

# The role of protein misfolding and aggregation in bse



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When a protein misfolds it changes its behavior and function. If it becomes hydrophobic after once being polar. The properties and functionality of the protein are no longer useful to the organism and disaster results. PrP<sup>Sc</sup> is hydrophobic, it avoids water inside of the cell...it attracts and attaches other proteins to misfolds and become hydrophobic ...Misfolding spreads because the PrP<sup>Sc</sup> act as chaperone proteins to convert PrP<sup>C</sup> TO PrP<sup>Sc</sup> and cannot be converted back to PrP<sup>C</sup>.

The normal homeostasis would be reached and health regained by PROTEASOMES (protein destroyers) eating the corrupt PrP<sup>Sc</sup> proteins BUT.. These are not recognized by the proteasomes and so are not destroyed. They keep multiplying and they clump together and aggregate inside the cell and the cell stops doing its normal work and eventually it dies. Prion - Wikipedia, the free encyclopedia. (n. d. ). Retrieved November 23, 2013, from <http://en.wikipedia.org/wiki/PrPSc> How enzymes work Enzymes are complex proteins whose main function is to reduce or speed up the energy required for a reaction to occur.

This happens thru the enzymes ability to break or form a bond within a substance that results in 1 or 2 new substances without changing the protein configuration of the enzyme itself - this keeps the enzyme available to continue its work. The area on the enzyme where the work takes place is called the active site. The specific molecule that becomes transformed is called a substrate.

It seems to me that enzymes with the suffixes dehydrogenase break up substances and synthetase combine substances to make new products.

Anaerobic Glycolysis occurs when there is continuing muscle activity. This produces some ATP for continued muscle work but not a whole lot. Lactic acid builds and eventually the muscles get fatigued and activity must stop. The blood then diffuses this lactate to the liver where it is converted back to glucose and enters into the citric acid cycle and more ATP is created.

If a certain enzyme were to be lacking in the citric acid cycle it would grind it to a halt, ATP energy would not be produced and cell death would occur. One of the assisting molecules that help the electrons cross the intercellular membrane of the mitochondria. The electrons are then passed from enzyme to enzyme in the inner membrane of the mitochondrion, in an energy gradient and they lose some of their energy at each step. This transfer which causes in a high concentration of  $H^+$  protons is what results in the phosphorylation of ADP to ATP (and energy).

The final transfer involves the combining of electrons and  $H_2$  atoms with oxygen. This forms water. The molecules that take part in the transport of these electrons are referred to as the electron transport chain. Oxaloacetate is the first substrate to bind to the enzyme. This induces the enzyme to change its conformation, and creates a binding site for the acetyl-CoA. Only when this acetyl-CoA has formed will another conformational change cause thioester hydrolysis and release coenzyme A. This ensures that the energy released from the thioester bond cleavage will drive the condensation. Oxaloacetate will be regenerated after the completion