versus \$1.39 per quarter of a gallon).

Vegetable oils deserve a more detailed analysis. For the purposes of this report, we have divided them into two groups: 1. Natural processed oils (olive, palm, palm kernel, coconut and groundnut oil) where the oil is obtained by pressing the fruit or heating/boiling it, and 2. Solvent-extracted seed oils (soybean, canola, sunflower

and cottonseed). This division almost replicates saturated versus unsaturated fats, with the exception of olive oil, which is mostly monounsaturated.

Natural oil consumption has been growing at 1.3% a year over the last 50 years; solvent-extracted oils at 2.3% a year. Natural oils represent 35% of all vegetable oil consumption; these oils have a high level of saturated fats (66% of total fat at current global consumption levels) and low omega-6 (11%). The balance—65%—is made up by solvent-extracted oils, which are low in saturated fats (14% of total fat) and high in omega-6 (44%). Palm oil dominates in the first group, soybean oil in the second. Together they account for over 60% of global demand.

Country or regional dominance in these oils varies substantially: Europe dominates in olive oil exports, Ukraine and Russia in rapeseed oil, Philippines and Indonesia in coconut oil, Malaysia and Indonesia in palm and palm kernel oil, Argentina, Brazil, the U.S. and Europe in soybean oil, and Canada in rapeseed (canola) oil.

Soybean oil versus palm oil

Soybean oil has a higher cost of production than palm oil, but the supply of raw materials—soybeans—can be managed quite effectively. Argentina, for example, produces two crops a year, and production can be tailored to the expected demand: crops can be planted and then collected just a few months later. The raw material of palm oil takes much longer to develop: a palm tree does not produce fruit for the first three years and reaches peak production only 8-10 years

after planting. Yet, production costs for palm oil in Malaysia and Indonesia are 35% lower than those for soybean oil in Argentina and Brazil.

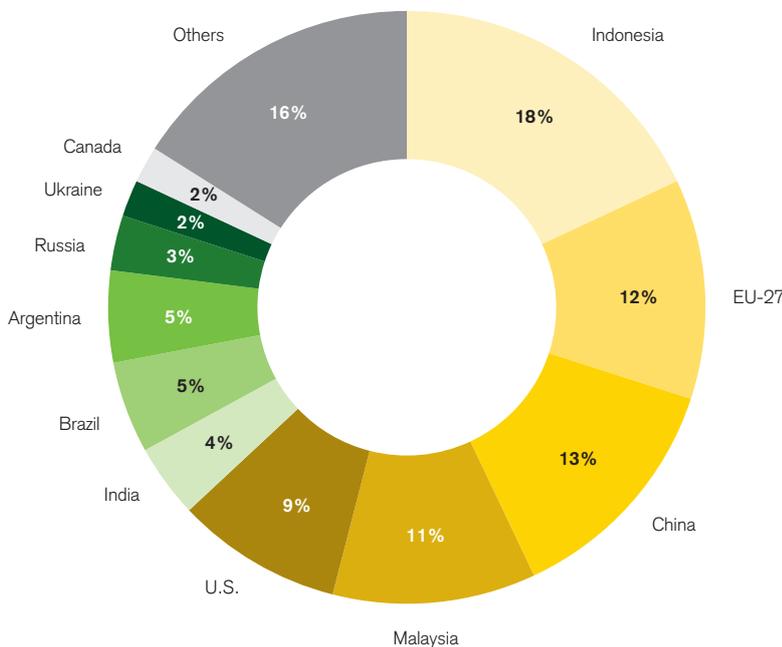
Oil palms are productive year round and they generate several times the yield of most other vegetable oils. As a consequence, communities that have adopted oil palm as a major source of activity have generally experienced positive financial and socio-economic outcomes, and in some cases a significant increase in net income. The economic attractiveness of palm oil has also led to concerns about its environmental impact: oil palm expansion has replaced large areas of tropical forest in both Malaysia and Indonesia. However, many of the largest companies in this space have committed to the standards of the RSPO (Roundtable on Sustainable Palm Oil), which aims to ensure that the palm oil plantations are developed in accordance with a code of environmental and social responsibility. Several food manufacturers are also aiming to use only RSPO certified palm oil in their products. A study on the conservation of tropical biodiversity by Bateman et al. published in June 2015 suggests that the market is willing to pay a 15-56% premium for certified sustainable palm oil.

While it's worth mentioning the environmental impact of certain products, this topic is beyond the scope of this report. We are focusing on the nutritional and health benefits (or negative effects of fats), not how they are obtained. Consequently, we do not delve into the details of the heavily chemically engineered process of extracting oil from rapeseed (canola) or cottonseed or questions over whether existing meat production systems are humane. We do not consider these factors irrelevant in the decision of what we eat or what we should eat; they are simply too complex to tackle in this report.



Figure 49

Main producers of edible oils by country



Source: Oil World



The evolution of fat

Using data from FAOSTAT and breaking up each basic food into its main fat components—saturated, monounsaturated, omega-6 and omega-3 polyunsaturated—we can develop a clear picture of what has changed over the past fifty years. We do not include transfat as we are looking at raw foods and ingredients; transfats are used by the food industry when preparing baked goods, snacks, margarine among other products.

Between 1961 and 2011, daily food intake globally increased by 39% from 1.27kg (2.8 pounds) per person to 1.76kg (3.9 pounds). Using our own analysis based on FAOSTAT food balance sheets and Nutriodata estimate of the calorie breakdown for each food item, caloric intake per day rose 31%, but calories from fat increased globally by 57% and within this animal fat calories (mostly dairy, meat, fish, and eggs) showed a 27% increase. Calories from protein rose 31% and calories from carbohydrates rose 18%. Note that 1 gram of fat contains 9 calories of energy versus 4 for protein and carbohydrates.

This seems pretty logical. As more food became available for a growing world population and incomes rose, people began to consume more fat on a relative basis. Only the U.S. shows a trend that is quite different from that for the rest of the world.

In the case of the U.S., caloric intake per day rose 26% during the same period (note that NHANES, though, suggests almost no increase in caloric intake between 1971 and 2010) while calories from fat increased 27%, but calories from animal fat declined 14%. On the other hand, “fat” calories from vegetable oils rose 156% in the same period. Calories from protein rose 10% and calories from carbohydrates rose 17%. The same numbers for Europe show an 11% increase in total calories, driven by a 38% increase in fat calories (a 17% increase

in animal fat calories) and a 16% increase in protein calories, but a 4% decline in carbohydrates calories.

In developing Asia and Latin America, large percentages of the poorest segments of the population experienced rising incomes and much lower inflation. This translated into a food consumption boom, in which fat was the most sought after macronutrient. In these two regions combined caloric intake rose 53% over the last fifty years, with calories from fat up 186% and those from animal fat up 275%. In Africa, caloric intake rose 31% with calories from fat up 39% and calories from animal fat up 34%.

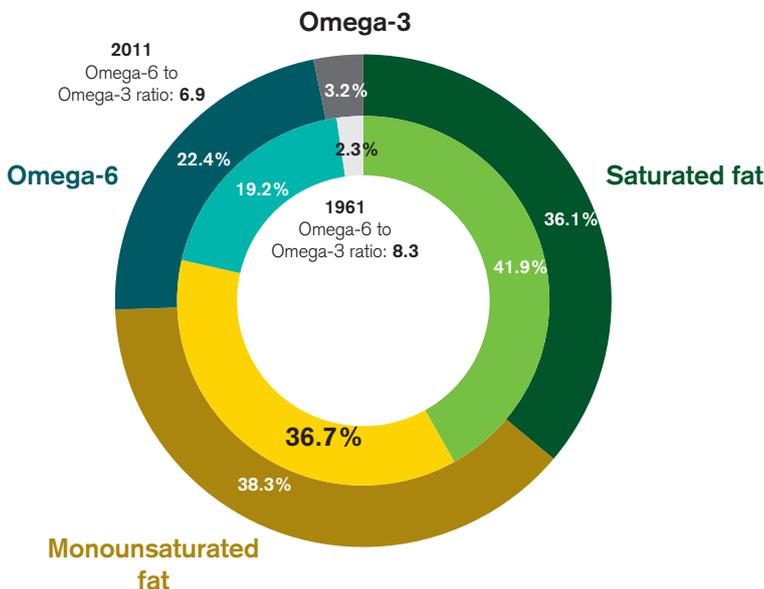
While overall calories consumed in the form of fat increased by 57% globally, saturated fat consumption rose by 38%, monounsaturated fat by 63%, omega-6 fatty acids by 89% and omega-3s by 125%. So in 2011, saturated fats accounted for 35% of all the fats consumed, versus 41% in 1961, monounsaturated 39% versus 38%, omega-6 23% versus 19%, and omega-3 3% versus 2%. These global trends, though, represent the average of very different regional patterns. If we divide the world very simplistically into developing and developed regions we can identify very different trends.

Rising wealth in Asia (China and South East Asia) from a very low level drove a sharp uptick in the consumption of saturated fat as a result of higher consumption of red meat, dairy and palm oil. In Asia for example, saturated fat consumption rose 79%, albeit from a small base.

Conversely, in the developed world, particularly in the U.S., the consumption of saturated fat declined. Meat (with the exception of poultry), eggs and dairy were shunned to follow the new dietary recommendations advocated by health organizations, doctors and scientists aimed at tackling the perceived key culprit in the heart attack epidemic: saturated fat. Saturated fat was ‘out’ and carbohydrates were ‘in’, as well as omega-6 and monounsaturated fats, via the wider adoption of vegetable oils. In the U.S., saturated fat consumption declined 2% and in Europe rose by just 14%⁴. Omega-6 intake rose 95% in the U.S. and 65% in Europe.

Figure 50

Global fat consumption 1961 versus 2011



Source: FAOSTAT

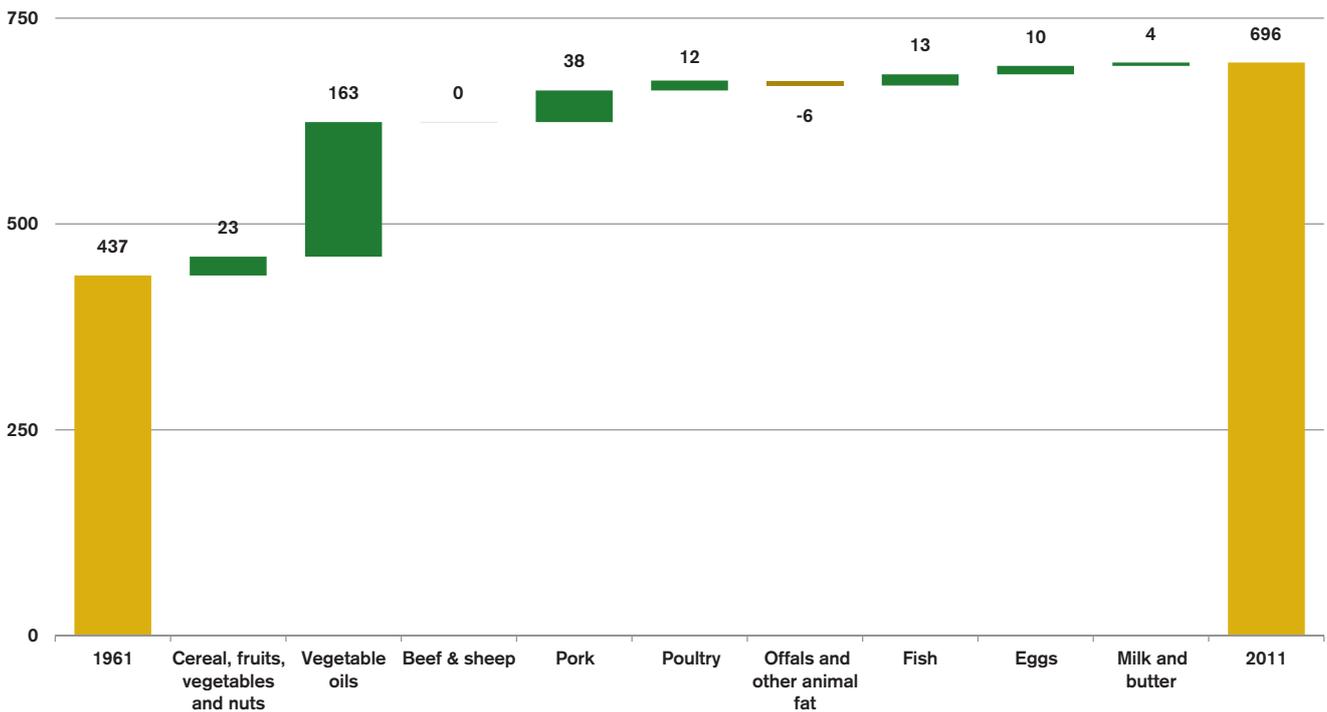
⁴ This estimates for the growth in saturated fat are quite conservative. A study by Popkin et al. in 2001 point to the fact that the type of meat we eat today is much leaner: from 25 gr of fat per 100 gr of meat to just 15 gr. If we were to adjust our 1961 and 2011 data for this, saturated fat consumption per capita would have declined 19% in the U.S. and 2% in Europe.





Figure 51

Global fat consumption – calories per day per capita 1961-2011



Source: Credit Suisse estimates based on FAOSTAT historic data, Nutrition Data

The new paradigm: Fat on the rise

Based on our research, recent medical papers, the trends we analyzed in consumer demand and key data from the “OECD-FAO Agricultural Outlook 2015-2025,” we have developed global and regional scenarios on the likely evolution of the consumption of fat, carbohydrates and protein. Here are our main forecasts:

- The drive towards increased fat consumption witnessed over the last fifty years will accelerate due to the combination of higher per capita wealth in developing countries and the gradual acceptance in the developed world that fat is at least not bad, if not actually healthy. Fat consumption per capita is set to grow from the 26% of total energy intake registered globally in 2011 (based on FAOSTAT data) to close to 31% by 2030, with the U.S. going from the current 40% to 47% and the rest of the developed world from 35% to 40%. We believe that in the case of the U.S., the FAOSTAT numbers are too high to start with as we discussed before; based on the more reliable NHANES data, we believe fat consumption in the U.S. will expand from the current 33% to 38%.
- Carbohydrates will decline from 60% of global energy intake in 2011 to 55% by 2030. Some will think that this is an aggressive forecast, as it took fifty years for carbohydrates to decline from 67% in 1961 to 60% in 2011. But we believe that the rising awareness of the link between excess carbohydrate consumption (and particularly sugar) and type 2 diabetes, cardiovascular issues and mental illnesses will most likely accelerate the historical trend.
For the U.S., we believe carbohydrates will decline from the current 51% of total caloric intake (based on NHANES) to 45%; for Europe we are likely to see a decline from 52% to 46%. Similar percentages apply to the rest of the developed world. If anything, we believe the decline of the percentage of energy we source from carbohydrates in the developed world could be sharper.
- Protein will grow just a little from 11% of daily energy intake globally to 12% by 2030 and should remain stable in the developed world and in the U.S. at 15-17%.
- Within fat, saturated fat is likely to experience the fastest growth, going from 9.4% in 2011 to 12.7% of daily energy intake by 2030, mono-unsaturated from 10.2% to 12.2%. We expect polyunsaturated omega-6 to decline slightly from 6% to 5.4% and omega-3 to grow from 0.50% to 0.55% (excluding supplements).
- Finally, we believe that calorie intake in the developed world—1.3 billion people or almost 20% of the total population—will decline from the current levels of 3,340 calories per day (using the FAO database) to 3,180; emerging markets instead—5.5 billion people or 80% of the total—will continue to catch up from the current 2,760 and get to 3,060 by 2030. We assume that 90% of these additional calories will come from the increase in fat consumption; saturated fat alone should account for two thirds of the increase in calorie intake.

The bottom line of these assumptions is that fat consumption per capita is likely to soar by 23% from now until 2030, protein by 12%, and carbohydrates will likely decline by 2%. This implies annual compound growth of 1.3% for fat consumption, compared to 0.9% over the last fifty years. Total demand for fat will be much higher—43% up for fat or 1.9% a year— given the 16% growth in the global population expected over the next fifteen years.





Figure 52

Regional estimates – calories per capita

	2011	2016	2021	2026	2030
East Asia & Pacific	3,046	3,101	3,158	3,216	3,263
Eastern Asia	3,043	3,099	3,158	3,217	3,266
Japan	2,719	2,692	2,665	2,639	2,618
Eastern Asia ex-Japan	3,071	3,133	3,196	3,260	3,313
Oceania	3,212	3,187	3,162	3,138	3,118
Australia	3,265	3,232	3,200	3,168	3,143
Oceania ex-Australia	3,037	3,037	3,037	3,037	3,037
Europe & Central Asia	3,323	3,290	3,257	3,225	3,199
Latin America & Caribbean	2,981	3,026	3,072	3,118	3,156
North America	3,617	3,545	3,475	3,406	3,352
Middle East & North Africa	3,157	3,188	3,220	3,253	3,279
South Asia	2,526	2,615	2,707	2,804	2,883
Southern Asia	2,473	2,564	2,660	2,759	2,841
India	2,455	2,567	2,685	2,808	2,911
Southern Asia ex-India	2,519	2,557	2,595	2,634	2,666
South-Eastern Asia	2,675	2,756	2,840	2,926	2,997
Sub-Saharan Africa	2,463	2,589	2,721	2,860	2,976
World	2,870	2,919	2,972	3,031	3,082

Source: FAOSTAT. Credit Suisse estimates

Figure 53

Regional estimates – percent total fat per capita

	2011	2016	2021	2026	2030
East Asia & Pacific	28	30	32	35	36
Eastern Asia	28	30	32	34	36
Japan	29	29	29	29	29
Eastern Asia ex-Japan	27	30	32	35	37
Oceania	40	41	42	43	43
Australia	42	43	44	44	45
Oceania ex-Australia	34	35	36	37	37
Europe & Central Asia	34	35	37	39	40
Latin America & Caribbean	28	31	33	36	38
North America	40	42	44	46	47
Middle East & North Africa	23	26	28	31	33
South Asia	20	20	21	22	23
Southern Asia	19	20	21	22	23
India	19	20	21	21	22
Southern Asia ex-India	20	21	23	24	26
South-Eastern Asia	20	21	22	22	23
Sub-Saharan Africa	19	20	22	23	24
World	26	27	28	30	31

Source: FAOSTAT. Credit Suisse estimates

Again within fat, we expect to see saturated fat, mono-unsaturated and omega-3 emerging as the clear winners. Here are the main drivers:

1. The substitution of saturated fat by sugar will reverse. Sugar accounts for 9% of all calories we consume today. This percentage is already declining and we will consume fatter milk, fatter dairy (e.g. butter) and cheeses.
2. In terms of real nutrients, most foods that are high in saturated fat are also high in monounsaturated fats (meat for example). So, high growth in saturated fats will drive healthy growth in monounsaturated fats.
3. The substitution of saturated fat with omega-6 oils or margarine should also reverse. This should be a less marked effect, as in most cases the consumers' perception of vegetable oils is quite positive and food manufacturing companies will still use the cheapest source of fat available. Vegetable oils—as we have seen before—have a price advantage: they provide the cheapest source of “fat” fuel. We expect sales of margarine to continue to decline.
4. The ultimate dismantling of transfats in the food chain will benefit saturated fats. Currently estimates are that the food industry in the U.S., Europe and Asia still uses transfats and that these account for 2.9% of our calorie intake in the U.S., 1.1% in Europe and 1.0% in Asia. We assume that transfats will be replaced by a combination of vegetable oils and saturated fat.
5. Consumption of better meat and dairy and more fish will drive an increase the daily intake of omega-3. The medical benefits of this fatty acid will ensure more consumption through omega-3 rich foods and particularly through supplements.

Bringing all these together, we believe that per capita consumption of saturated fat will soar by 38% by 2030, monounsaturated will increase by 24%, omega-6 polyunsaturated will decline 2% and omega-3 consumption will expand by 13% (excluding supplements). Total demand will be higher, again due to population growth: 60% higher for saturated fat (or 3.0% a year), up 44% for monounsaturated (2.3% a year); omega-6 up 13% (0.8% a year) and omega-3 up 31% (1.7% a year, ex-supplements).

Which foods?

What does this imply in terms of the demand for “fatty” foods: eggs, dairy, meat, fish and vegetable oils? Assuming preferences change driven by medical research and improved awareness, we can attempt to forecast what this implies in terms of food consumption rather than macronutrients. This is an informed guess; as reflecting our forecast on saturated or monounsaturated fat into actual food purchases assumes knowing consumer preferences fifteen years ahead, which is basically impossible.

To do this analysis, we reverse the model we developed to calculate the breakdown of each of the main “fat-rich” foods consumed in 2011 on a per-capita basis into the main fatty acids: saturated, monounsaturated, and polyunsaturated omega-6 and omega-3. We know—based on our top-down assumptions—the daily consumption levels of each

one of these main fatty acids in 2030 and we can adjust the consumption of different foods to match that distribution. This is an iterative process; the projected food consumption has to be based on logical assumptions and it has to match the top down forecast for fat and its sub-components.

We also checked this against historical growth rates taking into account that emerging markets' wealth is growing faster. This is a big driver of increased consumption of fat, particularly for dairy products and meat.

Our analysis highlights some clear winners, some neutral outcomes and some losers. We believe the winners will be eggs, dairy, red meat, and fish. The losers will be carbohydrates and particularly sugar. In the neutral camp we find the vegetable oils and chicken, which have experienced the highest growth in the last fifty years, driven by their value proposition and the desire to eat less saturated fat.

- We expect meat consumption to grow 23% over the next fifteen years. Within this, we see red meat improving its current perception of a source of “bad” (saturated) fat. Pork and chicken should grow slightly less. The move from non-fat to fat benefits pork and beef over chicken, but chicken remains the cheapest source of meat.
- On the oil front, we expect a little contraction (5–6%). The two leading processed oils: canola and soybean oil should decline. Both have a relatively positive image and will be able to counter the shift towards saturated fat and away from margarine and other products that rely on these oils. Palm should gradually improve its image and see the benefits of a trend towards “natural” oils. We expect it to grow by 10% on a per capita basis compared to 15% for olive oil. Coconut oil is mostly saturated and should grow faster from a very low base, as saturated fat becomes a “good” fat.
- Fish should grow in line with historical trends and so should nuts. Eggs should be the big winner: the cholesterol-heart link will gradually disappear from people's minds and eggs are an amazing source of both fat and protein. We expect egg consumption to grow at a rate of 4% a year. By 2030, we expect the world to consume close to 300 eggs a year per capita (just over five eggs a week).
- Finally, on the dairy front, we expect butter and cheese to continue to grow at a fast pace, as has been the case in the last 3-5 years. Milk and milk-related products should grow by 50% or 2.5% a year. Butter should continue to replace margarine and benefit from the full ban on transfats. Medical research shows that full-fat dairy is either beneficial or neutral in terms of CVD risk.





Geographical differences

In Figures 54 to 59, we show the expected changes in fat consumption versus historical trends for the world, the U.S., China, Europe, India and Australia.

For the U.S., we forecast meat consumption to trend higher, again with red meat in the lead. Eggs consumption should grow to 350 a year (almost one a day) from 235 currently. Among the solvent-extracted oils, we expect a small decline, with soybean oil still as the best selling, including the new high-oleic versions. Butter should grow faster as the market learns the benefits of eating butter and the negative perception linked to saturated fats gradually disappears. We will analyze shortly how these forecasts compare to historical consumption levels.

For Europe, we expect red meat consumption per capita to grow 9%, and poultry 19%. The levels we forecast for red meat consumption in 2030 are 13% below 1990 levels. Fish consumption should grow slower at 12%, as consumption is already at a relative high level and availability is more constrained. Milk, cheese and yogurt should expand 11% or 0.7% a year, but butter consumption should grow 4% a year. The 5.5 kg per capita we assume for 2030 is almost 10% below the consumption levels in 1990.

Egg consumption should skyrocket too and expand by 42% or over 2% a year to 300 eggs a year per person. This is over 40% higher than the previous record levels of 1985, but the softening of the stance on fat and the complete dismissal of the perceived negative effects of cholesterol intake should ensure strong growth in this food item. Finally, vegetable oil consumption on a per capita

basis should remain basically flat: solvent-extracted oils should decline 10%; natural oils should grow by 30% or more, albeit from a smaller basis.

China experienced a massive food consumption boom in the last fifty years with the quantity of food per capita consumed doubling from 1961 to 2011. We expect it to expand further by 13% from now until 2030. Vegetable oil consumption should grow by 16%; meat by 29% or 1.7% a year; and fish by less than 10%, as consumption is already 77% higher than the world's average. Milk products should be the big winners continuing a trend that started a decade ago. We expect consumption of milk products to grow 55% or almost 3% a year.

The Indian diet is light on meat, fish and eggs; we expect this to remain the case. Food consumption overall should expand by 1% a year, versus a 0.7% average increase over the past fifty years. Vegetable oils should grow 13%; red meat should remain flat, but the poultry segment should expand by over 2% a year.

Who should be the main winners? Eggs, milk and fish. Egg consumption should grow 4-5% a year to a still low level of 85 eggs per person a year. Fish could expand from a low level of 5kg per person to 10kg (still well below the world average). Finally dairy products should show strong growth: milk related products up 50% or almost 3% a year and butter/ghee up 55% or 3% a year.

For Australia, we expect beef consumption to grow by 10%; fish consumption should grow more slowly at 5%, as Australians already consume a relatively high level of fish. Egg consumption should almost double to 210 a year per person, 10% above the levels consumed in the 1960s. Milk, cheese and yogurt should expand 3% overall, as consumption is already relatively high. Butter consumption should grow by 35% and reach 5.5kg per person a year; less than half the levels consumed in 1960. Finally, vegetable oil consumption on a per capita basis should remain flat, with palm oil best positioned to grow and gain further market share. We expect consumption of canola oil—which is the most popular one—to decline slightly.

Are these forecasts realistic?

These forecasts cannot by definition be fully accurate. We are looking over a fifteen year span and assessing changes in consumer behavior. However, our estimates in terms of individual food consumption should be close—within a 10% upper and lower limit—to the most likely outcome. Historical trends have been quite clear over the past 10-20 years and some of the changes we forecast are already gradually happening.

A few points will give a sense of the magnitude of the change:

- Butter sales were up 14% year on year in the U.S in 2014 and are up 6% in the first 3 months of this year; note that butter imports were up 150%. Butter volume sales in the U.K. in 2014 were up 9% year on year
- Sales of nuts in the U.S. grew at an annual rate of almost 6% over the last three years
- Sales of eggs in the U.S. were up 2% in the twelve months ending July this year in volume terms, and organic eggs are up 21%; in the U.K. eggs sales were up 2% in 2014 over 2013
- Whole milk volume sales in the U.S. in first half of 2015 were up 11%, while skim milk was down 14%.
- Durum pasta sales in Western Europe were down 13% in the last five years, with Italy showing a 25% decline. In the U.S. volumes are down 6%. Bread consumption in France is declining at a rate of 1.5% a year.

Beyond these recent trends, we can also test how sensible our forecasts are, in three more ways:

1. Compare annual growth rates for the period 1961–2011 with those we forecast for the next fifteen years.
2. Check consumption levels of each “fat-rich” food relative to its long term historical trend (e.g. 100 years);
3. Identify the key quality drivers of “consumers’ food demand” and test our forecast relative to the outlook for each.

1. New and historical “fat” growth trends

We clearly believe that over the next fifteen years there will be an acceleration in the historical growth trend of fat consumption. Our bottom-up forecast for food consumption suggests that fat intake will grow at 1.3% a year from now until 2030 versus 0.9% a year between 1961 and 2011. Within this saturated fat should grow at 2% a year versus an historical trend of 0.6%. It is big change, but this whole report supports this conclusion.

How do our numbers compare to the 2015-25 outlook published this year by the OECD and FAO? On the meat front, OECD-FAO is forecasting 17% growth between 2014 and 2024. This implies 1.6% a year versus our estimate of 1.3% a year on a per capita basis. Adjusted for population growth—almost 1% a year—we have 2.3% a year, clearly much higher. For milk, OECD-FAO is projecting 2.5% annual growth; we expect total consumption per capita—including milk products like yogurt and cheese—to grow by 2.6% a year on a per capita basis or 3.6% for total demand. Finally for fish, OECD-FAO expects total aquaculture and capture to grow at 1.6% a year; we estimate total consumption per capita to grow at almost 1.6% a year or 2.5% for total demand.

Based on these comparisons, some people might find our estimates too high relative those developed by the OECD and FAO. However, there are two important issues to consider. First, we are projecting consumption; OECD-FAO is projecting production. This is a little like comparing apples and oranges. Stocks of processed meat or powder milk for example can affect the balance between consumption and production. Second, we are using FAO data which are not adjusted for waste (supply data). Over the years, we have become more efficient in avoiding waste particularly in emerging markets which account for 80% of the world population (for example with introduction of refrigerators). So true consumption might have been growing faster than suggested by the historical trends we have shown previously (FAOSTAT) and in the OECD-FAO outlook report.

2. Consumption of specific foods vs. historical levels

We provided a few historical reference points for Europe and Australia; the same are not relevant for India and China given the economic boom these countries have experienced in the last 20-30 years. The U.S. is a clear example of a country that changed “fat” direction in the 1960s and is now likely to reverse a fifty-year trend. So, it might be worth checking how realistic our assumptions on food consumption relative to historical trends are. Leveraging the USDA database on food consumption that goes back to 1909, we can check how our forecast for food consumption for 2030 stacks up relative to historical patterns.

For all the foods we could trace back to 1909, we checked for the points of highest and lowest consumption: the starting point at 1909, a mid-point at 1961 and the latest available data (usually 2011 or 2012).

In the case of butter for example, we can see that our forecast of 8.4 pounds a year is 53% below the 18



Figure 54

Global food consumption evolution forecast by fat type

	Percentage daily calories			Annual growth	
	1961	2011	2030	2011/1961	2030/2011
Saturated	41%	35%	41%	0.6%	2.0%
Monounsaturated	38%	39%	39%	1.0%	1.3%
Omega-6	19%	23%	17%	1.3%	-0.2%
Omega-3	2%	3%	3%	1.6%	0.7%
Total	100%	100%	100%	0.9%	1.3%
Ratio omega-6/omega-3	8.6	7.2	6.1		

Source: FAOSTAT, Credit Suisse estimates

Figure 55

U.S. food consumption evolution forecast by fat type

	Percentage daily calories			Annual growth	
	1961	2011	2030	2011/1961	2030/2011
Saturated	42%	32%	35%	-0.05%	0.9%
Monounsaturated	37%	35%	36%	0.4%	0.7%
Omega-6	19%	29%	25%	1.3%	-0.2%
Omega-3	2%	4%	4%	1.6%	0.0%
Total	100%	100%	100%	0.5%	0.5%
Ratio omega-6/omega-3	8.1	7.2	6.9		

Source: FAOSTAT, Credit Suisse estimates

Figure 56

China food consumption evolution forecast by fat type

	Percentage daily calories			Annual growth	
	1961	2011	2030	2011/1961	2030/2011
Saturated	29%	35%	38%	4.4%	2.1%
Monounsaturated	36%	42%	42%	4.4%	1.6%
Omega-6	31%	20%	17%	3.1%	1.0%
Omega-3	3%	3%	3%	4.0%	1.0%
Total	100%	100%	100%	4.0%	1.7%
Ratio omega-6/omega-3	9.3	6.0	6.1		

Source: FAOSTAT, Credit Suisse estimates



Figure 57

Europe food consumption evolution forecast by fat type

	Percentage daily calories			Annual growth	
	1961	2011	2030	2011/1961	2030/2011
Saturated	43%	35%	38%	0.3%	1.1%
Monounsaturated	39%	43%	43%	0.8%	0.7%
Omega-6	16%	19%	17%	1.0%	-0.1%
Omega-3	2%	3%	2%	1.2%	0.3%
Total	100%	100%	100%	0.6%	0.7%
Ratio omega-6/omega-3	8.4	7.6	7.1		

Source: FAOSTAT. Credit Suisse estimates

Figure 58

India food consumption evolution forecast by fat type

	Percentage daily calories			Annual growth	
	1961	2011	2030	2011/1961	2030/2011
Saturated	36%	37%	43%	1.2%	2.5%
Monounsaturated	38%	36%	34%	1.0%	1.4%
Omega-6	23%	24%	19%	1.2%	0.5%
Omega-3	2%	3%	3%	2.1%	1.2%
Total	100%	100%	100%	1.2%	1.6%
Ratio omega-6/omega-3	11.0	7.2	6.3		

Source: FAOSTAT. Credit Suisse estimates

Figure 59

Australia food consumption evolution forecast by fat type

	Percentage daily calories			Annual growth	
	1961	2011	2030	2011/1961	2030/2011
Saturated	49%	37%	38%	-0.5%	0.3%
Monounsaturated	40%	44%	44%	0.3%	0.1%
Omega-6	9%	16%	15%	1.3%	-0.2%
Omega-3	2%	3%	3%	1.4%	-0.1%
Total	100%	100%	100%	0.1%	0.1%
Ratio omega-6/omega-3	5.7	5.3	5.1		

Source: FAOSTAT. Credit Suisse estimates



3. Consumer: key drivers

pounds consumed in 1909, just 12% more than what Americans consumed in 1961 and 49% higher than the 5.6 pounds they consumed on average in 2012.

In the case of beef, Americans consumed 59 pounds a year in 1909; our forecast for 2030 of 72 pounds is 24% higher than that and 9% higher than the mid-point in 1961. However this forecast is still 23% lower than the record 94 pounds per capita consumed in 1976. Our forecast for overall meat consumption of 180 pounds per person for 2030 is 40% higher than in 1961, but just 6% higher than the record high of 170 pounds per person in 2004.

Finally, for eggs, our forecast of 350 eggs per year per person for 2030 is 23% higher than consumption levels in 1909 of 284 eggs per person, and 10% higher than in 1961 but exactly the same as Americans consumed in 1957 and 14% below the record 405 eggs per capita consumed in 1945. Note that the implied growth forecast for egg consumption is 2.2% a year, consistent with the trend of the last 12 months.

Another way to test our forecast—in a more qualitative way—is to develop a “value” matrix which takes into account the results of our consumer survey and the likely impact of medical research on the topic. We believe that factors such as Natural Origin, Healthier or Sensory/Taste experience are key to the long-term success of any product in the food sector. How does each of the “fat-rich” foods we analyzed above fare relative to this matrix?

Several “fatty-foods” score well on three or more of these attributes: olive oil, fish, nuts, dairy products (cheese and yogurt) and eggs. Red meat, chicken and vegetable oils are more in a neutral zone, but they stand to benefit from the overall trend towards consuming more fat and in the case of red meat more saturated and monounsaturated fats.

There are no clear losers within the fat group. This is pretty logical as the main shift will be from carbohydrates to fat. The correction of one major nutritional mistake—if not the biggest—is finally under way on a global basis.



Figure 60

Key drivers – consumers

	Natural origin / less processing	Taste	Packaging / easy to use / perishable	Ethical	Price	Expected-change in health perception
Butter	●	●	●	●	●	↑
Margarine	●	●	●	●	●	↓
Beef	●	●	●	●	●	↑
Pork	●	●	●	●	●	→
Chicken	●	●	●	●	●	→
Eggs	●	●	●	●	●	↑
Fish	●	●	●	●	●	→
Nuts	●	●	●	●	●	→
Soybean oil	●	●	●	●	●	↓
Canola oil	●	●	●	●	●	↓
Sunflower oil	●	●	●	●	●	↓
Olive oil	●	●	●	●	●	→
Palm oil	●	●	●	●	●	→
Coconut oil	●	●	●	●	●	↑
Milk	●	●	●	●	●	↑
Cheese	●	●	●	●	●	↑

Source: Credit Suisse

Glossary

AHA – American Heart Association. It is the oldest and largest voluntary organization in the U.S. dedicated to fighting heart disease and stroke. It funds research, advocates public health policies, and provides public health education in a variety of ways.

Case control or cohort study – a type of epidemiological study where subjects diagnosed with a disease or condition are compared to healthy controls and risk factors (e.g., diet, exercise, serum cholesterol) are assessed, usually retroactively. This type of study can be relatively inexpensive since subjects are often assessed only once and are not followed over time.

Clinical trial – a type of study in which participants are assigned to receive one or more interventions so that researchers can evaluate the effects of the interventions on health-related outcomes. A “randomized” trial is one that assigns participants to different study arms by chance. A “controlled” trial has a control group that does not receive the intervention(s). A “randomized controlled clinical trial” is considered the gold standard of clinical trials and of scientific evidence generally.

CVD – Cardiovascular disease is a general term that describes a disease of the heart or blood vessels. Blood flow to the heart, brain or body can be reduced as the result of a blood clot (thrombosis), or by a build-up of fatty deposits inside an artery that cause the artery to harden and narrow (atherosclerosis)

CHD – Coronary heart disease is a form of cardiovascular disease. A build up of fatty deposits called plaque (plak) builds up inside the coronary arteries.

Epidemiological study – a type of study that identifies the incidence of disease or some other condition across a population. Nutritional epidemiology involves assessing the diet of a population, sometimes periodically, and correlating that information with eventual health outcomes. These studies can demonstrate associations but not causation. Also known as an “observational” study.

Fatty Acids – chains of carbon atoms surrounded by hydrogen atoms. Individual fatty acids can be saturated or unsaturated. Three fatty acids bound together like a pitchfork are called triglycerides.

HDL-cholesterol – the type of cholesterol in high density lipoproteins that is known as “good” because people with higher levels tend to have a lower risk for heart disease. HDL-cholesterol is a fraction of total cholesterol.

LDL-cholesterol – the type of cholesterol in low density lipoproteins that is known as “bad” because people with very high levels tend to have a higher risk of heart disease.

Monounsaturated fats – fats in which the fatty acids contain only one double bond. The most common monounsaturated fat is called “oleic,” the type most abundant in olive oil.

Polyunsaturated fats - fat in which the fatty acids contain multiple double bonds. Polyunsaturated fats include vegetable oils, such as soybean, corn, safflower, sunflower, cottonseed and rapeseed, the main oil in canola.

Saturated fats - the fats that have no double bonds in the fatty acids they contain. These fats are found predominantly in animal foods, such as eggs, dairy, and meat, as well as in palm and coconut oils.

Trans fats – the fats that contain fatty acids with a double bond in the “trans” configuration. A “trans” bond creates a molecule in a zigzag shape, allowing adjacent fatty acids to lie nearly against each other, resulting in a fat that can be a solid at room temperature. The other type of double bond, called “cis,” creates U-shaped molecules that cannot stack together and therefore create oils.

Triglycerides – a form of fatty acids circulating in the blood. Triglycerides are comprised of three fatty acids joined together at their ends by a glycerol molecule, in the shape of a pitch-fork. Since the 1940s, high triglycerides have been considered a biomarker for heart disease.

Unsaturated fats – the fats with fatty acids that contain either one double bond (mono-unsaturated) or more (polyunsaturated).

WHO – The World Health Organization. Its primary role is to direct and coordinate international health within the United Nations’ system. The main areas of work are health systems; promoting health through the life-course; non-communicable and communicable diseases; corporate services; preparedness, surveillance and response.



Research **Institute**

Thought leadership from Credit Suisse Research
and the world's foremost experts



Fat: The New
Health Paradigm
Appendix

Guidelines

Figure 62

Nutritional guidelines: relevant organizations

Organization	Saturated fat	Other fats	Meat	Fish	Sugar	Other
AHA	The American Heart Association recommends aiming for a dietary pattern that achieves 5% to 6% of calories from saturated fat. Replace foods high in saturated fats with foods high in monounsaturated and/or polyunsaturated fats. This means eating foods made with liquid vegetable oil but not tropical oils. It also means eating fish and nuts. Also replace some of the meat with beans or legumes	The American Heart Association recommends cutting back on foods containing partially hydrogenated vegetable oils to reduce trans fat and preparing lean meats and poultry without added saturated and trans fat. Select fat-free, 1 percent fat and low-fat dairy products	Choose lean meats and poultry without skin and prepare them without added saturated and trans fat. Select meat substitutes such as dried beans, peas, lentils or tofu (soy-bean curd) in entrees, salads or soups.	The American Heart Association recommends eating fish (particularly fatty fish) at least two times (two servings) a week. Each serving is 3.5 ounce cooked, or about ¾ cup of flaked fish. Fatty fish like salmon, mackerel, herring, lake trout, sardines and albacore tuna are high in omega-3 fatty acids.	Limiting the amount of added sugars you consume to no more than half of the daily discretionary calories allowance. For most American women, that's no more than 100 calories per day. For men, it's 150 calories per day. The AHA recommendations focus on all added sugars, without singling out any particular types such as high-fructose corn syrup	
Official HHS and USDA – 7th Edition of Dietary Guidelines for Americans 2010 (current) (1)	Consume less than 10% of calories from saturated fatty acids by replacing them with monounsaturated and polyunsaturated fatty acids	Use oils to replace solid fats where possible Consume less than 300 mg per day of dietary cholesterol Keep trans fatty acid consumption as low as possible by limiting foods that contain synthetic sources of trans fats, such as partially hydrogenated oils, and by limiting other solid fats.	Choose a variety of protein foods, which include seafood, lean meat and poultry, eggs, beans and peas, soy products, and unsalted nuts and seeds	Increase the amount and variety of seafood consumed by choosing seafood in place of some meat and poultry.	Reduce the intake of calories from added sugars	Choose foods that provide more potassium, dietary fiber, calcium, and vitamin D, which are nutrients of concern in American diets. These foods include vegetables, fruits, whole grains, and milk and milk products.
Official HHS and USDA – 8th Edition of Dietary Guidelines for Americans 2015 (to be published in the fall of 2015 but Advisory Report available).	The committee recommends taking in less than 10% of total daily calories from saturated fat. Sources of saturated fat should be replaced with unsaturated fat, particularly polyunsaturated fatty acid	Previously, the Dietary Guidelines for Americans recommended that cholesterol intake be limited to no more than 300 mg/day. The 2015 committee is not bringing forward this recommendation because they consider evidence shows no appreciable relationship between consumption of dietary cholesterol and serum cholesterol	Reduce red and processed meats. Lean meat, chicken and turkey are no longer favored foods.	Increase the amount and variety of seafood consumed	Maximum of 10 percent of total calories from added sugar per day. Added sugars should be reduced in the diet and not replaced with low-calorie sweeteners, but rather with healthy options, such as water in place of sugar-sweetened beverages.	On average, the U.S. diet is low in vegetables, fruit and whole grains, and too high in calories, saturated fat, sodium, refined grains and added sugars. Under-consumption of vitamin D, calcium, potassium and fiber are of public health concern for the majority of the U.S. population
CINDI Dietary Guide, WHO Regional Office for Europe	Saturated fat should supply less than 10% of total energy intake. Replace most saturated fats with unsaturated vegetable oils or soft margarines. See (3) four country detail.	WHO recommends that healthy diets contain at most 30% of their energy from fat. (2) Saturated fat should supply less than 10% of total energy intake. Polyunsaturated fat should constitute around 7% of total energy. The balance of dietary fat should be monounsaturated	Replace fatty meat and meat products with beans, legumes, lentils, fish, poultry or lean meat.	A weekly intake of oily fish reduces the risk of cardiovascular diseases in many different ways	Select foods that are low in sugar, and eat refined sugar sparingly, limiting the frequency of sugary drinks and sweets.	

Sources: AHA <http://www.heart.org>

Dietary guidelines for Americans. <http://www.health.gov/dietaryguidelines/>

CINDI Dietary Guide, WHO Regional Office for Europe fat annex http://www.euro.who.int/__data/assets/pdf_file/0010/119926/E70041.pdf

- (1) The Dietary Guidelines for Americans Report is used by many public health agencies to drive public health recommendations programs (think the USDA school lunch program and the MyPlate icon). The Dietary Guidelines apply to all Americans ages 2 and older. By 2020, the Dietary Guidelines for Americans hope to also cover guidelines for infants.
- (2) CINDI Dietary Guide, WHO Regional Office for Europe fat annex
- (3) Fat / Saturated Fat guidelines across Europe

Figure 63

Nutritional guidelines by country

Country	Saturated fat	Other fats	Meat	Fish	Sugar	Other (taxes)
China – Ministry of Health	NA	Consume no more than 25 grams of fat per day	Consume 125-200 grams of fish, poultry, meat, eggs or other animal foods per day	Consume 50 grams of fish or shrimp per day	Limit sugar consumption	
Japan – Ministry of Health, Labour and Welfare and Ministry of Agriculture, Forestry and Fisheries.	Saturated fat should represent less than 7% of daily energy consumed	Avoid oily and fatty foods and make a balanced choice of fat from animal, plant, and fish. Total fat should represent 20-30% of the daily energy intake	3-5 servings fish and meat dishes per day	NA	Consume confectionery moderately	In January 2008, Japan passed the "Metabo Law". The law requires men and women between the ages of 45 and 74 to have their waistlines examined once a year and potentially seek medical treatment if their measurements fall outside established ranges.
Indonesia – Ministry of Health	NA	Obtain no more than a quarter of total energy intake from fats or oils	NA	NA	NA	
India – National Institute of Nutrition	Adults should choose low-fat, protein-rich foods such as lean meat, fish, pulses and low-fat milk.	Edible fat intake should not exceed 40 grams and total fat intake should be limited to levels at which fat will provide no more than 20% of total energy. The use butter should not be a regular daily feature	Include foods of animal origin such as milk, eggs and meat, particularly for pregnant and lactating women and children.	NA	The intake of sugar and sweets should be restricted. Limit to 4 (5 gr) portions a day	
Australia – National Health and Medical Research Council	Limit intake of foods high in saturated fat such as many biscuits, cakes, pastries, pies, processed meats, commercial burgers, pizza, fried foods, potato chips, crisps and other savory snacks.	Replace high fat foods which contain predominately saturated fats such as butter, cream, cooking margarine, coconut and palm oil with foods which contain predominately polyunsaturated and monounsaturated fats such as oils, spreads, nut butters/pastes and avocado	Eat 1-3 servings per day of lean meat and poultry, fish, eggs, nuts and seeds, and legumes/bean. A maximum of 455g of lean, cooked, red meat per week is recommended.	Around 2 serves of fish per week is recommended.	Limit intake of foods and drinks containing added sugars such as confectionery, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks.	
Canada – Federal Ministry of Health (Health Canada)	A diet low in saturated fat and trans fat can help reduce the risk of cardiovascular disease.	A diet low in saturated fat and trans fat can help reduce the risk of cardiovascular disease. Drink skim, 1% or 2% milk each day. Limit butter, hard margarine, lard and shortening.	Select lean meat. Trim the visible fat from meats. Remove the skin on poultry. Use cooking methods such as roasting, baking or poaching that require little or no added fat.	Eat at least two servings of fish each week. Choose fish such as char, herring, mackerel, salmon, sardines and trout.	Canada's Food Guide recommends eating foods low in sugar to help limit extra calories in the diet.	
Brazil – Ministry of Health and the Center for Epidemiological Research in Nutrition and Health of the University of São Paulo	Consumption of excessive amounts of saturated fats. Increases the risk of several chronic diseases.	Use oils and fats in small amounts for seasoning and cooking foods. As long as they are used in moderation oils and fats contribute toward diverse diets without rendering them nutritionally unbalanced.	Animal foods are usually good sources of proteins, vitamins and minerals, but contain no dietary fibre, and when fatty, are energy-dense	Because of the low fat content and because they have a high proportion of healthy fats (unsaturated fat), fish, as well as vegetables, are excellent substitutes for red meats.	When consumed in large amounts, sugar increases the risk of dental caries as well as obesity, and other chronic diseases	
Mexico – Ministry of Health	Saturated fat recommended to be no more 7% of total energy input. Saturated fats (not cholesterol) are cause of arterosclerosis.	Total fat recommended to be 30% energy input. Polyunsaturated fats like corn, peanut, sunflower and monosaturated fats like olive oil are recommended for cooking.	Lean meat and particularly chicken and turkey (without skin) are recommended over red meat	Fish is recommended over red meat	Limit refined sugar consumption	On January 2014, the Mexican Government enacted a \$1 MXN per liter tax, (around 0.08 USD), on sodas along a tax of 5% on junk-food.
Denmark – Ministry of Food, Agriculture and Fisheries	Limit saturated fat to 10% of daily energy intake (including "hard fat", i.e. saturated fat and trans fatty acids)	Limit total fat to 30% of daily energy intake.	Choose lean meats and cold meats.	At least 2 servings/per week (or 350g/week) of fish are recommended	Limit sugar consumption to a maximum of 10% of daily energy intake.	On October 2011, Denmark introduced a tax on foods containing more than 2.3% saturated fat. In November 2012 the tax was abolished as, according to the tax Ministry, it failed to change Danes' eating habits and encouraged cross border trading.
Hungary – Ministries of Health, Agriculture and Education	Decrease saturated fat sources (animal origin)	Avoid to use fat for food preparation, and avoid visibly fat rich foods.	Eat lean meats prepared with small amounts of fat regularly. Look for lean cold cuts.	Eat sea fish regularly, at least once a week	Avoid the frequent consumption of foods or drinks rich in added sugar.	On September 2011 the Hungarian government implemented a law imposing special taxes on foods with high fat, salt and sugar content. Tax is still in effect.

Sources: Ministry of Health issued "China's Dietary Guidelines (2007)". The Central People's Government of the People's Republic of China (in Chinese). http://www.gov.cn/xwfb/2008-01/15/content_858517.htm

Food Guide. Ministry of Agriculture, Forestry and Fisheries (in Japanese).

Overview of Dietary Reference Intakes for Japanese <http://www.mhlw.go.jp/file/06-Seisakujouhou-10900000-Kenkoukyoku/overview.pdf>

Indonesia. Kusharto CM, Hardinsya, Rimbawan. Nutritional Guidelines for Indonesia.

India's National Institute of Nutrition publishes the Dietary Guidelines for Indians - <http://ninindia.org/DietaryguidelinesforIndians-Finaldraft.pdf>

Australian Dietary Guidelines: https://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n55a_australian_dietary_guidelines_summary_131014.pdf

Eating Well with Canada's Food Guide: http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/pubs/res-educat-eng.pdf

http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/food-guide-aliment/view_eatwell_vue_bienmang-eng.pdf

Dietary Guidelines for the Brazilian Population 2014. http://189.28.128.100/dab/docs/portaldab/publicacoes/guia_alimentar_populacao_ingles.pdf

Mexico - Guia de Alimentacion Saludable. <http://www.imss.gob.mx/sites/all/statics/salud/guia-alimentos.pdf>

Official regulation (NORMA Oficial Mexicana NOM-043-SSA2-2012, Servicios básicos de salud. Promoción y educación para la salud en materia alimentaria. Criterios para brindar orientación) http://www.dof.gob.mx/nota_detalle.php?codigo=5285372&fecha=22/01/2013

Denmark Official Dietary Guidelines. <http://altomkost.dk/raad-og-anbefalinger/de-officielle-kostraad/>

Dietary guidelines for the adult population in Hungary (Hungarian) - http://www.oefi.hu/tap_fejn.pdf

References and Further Readings

1. A Science Advisory From the American Heart Association Nutrition Subcommittee of the Council on Nutrition, Physical Activity, and Metabolism; Council on Cardiovascular Nursing; and Council on Epidemiology and Prevention, 2009.
2. A.P. Jain, K.K. Aggarwal and P.Y. Zhang: Omega-3 fatty acids and cardiovascular disease, 2015.
3. American Heart Association: http://www.heart.org/HEARTORG/Conditions/Cholesterol/WhyCholesterolMatters/Why-Cholesterol-Matters_UCM_001212_Article.jsp: Why Cholesterol Matters.
4. Angela Liou Y, Innis S: Dietary linoleic acid has no effect on arachidonic acid, but increases n-6 eicosadienoic acid, and lowers dihomo-gamma-linolenic and eicosapentaenoic acid in plasma of adult men. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, 2009 Apr.
5. Austin GL, Ogden LG, Hill JO: Trends in carbohydrate, fat, and protein intakes and association with energy intake in normal-weight, overweight, and obese individuals. *The American Journal of Clinical Nutrition*, 1971-2006; 2011.
6. Bard JM, Drouet L, Lairon D, Cazaubiel M, Marmonier C and Claire Bal dit Sollier: Effect of milk fat on LDL cholesterol and other cardiovascular risk markers in healthy humans: the INNOVALAIT project (269.3), 2014 Apr.
7. Bard JM, Drouet L, Lairon D, Cazaubiel M, Marmonier C, Bal dit Sollier C: Effect of milk fat on LDL cholesterol and other cardiovascular risk markers in healthy humans: the INNOVALAIT project. *The FASEB Journal*. 2014 Apr.
8. Beresford SA, Johnson KC, Ritenbaugh C, Lasser NL, Snetselaar LG, Black HR, Anderson GL, Assaf AR, Bassford T, Bowen D, Brunner RL, Brzycki RG, Caan B, Chlebowski RT, Gass M, Harrigan RC, Hays J, Heber D, Heiss G, Hendrix SL, Howard BV, Hsia J, Hubbell FA, Jackson RD, Kotchen JM, Kuller LH, LaCroix AZ, Lane DS, Langer RD, Lewis CE, Manson JE, Margolis KL, Mossavar-Rahmani Y, Ockene JK, Parker LM, Perri MG, Phillips L, Prentice RL, Robbins J, Rossouw JE, Sarto GE, Stefanick ML, Van Horn L, Vitolins MZ, Wactawski-Wende J, Wallace RB, Whitlock E: Low-fat dietary pattern and risk of colorectal cancer: The Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA*, 2006 Feb.
9. Biolo G, Tipton KD, Klein S, Wolfe RR: An abundant supply of amino acids enhances the metabolic effect of exercise on muscle protein. *American Journal of Physiology*, 1997 Jul.
10. Blasbalg TL, Hibbeln JR, Ramsden CE, Majchrzak SF, Rawlings RR: Changes in consumption of omega-3 and omega-6 fatty acids in the United States during the 20th century. *The American Journal of Clinical Nutrition*, 2011 May.
11. Boden G: Free fatty acids (FFA), a link between obesity and insulin resistance, February 1998.
12. Bonthuis M, Hughes MC, Ibiebele TI, Green AC, van der Pols JC: Dairy consumption and patterns of mortality of Australian adults. *European Journal of Nutrition*, 2010 Jun.
13. Boris Hansel, Hospital Bichat-Claude Bernard, Université Paris-Diderot, Paris, France, Philippe Giral, Hospital de la Pitié-Salpêtrière, Université Pierre et Marie Curie, Paris, France: Dietary Cholesterol: Friend or Foe? Published by EDP Sciences 2015.
14. Caballero B: The Global Epidemic of Obesity: An Overview, 2007 May.
15. CDC: Long-term trends in Diabetes. 2014 Oct.
16. CDC: Trends in Intake of Energy and Macronutrients—United States, 1971-2000. 2004 Feb.
17. Charles M. Benbrook, Gillian Butler, Maged A. Latif, Carlo Leifert, Donald R. Davis: Organic production enhances milk nutritional quality by shifting fatty acid composition: a united states-wide, 18-month study. *Plos One*, 2013 Dec.
18. Chris Kressler; <http://chriskressler.com/cholesterol-doesn't-cause-heart-disease/>: Cholesterol doesn't cause heart disease.
19. Cohen MN, Crane-Kramer GMM: Ancient health: skeletal Indicators of Agricultural and Economic Intensification. Gainesville: U of Florida, 2007
20. Connye N. Kuratko, Coleen C. Nolan, Norman Salem, Jr: Long chain omega-3 fatty acids and cardiovascular disease, 2014.
21. Cordain L. Cereal grains: humanity's double-edged sword. *World Review of Nutrition and Dietetics* 1999.
22. Cunnane SC: Problems with essential fatty acids: time for a new paradigm? *Progress in Lipid Research*, 2003 Nov.
23. Dariush Mozaffarian, MD, MPH; Alberto Ascherio, MD, DrPH; Frank B. Hu, MD, PhD; Meir J. Stampfer, MD, DrPH; Walter C. Willett, MD, DrPH; David S. Siscovick, MD, MPH; Eric B. Rimm, ScD: Interplay Between Different Polyunsaturated Fatty Acids and Risk of Coronary Heart Disease in Men, 2005 Jan.
24. de Goede J, Geleijnse JM, Boer JM, Kromhout D, Verschuren WM: Linoleic acid intake, plasma cholesterol and 10-year incidence of CHD in 20,000 middle-aged men and women in the Netherlands, 2011 Aug.
25. de Goede J, Soedamah-Muthu, Trichia E, Geleijnse J, Kromhout D: Dietary intake of saturated fat by food source and incident coronary heart disease: the Zutphen Elderly Study. *Circulation*. 2015
26. de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N: Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation*, 1999 Feb 16; 99(6).
27. de Lorgeril M, Salen P, Martin JL, Monjaud I, Boucher P, Mamelle N: Mediterranean dietary pattern in a randomized trial: prolonged survival and possible reduced cancer rate. *Archives of Internal Medicine*, 1998 Jun 8; 158(11):1181-7.
28. de Souza RJ, Mente A, Maroleanu A, Cozma AI, Ha V, Kishibe T, Uleryk E, Budylowski P, Schünemann H, Beyene J, Anand SS: "Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies," *The BMJ*, July 2015.
29. Di Angelantonio E, Gao P, Pennells L, Kaptoge S, Caslake M, Thompson A, Butterworth AS, Sarwar N, Wormser D, Saleheen D, Ballantyne CM, Psaty BM, Sundström J, Ridker PM, Nagel D, Gillum RF, Ford I, Ducimetiere P, Kiechl S, Koenig W, Dullaart RP, Assmann G, D'Agostino RB Sr, Dagenais GR, Cooper JA, Kromhout D, Onat A, Tipping RW, Gómez-de-la-Cámara A, Rosengren A, Sutherland SE, Gallacher J, Fowkes FG, Casiglia E, Hofman A, Salomaa V, Barrett-Connor E, Clarke R, Brunner E, Jukema JW, Simons LA, Sandhu M, Wareham NJ, Khaw KT, Kauhanen J, Salonen JT, Howard WJ, Nordestgaard BG, Wood AM, Thompson SG, Boekholdt SM, Sattar N, Packard C, Gudnason V, Danesh J: Lipid-Related Markers and Cardiovascular Disease Prediction. *JAMA*, 2012 Jun.
30. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G: Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. *Archives of Internal Medicine*, 2006 Dec 11-25; 166(22):2437-45.
31. Earl S Ford and William H Dietz: Trends in energy intake among adults in the United States: findings from NHANES. *The American Journal of Clinical Nutrition*. 2013.
32. Ervin RB, Ogden CL: Trends in intake of energy and macronutrients in children and adolescents from 1999-2000 through 2009-2010. *NCHS Data Brief*. 2013 Feb.
33. Ervin RB, Wright JD, Wang CY, Kennedy-Stephenson J: Dietary intake of fats and fatty acids for the United States population: 1999-2000. *Adv Data*, 2004 Nov.
34. Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, Enrique Gómez-Gracia E, Ruiz-Gutiérrez V, Fiol M, Lapetra J, Lamuela-Raventós RM, Serra-Majem L, Pintó X, Basora J, M.D., Muñoz MA, Sorlí JV, Martínez JA, Martínez-González: primary prevention of cardiovascular disease with a mediterranean diet. *The New England Journal of Medicine*. 2015
35. Estruch R, Rose E, Martinez-Gonzalez MA: Mediterranean diet for primary prevention of cardiovascular disease. *The New England Journal of Medicine*, 2013.
36. FAO Corporate Documentary Repository: Fats and Oils in human nutrition: National indices of dietary fat supplies, 2008 Nov.
37. FAO: Fats and fatty acids in human nutrition. Report of an expert consultation. Global trends in production, intake and food consumption, 2010.

References and Further Readings (continued)

38. Farvid MS, PhD, Ding Ming, MS, Pan A, PhD, Sun Q, MD, ScD, Chiuve SE, ScD, Steffen LM, PhD, MPH, RD, FAHA, Willett WC, DrPH, Hu FB, MD, PhD: Dietary linoleic acid and risk of coronary heart disease: a systematic review and meta-analysis of prospective cohort studies, 2014 Aug.
39. Farzadfar F, Finucane MM, Danaei G, Pelizzari PM, Cowan MJ, Paciorek CJ, Singh GM, Lin JK, Stevens GA, Riley LM, Ezzati M; Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group (Cholesterol): National, regional, and global trends in serum total cholesterol since 1980: systematic analysis of health examination surveys and epidemiological studies with 321 country-years and 3.0 million participants. *Lancet*, 2011 Feb.
40. Fine EJ, Segal-Isaacson CJ, Feinman RD, Herszkopf S, Romano MC, Tomuta N, Bontempo AF, Negassa A, Sparano JA: Targeting insuling inhibition as a metabolic therapy in advanced cancer: a pilot safety and feasibility dietary trial in 10 patients. *Nutrition*. 2012.
41. Food and Nutrition Board, Institute of Medicine: *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients)*. Washington, D.C.: National Academies Press, 2005
42. Forsythe CE, Phinney SD, Feinman RD, Volk BM, Freidenreich D, Quann E, Ballard K, Puglisi MJ, Maresh CM, Kraemer WJ et al: Limited effect of dietary saturated fat on plasma saturated fat in the context of a low carbohydrate diet. *Lipids*, 2010.
43. Forsythe, CE, Phinney SD, Fernandez ML, Quann EE, Wood RJ, Bibus DM, Kramer WJ, Feinman RD, Volek JS: Comparison of low fat and low carbohydrate diets on circulating Fatty Acid composition and markers of inflammation. *Lipids*, 2008.
44. Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S: A randomized trial of a low-carbohydrate diet for obesity. *The New England Journal of Medicine*, 2003.
45. Frantz ID Jr, Dawson EA, Ashman PL, Gatewood LC, Bartsch GE, Kuba K, Brewer ER: Test of effect of lipid lowering by diet on cardiovascular risk. The Minnesota Coronary Survey. *Arteriosclerosis*, 1989 Jan-Feb.
46. Frantz ID Jr, Dawson EA, Ashman PL, Gatewood LC, Bartsch GE, Kuba K, Brewer ER: Test of effect of lipid lowering by diet on cardiovascular risk. The Minnesota Coronary Survey. *Arteriosclerosis*, 1989 Jan-Feb.
47. Fryar CD, Carroll MD, Ogden CL: Prevalence of overweight, obesity, and extreme obesity among adults: united states, trends 1960-1962 through 2009-2010. *NCHS Health E-Stat*. 2012 Sep.
48. G. A. Rose, D.M., M.R.C.P., W. B. Thomson, M.D., M.R.C.P. and R. T. Williams, M.B., M.R.C.P.: Corn Oil in Treatment of Ischaemic Heart Disease, 1965 Jun.
49. German JB, Gibson RA, Krauss RM, Nestel P, Lamarche B, van Staveren WA, Steijns JM, de Groot LC, Lock AL, Destailats F: A reappraisal of the impact of dairy foods and milk fat on cardiovascular disease risk. *European Journal of Nutrition*, 2009 Jun.
50. Gu Q, Paulose-Ram R, Burt VL, Kit BK: Prescription cholesterol-lowering medication use in adults aged 40 and over: united states, 2003-2012. *NCHS Data Brief*, 2014 Dec.
51. Gunnars K: 6 Reasons Why Vegetable Oils are Toxic.
52. Guyenet S: Gluten sensitivity: celiac disease is the tip of the iceberg, December 8, 2008.
53. Guyenet SJ, Schwartz MW: Regulation of food intake, energy balance, and body fat mass: implications for the pathogenesis and treatment of obesity. *The Journal of Clinical Endocrinology & Metabolism*, 2012 Mar.
54. Hall KD, Bemis T, Brychta R, Chen KY, Courville A, Crayner EJ, Goodwin S, Guo J, Howard L, Knuth ND, Miller BV, Prado CM, Siervo M, Skarulis MC, Walter M, Walter PJ, Yannai L: Calorie for calorie, dietary fat restriction results in more body fat loss than carbohydrate restriction in people with obesity. *Cell Metabolism*. 2015 Sep.
55. Harika RK, Eilander A, Alsema M, Osendarp SJ, Zock PL: Intake of Fatty Acids in General Populations Worldwide Does Not Meet Dietary Recommendations to Prevent Coronary Heart Disease: A Systematic Review of Data from 40 Countries. *Annals of Nutrition and Metabolism*, 2013 Apr.
56. Harnack LJ, Jeffery RW, Boutelle KN: Temporal trends in energy intake in the United States: an ecologic perspective. *The American Journal of Clinical Nutrition*, 2000 Jun.
57. Harris WS, Poston WC, Haddock CK.: Tissue n-3 and n-6 fatty acids and risk for coronary heart disease events, 2007 Jul.
58. Haug A, Hostmark AT, Harstad OM: Bovine milk in human nutrition—a review. *Lipids Health Discussion*, 2007 Sep.
59. Haug A, Hostmark AT, Harstad OM: Bovine milk in human nutrition—a review. *Lipids in Health and Disease*. 2007 Sep.
60. Hayward RA, Krumholz HM: Three Reasons to Abandon Low-Density Lipoprotein Targets. *Circulation Cardiovascular Quality and Outcomes Journal*, 2012 Jan.
61. Hoenselaar R: Saturated fat and cardiovascular disease: the discrepancy between the scientific literature and dietary advice. *Nutrition* 2012 Feb.
62. Holt BM, Formicola V: Hunters of the Ice Age: the biology of upper paleolithic people. *American Journal of Physical Anthropology* 2008(suppl): Cohen MN. *Health and the rise of civilization*. New Haven, Conn.: Yale University Press, 1989.
63. Hooper L., Martin N., Abdelhamid A and Davey Smith G: Reduction in saturated fat intake for cardiovascular disease. *The Cochrane Collaboration*, 2015.
64. Houston DK, Ding J, Lee JS, Garcia M, Kanaya AM, Tylavsky FA, Newman AB, Visser M, Kritchevsky SB; Health ABC Study: Dietary fat and cholesterol and risk of cardiovascular disease in older adults: the Health ABC Study. *Nutrition Metabolism Cardiovascular Diseases*, 2011 Jun.
65. Howard BV, Van Horn L, Hsia J, Manson JE, Stefanick ML, Wassertheil-Smoller S, Kuller LH, LaCroix AZ, Langer RD, Lasser NL, Lewis CE, Limacher MC, Margolis KL, Mysiw WJ, Ockene JK, Parker LM, Perri MG, Phillips L, Prentice RL, Robbins J, Rossouw JE, Sarto GE, Schatz IJ, Snetselaar LG, Stevens VJ, Tinker LF, Trevisan M, Vitolins MZ, Anderson GL, Assaf AR, Bassford T, Beresford SA, Black HR, Brunner RL, Brzyski RG, Caan B, Chlebowski RT, Gass M, Granek I, Greenland P, Hays J, Heber D, Heiss G, Hendrix SL, Hubbell FA, Johnson KC, Kotchen JM: Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA*, 2006 Feb.
66. <http://www.dailymail.co.uk/health/article-3176558/It-s-healthier-cook-LARD-sunflower-oil-Extraordinary-experiment-shows-ve-told-cooking-oils-wrong.html>.
67. <http://www.ers.usda.gov/amber-waves/2014-june/trends-in-us-per-capita-consumption-of-dairy-products,-1970-2012.aspx>.
68. http://www-who.int/nutrition/topics/3_foodconsumption/en/print.html: Diet, Nutrition and the Prevention of Chronic Diseases; Global and regional food consumption patterns and trends, *World Health Organization*. 28 January- 1 February 2002.
69. Hu FB, Stampfer MJ, Manson JE, Grodstein F, Colditz GA, Speizer FE, Willett WC: Trends in the Incidence of Coronary Heart Disease and Changes in Diet and Lifestyle in Women. *New England Journal of Medicine*, 2000 Aug.
70. Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA, Hennekens CH, Willett WC.: Dietary Fat Intake and the Risk of Coronary Heart Disease in Women. *New England Journal of Medicine*, 1997 Nov.
71. Hu FB, Stampfer MJ, Manson JE, Rimm E. Colditz GA, Rosner BA, Hennekens CH, Willett WC: Dietary fat intake and the risk of coronary heart disease in women. *The New England Journal of Medicine*, 1997.
72. Hulbert AJ: Metabolism and longevity: is there a role for membrane fatty acids? *Integrative and Comparative Biology*, 2010 Nov.
73. Hussain TA, Mathew TC, Dashti AA, Asfar S, Al-Zaid N, Dashti HM: Effect of low-calorie versus low-carbohydrate ketogenic diet in type 2 diabetes. *Elsevier*. 2012.
74. IBD in EPIC Study Investigators: Linoleic acid, a dietary n-6 polyunsaturated fatty acid, and the aetiology of ulcerative colitis: a nested case-control study within a European prospective cohort study. *Gut*, 2009 Dec.

References and Further Readings (continued)

75. Jacobs D, Blackburn H, Higgins M, Reed D, Iso H, McMillan G, Neaton J, Nelson J, Potter J, Rifkind B: Mortality Associations: Report of the Conference on Low Blood Cholesterol: Mortality Associations. *Circulation* 1992;86:1046-1060.
76. Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Balter K, Fraser GE, Goldbourt U, Hallmans G, Knekt P, Liu S et al: Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. *The American Journal of Clinical Nutrition*, 2009.
77. Jenkins DJ, Kendall CW, McKeown-Eyssen G, Josse RG, Silverberg J, Booth GL, Vidgen E, Josse AR, Nguyen TH, Corrigan S et al: Effect of a low-glycemic index or a high-cereal fiber diet on type 2 diabetes: a randomized trial. *JAMA*, 2008.
78. Jenkins DJ, Srichaikul K, Kendall CW, Sievenpiper JL, Abdunour S, Mirrahimi A, Meneses C, Nishi S, He X, Lee S, So YT, Esfahani A, Mitchell S, Parker TL, Vidgen E, Josse RG, Leiter LA.: The relation of low glycaemic index fruit consumption to glycaemic control and risk factors for coronary heart disease in type 2 diabetes. *Diabetologia*, 2011 Feb.
79. Jensen RG: Lipids in human milk. *Lipids*, 1999 Dec.
80. Kabagambe EK, Baylin A, Siles X, Campos H "Individual saturated fatty acids and nonfatal acute myocardial infarction in Costa Rica," *European Journal of Clinical Nutrition*, 2003 Nov.
81. Kerckhoffs DA, Brouns F, Hornstra G, Mensink RP: Effects on the Human Serum Lipoprotein Profile of β -Glucan, Soy Protein and Isoflavones, Plant Sterols and Stanols, Garlic and Tocotrienols. *Journal of Nutrition*. 2002 Sep.
82. Komlos J: Underperformance in Affluence: The Remarkable Relative Decline in the U.S. Heights in the Second Half of the 20th Century, April 2007.
83. Konie R: The Ugly Truth About Vegetable Oils (and why they should be avoided).
84. Kratz M, Baars T, Guyenet S: The relationship between high-fat dairy consumption and obesity, cardiovascular, and metabolic disease. *Eur J Nutr*. 2013 Feb.
85. Kratz M1, Baars T, Guyenet S: The relationship between high-fat dairy consumption and obesity, cardiovascular, and metabolic disease. *European Journal of Nutrition*, July 2012.
86. Krauss RM: Webinar Series: Setting the record straight on cholesterol, saturated fat and heart disease risk, July 2015.
87. Kris-Etherton P, Fleming J, Harris WS: The debate about n-6 polyunsaturated fatty acid recommendations for cardiovascular health. *Journal of the American Dietetic Association*, 2010 Feb.
88. Kris-Etherton PM, Taylor DS, Yu-Poth S, Huth P, Moriarty K, Fishell V, Hargrove R, Zhao G, Etherton TD: Polyunsaturated Fatty Acids in the Food Chain in the United States," *American Journal of Clinical Nutrition* 71. 2001 Jan.
89. Krumholz HM, Seeman TE, Merrill SS, Mendes de Leon CF, Vaccarino V, Silverman DI, Tsukahara R, Ostfeld AM, Berkman LF: Lack of Association Between Cholesterol and Coronary Heart Disease Mortality and Morbidity and All-cause Mortality in Persons Older Than 70 Years, *JAMA* 272, no. 17 (November 2, 1994): 1335-40, 1994 Nov.
90. Kumar V, Atherton P, Smith K, Rennie MJ: Human muscle protein synthesis and breakdown during and after exercise. *Journal of Applied Physiology*, 2009 Jun.
91. Lecerf JM, de Lorgeril M: Dietary cholesterol: from physiology to cardiovascular risk. *British Journal of Nutrition*, 2011 Jul.
92. Leren P: The effect of plasma cholesterol lowering diet in male survivors of myocardial infarction: a controlled clinical trial. *Acta Medica Scandinavica, Supplementum*, 1966; 466:1-92.
93. Leren P: The Oslo diet-heart study: eleven-year report. *Circulation* 42 (1970) 935-942
94. Lima LM, Carvalho MDG, Sousa MO: Apo B/apo A-I ratio and cardiovascular risk prediction, June 2007.
95. Lopez-Garcia E, Schulze MB, Manson JE, Meigs JB, Albert CM, Rifai N, Willett WC, Hu FB: Consumption of (n-3) Fatty Acids Is Related to Plasma Biomarkers of Inflammation and Endothelial Activation in Women, 2004 Jul.
96. Lukas Schwingshackl and Georg Hoffmann: Monounsaturated fatty acids and risk of cardiovascular disease: synopsis of the evidence available from systematic reviews and meta-analyses. *Nutrients*. 2012.
97. Maletta H: What we eat: Changing patterns of food consumption around the world, 2014 Sep.
98. McNeil NI: The contribution of the large intestine to energy supplies in man. *The American Journal of Clinical Nutrition*, 1984 Feb.
99. Micha R, Khatibzadeh S, Shi P, Fahimi S, Stephen Lim et al: <http://www.bmj.com/content/348/bmj.g2272>: Global, regional, and national consumption levels of dietary fats and oils in 1990 and 2010: a systematic analysis including 266 country-specific nutrition surveys. *The BMJ*. 2014 Apr.
100. Micha R, Mozaffarian D: Saturated fat and cardiometabolic risk factors, coronary heart disease, stroke and diabetes: a fresh look at the evidence. *Lipids*. 2010
101. Micha R, Wallace SK, Mozaffarian D: Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. *Circulation*, 2010 Jun 1.
102. Mielke MM, Zandi PP, Sjögren M, Gustafson D, Ostling S, Steen B, Skoog I: High total cholesterol levels in late life associated with a reduced risk of dementia. *Neurology*, May 2005 May.
103. Mielke MM, Zandi PP, Sjögren M, Gustafson D, Ostling S, Steen B, Skoog I: High total cholesterol levels in late life associated with a reduced risk of dementia. *Neurology*, 2005 May.
104. Mosley M, Daily Mail: Why it's healthier to cook with LARD than sunflower oil: Extraordinary experiment shows everything we've been told about cooking oils is wrong, 2015 Jul.
105. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC: Trans Fatty Acids and Cardiovascular Disease. *The New England Journal of Medicine*. 2006 Apr.
106. Mozaffarian D, Micha R, Wallace S: Effects on Coronary Heart Disease of Increasing Polyunsaturated Fat in Place of Saturated Fat: A Systematic Review and Meta-Analysis of Randomized Controlled Trials, 2010 Mar.
107. Mozaffarian D, Rimm EB, Herrington DM: Dietary fats, carbohydrate, and progression of coronary atherosclerosis in postmenopausal women. *The American Journal of Clinical Nutrition* 2004 Nov.
108. Mozaffarian D: Diverging global trends in heart disease and type 2 diabetes: the role of carbohydrates and saturated fats. *Lancet Diabetes Endocrinol*. 2015 Aug.
109. Muhlhauser BS and Ailhaud GP: Omega-6 polyunsaturated fatty acids and the early origins of obesity, 2013 Feb.
110. Nettleton JA, Legrand P, Mensink RP: ISSFAL 2014 Debate: It Is Time to Update Saturated Fat Recommendations. *Annals of Nutrition and Metabolism*, 2015.

References and Further Readings (continued)

111. Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, Mul-lary EC, Biryukov S, Abbafati C, Abera SF, Abraham JP, Abu-Rmeileh NM, Achoki T, AlBuhairan FS, Alemu ZA, Alfonso R, Ali MK, Ali R, Guz-man NA, Ammar W, Anwar P, Banerjee A, Barquera S, Basu S, Ben-nett DA, Bhutta Z, Blore J, Cabral N, Nonato IC, Chang JC, Chowdhury R, Courville KJ, Criqui MH, Cundiff DK, Dabhadkar KC, Dandona L, Davis A, Dayama A, Dharmaratne SD, Ding EL, Durrani AM, Estegha-mati A, Farzadfar F, Fay DF, Feigin VL, Flaxman A, Forouzanfar MH, Goto A, Green MA, Gupta R, Hafezi-Nejad N, Hankey GJ, Harewood HC, Havmoeller R, Hay S, Hernandez L, Husseini A, Idrisov BT, Ikeda N, Islami F, Jahangir E, Jassal SK, Jee SH, Jeffreys M, Jonas JB, Ka-bagambe EK, Khalifa SE, Kengne AP, Khader YS, Khang YH, Kim D, Kimokoti RW, Kinge JM, Kokubo Y, Kosen S, Kwan G, Lai T, Leinsalu M, Li Y, Liang X, Liu S, Logroscino G, Lotufo PA, Lu Y, Ma J, Mainoo NK, Mensah GA, Merriman TR, Mokdad AH, Moschandreas J, Naghavi M, Naheed A, Nand D, Narayan KM, Nelson EL, Neuhouser ML, Nisar MI, Ohkubo T, Oti SO, Pedroza A, Prabhakaran D, Roy N, Sampson U, Seo H, Sepanlou SG, Shibuya K, Shiri R, Shiuie I, Singh GM, Singh JA, Skirbekk V, Stapelberg NJ, Sturua L, Sykes BL, Tobias M, Tran BX, Trasande L, Toyoshima H, van de Vijver S, Vasankari TJ, Veerman JL, Velasquez-Melendez G, Vlassov VV, Vollset SE, Vos T, Wang C, Wang X, Weiderpass E, Werdecker A, Wright JL, Yang YC, Yatsuya H, Yoon J, Yoon SJ, Zhao Y, Zhou M, Zhu S, Lopez AD, Murray CJ, Gakidou E: Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2014 Aug.
112. Novak EM, Dyer RA, Innis SM: High dietary ω -6 fatty acids contrib-ute to reduced docosahexaenoic acid in the developing brain and inhibit secondary neurite growth. *Brain Research*, 2008 Oct.
113. OECD-FAO Agricultural Outlook 2015-2024; 2015.
114. Oh K, Hu FB, Manson JE, Stampfer MJ, Willett WC.: Dietary fat intake and risk of coronary heart disease in women: 20 years of follow-up of the nurses' health study. *American Journal of Epidemiol-ogy*, 2005 Apr.
115. Okayama A, Ueshima H, Marmot MG, Nakamura M, Kita Y, Yamakawa M.: Changes in Total Serum Cholesterol and Other Risk Factors for Cardiovascular Disease in Japan 1980-1989. *Internat-ional Journal of Epidemiology*, 1993 Dec.
116. O'Neil CE, Nicklas TA, Keast DR, Fulgoni VL: Ethnic disparities among food sources of energy and nutrients of public health concern and nutrients to limit in adults in the United States: NHANES 2003-2006. *Food and Nutrition Research*, 2014 Nov.
117. Patterson E, Wall R, Fitzgerald GF, Ross RP, Stanton C: Health implica-tions of high dietary omega-6 polyunsaturated Fatty acids, 2012 Apr.
118. Petousis-Harris H: Saturated Fat Has Been Unfairly Demonised: Yes, Primary Health Care 3, no. 4 (December 1, 2011): 317-19, December 1, 2011.
119. Popovich DG, Jenkins DJA, Kendall CWC, Dierenfeld ES, Car-roll RW, Tariq N, Vidgen E: The western lowland gorilla diet has implications for the health of humans and other hominoids. *Journal of Nutrition*, 1997 Oct.
120. Prentice RL, Caan B, Chlebowski RT, Patterson R, Kuller LH, Ock-ene JK, Margolis KL, Limacher MC, Manson JE, Parker LM, Paskett E, Phillips L, Robbins J, Rossouw JE, Sarto GE, Shikany JM, Stefanick ML, Thomson CA, Van Horn L, Vitolins MZ, Wactawski-Wende J, Wallace RB, Wassertheil-Smoller S, Whitlock E, Yano K, Adams-Campbell L, Anderson GL, Assaf AR, Beresford SA, Black HR, Brunner RL, Brzyski RG, Ford L, Gass M, Hays J, Heber D, Heiss G, Hendrix SL, Hsia J, Hubbell FA, Jackson RD, Johnson KC, Kotchen JM, LaCroix AZ, Lane DS, Langer RD, Lasser NL, Henderson MM: Low-fat dietary pattern and risk of invasive breast cancer: The Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA*, 2006 Feb.
121. Ptaschitz G, Strand E, Norekvål TM, Dierkes J, Dahl L, Svingen GFT, Assmus J, Schartum-Hansen H, Øyen J, Pedersen EKR, Drevon CA, Tell GS, Nygård O: Dietary Intake of Saturated Fat Is Not Associated with Risk of Coronary Events or Mortality in Patients with Established Coronary Artery Disease. *American Society for Nutrition*, 2014 Dec.
122. Ptaschitz NG, Strand E, Norekvål TM, Dierkes J, Dahl L, Svingen GFT, Assmus J, Schartum-Hansen H, Øyen J, Pedersen EKR, Drevon CA, Tell GS, Nygård O: Dietary Intake of Saturated Fat Is Not Associated with Risk of Coronary Events or Mortality in Patients with Established Coronary Artery Disease. *The Journal of Nutrition*, 2014.
123. Rahilly-Tierney CR, Spiro A, Vokonas P, Gaziano JM: Relation between high-density lipoprotein cholesterol and survival to age 85 years in men (from the VA Normative Aging Study). *American Journal of Cardiology*, 2011 Apr 15.
124. Rajwinder KH, Eilander A, Alssema M, Osendarp SJM, Peter L Zock: Intake of fatty acids in general populations worldwide does not meet dietary recommendations to prevent coronary heart disease: a systematic review of data from 40 countries. *Annals of Nutrition and Metabolism*, 2013.
125. Ramsden CE, Hibbeln JR, Majchrzak SF, Davis JM: n-6 fatty acid-specific and mixed polyunsaturate dietary interventions have different effects on CHD risk: a meta-analysis of randomised controlled trials, 2010 Dec. <http://www.ncbi.nlm.nih.gov/pubmed/21118617>
126. Ramsden CE, Zamora D, Leelarthaeapin B, Majchrzak-Hong SF, Faurot KR, Suchindran CM, Ringel A, Davis JM, Hibbeln JR: Use of dietary linoleic acid for secondary prevention of coronary heart dis-ease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. *BMJ*, 2013 Feb.
127. Ravnskov U: The Cholesterol Myths: Exposing the Fallacy that Cho-lesterol and Saturated Fat Cause Heart Disease. Washington DC: *New Trends Publishing, Inc.*; 2000.
128. Rong Y, Chen L, Zhu T, Song Y, Yu M, Shan Z, Sands A, Hu FB, Liu L: Egg consumption and risk of coronary heart disease and stroke: dose-response meta-analysis of prospective cohort studies. *BMJ*, 2013 Jan.
129. Russo GL: Dietary n-6 and n-3 polyunsaturated fatty acids: From biochemistry to clinical implications in cardiovascular prevention. *Biochem Pharmacol*, 2009 Mar.
130. Ruxton CH, Reed SC, Simpson MJ, Millington KJ: The health bene-fits of omega-3 polyunsaturated fatty acids: a review of the evidence. *Journal of Human Nutrition and Dietetics*, 2004 Oct.
131. Saint Onge JM, Krueger PM, Rogers RG.: Historical trends in height, weight, and body mass: Data from U.S. Major League Baseball play-ers, 1869-1983. *Economics & Human Biology*, 2008 Dec.
132. Saslow LR, Kim S, Daubenmier JJ, Moskowitz JT, Phinney SD, Goldman V, Murphy EJ, Cox RM, Moran P, Hecht FM: A randomized pilot trial of a moderate carbohydrate diet compared to a very low car-bohydrate diet in overweight or obese individuals with type 2 diabetes mellitus or prediabetes. *Plos One*. 2014 Apr.
133. Schatz IJ, Masaki K, Yano K, Chen R, Rodriguez BL, Curb JD: Cho-lesterol and all-cause mortality in elderly people from the Honolulu Heart Program: a cohort study. *Lancet*, 2001 Aug.
134. Simopoulos AP: Omega-3 fatty acids in health and disease and in growth and development. *The American Journal of Clinical Nutrition*, 1991.
135. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM: Meta-analysis of pro-spective cohort studies evaluating the association of saturated fat with cardiovascular disease. *The American Journal of Clinical Nutri-tion*, 2010 Mar.
136. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM: Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *The American Journal of Clinical Nutrition*, 2010.
137. Skerrett PJ: FDA gets with the evidence, proposes that trans fats are not "safe." *Harvard Health Publications*, 2013.
138. Sniderman AD, Williams K, Contois JH, Monroe HM, McQueen MJ, de Graaf J, Furberg CD: A meta-analysis of low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and apolipoprotein b as markers of cardiovascular risk. *Circulation: Cardiovascular Quality and Outcomes*, 2011 May.

References and Further Readings (continued)

139. Soderstrom L, Rosenblad A, Adolfsson ET, Wolk A, Hakansson N, Bergkvist L: A high energy intake from dietary fat among middle-aged and older adults is associated with increased risk of malnutrition 10 years later. *British Journal of Nutrition*. 2015
 140. Spence JD, Judd S, Howard V, Safford M, Howard G. Session Title: Community/Risk Factors Oral Abstracts I. Abstract 83: Effect of Dietary Cholesterol and Egg Consumption on Mortality and Cardiovascular Risk in the REGARDS Study, 2015.
 141. Studer M, Briel M, Leimenstoll B, Glass TR, Bucher HC: Effect of different antilipidemic agents and diets on mortality: a systematic review. *Archives of Internal Medicine*, 2005 Apr.
 142. Takeoka GR, Full GH, Dao LT: Effect of Heating on the Characteristics and Chemical Composition of Selected Frying Oils and Fats. *Western Regional Research Center, Agricultural Research Service, U.S. Department of Agriculture*, 1997.
 143. Tani S, Saito Y, Anazawa T, Kawamata H, Furuya S, Takahashi H, Iida K, Matsumoto M, Washio T, Kumabe N, Nagao K, Hirayama A: Low-density lipoprotein cholesterol/apolipoprotein B ratio may be a useful index that differs in statin-treated patients with and without coronary artery disease: a case control study. *International Heart Journal*, 2011.
 144. Tarrago-Trani MT, PhD, Phillips KM, PhD, Lemar LE, MS, Holden JM, MS: New and existing oils and fats used in products with reduced trans-fatty acid content, 2006 Jun.
 145. Thanassoulis G, Williams K, Ye K, Brook R, Couture P, Lawler PR, de Graaf J, Furberg CD, Sniderman A: Relations of change in plasma levels of LDL-C, non-HDL-C and apoB with risk reduction from statin therapy: a meta-analysis of randomized trials. *Journal of the American Heart Association*, 2014.
 146. U.S. Department of Agriculture, Interactive Nutrient Content of the U.S. Food supply. <http://65.216.150.146/NFSDatabase/QueNut.asp>.
 147. Uffe Ravnskov, M.D., Ph.D: The Cholesterol Myths: 2000.
 148. United States Environmental Protection Agency and Office of Air Quality Planning and Standards: Economic Impact Analysis for the Final Vegetable Oil Processing NESHAP, 2001 Jan.
 149. Virtanen JK, Mursu J, Tuomainen TP, Virtanen HE, Voutilainen S: Egg consumption and risk of incident type II diabetes in men: the Kuopio Ischaemic Heart Disease Risk Factor Study, *The American Journal of Clinical Nutrition*, 2015 May.
 150. Volek JS, Feinman RD: Carbohydrate restriction improves the features of Metabolic Syndrome. Metabolic Syndrome may be defined by the response to carbohydrate restriction. *Nutrition and Metabolism*, 2005.
 151. Volek JS, Phinney SD, Forsythe CE, Quann EE, Wood RJ, Puglisi MJ, Kraemer wJ, Bibus DM, Fernandez ML, Feinman RD: Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet. *Lipids* 2009.
 152. Volek JS, Volk BM, Phinney SD: The twisted tale of saturated fat, 2012.
 153. Vos E: a-Linolenic acid, linoleic acid, coronary artery disease, and overall mortality. *Department of Nutritional Sciences Faculty of Medicine University of Toronto*, 2003 Feb.
 154. Waddington E, Sienuarib K, Puddeya I, Croft K: Identification and Quantitation of Unique Fatty Acid Oxidation Products in Human Atherosclerotic Plaque Using High-Performance Liquid Chromatography, May 2001.
 155. Walldius G, Jungner I, Aastveit AH, Holme I, Furberg CD, Sniderman AD: The apoB/apoA-I ratio is better than the cholesterol ratios to estimate the balance between plasma proatherogenic and antiatherogenic lipoproteins and to predict coronary risk. *Clinical Chemistry and Laboratory Medicine*, 2004.
 156. Walldius G, Jungner I, Holme I, Aastveit AH, Kolar W, Steiner E: High apolipoprotein B, low apolipoprotein A-I, and improvement in the prediction of fatal myocardial infarction (AMORIS study): a prospective study. *Lancet*, 2001.
 157. William S. Harris, PhD, FAHA, Chair; Dariush Mozaffarian, MD, DrPH, FAHA; Eric Rimm, ScD, FAHA; Penny Kris-Etherton, PhD, FAHA; Lawrence L. Rudel, PhD, FAHA; Lawrence J. Appel, MD, MPH, FAHA; Marguerite M. Engler, PhD, FAHA; Mary B. Engler, PhD, FAHA; Frank Sacks, MD, FAHA: Omega-6 Fatty Acids and Risk for Cardiovascular Disease
 158. Wright JD, Wang CY: Trends in Intake of Energy and Macronutrients in Adults From 1999-2000 Through 2007-2008. *NCHS Data Brief*, 2010 Nov.
 159. Wu JH, Lemaitre RN, King IB, Song X, Psaty BM, Siscovick DS, Mozaffarian D: Circulating omega-6 polyunsaturated fatty acids and total and cause-specific mortality: the Cardiovascular Health Study, 2014 Aug.
 160. Yamagishi K, Iso H, Tsugane S: Saturated Fat Intake and Cardiovascular Disease in Japanese Population. *Journal of Atherosclerosis and Thrombosis*, 2015 Jan.
 161. Yamagishi K, Iso H, Yatsuya H, Tanabe N, Date C, Kikuchi S, Yamamoto A, Inaba Y, Tamakoshi A: Dietary intake of saturated fatty acids and mortality from cardiovascular disease in Japanese: the Japan Collaborative Cohort Study for Evaluation of Cancer Risk Study. *The American Journal of Clinical Nutrition*, 2010 Oct; 92.
 162. Yancy WS, Foy M, Chalecki AM, Vernon MC, Westman EC: A low-carbohydrate, ketogenic diet to treat type 2 diabetes. *Nutrition and Metabolism*. 2005.
 163. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L: Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*, 2004 Sep.
- Books:**
1. Campbell CT, Campbell TM: The China Study: The most comprehensive study of nutrition ever conducted and the startling implications for diet, weight loss and long-term health. Dallas, TX: BenBella, 2005.
 2. Feinman RD: The world turned upside down. *NMS Press- Duck in a Boat LLC*, 2014.
 3. Jaminet P, Shou-Ching J: Perfect Health Diet: Regain Health and Lose Weight by Eating the Way You Were Meant to Eat. New York: *Scribner*, 2012.
 4. Perlmutter D, Loberg K: Grain Brain, The Surprising Truth about Wheat, Carbs, and Sugar- Your Brain's Silent Killers, 2013.
 5. Smoller J, Wassertheil-Smoller S : Biostatistics and Epidemiology: A Primer for Health and Biomedical Professionals. New York: Springer-Verlag, 2004.
 6. Talbot G: Reducing saturated fats in foods. Oxford, England: Woodhead Pub., 2011.
 7. Taubes G: Good Calories, Bad Calories. New York: *Anchor Books*; 2007.
 8. Teicholz N: The Big Fat Surprise. Why Butter, Meat & Cheese Belong in a Health Diet. New York: *Simon & Schuster*; 2014.
 9. Volek JS, Phinney SD: the art and science of low-carbohydrate living. Charleston, S.C. *Beyond Obesity*, 2011.

Also published by the Research Institute



Opportunities in an urbanizing world
April 2012



Family businesses: Sustaining performance
September 2012



The shale revolution
December 2012



Sugar Consumption at a crossroads
September 2013



Latin America: The long road
February 2014



Emerging Consumer Survey 2014
February 2014



Global Investment Returns Yearbook 2014
February 2014



Emerging capital markets: The road to 2030
July 2014



The Success of Small Countries
July 2014



The CS Gender 3000: Women in Senior Management
September 2014



Global Wealth Report 2014
October 2014



Emerging Consumer Survey 2015
January 2015



Global Investment Returns Yearbook 2015
February 2015



The Success of Small Countries and Markets
April 2015



The Family Business Model
July 2015

Imprint

PUBLISHER

CREDIT SUISSE AG

Research Institute
Paradeplatz 8
CH-8070 Zurich
Switzerland
cs.researchinstitute@credit-suisse.com

AUTHORS

Stefano Natella
Vamil Divan
Marcela Giraldo

CONTRIBUTORS

Alainn Bailey
Muriel Chen
Utkarsh Goklani
Charlie Mills
Rob Moskow
Vinit Sinha
Ting Min Tang
Priscilla Tjitra

EDITORIAL DEADLINE

September 16, 2015

General disclaimer / Important information

This document was produced by and the opinions expressed are those of Credit Suisse as of the date of writing and are subject to change. It has been prepared solely for information purposes and for the use of the recipient. It does not constitute an offer or an invitation by or on behalf of Credit Suisse to any person to buy or sell any security. Nothing in this material constitutes investment, legal, accounting or tax advice, or a representation that any investment or strategy is suitable or appropriate to your individual circumstances, or otherwise constitutes a personal recommendation to you. The price and value of investments mentioned and any income that might accrue may fluctuate and may fall or rise. Any reference to past performance is not a guide to the future.

The information and analysis contained in this publication have been compiled or arrived at from sources believed to be reliable but Credit Suisse does not make any representation as to their accuracy or completeness and does not accept liability for any loss arising from the use hereof. A Credit Suisse Group company may have acted upon the information and analysis contained in this publication before being made available to clients of Credit Suisse. Investments in emerging markets are speculative and considerably more volatile than investments in established markets. Some of the main risks are political risks, economic risks, credit risks, currency risks and market risks. Investments in foreign currencies are subject to exchange rate fluctuations. Any questions about topics raised in this piece or your investments should be made directly to your local relationship manager or other advisers. Before entering into any transaction, you should consider the suitability of the transaction to your particular circumstances and independently review (with your professional advisers as necessary) the specific financial risks as well as legal, regulatory, credit, tax and accounting consequences. This document is issued and distributed in the United States by Credit Suisse Securities (USA) LLC, a U.S. registered broker-dealer; in Canada by Credit Suisse Securities (Canada), Inc.; and in Brazil by Banco de Investimentos Credit Suisse (Brasil) S.A.

This document is distributed in Switzerland by Credit Suisse AG, a Swiss bank. Credit Suisse is authorized and regulated by the Swiss Financial Market Supervisory Authority (FINMA). This document is issued and distributed in Europe (except Switzerland) by Credit Suisse (UK) Limited and Credit Suisse Securities (Europe) Limited. Credit Suisse Securities (Europe) Limited and Credit Suisse (UK) Limited, both authorized by the Prudential Regulation Authority and regulated by the Financial Conduct Authority and the Prudential Regulation Authority, are associated but independent legal entities within Credit Suisse. The protections made available by the Financial Conduct Authority and/or the Prudential Regulation Authority for retail clients do not apply to investments or services provided by a person outside the UK, nor will the Financial Services Compensation Scheme be available if the issuer of the investment fails to meet its obligations. This document is distributed in Guernsey by Credit Suisse (Channel Islands) Limited, an independent legal entity registered in Guernsey under 15197, with its registered address at Helvetia Court, Les Echelons, South Esplanade, St Peter Port, Guernsey. Credit Suisse (Channel Islands) Limited is wholly owned by Credit Suisse AG and is regulated by the Guernsey Financial Services Commission. Copies of the latest audited accounts are available on request. This document is distributed in Jersey by Credit Suisse (Channel Islands) Limited, Jersey Branch, which is regulated by the Jersey Financial Services Commission for the conduct of investment business. The address of Credit Suisse (Channel Islands) Limited, Jersey Branch, in Jersey is: TradeWind House, 22 Esplanade, St Helier, Jersey JE4 5WU. This document has been issued in Asia-Pacific by whichever of the following is the appropriately authorised entity of the relevant jurisdiction: in Hong Kong by Credit Suisse (Hong Kong) Limited, a corporation licensed with the Hong Kong Securities and Futures Commission or Credit Suisse Hong Kong branch, an Authorized Institution regulated by the Hong Kong Monetary Authority and a Registered Institution regulated by the Securities and Futures Ordinance (Chapter 571 of the Laws of Hong Kong); in Japan by Credit Suisse Securities (Japan) Limited; this document has been prepared and issued for distribution in Singapore to institutional investors, accredited investors and expert investors (each as defined under the Financial Advisers Regulations) only, and is also distributed by Credit Suisse AG, Singapore Branch to overseas investors (as defined under the Financial Advisers Regulations). Credit Suisse AG, Singapore Branch may distribute reports produced by its foreign entities or affiliates pursuant to an arrangement under Regulation 32C of the Financial Advisers Regulations. Singapore recipients should contact Credit Suisse AG, Singapore Branch at +65-6212-2000 for matters arising from, or in connection with, this report. By virtue of your status as an institutional investor, accredited investor, expert investor or overseas investor, Credit Suisse AG, Singapore Branch is exempted from complying with certain compliance requirements under the Financial Advisers Act, Chapter 110 of Singapore (the "FAA"), the Financial Advisers Regulations and the relevant Notices and Guidelines issued thereunder, in respect of any financial advisory service which Credit Suisse AG, Singapore branch may provide to you. ; elsewhere in Asia/Pacific by whichever of the following is the appropriately authorized entity in the relevant jurisdiction: Credit Suisse Equities (Australia) Limited, Credit Suisse Securities (Thailand) Limited, Credit Suisse Securities (Malaysia) Sdn Bhd, Credit Suisse AG, Singapore Branch, and elsewhere in the world by the relevant authorized affiliate of the above.

This document may not be reproduced either in whole, or in part, without the written permission of the authors and Credit Suisse. © 2015 Credit Suisse Group AG and/or its affiliates. All rights reserved

CREDIT SUISSE AG
Research Institute
Paradeplatz 8
CH-8070 Zurich
Switzerland
cs.researchinstitute@credit-suisse.com
www.credit-suisse.com/researchinstitute

