Angiogenesis is a multi-step process, which includes endothelial cell proliferation, migration, basement membrane degradation, and new lumen organization. The development of new blood vessels is controlled by a local balance between stimulators and inhibitors of new vessel growth (Kerbel 2008). A finely tuned equilibrium exists between physiological anti-angiogenic and pro-angiogenic factors (Folkman 2000, Kerbel and Folkman 2002).

In pathological states such as chronic inflammation and tumor growth, there is an imbalance between endogenous stimulator and inhibitor levels, leading to an "angiogenic switch". Aberrant angiogenesis is involved in invasion/metastasis as well as malignant tumors (Brooks *et al.,* 1998).

These vessels are the primary route, by which tumor cells leave the primary tumor site and enter the circulation, and as such vascular density can provide an indicator of metastatic potential (Folkman and Shing 1992). Specifically, highly vascular primary tumors are generally regarded as having a higher incidence of metastasis than poorly vascularized tumors. Tumor angiogenesis is activated by the production of angiogenic stimulators including: members of the fibroblast growth factors (FGF) and vascular endothelial growth factor (VEGF) families. On the other hand, angiogenesis inhibitors such as angiostatin (a 38 KDa internal fragment of plasminogen), and endostatin have been shown to modulate tumor growth in primary tumor sites or metastases. Thus, angiogenic inhibitors have been considered as potential anticancer drugs (Crawford *et al.*, 2009).

Cancer cells (probably like all tissues) secrete substances that promote the formation of new blood vessels; a process called **angiogenesis**. Over a dozen substances have been identified that promote angiogenesis. A few examples are: