β-Carotene: Biological Functions and Mechanisms (Postprint)

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Date: 2018-12-24T00:00:00+00:00

Abstract

β-carotene is the most abundant carotenoid in mammalian tissues and serves as an important precursor of vitamin A. β-carotene and its metabolites act as key regulatory signaling factors in tissue metabolism, thereby exerting numerous beneficial effects in mammals (including humans). This article provides an overview of the effects of β-carotene on animal production performance, immune function, and reproductive performance, as well as the underlying mechanisms.

Full Text

Biological Functions and Mechanisms of β-Carotene

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Abstract: β-Carotene is the most abundant carotenoid in mammalian tissues and a crucial precursor of vitamin A. β-carotene and its metabolites serve as key regulatory signaling factors in tissue metabolism, exerting numerous beneficial effects in mammals, including humans. Although β-carotene is considered a safe form of vitamin A, its tightly controlled intestinal absorption mechanism means that adverse effects can arise from inappropriate intake under certain conditions. This review summarizes the metabolism of β-carotene and clearly distinguishes its potential beneficial versus harmful impacts on animal health, thereby providing a theoretical basis for determining appropriate β-carotene intake levels across different animal species.

Keywords: β-carotene; performance; immune function; reproductive performance

1 Structure and Properties of β-Carotene

Carotenoids in nature are highly diverse and can be classified into carotenes and xanthophylls based on their chemical structure [1]. Carotenes, such as β-carotene, α-carotene, and β-cryptoxanthin, are non-oxygenated carotenoids with linear or cyclic hydrocarbons at one or both ends of the molecule, while xanthophylls (e.g., zeaxanthin, meso-zeaxanthin, astaxanthin, and canthaxanthin) are oxygenated derivatives of carotenes [2]. β-Carotene was first discovered in carrots along with three isomers (α, β, γ), among which β-carotene exhibits the highest activity and is most widely distributed in nature [3].

The link between β-carotene and vitamin A was established by Von Euler et al. [4], who further demonstrated that crystalline carotene possesses vitamin A activity. Moore [5] subsequently showed that β-carotene can be converted to vitamin A in rats. Based on their ability to serve as vitamin A precursors, carotenoids are classified as either provitamin A or non-provitamin A. Provitamin A carotenoids and their metabolites (retinoids) generated through enzymatic and non-enzymatic cleavage can produce vitamin A, with β-carotene being the most abundant provitamin A in nature [6]. β-Carotene is a short-chain molecule containing 40 carbon atoms, 15 conjugated double bonds, and two β-ionone rings at its structural termini. These characteristics render β-carotene highly hydrophobic and nonpolar in nature. In animals, polar carotenoids appear to be more readily absorbed than nonpolar ones [7], though carotenoids as a whole exhibit strong hydrophobic properties.

2 Absorption, Transport, Metabolism, and Deposition of β-Carotene in Animals

2.1 Intestinal Absorption of β-Carotene Most kinetic parameters for β-carotene digestion have been derived from non-ruminant studies. Since β-carotene is transported together with lipids in the body, its transfer and absorption in the small intestine are strongly influenced by dietary fat type and content. The nonpolar nature of β-carotene determines its location within the micellar core during transport, with transfer efficiency from emulsion to chylomicrons ranging from 12% to 18% [8]. When β-carotene intake is high or fat intake is low, the transfer from emulsion to chylomicrons becomes rate-limiting in the small intestine. The small intestine is primarily responsible for absorbing lipids, fat-soluble vitamins, and β-carotene, and subsequently delivering them to peripheral tissues.

Research indicates that even though the human intestine highly expresses β-carotene-15,15' -oxygenase (CMO , BCMO1, BCO1), it does not completely convert ingested β-carotene to vitamin A in the gut. Instead, 17%-45% of consumed β-carotene is released into peripheral circulation in its intact, uncleaved form [9]. Studies have shown that variable CMO enzyme activity associated with multiple polymorphisms in the CMO gene may be responsible for lower β-carotene cleavage efficiency in certain individuals [10]. Rodents such as mice demonstrate highly efficient intestinal uptake of β-carotene, with the provitamin A carotenoid detectable in circulation only upon supraphysiological intake [11]. Other animal models, including Mongolian gerbils, silkworms, and ruminant calves, can also absorb intact β-carotene, with provitamin A distribution in serum and tissues similar to that in humans. Although the mechanisms of β-carotene absorption and transport in ruminants remain unclear, these animals serve as excellent models for studying carotenoid transport. The process is more complex in ruminants than non-ruminants due to rumen microbial modification and resynthesis of fats before reaching the duodenum. Notably, xanthophylls appear in calf serum earlier than β-carotene before rumen development [12].

2.2 Transport of β-Carotene in Animal Serum Due to its high lipophilicity and nonpolarity, β-carotene transport is closely associated with various lipoproteins in circulation. It can enter the hydrophobic cores of lipoprotein particles such as chylomicrons and their remnants [very low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), and low-density lipoprotein (LDL)], as well as other lipids like cholesterol esters and retinyl esters [13]. These lipoproteins facilitate β-carotene transfer from the intestinal barrier to various body tissues and its inter-tissue transport. Different animal species utilize different lipoprotein classes for β-carotene transport; for instance, high-density lipoprotein (HDL) is the primary carrier of β-carotene in bovine blood circulation [14].

Overall, although the order of lipoprotein transport for different provitamin A carotenoids varies significantly among species, β-carotene can bind to and be transported by various circulating lipoproteins. Studies show that β-carotene can enter all lipoprotein types to varying degrees, with HDL accounting for approximately 82%, LDL for 12%, and VLDL for 0.3% [15]. In rats, serum β-carotene primarily binds to larger VLDL and LDL particles [16]. Research by Pei Lingpeng [17] found that most carotenoids such as β-cryptoxanthin are mainly distributed in LDL and HDL, while approximately 53% of lutein and zeaxanthin bind to HDL, 31% exist in LDL, and about 16% associate with VLDL. The fate and ultimate metabolism of β-carotene largely depend on its affinity for different lipoproteins. Gugger et al. [18] investigated intracellular transport mechanisms of β-carotene between organelles and found that its intracellular transport is not regulated by cytoplasmic transport proteins but may be controlled by vesicular transport or membrane-bound proteins.

2.3 Metabolism of β-Carotene in Animals Retinaldehyde formed from provitamin A cleavage can be oxidized by retinaldehyde dehydrogenase to produce all-trans retinoic acid, the biologically active form of vitamin A. Retinoic acid functions not only as a transcriptional regulator but also as a ligand for specific nuclear receptors—retinoic acid receptors (RAR) or retinoid X receptors (RXR)—forming homodimers or heterodimers to regulate the transcription of hundreds of target genes [19]. When retinoic acid production exceeds certain limits in tissues, transcriptionally inactivating enzymes belonging to the cytochrome P450 family can oxidatively degrade it to produce more polar compounds such as 4-hydroxy or 4-oxo retinoic acid [20] (Figure 1 [Figure 1: see original paper]).

CMO : β-carotene-15,15 -oxygenase; **CMO** : β-carotene-9 ,10 -oxygenase; **ADH**: alcohol dehydrogenase; **RDH**: retinol dehydrogenases; **REH**: retinol esterification; **LRAT**: retinol acyltransferase; **ALDH1**: aldehyde dehydrogenase 1; **RALDH**: retinoic aldehyde dehydrogenase; **Cyp26**: cytochrome P450 26 family enzyme.

Figure 1 Metabolism of β-carotene [2]

As shown in Figure 1, CMO symmetrically oxidatively cleaves the 15,15 double bond of β-carotene to produce two molecules of retinaldehyde. Retinaldehyde can be oxidized to retinoic acid by aldehyde dehydrogenase (ALDH1) or retinaldehyde dehydrogenase (RALDH), and further oxidized by cytochrome P450 26 family enzymes (Cyp26) to more polar compounds including 4-oxo retinoic acid, which is considered transcriptionally inactive. Alternatively, various forms of alcohol dehydrogenase (ADH) from the medium-chain dehydrogenase/reductase (MDR) family and retinol dehydrogenases (RDH) from the short-chain dehydrogenase/reductase (SDR) family can reduce retinaldehyde to retinol, which can then be esterified to retinyl esters by lecithin retinol acyltransferase (LRAT).

Additionally, apocarotenals can be produced from β-carotene. Cleavage at the 9 ,10 double bond is catalyzed by β-carotene-9 ,10 -oxygenase 2 (CMO or BCMO2 or BCO2), producing β-apo-10 -carotenal and β-ionone. Asymmetric cleavage at other double bonds can occur non-enzymatically or be enzyme-catalyzed. Figure 1 illustrates some potential apocarotenoids produced through asymmetric cleavage of β-carotene. Dashed arrows indicate that apocarotenoids can ultimately be converted to one molecule of retinaldehyde, though this conversion mechanism remains incompletely elucidated.

In both humans and mice, β-carotene cleavage enzymes such as CMO and CMO are expressed in various adult tissues including liver and adipose tissue, as well as in developing tissues such as placenta, yolk sac, and embryo [21]. These enzymes can convert β-carotene to vitamin A in situ, suggesting that β-carotene can serve as a local source of retinoids in various body locations. CMO is a cytosolic enzyme with strong substrate specificity that interacts only with carotenoids possessing at least one unsubstituted β-ionone ring and represents the primary enzyme for β-carotene cleavage to vitamin A in adult tissues [22]. In vitro studies have found that retinaldehyde and retinoic acid formed through CMO -mediated β-carotene cleavage may influence lipid metabolism in adipocytes by regulating peroxisome proliferator-activated receptor (PPARγ) and retinoic acid receptor (RAR) signaling pathways [23]. However, whether

CMO similarly affects lipid metabolism in various tissues and whether this effect is independent of its β-carotene cleavage capacity remains unclear. β-Carotene can also be asymmetrically cleaved by CMO to produce β-ionone rings and apocarotenoids that ultimately convert to one molecule of retinaldehyde [2], though the conversion mechanism is not fully understood.

2.4 Deposition of β-Carotene in Animals In animals, β-carotene is primarily stored in the liver, with smaller amounts deposited in adipose tissue, adrenal glands, and skin [16]. The distribution and storage locations of β-carotene vary considerably among animal tissues. Shapiro et al. [24] supplemented rats with β-carotene and detected its presence in the liver but not in adipose tissue, suggesting that β-carotene does not simply accumulate in fat but may bind to a specific β-carotene-binding protein whose lipophobic properties reduce β-carotene deposition in adipose tissue. Research also found that in Sanhuang chickens, β-carotene mainly deposits in the small intestine and liver [25], while in breeding hens, carotenoid content in abdominal fat exceeded that in the liver, possibly due to differential carotenoid distribution across growth stages. During development, carotenoids primarily distribute to the liver, adipose tissue, blood, skin, and feathers, gradually shifting to reproductive organs after sexual maturity [26]. Different carotenoids also show markedly different distributions among animal species: β-carotene content is relatively high in sheep and goat livers, while xanthophylls are more abundant in adipose tissue and serum; in cattle, β-carotene dominates serum and adipose tissue, with high xanthophyll content in adipose tissue but low β-carotene levels in the liver [27].

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3 Biological Functions of β-Carotene

3.1 Effects of β-Carotene on Animal Performance Research demonstrates that dietary β-carotene supplementation in dairy cows can improve both milk quality and yield. Heat-stressed cows supplemented with 400 mg β-carotene daily increased milk production by 11%, while those receiving 300 mg daily showed a 6.4% increase, indicating that β-carotene enhances both milk yield and quality [28]. He Wenjuan [29] found that adding β-carotene to the diet of Chinese Holstein cows did not significantly affect milk yield, milk composition, or somatic cell count during early lactation when vitamin A was adequate, but milk production increased to varying degrees three months postpartum. Xia Yun et al. [30] supplemented Australian Holstein cows with 90 mg/d β-carotene and observed that milk yield increased by 11.03% on day 20 and 13.83% on day 40 compared to the control group, with significantly improved milk fat content. Sun Shengxiang [31] reported that supplementing dairy cows with 900 mg β-carotene per day maximized milk yield increase and maintained higher lactation levels for a certain period, with significant improvements in milk fat, protein, and dry matter content. Wu Hongjiu et al. [32] found that adding varying concentrations of β-carotene to Chinese Holstein cow diets significantly increased milk yield, milk fat percentage, and milk protein percentage compared to the control group. Oliveira et al. [33] supplemented cows with 1.2 g β-carotene daily and observed that milk protein content increased from 2.90% to 2.96%, while the ratio of fat to protein greater than 1.5 decreased from 22.6% to 6.5%. Ma Jifeng et al. [34] reported that adding β-carotene to dairy cow basal diets increased milk yield by 3.53%, 9.06%, and 13.39% compared to the control group.

The coloration effect of β-carotene influences beef quality in cattle. β-Carotene deposited in adipose tissue can cause yellowing of body fat and reduce beef quality grades, so β-carotene intake should be reduced during the late fattening period of beef cattle [35]. Research also indicates that feeding low doses of vitamin A and carotene promotes beef marbling formation [12].

3.2 Effects of β-Carotene on Animal Immune Function β-Carotene enhances humoral immunity, cellular immunity, and nonspecific immune function in animals, thereby improving disease resistance. Dietary β-carotene supplementation increases serum lysozyme activity. Chew et al. [36] found that β-carotene stimulates lymphocyte proliferation and enhances cell-mediated humoral immune responses, exerting positive effects on immune reactions. He Wenjuan [29] observed that adding β-carotene to Chinese Holstein cow diets reduced the incidence of retained placenta, metritis, and mastitis during the first three months postpartum. Cucco et al. [37] reported that β-carotene supplementation in poultry promoted growth and improved immunity. Ma Sihui et al. [38] found that dietary β-carotene in mice alleviated cyclophosphamide-induced immunosuppression and enhanced immune function by increasing cytokine and immunoglobulin levels in immunosuppressed mice. Ma Jifeng et al. [34] demonstrated that β-carotene supplementation reduced milk somatic cell counts by 18.54%, 35.27%, and 46.10% compared to the control group. Nishijima et al. [39] fed dried carrots to Japanese Black cattle and found increased IgA and IgG concentrations in colostrum of β-carotene-deficient cows. When serum retinol concentration increased to 100 ng/mL during the last week before calving, the incidence of mastitis during early lactation decreased by 60% [40]; however, serum β-carotene concentration was not associated with retained placenta or mastitis. Numerous studies have confirmed that β-carotene supplementation positively influences animal immune function.

3.3 Effects of β-Carotene on Animal Reproductive Performance Dietary β-carotene may be associated with reproductive performance, particularly as ruminant ovaries, especially the corpus luteum, contain high β-carotene concentrations. β-Carotene deficiency may cause delayed ovulation, luteal insufficiency, and increased incidence of ovarian cysts in dairy cows [41]. Numerous studies have investigated adjusting dairy cow nutrition to improve fertility. The benefits of β-carotene supplementation for reproductive performance may relate to its conversion to vitamin A, particularly in the uterus and ovaries [42]. Serum β-carotene concentration is associated with progesterone secretion by luteal cells;

cows that ovulated during the first postpartum wave had higher mean serum β-carotene concentrations three weeks prepartum than non-ovulating cows. Supplementing β-carotene (500 or 2,000 mg/d) during the prepartum period significantly increased the number of cows ovulating during the first postpartum wave [43]. Supplementing heat-stressed cows with 400 mg β-carotene for over 90 days during the first 120 days postpartum improved pregnancy rates [28]. Researchers in the United States and Germany found that β-carotene supplementation shortened age at puberty, increased pregnancy rates, promoted uterine repair and ovulation, and reduced ovarian cyst incidence and early embryonic mortality. The importance of β-carotene for reproduction has been extensively studied in Japan, where cows with ovarian cysts showed significantly lower serum carotene concentrations [(11$±2)$\mu g/dL]thanhealthycows[$(33±$4) g/dL]. Serum β-carotene concentration also correlated with embryo quality in superovulated Japanese Black cattle; when serum β-carotene exceeded 200 g/dL, corpus luteum and total recoverable embryo numbers tended to increase, with significantly more normal transferable embryos [44]. β-Carotene supplementation also improved fertility in other animals; adding vitamin A (4,000 IU) and β-carotene (100 mg/kg) increased β-carotene content in adrenal glands and corpora lutea of gilts [45]. β-Carotene also enhances rumen function, as in vitro supplementation significantly improved rumen bacterial growth and cellulose digestion capacity [46].

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4 Summary

Research demonstrates that β-carotene functions not only as a vitamin A precursor but also as an antioxidant and anticancer agent in the body, exerting varying degrees of positive effects on animal performance, reproductive function, and immune capacity. Studies on β-carotene supplementation levels in animals have increased annually, though current evidence remains insufficient to establish optimal supplementation strategies. Further research is needed to determine β-carotene requirements for animals at different production stages. While relatively in-depth studies have examined the conversion, transport, deposition, and metabolism of carotenoids and vitamin A, the antioxidant and anticancer biological functions of β-carotene require deeper investigation.

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