Essentiality of n-6 fatty acids

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It was with great interest I read the recent JBC Reflections by William L. Smith (1). However, I was a bit provoked by his statement in the abstract that “Their essentiality (i.e. n-6 fatty acids) is largely due to their function as prostaglandin precursors.” This is not correct.

The major and early deficiency symptoms of n-6 fatty acids (n-6FA) are growth defects, scaly skin, and increased transepidermal water loss (TEWL), and they are ALL related to proper levels of linoleic acid-derivatives in the skin.

In short, some important arguments are as follows.

1) The growth defect of n-6FA-deficient rats is due to increased TEWL (and thereby loss of energy), and no growth and skin defects are seen when raising rats in high humidity (2).

2) Dietary arachidonic acid can cure the growth defects and the TEWL, but this is due to retro-conversion of arachidonic acid to linoleic acid (3, 4).

3) Dietary columbinic acid, which is not an arachidonic acid precursor, can cure the well-known n-6FA deficiency symptoms.

4) Mice deficient in δ-6-desaturase do not develop the well-described n-6FA deficiency symptoms.

5) Severe inhibition of COX enzymes (e.g. aspirin) does not result in n-6FA deficiency symptoms.

6) Knockout of COX enzymes or receptors for various prostaglandins, leukotrienes, and other arachidonic acid derivatives does not result in mice having well-known n-6FA deficiency symptoms.

7) Recent research provides plausible molecular mechanisms for the essentiality of linoleic acid in maintaining a low TEWL (5).

Surely, there must also be deficiency symptoms related to arachidonic acid, but they have not been clearly described yet.

References


