Background
Gastric cancer (gastric carcinoma) cells have been found to have higher levels of a mutated form of the matrix metalloproteinase Epilysin (MMP-28). The study of metalloproteinases like epilysin focuses on structurally similar enzymes that include a metal core. These proteins play important roles in breaking down other material found in the spaces between individual cells including other proteins, growth factors.

Figure 1: Overexpression of MMP28 protein in gastric cancer is correlated with tumor aggressiveness and poorer prognosis (Jian, et al. 2016).

Introduction
Epilysin is one of the members of the matrix metalloproteinase (MMP) family. Epilysin has been shown to be expressed in many human tissues such as testes, lung, intestine, heart, brain, and keratinocytes in skin. The protein is made up of 520 amino-acids which include a signal peptide, a prodomain with an unusual cysteine-switch region followed by the furin cleavage site, a catalytic domain, a hinge-region and a hemopexin domain (see Figure 5).

Epilysin is responsible for restructuring of the basement membrane and adhesive proteins between karyocytes to supply newly formed cells. In different cancer tissues, increased expression of mutant epilysin results in activation of tumor growth factors that promote cell division and growth while also promoting stable and irreversible epithelial to mesenchymal transition (EMT) accompanied by loss of cell surface adhesion.

Figure 2: Three-dimensional structure of Epilysin (MMP28). Sites indicated in yellow are zinc ions (Kaluri, R., et al. 2009).

Epilysin has been shown to be one of the important precursors for fibroblasts that begin during the course of organ fibrosis. Matrix metalloproteinases degrade the extracellular matrix, which release a variety of inflammatory signals which lead to organ fibrosis, the thickening and scarring of connective tissues. Therefore epithelial cells are important precursors for fibroblasts.

Epithelial to mesenchymal transition is a fundamental biological process where epithelial cells lose their polarity and adopt morphology appropriate for migration (and, if carcinoma, metastasis). The second type of EMT which occurs in the epithelial tissues is mediated by inflammatory cell and fibroblast which release a variety of inflammatory signals which lead to organ fibrosis.

Figure 3: Immunologically labeled tissue showing expression of wild-type and mutant Epilysin (https://www.researchgate.net/figure/24306604_fig3_Expression-of-MMP-28-in-stably-transfected-SW1353-cells-SW1353-cells-were-stably).