LETTER TO THE EDITOR

Cyclooxygenase-2, prostaglandin E2 and acute myeloid leukemia

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Dear Sir,

We read with a great interest the excellent article by Chien et al. (1) demonstrating that vascular endothelial growth factor-C (VEGF-C) promotes angiogenesis in acute myeloid leukemic (AML) cells by induction of cyclooxygenase (COX)-2 and the subsequent prostaglandin E2 production. This very interesting study answers a currently unsolved question: Why only a subset of AML blasts express COX-2?

During the past decade, several studies have reported the involvement of lipidic mediators (including COX-derived metabolites) on the growth of human myeloid/erythroid progenitors and blood cell lines (2,3). More recently, freshly isolated AML blasts were reported to secrete prostaglandin E2 (4) and to express functional EP2 receptors (5,6). While AML blasts expressed COX-2 transcripts but not the corresponding protein (4), lipopolysaccharide stimulated COX-2 protein expression through the toll-like receptor-4 pathway in a subset of AML blasts (7). Chien et al. (1) reported that 50% of bone marrow specimens from AML patients express COX-2 with a strong correlation between COX-2 and VEGF-C. Of interest, the expression of VEGF-C and VEGF receptors is positively regulated by LPS in HL-60 cells (a M3 AML cell line) during macrophage differentiation (8).

In conclusion, past studies have highlighted that lipidic mediators might act on tumor growth by altering the local cytokine network (9,10). The interesting study of Chien et al. (1) returns the situation and shows that angiogenic factors (such as VEGF-C) might act on tumor growth by altering the lipidic network.

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References


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