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Linoleic and alpha-linolenic acids, obtained from plant material in the diet are the precursors in tissues of two families with opposing effects which are referred to as "essential fatty acids" (EFA): arachidonic acid (AA) and pentaene (eicosapentaenoic acid: EPA) and hexaene (docosahexaenoic acid: DHA) acids.

The role of EFA is crucial, without a source of AA or compounds which can be converted into AA, synthesis of prostaglandins (PGs) by a cyclooxygenase (COX) enzyme would be compromised, and this would seriously affect many normal metabolic processes. COX, also known as prostaglandin endoperoxide synthase (Pghs) or as prostaglandin G/H synthase, is a key membrane bound enzyme responsible for the oxidation of AA to PGs.

Two COX isoforms have been identified, COX-1 and COX-2 that form PGH₂, a common precursor for the biosynthesis of thromboxane A₂ (TxA₂), prostacyclin (PGI₂) and PGs (PGD₂, PGE₂, PGF₂alpha. COX-1 enzyme is expressed constitutively in most cells and tissues. Its expression remains constant under either physiological or pathological conditions controlling synthesis of those PGs primarily involved in the regulation of homeostatic functions.

In contrast, COX-2 is an intermediate response gene that encodes a 71-kDa protein. COX-2 is normally absent from most cells but highly inducible in certain cells in response to inflammatory stimuli resulting in enhanced PG release. PGs formed by COX-2 primarily mediate pain and inflammation but have multiple effects that can favour tumorigenesis. They are more abundant in cancers than in normal tissues from which the cancers arise.

COX-2 is a participant in the pathway of colon carcinogenesis, especially when mutation of the APC (Adenomatous Polyposis Coli) tumour suppressor gene is the initiating event. In addition, COX-2 up-regulation and elevated PGE₂ levels are involved in breast carcinogenesis. It seems that there is a correlation between COX-2 level of expression and the size of the tumours and their propensity to invade underlying tissue.

Inhibition by non-steroidal anti-inflammatory drugs (NSAIDs) of COX enzymes which significantly suppress PGE₂ levels, reduced breast cancer incidence and protected against colorectal cancer. Therefore it is suggested that consumption of a diet enriched in n-3 PUFA (specifically EPA and DHA) and inhibition of COX-2 by NSAIDs may confer cardioprotective effects and provide a significant mechanism for the prevention and treatment of human cancers.

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