Inhibition of the soluble extracellular domain of E-cadherin as a novel anti-cancer drug

Neutralization of the extracellular fragment of the adhesion molecule E-cadherin selectively induces epithelial-derived tumor cells to undergo apoptotic cell death while having no effect on normal healthy cells.

Background:
One of the greatest challenges encountered in the development of novel anticancer therapies is the inability of the therapeutic agents to distinguish between normal healthy tissues and aberrant cancerous cells. The non-selective cytotoxicity and narrow therapeutic index of these chemotherapeutic agents results in an unwanted systemic drug toxicity that is debilitating to patients and heightens overall patient mortality.

Technology Description:
Research done by Dr. Brouxhon et al at Stony Brook University selectively kills tumor cells and spares normal non-cancerous cells by acting upstream of the HER2 receptor and therefore captures a wider array of downstream targets. Specifically, exogenous application of a monoclonal or polyclonal antibody against sEcad, selectively killed a representative panel of human and mouse tumor cell lines by inducing cellular apoptosis (breast and skin). Similar findings were found in mouse lung cancer cells. In contrast, non-cancerous cells, including normal human breast epithelial cells, normal human and mouse keratinocytes, mouse 3T3 fibroblasts and human HUVEC endothelial cells remaine protected from this anti-sEcad-mediated programmed cell death.

Patents / Publications:
• Patent Pending

Advantages
• Highly specific and effective targeting of tumor cells while leaving non-tumor cells protected

Applications
• Anti-cancer therapeutics

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