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Enzyme Engineering

ISSN: 2329-6674

Commentary

Brief Note on Enzyme Inhibition

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DESCRIPTION

A compound that binds to an enzyme and reduces its activity is known as an enzyme inhibitor. The process is known as enzyme inhibition. By binding to the active sites of enzymes, inhibitors decrease the compatibility of substrate and enzyme, resulting in the suppression of the formation of Enzyme-Substrate complexes, prohibiting catalysis of processes and lowering (sometimes to zero) the quantity of product generated by a reaction. The concentration of inhibitor molecules increases, the rate of enzyme activity reduces. Hence the amount of product produced during the biochemical process is inversely proportional to the concentration of inhibitor molecules. Many drugs are enzyme inhibitors, which kill pathogens or create metabolic imbalances by decreasing enzyme activity. They are also used in insecticides. Inhibitors are not the only substances that bind to enzymes; enzyme activators attach to enzymes and boost their enzymatic activity. The enzyme substrates bind to enzymes and are transformed into products during the enzyme's regular catalytic cycle.

Types of enzyme inhibition

Reversible inhibition and irreversible inhibition are the two basic kinds of enzyme inhibition. The strength of the interaction between the enzyme and the inhibitor determines the difference. Furthermore, reversible inhibition can be classified as competitive, non-competitive, uncompetitive based on where the inhibitor binds to the enzyme. Both reversible and irreversible Inhibitors are substances that attach to an enzyme and inhibit its action. Another way to do this is to connect to an enzyme permanently. These inhibitors are said to as irreversible inhibitors. Other inhibitors which can temporarily bind to an enzyme are known as reversible inhibitors. Reversible inhibitors attach to either an active site or another location on the enzyme. Based on the binding region they are divided into 3 types there are competitive inhibitors, non-competitive inhibitors, un-competitive.

Competitive inhibitors: Competitive inhibitors compete with the substrate at the active site, raising Km (the michaelis-menten constant). However, Vmax remains unaltered since the reaction may still be completed with sufficient substrate concentration. Because of the rise in Km, the graph plot of enzyme activity vs. substrate concentration would move to the right, and the line weaver-burke plot would be steeper when compared to no inhibitor.

Non-competitive inhibitors: Non-competitive inhibitors attach to another site on the enzyme and hence reduce Vmax. KM value, on the other hand, remains unaltered. When compared to no inhibitor, this is evidenced by a smaller maximum on a graph mapping enzyme activity versus substrate concentration and a greater y-intercept on a line weaver-burke plot.

Uncompetitive inhibitors: Uncompetitive inhibition occurs when the inhibitor attaches to the enzyme-substrate complex after the substrate has bound and stops the reaction from taking place.

Allosteric inhibition

Michaelis-menten enzymes have a hyperbolic curve, whereas allosteric enzymes have a sigmoidal curve. This is because most allosteric enzymes include several subunits that can interact with one another when the substrate binds to the enzyme. Inhibition can affect K0.5, the substrate concentration for half-saturation, or Vmax, or both. This causes the curve to move to the right, and in the case of decreasing Vmax, it shifts downward. Allosteric enzymes have two states: the "T" state, which has a low affinity, and the "R" state, which has a high affinity. Inhibitors act by preferentially attaching to an allosteric enzyme's T state, forcing the enzyme to remain in this low affinity state. This is highly effective for limiting the quantity of an enzyme's product since the product may then block the same kind of enzyme to guarantee that the amount of product is not excessive. This is also known as feedback inhibition. For example, ATP allosterically inhibits pyruvate kinase to prevent more pyruvate production, resulting in reduced ATP generation. Furthermore, citrate, which is an intermediary in the Krebs cycle, inhibits phosphofructokinase allosterically. This indicates that glycolysis will be inhibited when Krebs cycle generates a lot of ATP.

Phosphorylation is another mechanism for inhibiting enzymes. This is commonly accomplished by the activity of kinase

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Received: 10-Jan-2022, Manuscript No. EEG-22-0001; **Editor assigned:** 12-Jan-2022, PreQC No. EEG-22-0001 (PQ); **Reviewed:** 26-Jan-2022, QC No. EEG-22-0001; **Revised:** 31-Jan-2022, Manuscript No. EEG-22-0001 (R); **Published:** 07-Feb-2022, DOI: 10.35841/2329-6674.22.11.1000172

Citation: Salcedo A (2022) Brief Note on Enzyme Inhibition. Enz Eng.11: 172.

enzymes. Their mechanism is either to block or activate an enzyme. Kinase enzymes remove a phosphate group from ATP and bind it to the enzyme. When this increases enzyme activity, a cascade reaction occurs, allowing for a huge response to be created from a little stimulus. Enzymes can also be secreted in an inactive condition, which is referred to as zymogens.

Zymogens are a useful mechanism for securely transporting enzymes to different sites without the enzyme becoming active and completing its activity along the route. Because of the inclusion of amino acids in the protein, they stay inactive. To activate a zymogen, another enzyme must break off these extra amino acids. The pancreas, for example, produces chymotrypsinogen, but it is inactive and cannot function. When the enzyme chymotrypsin reaches the intestines, another enzyme (trypsin) cleaves off the extra amino acids to generate the active version, chymotrypsin.

CONCLUSION

Pharmaceutical medications are the most common therapeutic use of enzyme inhibition. ACE inhibitors are a frequent hypertension therapy. The function of the angiotensin-converting enzyme converts angiotensin I to angiotensin II (ACE). Angiotensin II, on the other hand, has several side effects that result in higher blood pressure. As a result, ACE inhibitors are meant to reduce ACE's function competitively, resulting in less angiotensin II production and lower blood pressure.