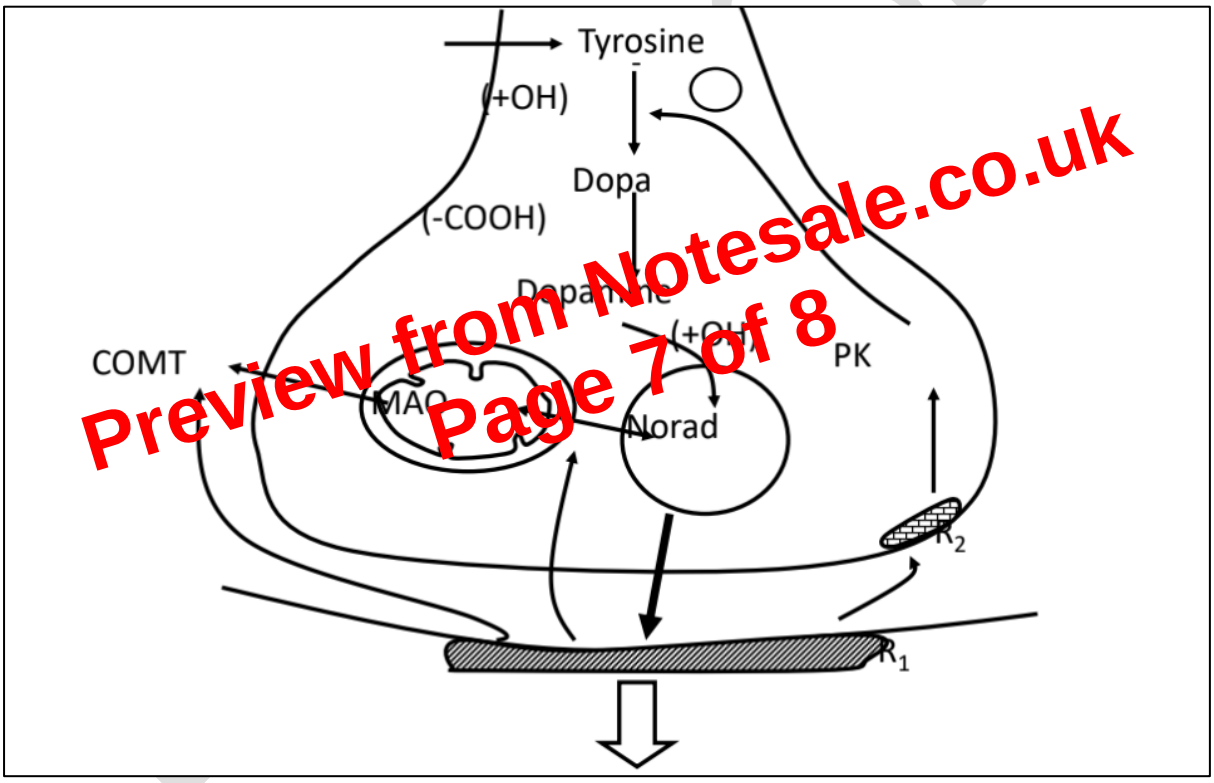
Noradrenergic synapses contain the neurotransmitter noradrenaline (NorAd). The synthesis of NorAd is done by the following steps:

- **Tyrosine** is converted 1,2-dihydroxyphenylalnine (**DOPA**) through **tyrosine hydroxylase**,
- **DOPA** is then converted into **dopamine** through **DOPA decarboxylase**,
- **dopamine** is finally converted into **NorAd** by **dopamine β-hydroxylase** (NorAd can be further converted into adrenaline by the action of phenylethanolamine N-methyltransferase).

Vesicular monoamine transporters (**VMT**) load NorAd into vesicles in the nerve endings, when calcium influx is generated, exocytosis of NorAd is achieved, at the level of the synaptic cleft, NorAd binds to its noradrenergic receptors in the post synaptic element, excess NorAd undergoes reuptake into in the presynaptic element through Norepinephrine Transporter (**NET**) found on the presynaptic element or it can be catabolized by monoamine oxidase (**MAO**) [intracellularly inside a mitochondrion] or through catechol-O-methyltransferase (**COMT**) [at the synaptic cleft]. There are also receptors at the presynaptic element for NorAd which inhibits the biosynthesis of NorAd through the G_i protein signaling pathway.



Usually, neurotransmitters are terminated from the synaptic cleft through **diffusion**, **degradation** or **reuptake.**

Biogenic amine and neuropeptides influence the metabolism of the post synaptic element and have a longer influence than other neurotransmitters. They activate a signaling pathway that involves the function of a kinase (through production of cAMP) that phosphorylates proteins that closes K^+ channels which leads to longer times of stimulation and longer recuperation (to return to resting potential) times.