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# Human Health Effects of Fatty Acids in Beef<sup>1</sup>

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The popular perception of fats is that they not only make you fat, but they also increase the risk of a number of health problems such as heart diseases, stroke, diabetes, and some cancers. However, fats are essential to human health. To date numerous reports have been written describing the influence of dietary fats on humans and animals. The objective of this paper is to describe the importance of individual fatty acid on human health.

The white material called fat on a cut of meat is adipose tissue. This adipose tissue is a form of connective tissue, which functions to surround adipose cells that serve as heat insulators and reserve energy supplies (Allen, 1976). These adipose cells are distended with lipids, water, and other constituents. Lipids will constitute 80% to 90% of the volume of adipose tissue in finished beef animals. Five to 15% is water and the remainder is protein, minerals, and carbohydrates. However, such proportions vary with stage of growth and state of nutritional change in the animal (Allen, 1976).

Often the terms "fat" and "lipid" are used interchangeably. Speaking, in a strict chemical sense, the term "fat" refers to lipids, which are the major components of adipose tissue. Fatty acids are a type of lipid.

A fatty acid is a chain of carbon atoms with hydrogen atoms attached. The first obvious classification of fatty acids is by carbon chain length, which may range from one (C1) to 30 (C30) carbon atoms. For instance, C12 represents a fatty acid with 12 carbon atoms.

Fatty acids of C1 to C6 length exist in a free form. The longer carbon chain fatty acids can exist in a free form or esterified as triglyceride (3 fatty acids bound to a glyceride molecule). Fatty acids located within the adipose tissue in ruminants occur almost entirely as triglycerides with a predominance of C16 and C18 fatty acids.

Fatty acids may be saturated, monounsaturated, or polyunsaturated. A saturated fatty acid is a molecule that has no double bonds in the carbon

chain and carries the maximum number of hydrogen atoms. The number following the colon that refers to the length of the carbon chain indicates the degree of saturation. Thus, C12:0 refers to a fatty acid with 12 carbon atoms and no double bonds (a saturated fatty acid). When unsaturation occurs, one or more hydrogen atoms are removed and a double bond between two carbons is formed (C=C). For instance, C16:1 represents a monounsaturated fatty acid composed of 16 carbons with 1 unsaturation point (removal of two hydrogen atom; mono means one), while C18:3 represents a polyunsaturated fatty acid composed of 18 carbons with 3 unsaturation points (removal of 6 hydrogen atoms, poly means many).

Both number and location of the double bond are important for a fatty acid's biological function and stability. For instance, saturation increases stability. Fats can become oxidized or rancid when exposed to oxygen. Polyunsaturated fatty acids spoil because the double bonds are unstable. Saturated fats are more resistant to oxidation and thus less likely to become rancid.

When double bonds are present, the fatty acids may be present in either a *cis*- or *trans*- configuration, giving different shapes to the fatty acid.

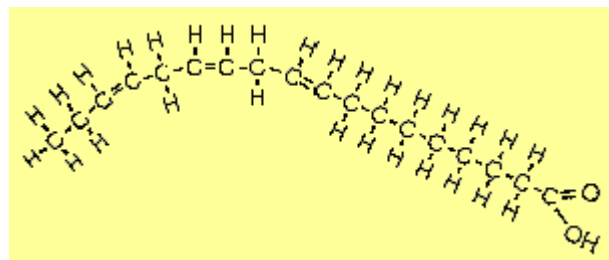


Fig.1

Shown above is the essential fatty acid, alpha-linolenic acid (C18:3 *cis*) (the notation C18:3 tells us that the fatty acid has 18 carbons in the carbon chain and 3 points of unsaturation due to 3 points between carbon atoms with double bonds). Note that at the double bonds, the missing hydrogen atoms all come from one side. This is the *cis*- configuration (18:3 *cis*). Unsaturated fatty acids rarely

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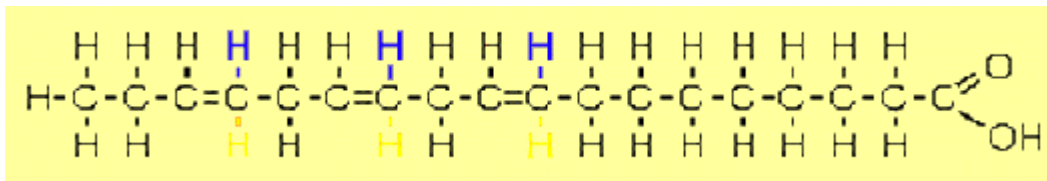


Fig.2

appear in the trans-form in nature, except in small amounts in ruminant products (dairy, beef). However, when unsaturated fatty acids are overheated as in industry, the hydrogen atoms can jump from side to side. This is most commonly found in partially hydrogenated vegetable oils. The picture below shows alpha-linolenic acid in its trans-configuration (C18:3 trans).

The gray Hs in the above illustration show where the hydrogen atoms were before heating. The black Hs show the spots they moved into. Notice that they flipped sides. Even though the same number of carbon and hydrogen atoms is in the molecule, this is not at all the same fatty acid. Because there is now a balanced number of hydrogen atoms on each side of the molecule, it no longer bends, but is straight, like a saturated fatty acid. Also, its melting point has risen dramatically. In our bodies, it acts more like a saturated fatty acid than an unsaturated fatty acid. Most trans-fatty acids do not have beneficial properties compared to cis-fatty acids.

Saturated fatty acids are the main fatty acids found in meat and dairy products. Unsaturated fats are most commonly found in the oils of vegetables, such as avocado, and also in nuts and fish. Unsaturated fats are considered the healthiest dietary fats.

Another classification of fats is the "essential fatty acids". These are the polyunsaturated fatty acids that cannot be produced in the human body and must be obtained from the diet, since they are needed for basic functions in our bodies. Present in every healthy cell of the body, they are critical for normal growth and functioning of the cells, muscles, nerves, and organs. Deficiency of essential fatty acids is linked to a variety of symptoms including rough and scaly skin and dermatitis (omega-6 fatty acid) and scaly and hemorrhagic dermatitis, hemorrhagic folliculitis of the scalp, impaired wound healing and growth retardation (omega-3 fatty acids). Adequate intakes of essential fatty acids may protect against heart diseases and diabetes (DRI, 2002). It has been estimated that as much as 50% of the American population may consume insufficient quantities of essential fatty acids (Hu et al., 1999).

There are two types of essential fatty acids important to human health – the omega-6 and the omega-3 fatty acids. The term "omega" refers to the position of the first double bond in the fatty acid, counting from the methyl end (O=C—OH) of the carbon chain. An omega-3 fatty acid has the first double bond on the third carbon atom, and an omega-6 fatty acid has the first double bond on the sixth carbon atom. Omega-3 and omega-6 fatty acids cannot be converted from one form to the other. Both have to be present in the diet in a proper balance for a good health.

The metabolism of omega-6 and omega-3 fatty acids in our bodies requires the same enzymes system, resulting in competition between the two families. An excess of one family of fatty acids (usually omega-6) can interfere with the metabolism of the other (usually omega-3), reducing the production of omega-3-derived long-chain fatty acids, and altering their biological effects (Horrobin and Manku, 1990; Emken, 1995). For instance, linoleic (C18:2; omega-6) and alpha-linolenic (C18:3; omega-3) fatty acids are both essential to humans. From these fatty acids, enzymes in the body can produce other, longer chain fatty acids such as arachidonic acid (AA, C20:4) omega-6, and docosahexaenoic acid (DHA, 22:6) and eicosapentaenoic acid (EPA, C20:5) omega-3, respectively. These two fatty acids work together in a competitive balance to regulate blood clotting, immune response, and inflammatory processes. The AA fatty acid aids in the constriction of blood vessels and the formation of blood clots, and EPA aids in the relaxation of blood vessels and inhibits blood clotting. Although they have opposite functions, both are necessary to maintain a healthy balance between excessive bleeding and excessive blood clotting (Clandinin, 2000).

With changes in food production and diet, the consumption of omega-6 has risen in this country while omega-3 consumption has fallen. Ideally, intake of omega-6 fatty acids should be no more than 10 times that of omega-3 fatty acids (DRI, 2002), some believe it should be even lower at 4:1 (Simopoulos, 1999). Today in the typical U.S. diet, omega-6 intake is up to 20-30 times more than omega-3 intake (Simopoulos, 1999).

About 80% of the fatty acid in beef is composed of palmitic (C16:0), stearic (C18:0), and oleic acid (C18:1). The remaining 20% is distributed among 30 different fatty acids. Inference of the health effect of fatty acids is based on experimental diets enriched with selected fatty acids.

Oleic acid (C18:1) is the primary mono-unsaturated fatty acid in beef and accounts for about 33% of the fatty acid in beef. It is also found in rich amounts in olive, canola, and peanut oils. Available evidence indicates that while most saturated fatty acids raise serum cholesterol concentrations the monounsaturated oleic acid does not (Denke, 1994). For practical purposes, it is convenient to use the neutrality of oleic acid as a baseline with which to judge the responses of other fatty acids. The fact that the body synthesizes a large quantity of oleic acid suggests that it has a variety of biological uses, and to this extent the concept of the neutrality of oleic can be extended to imply safety (Grundy, 1994). In several studies on the relative carcinogenicity of fatty acids or their ability to suppress the immune system, oleic acid was the fatty acids with the least negative effect (Grundy, 1994). One reason why oleic acid may not raise serum cholesterol concentrations is because it is a favored substrate for the liver enzyme that converts cholesterol to an inactive form (the Acyl CoA transferase: cholesterol acyltransferase) (Grundy, 1994).

Palmitic acid (C16:0) is a saturated fatty acid accounting for about 27% of the fatty acids in beef. There is very strong evidence that palmitic acid raises serum cholesterol levels (Grundy, 1994) and that this occurs predominantly by increasing bad cholesterol (LDL) levels. This fatty acid accounts for most of the cholesterol-raising activity from beef, thereby increasing the risk of atherosclerosis, cardiovascular disease, and stroke (Nicolosi et al., 1998).

Stearic acid (18:0) is a saturated fatty acid accounting for about 18% of the fatty acid in beef. Several studies have shown that the stearic acid effect on total cholesterol is minimal and not detrimental to human health (Bonanome et al., 1988; Zock et al., 1992; Kris-Etherton et al., 1993; Judd et al., 2002). For practical purposes, stearic acid is essentially neutral in its effects on serum total cholesterol, similar to oleic acid (Grundy, 1994). It is not clear why stearic acid does not raise cholesterol level as do other saturated fatty acids. A possible reason could be that it is rapidly absorbed into body tissue compared with other saturated fatty acids (Grundy, 1994). However, it has been observed in dogs, rats, and hamsters that stearic acid or stearic acid-rich glycerides are absorbed less efficiently

than saturated fatty acids of shorter chain length or their glycerides (Kitchevsky, 1994). Some investigators have speculated that stearic acid may be thrombogenic (causes blood clotting). This effect has not been proven (Grundy, 1994). Also, the effects of stearic acid on hypertension, cancer, obesity, and other illnesses are unknown (Hu et al., 1999).

Beef is also a source of two saturated fatty acids, lauric (C12:0) and myristic (C14:0), that are related to human health issues. Lauric and myristic fatty acids are responsible for raising bad cholesterol levels in blood serum (Grundy, 1994) and have been shown to be strongly correlated with early heart attack (Kromhout et al., 1995). However, the percentages of lauric (less than 1%) and myristic (2-3%) acids in beef are small. Data suggest that lean beef has no more cholesterol-raising effect than chicken or fish and therefore, lean beef need not to be eliminated from cholesterol-lowering diets (Denke, 1994).

Fatty acids with an odd number of carbon atoms such as pentadecanoic (C15:0) and heptadecanoic acid (C17:0) (less than 1% and 1-2% of fatty acids in beef respectively), are produced primarily from accumulation of a 3-carbon fatty acid end product that occurs due to the lack of vitamin B12. Odd-chain fatty acids can build up in membrane lipids of nervous tissue, resulting in altered myelin integrity and demyelination, leading eventually to impaired nervous system functioning (Frenkel et al., 1973).

Elaidic acid (C18:1) (2-5% of the fatty acid in beef), is a monounsaturated trans-fatty acids, which can raise bad cholesterol (LDL) in serum (Abbey and Nestel, 1994; Muller et al., 1999, Judd et al., 2002). Trans-fatty acids, as mention above, may behave similar to saturated fatty acids. Studies have shown that foods enriched in C18:1-trans resulted in higher bad cholesterol (LDL) levels compared with C18:1-cis (Nicolosi, et al., 1998; Judd et al., 2002). Whereas C18:1-trans raised bad cholesterol (LDL) equivalent to saturated fatty acids, it had no effects on good cholesterol (HDL) (Judd et al., 2002).

Other monosaturated fatty acids are palmitoleic acid (C16:1) (about 2%-3%), transvaccenic acid (C18:1 trans-11) (3%-4%), and vaccenic acid (C18:1 cis-11) (1-2%). Transvaccenic acid is important in the human bodies' production of conjugated linolenic acids (CLA) which is discussed later. Palmitoleic acid (C16:1 cis) is a mono-unsaturated fatty acid also found in rich amounts in macadamia nuts, olive, canola, and peanut oils. This mono-unsaturated fatty acid is beneficial in reducing bad cholesterol (LDL) and it behaves like a saturated

and not as a saturated fatty acid in its effect on LDL cholesterol (Nestel et al., 1994). It also reduces the fat deposition in blood vessels and reduces blood clot formation (Grundy, 1994). Vaccenic acid has no known importance to human health. However, all these C:18 fatty acids may be elongated and desaturated in adipose tissue to produce long chain fatty acids (C22 and C20), which are beneficial for human health (Burdge and Wootton, 2002).

The major important polyunsaturated fatty acids found in beef are linoleic acid (C18:2) (about 3.5%), alpha-linolenic acid (C18:3) (1.5%), arachidonic acid (C20:4) (about 1%), eicosapentaenoic acid (EPA) (C20:5) (<1%), docosapentaenoic acid (DPA-3) (C22:5) (<1%), and docosahexaenoic acid (DHA) (C22:6) (<1%) (Enser et al., 1998). Alpha-linolenic acid (C18:3) is classified as a short-chain omega-3 fatty acid and is also found in nuts and seeds. Eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA-3), and docosahexaenoic acid (DHA) are found predominantly in foods of marine origin and are classified as long-chain omega-3 fatty acids. The meat and milk of grazing animals has been reported to contain significantly more omega-3 fatty acids than does meat and milk of animals fed conserved forages and grains. This higher content of omega-3 fatty acids may be beneficial to human health (Kelly et al., 1988; Dhiman, 1999).

Linoleic acid (C18:2) (about 3.5%) is also found in corn, sunflower oil, safflower oil and soybeans. Arachidonic acid (C20:4) (about 1%) is found in brain, liver, glandular and egg lipids. Both of these fatty acids belong to the omega-6 family of fatty acids.

The omega-3 fatty acids present in pastures, like the alpha-linolenic acid (C18:3), appear to have little direct value for human health. However, the human body can add 2 or 4 carbons to these 18-carbon chain fats to produce 20- or 22-carbon chain omega-3 fatty acid. Thus, alpha-linolenic acid (C18:3) is a precursor for EPA (C20:5) and DHA (C22:6) fatty acids, which are important for human health. It has been suggested that alpha-linolenic acid has a beneficial effect on cardiovascular heart disease (Ascherio et al., 1999; Hu et al., 1999). However, other studies reported no evidence of alpha-linolenic acid having a positive effect on cardiovascular heart disease (Renaud et al., 2002; Sanderson et al., 2002). Although alpha-linolenic acid supplementation causes an increase in the blood and plasma levels of alpha-linolenic acid, EPA and DPA, no benefit has shown on either risk factors for cardiovascular diseases or on the secondary prevention of cardiovascular heart disease (Sanderson et al., 2002). More studies have to be done to deter-

mine alpha-linolenic acid beneficial effects on human health. Alpha-linolenic acid (C18:3) may help balance linoleic acid (C18:2) and be beneficial (Baer, 2003 personal communication).

For many years linoleic acid (C18:2; omega-6) was thought to be the preferable fatty acid for the diet because it was considered to be the most effective cholesterol-lowering fatty acid. However, despite an increase in linoleic acid intake (from about 4% to 7%), there has been a growing reservation about recommending its consumption, due to no proven long-term safety (Grundy, 1994). In humans, high supplemental intakes of linoleic fatty acids can lower good cholesterol concentration and may increase the risk for cholesterol gallstones. In addition, the presence of linoleic acid in bad cholesterol lipids makes them more prone to oxidation, which could promote atherosclerosis. Because of these detrimental effects, current recommendations have been moderated and now caution that intakes of this fatty acid should not exceed current concentrations (about 7% of total energy intake) (Grundy, 1994). Surprisingly, recent information from the American Heart Association indicates that linoleic acid has a noticeable effect on lowering cholesterol further than oleic and palmitic acids when plasma cholesterol levels are high (>200 mg/dL). They suggest that at a 10 % calorie intake in the form of polyunsaturated fats, linoleic acid achieves a maximal effect on cholesterol lowering. It also has been suggested (Iso et al., 2002) that a higher intake of linoleic acid appear to protect against stroke, possibly through potential mechanism of decreased blood pressure, reduced platelet aggregation, and enhance deformability of erythrocyte cells. More studies need to be done to determine the effect of linoleic acid in human health.

Linoleic acid (C18:2; omega-6) and alpha-linolenic acid (C18:3; omega-3), are plant fatty acids that can be transformed to CLA (conjugate linolenic acid) by bacteria in the rumen (Kepler et al. 1966). CLA is a collective term describing a mixture of positional and conjugated isomers of linoleic acid (C18:2) involving a double bond at positions 8 and 10, 9 and 11, 10 and 12, or 11 and 13 (Eulitz et al., 1999). Each of these positional C18 isomers can occur in cis-trans, trans-cis, cis-cis, and trans-trans forms (Eulitz et al., 1999). In beef and milk samples, the cis 9-trans 11 and the cis10-trans12 CLA are the predominant forms. Interest in CLA research has increased in the past few years as a result of reports of CLA consumption providing several health benefits (Kramer, 1998)). Because plants do not synthesize CLA, ruminant fats in milk or meat are the primary dietary CLA sources for humans (Herbein et al., 2000). The predominant CLA

in ruminant fats is the cis-9, trans-11 isomer that accounts for more than 80% of total CLAs (Chin et al., 1992). It has been found that CLA is an antioxidant, which also reduces circulating cholesterol in mice (West et al., 1998). Other literature reports that CLA has a positive effect by reducing cardiovascular risk, protects against atherosclerosis, is anti-carcinogenic, reduces intake, reduces body content of adipose tissue and lipid, and enhances the immune system (Lee et al., 1994; Nicolosi et al., 1997; Ip et al., 1991, 1994; West et al., 1998; Cook, 1991).

The CLA (C18:2 trans10, cis12) appears in smaller quantity and seems to be involved in reduction of fatty acids synthesis in cows and mice (Griinari et al., 1998; Park et al., 1999). However, no direct effect of this CLA on human health has been observed. French and co-workers (2000) reported that decreasing the proportion of concentrate (grain) in the diet, which effectively increased grass intake, causes a linear increase in CLA concentrations in intramuscular fat in steers.

It has been found that stearic acid (C18:0) cannot be stored well in tissue. It is converted to oleic acid (cis-18:1) apparently so body fat can be maintained in a "liquid state" at body temperature (Herbein et al., 2000). The enzyme that adds a double bond to stearic acid to form oleic acid is SCD (stearoyl-CoA desaturase or  $\Delta^9$ -desaturase). Most tissues of ruminants, mice, rat, and chicken, have SCD, especially in the intestines, liver, adipose tissue, and mammary glands. This enzyme has also been detected in humans. However, the distribution of SCD in humans is unknown. In humans, the liver is the principal tissue containing SCD and presumably also has the highest SCD activity (Turpeinen et al., 2001). SCD is important because it can add a cis-9 double bond to convert trans-vaccenic acid (TVA) (trans-18:1) to CLA. Therefore, beef fat is not only an excellent source of CLA, but also contains large amounts of TVA, which can be converted to CLA in the human body (Adlof et al., 2000; Turpeinen et al., 2001). Moreover, some studies confirmed that when a diet rich in TVA was fed to humans, mice, or rats, accumulation of CLA in serum fat or body tissue was detected in a much higher conversion pattern than feeding CLA itself (Salmine et al., 1998; Santora et al., 2000; Banni et al., 2001). Turpeinen et al. (2001) showed that the conversion rate over a range of TVA intakes (1.5, 3, and 4.5 g TVA/day) in human subjects was 19%, and that interindividual differences were prominent ranging from one non-responder to a conversion rate greater than 30% in another subject. Whether the amount of CLA formed from TVA in the diet will result in positive

human health benefits remains to be seen. Banni et al. (2001) showed that the CLA levels formed from TVA sources reduced the total number of cancer pre malignant lesions by about 50% in rats. TVA is a trans fatty acid that raises bad cholesterol in serum (Judd et al., 2002), however, the conversion to CLA is a benefit for human health.

It has been suggested that arachidonic acid (C20:4) is detrimental to human health (Barham et al., 2000). However, it promotes inflammation that is an important protective response when one is injured. It also forms the basis of anti-inflammatory prostaglandins that the body uses, to reduce inflammation (Fallon and Enig, 1996). The amount of arachidonic acid in beef is very low (less than 0.5% of total fat); thus, great amounts of beef have to be consumed to detect any contradictory effect.

Two other omega-3 fatty acids in beef, DHA (C22:6) and EPA (C20:5), have been reported to have health benefits (Mantzioris et al., 2000). These omega-3 fatty acids have been shown to prevent cancer (Hardman, 2002), and cardiovascular disease (Simopoulos, 2002), as well as being therapeutic for arthritis (Kremer, 2000), autoimmune disease (Harbige and Fisher, 2001), inflammatory effects (Grimm et al., 2002) and depression (Puri et al., 2001). DHA is also important during pregnancy for infant and brain development (Horrocks and Yeo, 1999; Ewinwright, 2002) and reduces the incidence of premature birth (Allen and Harris, 2002). EPA lowers blood cholesterol (Pal et al., 2002) and reduces blood clotting, allowing better blood circulation (Heller et al., 2002). Thus, there is a benefit from the production of additional DHA and EPA by the body's elongation and desaturation of shorter chain fatty acids (C18:3 omega-3; 18:2 omega-6; 18:1) in humans as mentioned above. There is a lot of popular discussion about the value of the omega-3 fatty acids for human health (Simopoulos and Robinson, 1999).

Erucic acid (C22:1; about 1% in beef fat) is a fatty acid that is apparently responsible for a favorable response of persons with nervous system disorders (Christensen et al., 1988). The administration of erucic acid in the diet will reduce the serum levels and brain accumulation of very long chain saturated fatty acids (such as C26:0) responsible for demyelination (Sargent et al., 2002; Rasmussen et al., 2002).

Accumulation of certain long-chain fatty acids is associated with degenerative diseases of the central nervous system, such as behenic acid (C22:0; about 1% in beef fat) and lignoceric acid (C24:0; about 1%) as well as that of the unsaturated mem-

bers of the C22 and C24 group. Accumulation occurs because enzymes needed to maintain turnover of those fatty acids are lacking (Lord and Braloley, 2001). Behenic acid has been detected to be a cholesterol-raising saturated fatty acids factor in humans (Cater and Denke, 2001).

In summary, individual fatty acids have diverse effects on human health. Heart diseases have been found to be favorably affected by the consumption of certain unsaturated fatty acids. Unsaturated fatty acids lower plasma total cholesterol and "bad" cholesterol levels when substituted for saturated fatty acids. However, trans-monounsaturated fatty acids were found to be intermediate between cis-monounsaturated fatty acid and long-chain saturated fatty acids in their effects on plasma total and "bad" cholesterol concentrations. Cholesterol levels are related to cardiovascular diseases. However, predominant beef fatty acids, such as oleic (C18:1 cis-9) and stearic (C18:0) appear to be essentially neutral in their effects on cholesterol levels. Moreover, when the effects of lean red meat (beef) vs. lean white meat (fish, poultry) consumption were compared on the good and total cholesterol serum levels, no significant difference ( $P < .001$ ) were present between treatment groups (Davidson et al., 1999). These researchers also observed that lean meat was a more important factor over whether the meat was white or red. Some of the monounsaturated (C18:1) fatty acids are important precursors of long chain fatty acids that are beneficial for human health. Long-chain omega-3-fatty acids (EPA and DHA) have an essential role in brain development in newborns and are vital to the healthy maintenance and function of the body. They also can help maintain long-term good health by reducing the risk of a number of diseases such as heart diseases, cancer, autoimmune diseases, inflammatory diseases, and depression. Plant fatty acids, such as linoleic acid (C18:2) and alpha-linolenic acid (C18:3), may be converted in the ruminant to CLA, which may provide several health benefits in humans relative to heart diseases, atherosclerosis, cancer, body lipid content, and immune system. Also beef is a good source of TVA, which the human body can convert to CLA. Meat of forage-raised animals contains significantly more omega-3-fatty acids, CLA, and unsaturated fatty acids, compared to meat of animals finished with stored forages or grain. Thus pasture-finished beef has the potential to compete as a health-promoting food with other health oriented products on the market.

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## References

- Abbey M. and Nestel P. J. 1994. Plasma cholesterol ester transfer protein activity is increased when trans-elaidic acid is substituted for cis-oleic acid in the diet. *Atherosclerosis* 106:99-107.
- Allen C. E., D. C. Beltz, D. A. Cramer, and R. G. Kauffman. 1976. *Biology of the fats in meat animals*. North Central Research Publication. No, 234. pp.1
- Allen K. G. and M. A. Harris. 2001. The role of n-3 fatty acids in gestation and parturition. *Exp. Biol. Med.* (Maywood) 226(6):498-506.
- Ascherio A. 2002. Epidemiologic studies on dietary fats and coronary heart disease. *Am. J. Med.* 113 (suppl) 9B:9S-12S
- Banni S., E. Angioni, E. Murru, G. Carta, M. P. Melis, D. Bauman, Y. Dong, and C. Ip. 2001. Vaccenic acid feeding increases tissue levels of conjugated linoleic acid and suppresses development of premalignant lesions in rat mammary gland. *Nutr. Cancer* 41:91-97.
- Barham J. B., M. B. Edens, A. N. Fonteh, M. M. Johnson, L. Easter, and F. H. Chilton. 2000. Addition of eicosanoic acid to gamma-linolenic acid-supplemented diets prevents serum arachidonic acid accumulation in humans. *J. Nutr.* 130:1925-1931.
- Bonanome A. and S. Grundy. 1988. Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. *N. Engl. J. Med.* 318:244-248.
- Burdge G. C. and Wootton S. A. 2002. Conversion of alpha-linolenic acid to eicosapentaenoic, docosapentaenoic and docosahexaenoic acids in young women. *Br. J. Nutr.* 88:411-420.
- Cater N. B. and M. A. Denke. 2001. Behenic acid is a cholesterol-raising saturated fatty acid in humans. *Am. J. Clin. Nutr.* 2001. 73(1):41-44.
- Chin S. F., Liu W., J. M. Storkson, and M. Pariza. 1992. Dietary sources of conjugate dienoic isomers of linoleic acid, a recognized class of anticarcinogens. *J Food Comps. Anal.* 5: 185-197.
- Clandinin M.T. 2000. Functional Aspects of n-6 and n-3 Fatty Acids. Science Policy Forum, 43<sup>rd</sup> Annual Meeting, Canadian Federation of Biological Societies, Ottawa, ON, Canada, June 22-25, 2000.

- Christensen E., T. A. Hagve, and B. O. Christophersen. The Zellweger syndrome: deficient chain-shortening of erucic acid (22:1 (n-9)). *Biochem. Biophys. Acta* 1988; 959(2):134-42.
- Cook, M. E., C. C. Miller, Y. Park, and M. Pariza. 1993. Immune modulation by altered nutrient metabolism: nutritional control of immune-induced growth depression. *Poult. Sci.* 72:1301-1305.
- Davidson M. H., D. Hunninghake, K. C. Maki, P. O. Kwiterovich Jr., and S. Kanfonek. 1999. Comparison of the effects of lean red meat vs. lean white meat on serum lipid levels among free-living persons with hypercholesterolemia. *Arch. Inter.Med.* 159:1331-1338.
- Denke M. A. 1994. Role of beef and beef tallow, an enriched source of stearic acid, in a cholesterol-lowering diet. *Am. J. Clin. Nutr.* 60 (Suppl):1044S-9S.
- Dhiman T. R. 1999. Factors enriching CLA concentration in ruminant food product. IFT Annual Meeting Technical Program Abstracts. 82-84 237.
- DRI. 2002. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients).
- Enser M, K. G. Hallett, B. Hewett, G. A. Fursey, J. D. Wood, and G. Harrington. 1998. Fatty acids content and composition of UK beef and lamb muscle in relation to production system and implications for human nutrition. *Meat Sci.* 49 (3):329-341.
- Eulitz, K., M.P. Yurawecz, N. Sehat, J. Fritsche, J.A.G. Roach, M.M. Mossoba, J.K.G. Kramer, R.O. Adlof, and Y. Ku. 1999. Preparation, separation, and confirmation of the eight geometrical *cis/trans* conjugated linoleic acid isomers 8, 10- through 11, 13-18:2. *Lipids* 34:873-877.
- Emken E. A. In: Proceedings from the Scientific Conference on Omega-3 Fatty Acids in Nutrition, Vascular Biology, and Medicine. Dallas, TX: American Heart Association, 1995, pp. 9-18.
- Fallon S. and M. G. Enig. 1996. "Tripping Lightly Down the Prostaglandin Pathways". *Price-Pottenger Nutr. Foundation Health J.* 20(3):5-8 and on [www.WestonAPrice.org](http://www.WestonAPrice.org)
- French P., C. Stanton, F. Lawless, E.G. O'Riordan, F.J. Monahan, P.J. Caffrey, and A. P. Moloney. 2000. Fatty acids composition, including CLA, of intramuscular fat from steers offered grass, grass silage, or concentrate-based diet. *J. Anim. Sci.* 78: 2849-2855.
- Frenkel E. P, Kitchens R. L, Johnson J. M. 1973. The effect of vitamin B12 deprivation on the enzymes of fatty acid synthesis. *J. Biol Chem.* 248:7450.
- Griinari, J. M., D. A. Dwyer, M. A. McGuire, D. E. Bauman, D. L. Palmquist and K. V. V. Nurmela. 1998. Trans-octadecenoic acids and milk fat depression in lactating dairy cows. *J. Dairy Sci.* 81:1251-1261.
- Grimm H., K. Mayer, P. Mayser, and E. Eigenbrodt. 2002. Regulatory potential of n-3 fatty acids in immunological and inflammatory processes. *Br. J. Nutr.* 87 Suppl 1:S59:67.
- Grundt S. M. 1994. Influence of stearic acid on cholesterol metabolism relative to other long-chain fatty acids. *Am. J. Clin. Nutr.* 60 (Suppl):986S-90S.
- Harbige L. S. and B. A. Fischer. 2001. Dietary fatty acid modulation of mucosally-induced tolerogenic immune responses. *Proc. Nutr. Soc.* 60(4): 449-456.
- Hardman W. E. 2002. Omega-3 fatty acids to augment cancer therapy. *J. Nutr.* 132 (11 Suppl):3508S-3512S).
- Heller AR, S. Fischer, T. Rossel, S. Geiger, G. Siegert, M. Ragaller, T. Zimmermann, and T. Koch. 2002. Impact of n-3 fatty acid supplemented parenteral nutrition on haemostasis patterns after major abdominal surgery. *Br J Nutr* 87 (Suppl 1):S95-101.
- Herbein J. H., J. J. Looor, and W. A. Wark. 2000. An opportunity for pasture-based dairy farms?. Mid-Atlantic dairy grazing field day and workshop; Abingdon, VA. July 11<sup>th</sup>.
- Horrobin D. F. and Manku M. S. 1990. *In Omega-6 Essential Fatty Acids*. Horrobin D. F. Ed. New York, NY: Alan R. Liss. p. p. 21-53.
- Horrocks L. A. and Y. K. Yeo. 1999. Health benefits of docosahexaenoic acid (DHA). *Pharmacol. Res.* 40(3):211-225.
- Hu F.B, M. J. Stampfer, J. E. Manson, E. B. Rimm, A. Wolk, G. A. Colditz, C. H. Hennekens, and W. C. Willett. 1999. Dietary intake of alpha-linolenic acid and risk of fatal ischemic heart disease among women. *Am J Clin Nutr* 69:890-897.
- Hu F.B, M. J. Stampfer, J. E. Manson, A. Ascherio, G. A. Colditz, F. E. Speizer, C. H. Hennekens, and W. C. Willett. 1999. Dietary saturated fats and their food sources in relation to the risk of coronary heart disease in women. *Am J Clin Nutr* 70; 1001-1008.
- Iso H., S. Sato, U. Umemura, M. Kudo, K. Koike, A. Kitamura, H. Imano, T. Okamura, Y. Naito, and T. Shimamoto. 2002. *Stroke.* 33:2086-2093.
- Ip C., S. F. Chin, J. A. Scimeca, and M. Pariza. 1991. Mammary cancer prevention by conjugated dienoic derivative of linoleic acid. *Cancer Res* 51: 6118-6124.
- Ip C., M. Singh, H. J. Thompson, and J. A. Scimeca. 1994. Conjugate linoleic acid suppresses mammary carcinogenesis and proliferative activity of the mammary gland in the rat. *Cancer* 54: 1212-1215.
- Judd J. T., D. J. Baer, B. A. Clevidence, P. Kris-Etherton, R. A. Muesing, and M. Iwane. 2002. Dietary cis and trans monounsaturated and saturated FA and plasma lipids and lipoproteins in men. *Lipids.* 37(2):123-31.
- Kelly M. L., Berry J. R., Dwyer DA, Griinari JM, Chouinard PY, Van Amburgh ME, Bauman D. E. 1998. Dietary fatty acid sources affect conjugated linoleic acid concentrations in milk from lactating dairy cows. *J. Nutr.* 128(5):881-5.
- Kramer K. G., N. Sehat, M. E. R. Dugan, M. M. Mossoba, M. P. Yurawecz, J. G. Roach, K. Eulitz, J. L. Aalhus, A. L. Schaefer, and Y. Ku. 1998. Distribution of conjugated linoleic acid (CLA) isomers in tissue lipid classes of pigs fed a commercial CLA mixture by gas chromatography and silver ion-high-performance liquid chromatography. *Lipids* 33: 549-558.



- Kremer J. M. 2000. N-3 fatty acid supplements in rheumatoid arthritis. *Am. J. Clin. Nutr.* 71(1 Suppl):349S-51S
- Kritchevsky D. 1994. Stearic acid metabolism and atherogenesis: history. *Am. J. Clin. Nutr.* 60 (Suppl):997S-1001S
- Kris-Etherton P. M., J. Deer, D. C. Mitchell, V. A. Mustad, M. E. Russell, E. T. McDennell, D. Slabsky, T. A. Pearson. 1993. The role of fatty acids saturation on plasma lipids, lipoproteins: I. Effects of whole food diets high in cocoa butter, olive oil, soybean oil, dairy butter, and milk chocolate on the plasma lipids of young men. *Metabolism* 42:121-9.
- Kepler C. R., K. P. Hirons, J. J. McNeill, and S. B. Tove. 1966. Intermediates and products of the biohydrogenation of linoleic acid by *Butyrivibrio fibrisolvens*. *J. Biol. Chem.* 241: 1350.
- Kromhout D, Menotti A, Bloemberg B, Aravanis C, Blackburn H, Buzina R, Dontas AS, Fidanza F, Giampaoli S, Jansen A, et al. 1995. Dietary saturated and *trans*-fatty acids and cholesterol and 25-year mortality from coronary heart disease: the Seven Countries Study. *Prev. Med.* 24: 308-15.
- Lord R. S. and J. A. Bralley. Copyright 2001 Metamatrix Inc. Metamatrix is a service mark registered with the United States Patent and Trademark Office ([www.metamatrix.com](http://www.metamatrix.com)).
- Mantzioris E., L. G. Clenad, R. A. Gibson, M. A. Neuman, M. Demasi, and M. J. James. 2002. Biochemical effects of a diet containing foods enriched with n-3 fatty acids. *Am. J. Clin. Nutr.* 72:42-48.
- Muller H., O. Jordal, I. Seljeflot, P. Kierulf, B. Kirkhus, O. Ledsaak and J. I. Pedersen. 1998. Effect on plasma lipids and lipoproteins of replacing partially hydrogenated fish oil with vegetable fat in margarine. *Br. J. Nutr.* 80:243-251.
- Nestel P., Clifton P. and Noakes M. 1994. Effects of increasing dietary palmitoleic acid compared with palmitic and oleic acids on plasma lipids of hypercholesterolemic men. *J. Lipid Res.* 35:656-662.
- Nicolosi R. J., E. J. Rogers, D. Kritchevsky, J. A. Scimeca, and P. J. Huth. 1997. Dietary conjugate linoleic acid reduces plasma lipoprotein and early aortic atherosclerosis in hypercholesterolemic hamster. *Artery* 22: 266-277.
- Nicolosi R. J., T. A. Wilson, E. J. Rogers, and D. Kritchevsky. 1998. Effects of specific fatty acids (8:0, 14:0, cis-18:1) on plasma lipoproteins, early atherogenic potential, and LDL oxidative properties in the hamster. *J. Lipids Res.* 39:1972-1980.
- Pal S., A. M. Thomson, C. D. Bottema, and P. D. Roach. 2002. Polyunsaturated fatty acids downregulate the low density lipoprotein receptor of human HepG2 cells. *J. Nutr. Biochem* 13(1):55-63.
- Park Y., J. M. Storkson, K. J. Albright, W. Liu, and M. W. Pariza, 1999. Evidence that the trans-10, cis-12 isomer of conjugated linoleic acid induces body composition changes in mice. *Lipids* 34: 235-241.
- Puri B. K., S. J. Counsell, G. Hamilton, A. J. Richardson, and D. F. Horrobin. 2001. Eicosapentaenoic acid in treatment-resistant depression associated with symptom remission, structural brain changes and reduced neuronal phospholipids turnover. *Int. J. Clin. Pract.* 55(8):560-563.
- Ramussen M., A. B. Moser, J. Borel, S. Khangoora, and H. W. Moser. 1994. Brain, liver, and adipose tissue erucic and very long chain fatty acids levels in adrenoleukodystrophy patients treated with glyceryl tri-erucate and trioleate oils (Lorenzo's oil). *Neurochem Res.* 19:1073-1082.
- Renaud S., and Lanzmann-Petithory D. 2002. Dietary fats and coronary heart disease pathogenesis. *Curr Atheroscler. Rep.* 4:419-424.
- Salminen, M. M., M. Mutanen, M. Jauhiainen, and A. Aro. 1998. Dietary *trans*-fatty acids increase conjugated linoleic acid levels in human serum. *Nutr. Biochem.* 9:93-98.
- Sanderson P., Y. E. Finnegan, C. M. Williams, P. C. Calder, G. C. Burdge, S. A. Wootton, B. A. Griffin, D. J. Millward, N. C. Pegge and W. J. E. Bemelmans. 2002. UK food standards agency  $\alpha$ -linolenic acid workshop report. *Br. J. Nutr.* 88:573-579.
- Santora J. E., D. L. Palmquist, and K. L. Roehrig. 2000. Trans-vaccenic acid is desaturated to conjugated linoleic acid in mice. *J. Nutr.* 130:208-215.
- Sargent J. R., Coupland K. and Wilson R. 1994. Nervonic acid and demyelinating diseases. *Med. Hypotheses.* 42:237-242.
- Simopoulos A. P. 1999. Essential fatty acids in health and chronic disease. *Am. J. Clin. Nutr.* 70 (suppl):506S-9S.
- Simopoulos A. P. and J. Robinson. 1999. The Omega Diet. The lifesaving nutritional program based on the diet of the island of Crete. Harper perennial publishers, Inc., NY.
- Simopoulos A. P. 2002. The importance of the ratio of omega-6/omega-3 essential fatty acids. *Biomed Pharmacother.* 56(8):365-79.
- Turpeinen A. T., M. Mutanen, A. Aro, I. Salminen, S. Basu, D. L. Palmquist, and M. Griinari. 2002. Bioconversion of vaccenic acid to conjugated linoleic acid in humans. *Am. J. Clin. Nutr.* 76:504-510.
- Wainwright P. E. 2002. Dietary essential fatty acids and brain function: developmental perspective on mechanisms. *Proc. Nutr. Soc.* 61(1):61-69.
- West D. B., J. P. Delany, P. M. Camet, F. Blohm, A. A. Truett, and J. Scimeca. 1998. Effect of conjugated linoleic acid on body fat and energy metabolism in the mouse. *Am. J. Physiol.* 275: R667-R672.
- Zock P. L. and Katan M. B. 1992. Hydrogenation alternatives: effects of *trans*-fatty acids and stearic acid versus linoleic acid on serum lipids and lipoprotein in humans. *J. Lipids Res.* 33: 399-410.