

Prion protein identified as a novel early pancreatic cancer biomarker

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Mad cow disease is caused by the accumulation of an abnormal protein, the prion, in the brain of an affected patient. Outside of the brain, very little is known about prions. Case Western Reserve University School of Medicine, researchers have, for the first time, identified the prion as a biomarker for pancreatic cancer. Pancreatic cancer is one of the most deadly cancers in humans; the five year survival rate is less than 10 percent.

Chaoyang Li, Ph.D., Wei Xin, M.D., and professor of pathology, Man-Sun Sy, Ph.D., discovered the mechanism by which prions causes tumors to grow more aggressively. They published these findings in the September issue of the <u>Journal of Clinical Investigation</u>.

Unlike normal cells, in human pancreatic <u>cancer</u> cells the prion is incompletely processed and binds to a molecule inside the cell known as filamin A. Filamin A is an important regulator of the cell's skeleton and its signaling machineries. The binding of the incompletely processed prion to filamin A disrupts the cell's organization and signaling. As a result, the tumor <u>cells</u> grow more aggressively. On the other hand, when the prion level is reduced, the tumor cell loses its ability to grow in tissue culture and in animals. Most importantly, Dr. Li, et al. found that a subpopulation of patients had incompletely processed prion protein in their pancreatic cancer. This subgroup of patients had significantly shorter survival compared to patients whose tumors do not have prion.

According to Dr. Sy, "Currently there is no early diagnostic marker for



<u>pancreatic cancer</u>. Detection of the incompletely processed prion may provide such a marker. Preventing the binding of prion to filamin A may open new avenues for therapeutic intervention of this deadly disease."

Next, Drs. Li and Sy will look to determine if this type of <u>prion</u> protein expression is seen in other types of cancer.

Source: Case Western Reserve University (<u>news</u>: <u>web</u>)

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