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It has been shown that VEGF is overexpressed in various tumours, including breast cancer, and VEGF levels are correlated with tumour-associated angiogenesis and vascular permeability^[@CR55]^.

An increase in the levels of VEGF was observed in a glucose-induced diabetic rat model^[@CR56]^.

Moreover, inhibition of VEGF expression was shown to be effective in treating diabetic retinopathy in diabetic rats^[@CR57]^.

Soluble VEGF-R1 was observed in the serum of patients with diabetic retinopathy^[@CR58]^.

The role of TGF- β in tumour angiogenesis has been shown in a number of studies^[@CR59],[@CR60]^.

VEGF is secreted by tumour cells under certain conditions. Similarly, the cytokine TGF- β is secreted by fibroblasts in a tumour microenvironment^[@CR61]^.

Tumour cells can secrete VEGF to increase vascular permeability, leading to the formation of vascular networks in the tumour^[@CR62]^.

Several mechanisms have been suggested to explain TGF- β -mediated angiogenesis. TGF- β has been shown to induce the EMT process

in cancer cells^{[@CR63],[@CR64]}. In the TGF- β -induced EMT process, the expression of Snail is upregulated, leading to activation of the EMT mechanism^[@CR65]. Vimentin also plays a role in induction of tumour angiogenesis and its expression is correlated with poor prognosis in breast cancer^[@CR66]. Conclusions {#Sec13} =====
Our data indicate that TGF- β induced EMT and upregulated VEGF-A expression. In addition, glucose-induced EMT increased expression of TGF- β , upregulated the expression of VEGF-A, and activated the VEGF-VEGFR system. Based on these findings,

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