OPINION

The Conversion of Glutamic Acid into Gamma-Amino Butyric Acid within Pancreatic Islet β Cells using as a Glutamic Acid Decarboxylase (GAD) Catalyzes

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ABSTRACT

Glutamic acid is a -amino acid that is employed in the production of proteins by practically all living things. It is non-essential in humans, which means it can be synthesized by the body. It's also an excitatory neurotransmitter in the vertebrate nervous system, and it's the most prevalent. In GABA-ergic neurons, it serves as a precursor for the synthesis of the inhibitory gamma-aminobutyric acid (GABA).

Glutamic Acid Decarboxylase (GAD) is divided into two isoforms, GAD_{65} and GAD_{67}, based on their molecular weights, and is expressed by two separate genes. GAD_{67} appears to be a cytosolic enzyme that is found throughout GABA-ergic neurons, including cell bodies, dendrites, and axonal processes. GAD_{65}, on the other hand, is mostly located in nerve terminals and may be tethered to the membrane of the neurotransmitter-containing vesicles. GAD regulation is complicated and not completely understood. PLP is a cofactor for GAD, and its association and dissociation play a key role in GAD activity modulation in the near term.

INTRODUCTION

Glutamic Acid Decarboxylase (GAD) enzymes catalyze the synthesis of GABA, a key transmitter in the central nervous system that also has functions in peripheral organs. GAD isoforms have been discovered to play unexpected new roles in autoimmune diseases such as neurological disorders and insulin-dependent diabetes in recent molecular investigations. The co-authors of this review covered genetics, cell biology, molecular immunology, and the role of GAD as auto antigens in human autoimmunity at the 1995 Frontiers in Medicine Symposium. Unique patterns of reactivates in both cellular and humoral immune responses have been discovered in studies on illness diagnosis, prediction, and prognosis. It will need more research to see if GAD compounds can be employed to treat autoimmune illnesses [1].

Conversion of Glutamic Acid into Gamma-Amino Butyric Acid

Gamma-Amino Butyric Acid (GABA) is a non-protein amino acid found throughout nature. It is made by the enzyme glutamate decarboxylase performing irreversible -Decarboxylation of Glutamate (GAD). Plants, animals, and microbes all contain GABA and GAD. GABA is found all over the body and plays a function in the regulation of cardiovascular disorders like blood pressure and heart rate, as well as the reduction of anxiety and discomfort. Although researchers previously manufactured GABA using a chemical approach, it has become less acceptable due to environmental pollution. For the manufacture of GABA, researchers currently use a more promising microbial approach. GABA is in high demand in the pharmaceutical and food industries [2].

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Enhanced Productivity of Gamma-Amino Butyric Acid

GABA (gamma-aminobutyric acid) is a non-protein amino acid found throughout nature. It is made by the enzyme glutamate decarboxylase performing irreversible -decarboxylation of glutamate (GAD). Plants, animals, and microbes all contain GABA and GAD. GABA is found all over the body and plays a function in the regulation of cardiovascular disorders like blood pressure and heart rate, as well as the reduction of anxiety and discomfort. Although researchers previously manufactured GABA using a chemical approach, it has become less acceptable due to environmental pollution. For the manufacture of GABA, researchers currently use a more promising microbial approach. GABA is in high demand in the pharmaceutical and food industries. Second, we increased GABA productivity by deleting the genomic region encoding the C-plug of GadC (a glutamate/GABA antiporter) to allow its transport channel to remain open at neutral pH, promoting L-Glu and GABA transportation. Third, by introducing the GroESL molecular chaperones, we increased the expression

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of soluble GadB, resulting in increased GABA productivity, with GABA and productivity acquired in one cycle. Finally, we stopped GABA breakdown by removing gadA and gadB from the E. coli genome, resulting in nearly minimal GABA degradation after 40 hours. The altered recombinant *E. coli* strain attained productivity in a bioreactor after the cascade system changes, with a conversion of L-Glu [3]. In comparison to the starting strain, productivity increased by around twofold. This rise reflects the highest GABA productivity achieved by whole-cell bioconversion employing L-Glu as a substrate in a single cycle to date, surpassing the productivity achieved by three consecutive conversion cycles [3].

Role of Glutamate Receptors

Although the excitatory amino acid glutamate and its receptors play critical roles in many CNS functions, its presence in peripheral tissues has remained a mystery. Using reverse transcriptase polymerase chain reaction, we found kainate, alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionate (AMPA), and N-methyl-D-aspartate (NMDA) receptor subtype mRNAs in pancreatic islets (RT-PCR). Kainate, AMPA, and NMDA all raise and depolarize single pancreatic beta-cells, according to intracellular calcium measurements and electrophysiological recordings [4].

Mapping of GAD genes

Glutamic Acid Decarboxylase (GAD) catalyses the formation of Gamma-Aminobutyric Acid (GABA), a key inhibitory neurotransmitter found in the Central Nervous System (CNS) and elsewhere. Autoantibodies linked to the development of insulin-dependent diabetes mellitus and the unusual stiff man syndrome have been found to target GAD in recent investigations. Different transcripts have been discovered in studies of GAD expression, implying that there are multiple GAD isoforms. In situ hybridization was used to map three separate genes to human and mouse chromosomes in this investigation. In a known conservation region, the GAD1 gene was found on human chromosome 2q31 and mouse chromosome 2D [5].

CONCLUSION

After glutainine injection, GABA might be released from beta cells into the islet-acinar portal system, enhancing CCK-stimulated exocrine secretion *via* GABA(A) receptors. GABA is a hormone that regulates pancreatic exocrine secretion in islet beta cells.

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