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METABOLISM OF ARACHIDONIC ACID, ITS CONCENTRATION IN ANIMAL PRODUCTS AND INFLUENCE ON INFLAMMATORY PROCESSES IN THE HUMAN BODY: A REVIEW

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Abstract

In this review paper we focused on the metabolism of arachidonic acid (AA), an *n-6* fatty acid. It can be metabolized to many compounds having a broad effect in the body. Their homeostasis and human health depend on the ratio of dietary *n-6* to *n-3* fatty acids. These compounds, taken together with the products of animal origin and vegetables should be better balanced in the human diet. This can be achieved by reducing arachidonic acid and its precursors through a diet that modifies the content of AA in animal products such as eggs, milk and meat.

Key words: arachidonic acid, inflammatory processes, omega-6 fatty acids, eicosanoids, metabolism of arachidonic acid

AA – arachidonic acid
ALA – α-linolenic fatty acid
COX, PGHS – cyclooxygenase
DGLA – dihomo-γ-linolenic acid
DHA – docosahexaenoic acid
DPA – docosapentaenoic acid
EPA – eicosapentaenoic acid
FFAs – unesterified fatty acids
GLA – γ-linolenic acid
LA – linoleic acid
LCFA – long chain fatty acids
LNA – linolenic acid
LOX – lipoxygenase
LT – leukotrienes

NSAIDs – nonsteroidal anti-inflammatory drugs
PG – prostaglandin
PGI – prostacyclin
PUFA – polyunsaturated fatty acids
TX – thromboxanes
5-HETE – 5-hydroxyeicosatetraenoic acid
5-HPETE – 5-hydroperoxyeicosatetraenoic acid

Arachidonic acid (AA), which is an *n-6* polyunsaturated fatty acid, is used in the animal body for a number of biological functions. It is a component of phospholipid membranes and is a precursor to an extensive group of compounds called eicosanoids. Three of them: prostaglandins, leukotrienes and thromboxanes are largely responsible for the regulation of inflammatory processes. In this study we described the metabolism of arachidonic acid to the previously mentioned biologically active compounds and their impact on the inflammatory response with particular emphasis on excessive immune response to the pathogen and the possibility of its modification by changing the diet and also the composition of animal meat.

Biosynthesis of arachidonic fatty acid and other long-chain polyunsaturated fatty acids

In addition to docosahexaenoic acid (C22:6 DHA), arachidonic fatty acid (C20:4) is the major component of cell membranes. It is esterified in the phospholipids of cell membranes in the sn-2 position, and is present in abundant quantities in nervous tissue such as brain, synapses, myelin, astrocytes and in the kidneys and the heart (Wainwright, 2002; Palmquist, 2009). Arachidonic fatty acid is also present at relatively high levels in cell membranes of erythrocytes, neutrophils, monocytes as well as in liver cells (Simpoulos, 2001), whereas DHA is found at high levels in only a few tissues outside the central nervous system. According to Spector (2000), the content of arachidonic acid in plasma phospholipids and triglycerides is 8% and 1.64% respectively, while docosahexaenoic acid content is only 2.4% and 0.35%. It should be noted that besides AA and DHA fatty acids, the main fatty acid which is found in both lipids is the linoleic acid (LA) from the *n-6* group (a precursor of arachidonic acid). It constitutes 21%–23% of total fatty acids. In conclusion, arachidonic acid is an essential unsaturated fatty acid, because it is required for normal development and functioning of the organism. The rest of the paper will present the effects of excessive consumption of AA and LA fatty acids.

The linoleic fatty acid (C18:2) from the *n-6* group as well as α-linolenic fatty acid (C18:3 ALA) from the *n-3* group are classified as essential fatty acids which means that these acids, due to the lack of adequate enzymes (Δ12- and Δ15-desaturase) cannot be synthesized in the organism of animals and humans and therefore must be supplied with food (Jelińska, 2005; Kolanowski, 2007). The exception are eicosapentaenoic acid (C20:5 EPA) and docosahexaenoic acid (C22:6) from the *n-3* group and arachidonic acid from the *n-6* group, which can be provided directly in the diet or synthesized in the body from linoleic and α-linolenic fatty acids (Cook and McMaster, 2002; Kolanowski, 2007). Because the organism of cats has limited ability

to synthesize arachidonic fatty acid due to the low activity of Δ6-desaturase enzyme, arachidonic fatty acid is also basic for these animals (Bauer, 1997). Conversion of essential fatty acids is more efficient and more intensive in animal tissues (Bezard et al., 1994; Palmquist, 2009). The amount of arachidonic acid in humans and animals depends largely on the composition of fatty acids supplied in the diet, as well as the proportion between different groups of fatty acids.

Dietary arachidonic acid, linoleic acid as well as other polyunsaturated fatty acids are esterified in the liver and then released into the bloodstream, bound to lipoproteins and distributed to all cells (Nowak, 2009). The uptake of plasma triglycerides by the cells through the action of lipase protein is followed by hydrolysis of triglycerides into free fatty acids. A small amount of AA, like other fatty acids, is released back into the bloodstream where it is bound to albumin. Over 90% of fatty acids circulate in the blood in the form of proteins attached to the plasma. The concentration of unbound free fatty acids (FFAs – unesterified fatty acids) is low but they are the most metabolically active lipid fraction. Recent research shows that plasma FFAs are a likely source of PUFA for the brain (Spector, 2000). According to Washizaki et al. (1994), also unesterified AA and DHA fatty acids introduced into the blood plasma are quickly taken up by the neurons and esterified into membrane phospholipids. It was found that the unesterifed fatty acids function as secondary messengers of signals from the extracellular environment through the cell membrane to the cell nucleus (Łoś-Rychalska and Czerwionka-Szaflarska, 2010). Moreover, they can directly affect the expression of genes which modulate the oxidation process of fatty acids, the synthesis of fatty acids, lipids and lipoproteins (Jump et al., 1994; Łoś-Rychalska and Czerwionka-Szaflarska, 2010) and the synthesis of biologically active substances that play a role in the development of inflammation (Jelińska, 2005; Jabłońska-Trypuć and Czerpak, 2009). Free fatty acids acting outside the cell can stimulate the activity of different receptors and thus may affect the secretion of insulin and hypothalamic neuropeptide Y and can influence initiation mechanisms of innate immunity response (Doege and Stahl, 2006). The majority of free fatty acid is transported into the cell by diffusion or by protein-mediated transport (Fig. 2) (McArthur et al., 1999). While the transport of long chain fatty acids (LCFA) into the cells of muscle tissue, liver, adipose tissue, heart and intestines, where the processes of metabolism and storage of LCFA are intensive, takes place only by protein-mediated pathway (Doege and Stahl, 2006).

In the cytoplasm of the cells free arachidonic fatty acid, like other free fatty acids, is activated to acyl-CoA (Coleman et al., 2002; Cook and McMaster, 2002). Thus activated fatty acids take part in further changes in the cell cycle, by which they are incorporated into triglycerides, cholesterol esters and phospholipids of cell membranes. AA can be stored as an energy reservoir in the form of triglyceryde in adipose tissue or can be used by cells as a source of energy through β-oxidation process in mitochondria (Spector, 2000; Gao et al., 2009). Arachidonic fatty acid released from membrane phospholipids may undergo resynthesis or can be converted to eicosanoids (Doege and Stahl, 2006). Transformations and the amount of arachidonic acid are controlled by membrane proteins and intracellular and extracellular receptors (Fig. 2). Arachidonic acid metabolism in physiological and experimental

conditions was studied by Brash (2001). It was found that the introduction of high concentrations of unbound free AA is rapidly taken up by the cells through passive diffusion and then directly metabolized to eicosanoids. Excessive intake of AA as well as LA fatty acid resulted in increased inflammatory proportions of AA in cell phospholipids (Palmquist, 2009) (Fig. 2). The concentration of AA in resting cells is kept at a low level (5 μM) but the release of 1% of AA in cell membranes may increase the level of AA within the cells to 50 μM (Brash, 2001).

The amount of arachidonic fatty acid depends on the amount of linoleic and α-linolenic fatty acids supplied through the diet. Just like α-linolenic acid, linoleic acid may undergo enzymatic transformations consisting in introduction of further double bonds from the carboxyl group (-COOH) with the participation of specific desaturase (Δ-6, Δ-5) and the hydrocarbon chain elongation by the enzyme elongase, leading to the formation of long derivatives with 20-22 carbon atoms in the chain (Stołyhwo-Szpajer et al., 2001; Sprecher, 2002; Dobryniewski et al., 2007; Walczewska et al., 2011).

Desaturases and elongases are enzymes associated with the microsomal lipid layer and its activation requires the presence of zinc atoms (Nakamura and Nara, 2004). Transformation of fatty acids ALA and LA depends mainly on the amount of desaturase, whose activity decreases in the case of zinc, vitamin B_6 and magnesium deficiency (Nakamura and Nara, 2004). Factors such as a diet high in artificial trans-fatty acids, the processes of aging, diabetes and hypertension as well as the use of anticoagulants reduce the activity of these enzymes (Kolanowski, 2007; Cichosz and Czeczot, 2011). Zinc is an essential cofactor in the metabolism of linoleic acid, at the site of desaturation to γ-linolenic acid. It is also thought to be essential in the metabolism of dihomo-γ-linolenic acid to arachidonic acid, which can affect the synthesis of prostaglandins (Darmstadt et al., 2000). On the other hand, in a study by Bolesławska et al. (2007) it was demonstrated that zinc supplementation in a daily diet has an effect on the metabolism of polyunsaturated essential fatty acids, which are followed by changes in fatty acid profile in blood plasma phospholipids. An increase in α-linolenic acid and also arachidonic acid was observed by Bolesławska et al. (2007). The enzyme Δ-6 desaturase converts linoleic acid (C18:2 *n-6*) to γ-linolenic acid (GLA C18:3 *n-6*). The same enzyme is involved in the metabolism of α-linolenic acid (C18:3 *n-3*) into octadecatetraenoic acid (Sprecher, 2002). Then, by the action of elongases, dihomo-γ-linolenic acid (DGLA C20:3 *n-6*) is converted from γ-linolenic fatty acid (GLA C18:3 *n-6*). As a result of Δ-5 desaturase actions the arachidonic acid (C20:4 *n-6*) is synthesized and the octadecatetraenoic acid is changed into eicosapentaenoic acid (C20:5 *n-3*) (Stołyhwo-Szpajer et al., 2001; Nakamura and Nara, 2004). Due to the fact that the elongation occurs faster than the desaturation, the amount of dihomo-γ-linolenic fatty acid is greater than that of γ-linolenic acid (Yang-Yi and Chapkin, 1998). According to Johnson et al. (1997), supplementation of dietary γ-linolenic acid increases the content of dihomo-γ-linolenic fatty acid in the tissues, resulting in a significant reduction in activity of Δ-5 desaturase, thus blocking the synthesis of arachidonic acid from the dihomo-γ-linolenic acid. The γ-linolenic acid, which is an intermediate product of enzymatic transformation of linoleic acid is abundant in primrose oil and blackcurrant seeds

(Jelińska, 2005). It seems that dietary supplementation of this oil could decrease the conversion of AA.

The enzyme Δ-6 desaturase converts AA to tetraeicosapentaenoic fatty acid (C24:5 *n-6*) and eicosapentaenoic acid is changed into tetradocosahexaenoic fatty acid (C24:6 *n-3*) (Nakamura and Nara, 2004). In the last stage of the biosynthesis of fatty acids the tetraeicosahexaenoic and tetradocosapentaenoic fatty acids are translocated from endoplasmic reticulum to the peroxisome, where 2 carbon atoms are removed through modified beta-oxidation, leading to the formation of docosapentaenoic (C22:5 *n-6*, DPA) and docosahexaenoic fatty acids (Sprecher, 2002; Nowak, 2009). For the incorporation of DHA and DPA to membrane phospolipids and triglycerides the retranslocation of these fatty acids to the endoplasmic reticulum is needed (Sprecher, 2002).

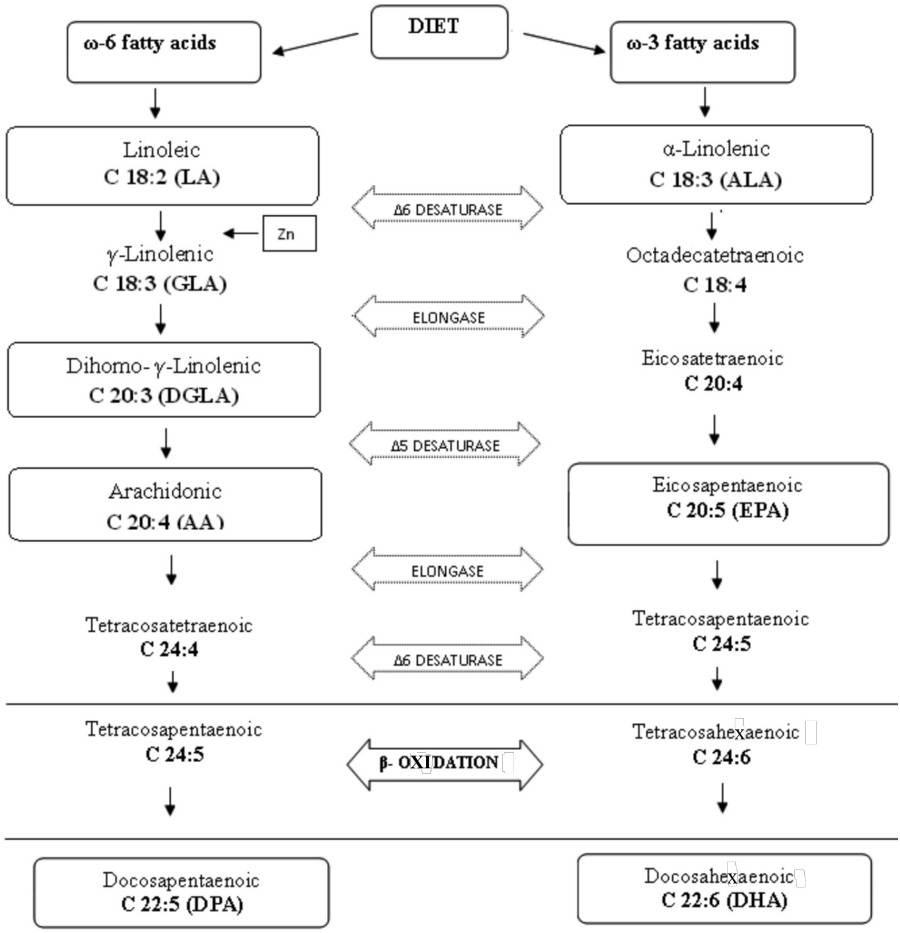

Figure 1. Biosynthesis of long-chain polyunsaturated fatty acids (adapted from Nowak, 2009; Walczewska et al., 2011)

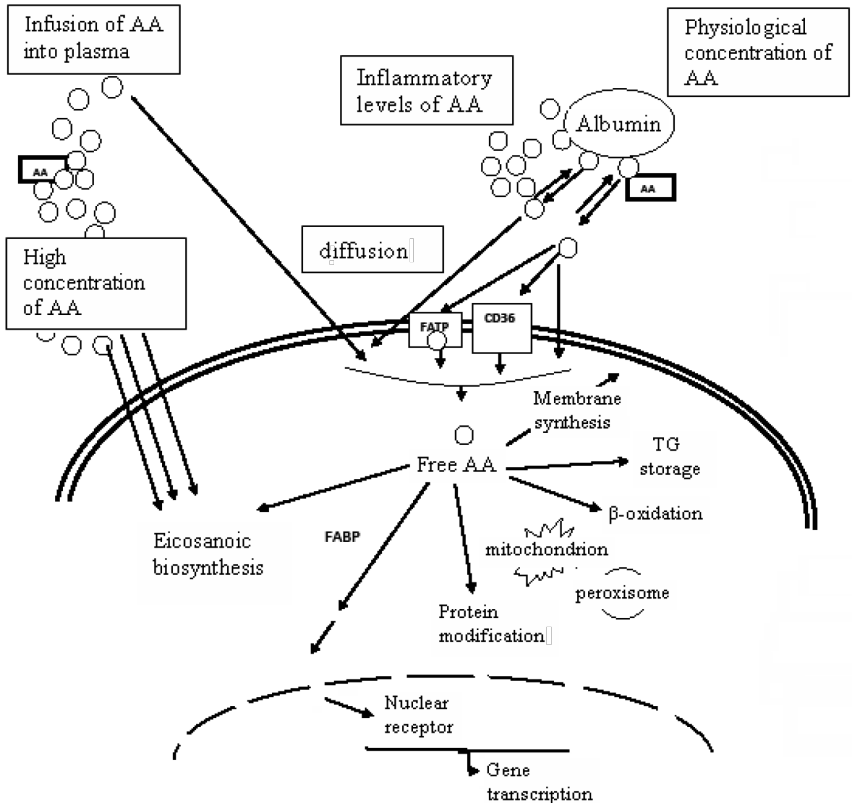

Figure 2. Mechanisms of uptake and action of arachidonic fatty acid (AA) in the cells (adapted from Brash, 2001; Doege and Stahl, 2006)

Different lipid metabolism, involving rumen bacteria, occurs in ruminants. This process has the main influence on the profile of fatty acids available for absorption and the use of the tissue (Jenkins et al., 2008). Unsaturated fatty acids taken with food by ruminants are converted into saturated fatty acids in the process of lypolysis and biohydrogenation.

The initial step in the metabolism of dietary lipids which entered the rumen is lypolysis. During this process the ester linkages of triacylglycerols, phospholipids and galactolipids are hydrolyzed by the rumen bacterial enzymes, releasing free fatty acids (Palmquist et al., 2005; Jenkins et al., 2008) (Fig. 3). Released unsaturated fatty acids consist mainly of linoleic (C18:2 *n-6*) and linolenic (C18:3 *n-3*) fatty acids. These two fatty acids are biohydrogenated by the rumen bacteria, leading to formation of saturated fatty acids.

The first stage in biohydrogenation of linoleic fatty acids is saturation which involves isomeration reactions. During the isomeration reactions LA is converted into isomer cis-9, trans-11 conjugated linoleic acid (CLA) which is reduced to vaccenic

(VA) fatty acid (Chilliard et al., 2007). In the next stage VA is changed into stearic fatty acid (C18:0). The linolenic (LNA) fatty acid is biohydrogenated following the same pattern. It starts with an isomeration reaction, followed by a sequence of reductions and ends with the formation of stearic fatty acid (Chilliard et al., 2007; Jenkins et al., 2008) (Fig. 3). The VA and CLA are the most important intermediaries formed during these transformations (Zymon and Strzetelski, 2007). The VA is a substrate for the production of cis-9, trans-11 C18:2 in the ruminant's own tissues (Scollan et al., 2006). In the tissues Δ-9 desaturate converts the stearic fatty acid into cis-9 C18:1.

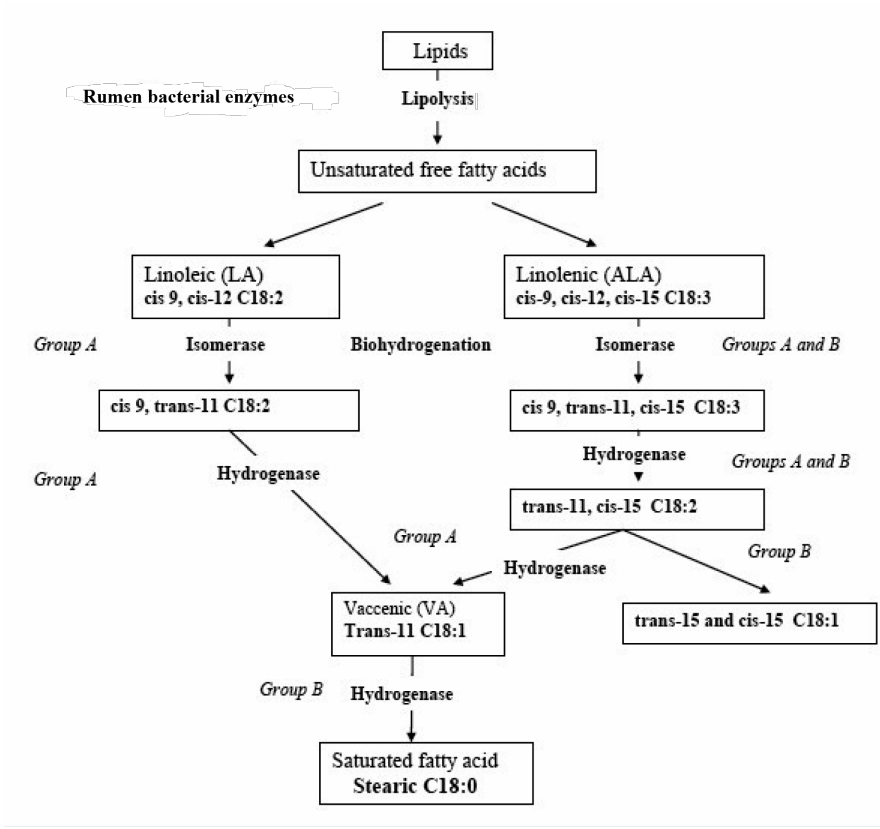

Figure 3. Metabolism of PUFA in the rumen (adapted from Zymon and Strzetelski, 2007; Chilliard et al., 2007)

Involved in biohydrogenation are ruminal bacteria which have been classified into two groups: A and B, based on their metabolic pathways. Group A includes bacteria that can hydrogenate PUFA into trans-11 C18:1 fatty acids. Group B are bacteria which can hydrogenate oleic fatty cis-9 C18:1 and its isomers as well as trans-11 C18:1 fatty acids into stearic fatty acid. In order to complete the biohydrogenation

of PUFA, the presence of both bacterial groups is required (Zymon and Strzetelski, 2007) (Fig. 3). Despite the rumen bacterial activity, increasing content of PUFA protected from ruminal biohydrogenation in animal feed increases the PUFA content in ruminant meat and milk (Scollan et al., 2006).

It is suggested that fish oil, as a rich source of DHA and EPA, inhibits the biohydrogenation of LA and ALA fatty acids, causing an accumulation of isomers trans C18:1 and trans C18:2 (Loor et al., 2005; Wąsowska et al., 2006). It was also found that the addition of LA prevented biohydrogenation of DHA and EPA (Wąsowska et al., 2006). In an earlier study with young bulls fed mixtures containing flax seed, an increase in the content of LNA and EPA fatty acids was recorded (Strzetelski, 2001). It is supposed that ruminants may have the ability to dehydrogenate and elongate LNA into DHA (Maciaszek and Strzetelski, 2005). However, the biohydrogenation of DHA and EPA is not completely understood (Jenkins et al., 2008).

After leaving the rumen, the free fatty acids from the particles of food and bacteria are desorbed by lysolecithin and bile salts. The micelles are formed and taken up by the epithelial cells of the jejunum. The free fatty acids are released from the small amounts of triglycerides and glycolipids reaching the intestines (Zymon and Strzetelski, 2007). Metabolism of fatty acids is started.

Arachidonic acid in the inflammatory process

Arachidonic acid as the main precursor of eicosanoids opens new possibilities for research on diseases characterized by a severe inflammatory process. Inflammation is an important physiological process aimed at the fastest possible removal of the pathogen and repair of the damage. It is connected with the changes in the blood vessels. Their enlargement, increased permeability and blood flow enable the appropriate proteins to migrate to the sites where the proinflammatory factor is present, and to begin repair processes. Eicosanoids, which include prostaglandin (PG), prostacyclin (PGI), thromboxanes (TX) and leukotrienes (LT) play a key role in the inflammatory process by influencing its course and duration (Calder, 2006). They are produced by neutrophils, macrophages, platelets, mast cells and endothelial cells and exhibit local activity (Paradowski et al., 2005). Controlled and mild inflammation is a very positive and necessary process of restoring the body's homeostasis. However, an escalation of immune response, changed to the chronic form may become a factor leading to pathological damage to tissues or organs (Calder, 2006). This happens in diseases such as rheumatoid arthritis, inflammatory bowel disease (e.g. Crohn's disease), type II diabetes, atherosclerosis, Alzheimer's disease, retinopathies, psoriasis, multiple sclerosis, chronic obstructive pulmonary disease, allergy and asthma (Calder, 2006; 2009). Pain, increased temperature, swelling, and impairment of motor function which accompany uncontrolled inflammatory processes may significantly worsen the patient's condition.

Eicosanoids, which are inflammatory mediators, can be synthesized from dihomo-γ-linolenic acid and eicosapentaenoic acid, but those arising from free arachidonic acid have the highest biological activity. Arachidonic acid, associated with phospholipid membranes in reaction to stimuli such as epinephrine, histamine, bradykinin, angiotensin II or thrombin by the action of phospholipase A_2 is released and under-

goes further biochemical changes. With the participation of the enzyme cyclooxygenase (COX), cyclic eicosanoids: prostaglandins, prostacyclins and thromboxanes are formed and the lipoxygenase (LOX) catalyzes the synthesis of leukotrienes. Prostaglandin synthase (COX, PGHS), being active in the endoplasmic reticulum leads to the formation of arachidonic acid to PGG_2, which is next reduced to PGH_2. PGH_2 is a substrate for further synthesis of prostaglandins, prostacyclins and thromboxanes. Leukotrienes are also formed through multistep synthesis. As a result of an oxygen molecule insertion to the free arachidonic acid by 5-lipoxygenase, 5-hydroperoxyeicosatetraenoic acid (5-HPETE) is produced. After that, 5-HPETE can be reduced to two compounds: 5-hydroxyeicosatetraenoic acid (5-HETE, with the participation of glutathione peroxidase) or leukotriene A_4 (LTA_4, by further action of 5-lipoxygenase). LTA_4 is the precursor for the synthesis of other leukotrienes.

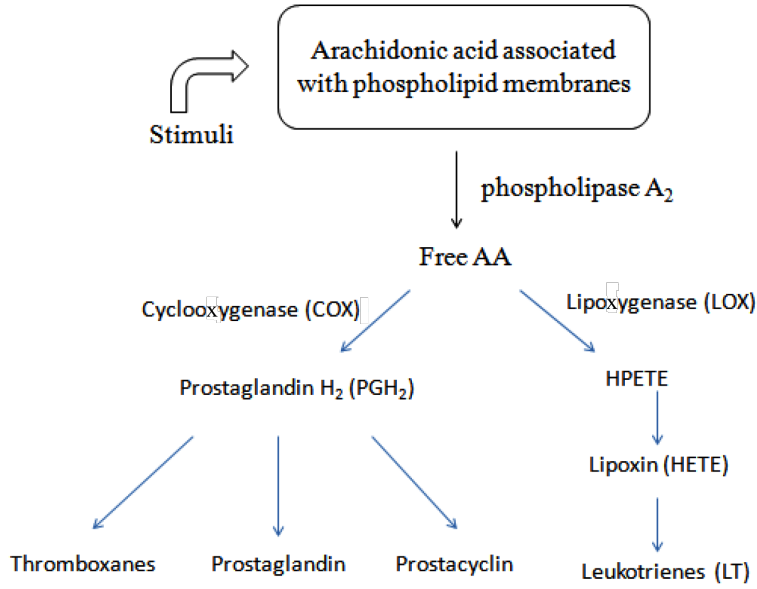

Figure 4. Scheme of eicosanoid formation

Eicosanoids exhibit a broad spectrum of activity in the course of the inflammatory response, often presenting opposite functions. They are not stored in cells, but synthesized from AA during the immune response. Due to their short half-life they are classified in the group of hormones acting locally. The strongest pro-inflammatory effect is observed in the case of prostaglandin G_2 and H_2 inducing platelet aggregation, and in the case of prostaglandin E_2 and F_2 (Dobryniewski et al., 2007). PGE_2 induces COX-2 in fibroblast cells, thereby stimulating its own production and also stimulates the production of interleukin-6 (strongly reinforcing the inflammatory process) by macrophages (Bagga et al., 2003), which cause fever, pain and conges-

tion in places where tissue impairing factor is present. One source of the excessively increased temperature in the inflammatory reaction is the action of prostaglandin E_2 in the hypothalamic thermoregulatory centre. Similarly, as a result of increased permeability of blood vessels under the influence of activity of bradykinin, histamine and PGE_2, increased volume of extracellular fluid causes swelling (Long et al., 1990). However, by promoting the formation of lipoxins as a result of increased activity of the enzyme 15-LOX, inhibiting the synthesis of TNF (Tumor Necrosis Factor) and IL-1, prostaglandin PGE_2 reveals its anti-inflammatory nature (Levy et al., 2001). Prostacyclin PGI_2 relaxes bronchioles and capillaries, reduces the activity of platelets and seals endothelium (Paradowski et al., 2005) which is totally opposed to the action of eicosanoids such as $T{\times}A_2$ (tromboxan A_2) and PGF_2. It is worth noting that the prostacyclin has a beneficial effect on cardiovascular function by preventing platelet aggregation, vessel dilator, and lowering blood pressure.

In the case of leukotrienes, especially LTB_4, there is observed a strong ability to induce an inflammatory response by increasing vascular permeability, local blood flow, chemotactic activity of leukocytes, bronchoconstriction, increasing the synthesis of TNF and IL-6 and the release of lysosomal enzymes (Calder, 2009). Other eicosanoids from this family: LTC_4, LTD_4, LTE_4 have similar biological activity. Their ability to contract airway smooth muscle in the lung and stimulate bronchoconstriction may induce asthma attacks, and the release of leukotrienes in large quantities may cause anaphylactic shock. These hormones are responsible for overproduction of mucus and inflow of eosinophilia during the development of an allergic reaction. Eosinophilic granulocytes contain a toxic protein which can be released at the infiltration and may lead to respiratory epithelial damage and intensification of inflammation (Bousquet et al., 1992).

In the treatment of inflammatory diseases, the most commonly used are nonsteroidal and steroidal substances that block the synthesis of eicosanoids and their receptors. The first group of anti-inflammatory drugs called NSAIDs includes e.g. aspirin or ibuprofen. The underlying mechanism of action of aspirin is based on the acetylation that prevents the synthesis of prostanoids formed with the participation of cyclooxygenase. Acetylsalicylic acid, which is the active ingredient of this drug, competes with AA for COX active site and inhibits its activity as a result of the hydrogen atom attachment to serine 530. Aspirin more strongly blocks the enzyme COX-1 than COX-2, which may contribute to the development of peptic ulcer disease. Due to the deficit of COX-1, the products of which have a protective effect on the mucosa of the stomach, lesions may start to occur (Czyż and Watała, 2005). This necessitates a search for safer anti-inflammatory drugs which inhibit COX-2. A similar mechanism of operation is also characteristic of other drugs. Their action is reversible and disappears at the time of dissolution. What raises more concern is the use of anti-inflammatory steroids. Their effectiveness in the treatment of many autoimmune diseases such as asthma attacks or their use as immunosuppressive agents after transplantation is extremely common. However, their chronic administration carries a high risk of many side effects. Adrenal insufficiency, increased susceptibility to bacterial, fungal and viral infections, diabetes, obesity, myopathy, glaucoma and osteoporosis may significantly worsen the patient's quality of life. In that case,

a well-balanced diet can become an important part of the treatment process. Adjusting consumption of fats which are involved in the inflammatory response is critical for people struggling with the disorder characterized by uncontrolled inflammatory response. As already mentioned arachidonic acid can be directly consumed together with foods of animal origin (Haug et al., 2010) or synthesized by the transformation of exogenous linoleic acid. In particular, much attention is paid to the fact that far too much *n-6* fatty acids are consumed in relation to *n-3*. While this ratio should be close to 1, in countries with the most scarce intake of *n-3* fatty acids, it is even 20–30:1 (Simopoulos, 2002). Such a strong imbalance increases the risk of cardiovascular diseases, cancer, inflammatory changes, and autoimmune disorders. Improved health status in asthma, rheumatoid arthritis and colorectal cancer have been observed at the *n-6/n-3* fatty acids ratio of 2.5/1 and 4/1, respectively (Simopoulos, 2002). Limited intake of *n-6* fatty acids or their precursors can minimize the effects of pathological changes associated with the development of the inflammatory response by inhibiting the synthesis of eicosanoids from arachidonic acid. Linoleic acid is the precursor of the family of *n-6* fatty acids and α-linolenic acid is a precursor to *n-3*. As a result of chemical changes in the endoplasmic reticulum, arachidonic acid is formed from LA. Enzymes that are involved in this process also catalyze the conversion of α-linolenic acid to EPA, which can then be converted to DHA. As a result of competition for the same biocatalysts, excessive intake of LA inhibits the production of EPA and DHA, and also leads to the overproduction of arachidonic acid. Conversely, increased consumption of ALA strongly reduces the formation of *n-6* fatty acids in favour of the desired *n-3* (Jelińska, 2005). Also, the high content of EPA inhibits phospholipase 2, which is responsible for the release of AA from phospholipids of cell membranes. EPA and DGLA also competes with AA for access to the cyclooxygenase and 5-lipoxygenase metabolites (Guivernau et al., 1994), enzymes that convert LA and ALA to eicosanoids (Jelińska, 2005).

Products of animal origin as a source of arachidonic acid and its precursors

The fatty acid composition in a typical human diet over the past 100 years has changed significantly, with a significant increase in the intake of *n-6* unsaturated fatty acids due to frequent presence of vegetable oils in the diet and the recommendation of a high intake of these fatty acids because of their cholesterol-lowering properties in serum (Simopoulos, 2002). Linoleic acid contained in the human diet, as a precursor is considered a major source of arachidonic acid. Meat and animal products contain less linoleic acid in comparison to some vegetable oils such as sunflower, soybean, corn or grape seed oils, where the level of this acid is 55–65%. Table 1 shows the composition of meat of different animal species with the characteristics of the total fat content and fatty acid levels of LA and AA.

The fat content of lean meat ranges from 1 g/100 g in turkey meat to 4.2 g/100 g in lamb meat. Whereas the level of arachidonic acid in beef and lamb meat is 30–40 mg/100 g and is 1.5–2.5 times lower compared to its content in chicken, duck, turkey and pig meat (43, 99, 74 and 54 mg/100 g, respectively) (Li et al., 1998). Similar relationships apply to the AA precursor linoleic acid, the levels of which are 3 times lower in beef and lamb meat than in chicken meat and pork.

Table 1. Content of fat, arachidonic acid and linoleic acid in the lean meat of different species of livestock (Li et al., 1998)

Fatty acids	Type of meat					
	beef	lamb	pork	chicken	duck	turkey
Total fat of meat (g/100 g)	1.4±0.2	4.2±1.0	2.0±0.3	2.3±0.5	1.7±0.3	1.0±0.1
Arachidonic acid (% of total acids)	3.0±0.6	1.2±0.3	3.6±0.3	2.5±0.3	9.3±1.8	9.2±0.9
Linoleic acid (% of total acids)	5.6±1.1	4.9±1.3	14.4±3.4	14.4±1.7	18.2±3.4	18.6±4.4

Taber et al. (1998) determined the fatty acid content and also the level of arachidonic and linoleic fatty acids in raw and cooked meat (beef, chicken, turkey, pork) and also in eggs. They showed the content of arachidonic and linoleic acids to be higher by up to 50–67% in cooked meat and boiled eggs in comparison with the raw products. The average content of linoleic and arachidonic acids in eggs is 1148 mg/100 g and 142 mg/100 g, respectively (Taber et al., 1998) but in milk the range is 2.0–7.0% and 0.03–0.09% (Chilliard et al., 2008).

Possibilities to reduce the content of arachidonic acid through nutrition in animal products

It is necessary to remember that one of the main factors determining the fatty acid composition of tissues, in addition to the diet, is animal species and breed. Meat from animals of primitive, often dual-purpose breeds has a higher fat content in muscle tissue. Other important factors affecting the quantity and composition of fatty acids is sex, age of the animal, feeding intensity and type of fodder used for fattening.

Using the example of the pig, we can conclude that the nutrition level affects not only the growth rate of pigs (Mason et al., 2005), but also the amount of fat deposited during growth period (Kristensen et al., 2002) and the fatty acid profile and palatability of meat (Cameron et al., 2000). Carcasses from animals fed semi- and *ad libitum* vs. restricted diets were characterized by more general fatness and a greater proportion of intramuscular fat in the *musculus longissimus dorsi* (MLD). The review of Skiba (2005) showed that dietary restrictions involving the lower levels of protein in the feed lead to greater carcass fatness and limitation of the amount of feed results in less fat deposition. Nutrients taken in feed are used by animals in the first place to cover everyday needs, to deposit protein and then fat. Therefore, if pigs are fed sparingly deposition of fat is lower, both in terms of subcutaneous and intramuscular fat (IMF) (Kristensen et al., 2002; Mason et al., 2005).

The principal method to modify the lipid profile of animal tissues is through dietary intake of fat. Several studies support the possibility of introducing dietary fatty acids to body tissues, which affect the quantitative and qualitative characteristics of meat. One of the ways of enriching the meat in polyunsaturated fatty acids (PUFAs) is through dietary oils and oilseeds (Flachowsky et al., 1997; Barowicz and Pieszka, 2001; Kouba et al., 2003; López-Bote et al., 2002; Daza et al., 2005; Smink et al., 2010; Skiba et al., 2011). Similar modifications in fatty acid composition can be accomplished in milk and eggs (Rego et al., 2005; Oliveira et al., 2010).

The addition of vegetable oils increases the energy level in the ration and it is the main source of unsaturated fatty acids in the diet. The sunflower, corn and soybean oils contain substantial amounts of linoleic acid (C18:2) in the range 55–65%, which in turn adversely affects the fatty acid profile of muscle tissue, and the *n-6* to *n-3* fatty acid ratio is narrowed. Most preferable in terms of linoleic acid content are olive, flaxseed and canola oils, where the level of this acid is 10.5, 16.2 and 21.1%, respectively. The rations for poultry, pigs and cattle raised in Poland are based on cereal grains, mainly barley, wheat and triticale, which are characterized by a high content of linoleic acid (C18:2) belonging to the family of *n-6* fatty acids. A consequence of this is the increased content of *n-6* unsaturated fatty acids (PUFA) in meat lipids, mainly linoleic acid (C18:2) together with a decreased level of *n-3* fatty acids, which is unfavourable from the perspective of consumer health. Most beneficial from the standpoint of human nutrition is the fatty acid profile of ruminant meat, especially meat from lambs and young cattle grazed with a limited amount of compound feed (Noci et al., 2005; Nuernberg et al., 2006). In older beef cattle, according to high biohydrogenation of unsaturated fatty acids, the use of fish oil is the most effective way of decreasing AA in meat together with increasing the level of *n-3* acids (Scollan et al., 2001). There were also trials concerning the protection of fatty acids from fish oil by microencapsulation and in dairy cattle feeding this caused a significant increase in the level of *n-3* acids in milk (Lacasse et al., 2002).

Table 2. Effect of fodder or feed additives on reducing the level of linoleic and arachidonic acids in animal products

Fodder or feed additives	Animal species/effect	Author/source
Linseed	dairy cattle/↓C18:2; ↓C20:4	Chilliard et al., 2008
	broiler chicken /↓C18:2; ↓C20:4	Azcona et al., 2008
Rape seed	broiler chicken /↓C18:2; ↓C20:4	Azcona et al., 2008
Rape oil	broiler chicken /↓C18:2; ↓C20:4	Nobar et al., 2007
Fish oil	pigs/↓C18:2	Skiba et al., 2011
	laying hens /↓C18:2; ↓C20:4	Carvalho et al., 2009
Linseed oil	laying hens/↓C18:2; ↓C20:4	Oliveria et al., 2010
	broiler chicken/↓C18:2; ↓C20:4	Nobar et al., 2007
	broiler chicken/↓C18:2; ↓C20:4	Shin et al., 2012
CLA	pigs /↓C18:2	Pieszka et al., 2006
	broiler chicken /↓C18:2	Shin et al., 2011
Pasture (grass)	beef cattle /↓C18:2	Noci et al., 2005
	lambs /↓C18:2	Nuernberg et al., 2006
Lard	pigs/↓C18:2	Skiba et al., 2011

Another way of changing the fatty acid composition of lipids, among others lowering the level of arachidonic and linoleic acids in the diet, is to add isomers of conjugated linoleic acid (CLA), which stimulates the activity of 9 SCD and Δ5 and 6 desaturase (Eder et al., 2002; Smith et al., 2002; Shin et al., 2012). The use of CLA in compound feeds for pigs and broiler chickens increased the levels of *n-3* fatty acids and decreased the levels of *n-6* acids (Pieszka et al., 2006; Shin et al., 2011).

Czauderna et al. (2003) demonstrated the ability of the diet containing selenium and CLA isomers to increase the level of CLA in rat muscles. Scientists also attempted to change the fatty acid profile using minerals such as copper and chromium; the addition of copper to the feed of growing pigs increased the content of saturated acids together with a decrease in the level of unsaturated fatty acids in blood (Dove and Haydon, 1992), but an oversupply of these elements creates toxicological concerns and that is why there is no practical application in nutrition.

In a recent study, Shin et al. (2012) showed that a mixture consisting of 2.45% flaxseed oil, 0.05% DHA and 2.5% olive oil significantly reduces the levels of AA and LA in breast and thigh muscle of broiler chickens. The same mixture containing EPA instead of DHA significantly decreased mRNA expression of Δ6-desaturase. It was therefore concluded that the addition of flaxseed oil, olive oil, EPA and DHA fatty acids would have the most beneficial impact on the profile of fatty acids in the meat of broiler chickens.

Summary

The modern human diet is characterized by intake of very high levels of *n-6* fatty acids, which causes an increase in the ratio of *n-6/n-3* acids, unfavourable for health. Recent studies have shown that excess of *n-6* fatty acids inhibits the metabolism of *n-3* fatty acids, which contributes to impaired physiological balance of biologically active compounds synthesized from them. As already mentioned, excessive intake of arachidonic acid and linoleic acid causes abnormal cell membrane permeability, blood coagulation and the overactive immune system, increases inflammation and may contribute to the development of neurodegenerative diseases. Maintaining the right proportion of both groups of polyunsaturated fatty acids is essential for maintaining homeostasis and may be extremely important in the treatment of diseases which are characterized by excessive intensification of the immune response, while administered drugs are not neutral and often lead to pathological changes. Overproduction of inflammatory factors may be limited by eating meat or eggs containing a reduced amount of their precursors, arachidonic and linoleic acids. A properly balanced diet can help to ensure the best possible profile of fatty acids in animal products, enabling products to maintain an appropriate level of synthesized lipids, and thus can improve or maintain our health.

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DOROTA BEDERSKA-ŁOJEWSKA, SYLWIA ORCZEWSKA-DUDEK, MAREK PIESZKA

Metabolizm kwasu arachidonowego, jego stężenie w produktach zwierzęcych i wpływ na procesy zapalne w organizmie człowieka – artykuł przeglądowy

STRESZCZENIE

W pracy poruszono problem metabolizmu kwasu arachidonowego należącego do grupy *n-6*. Na jego bazie powstaje wiele związków wykazujących szerokie działanie w organizmie. Ich prawidłowa homeostaza, a co za tym idzie, zdrowie człowieka zależne są od stosunku spożywanych w diecie kwasów *n-6* do *n-3*. Związki te przyjmowane wraz z produktami pochodzenia zwierzęcego i roślinnego powinny być lepiej zbilansowane w ludzkiej diecie. Jest to możliwe poprzez obniżenie zawartości kwasu arachidonowego oraz jego prekursorów na drodze żywieniowej w produktach pochodzenia zwierzęcego takich jak jaja, mleko czy mięso.