presynaptic membrane. The next step is that the enzyme glutamic acid decarboxylase metabolises the glutamate into GABA. The supply of GABA is maintained through the steps that occur by the GABA shunt which involves the enzyme GABA transaminase (GABA-T). This enzyme appears to be attached to the mitochondria and it catalyses the reaction between GABA and alpha ketoglutarate to form succinate semialdehyde and L-glutamate. This reaction is vital in the conservation of GABA as there is a very low concentration of glutamate found in the presynaptic neuron. Therefore a high concentration of glutamate needs to be present to ensure there is a continuous supply of the neurotransmitter GABA. Like catecholamines the rate of release is regulated by autoreceptors present on the presynaptic neuron. When GABA is released it binds to these receptors which subsequently decrease the release of the neurotransmitter. Once GABA has been synthesised it is actively transported into the vesicle by a transport protein and its release is triggered when there is an influx of calcium ions due to a nerve impulse.

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\text{Oxaloacetate} \xrightarrow{\alpha\text{-oxoglutarate}} \text{Glutamate} \xrightarrow{\text{GABA}} \text{GABA}
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After binding to post synaptic receptors and stimulating a response, the GABA neurotransmitter is inactivated as it is taken back into the presynaptic neurone through transport proteins that couples the influx of Na+ ions to the influx of GABA molecules. The key difference between GABA and other neurotransmitters is that for GABA there are multiple transport proteins on its presynaptic membrane. This allows for the rapid uptake of GABA once it has been released into the synaptic cleft. After the transmitter is inside the neuron the enzyme GABA transaminase converts the GABA into succinic semialdehyde. Following this succinate semialdehyde dehydrogenase catalyses the conversion of succinic semi-aldehyde to succinic acid which is fed into the Krebs cycle as an intermediate. As mentioned before the breakdown of GABA once it is taken back into the presynaptic neurone is essential as it allows glutamate to be formed which allows new GABA molecules to be synthesised. Studies with transgenic mice that lack membrane transporters show that the store of releasable transmitter is substantially decreased which shows that synthesis is unable to maintain the store if the recapture mechanism doesn’t work.