oncological diseases. SC-236 and its analogue celecoxib are used as the COX2 inhibitors in considering the effect of COX2 on tumor vessel density. Almost 78% reduction in tumor growth and lung metastasis after 28 days of treating tumors using mentioned inhibitor is observed, however the declared changes are not significant statically. Using terminaldeoxynucleotidyl transferase–mediated nick-end labeling (TUNEL) procedure showed that induced limitation of the tumor growth is not the consequence of the increased apoptosis however disrupted proliferation, angiogenesis and vascular density are the responsible causes of this effect (20,25).

Angiogenesis of other tumor models are reported to be inhibited by arresting endothelial cell cycles, increasing the expression of endostatin, or inducing apoptosis by decreasing endothelial growth factor (26,27). In the considered studies, using COX2 inhibitor did not lead to decreased expression of the endothelial growth factor. Using COX2 inhibitor (SC-236), yielded some changes in gene expression of Wilms' tumor which lead to increase or decrease in the expression of genes to suppress the vascular proliferation and stability.

Data regarding altered genes expression after administrating COX2 inhibitor are summarized in Table 2.

By considering all the studies performed on evaluating the changes in expression level of COX2 protein during Wilms' tumor a considered malignancy, increase in COX2 expression was the indication of its role in pathogenesis of Wilms' tumor and immonureactivity of COX2 protein was demonstrated in all stages of the tumor development thus Cox2 inhibitors could be considered as a therapeutic agent.

Table 2. Altered expression of various genes in Wilms' tumor due to inhibition effect of SC-236.

Altered genes expression	Changes after SC-236 applying
Perlecan	↑fold 1.8
Collagen IV	↑3.6fold
Tissue inhibitor of metalloproteinase-1	↑fold 1.4
Desmin	↓fold 0.62
,EDG1	↓fold 0.42
CXCR4	↓fold 0.68
Spondin-1	↓fold 0.26
CCM1	↓fold 0,77
Pleiotrophin	↓fold 0.72
CXCL14	↓fold 0.32

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Conflict of Interest

The authors declare no conflict of interest.

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