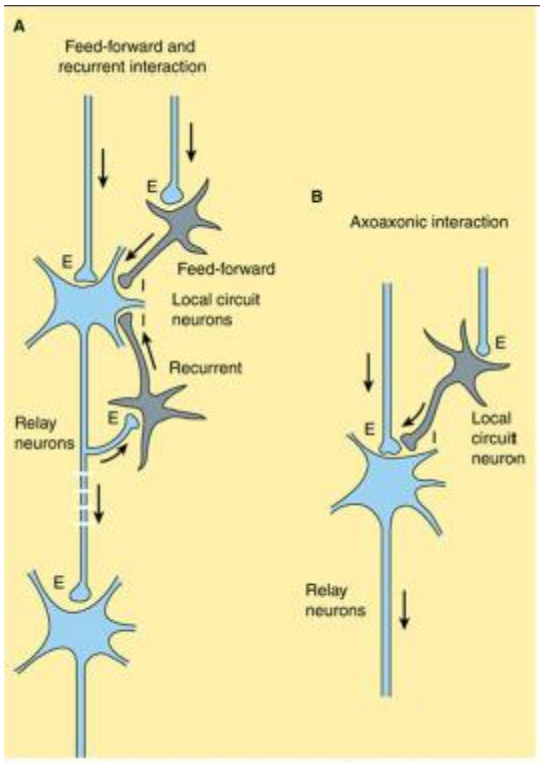

a. Source: Katzung BG, Masters SB, Trevor AJ: Basic & Clinical Pharmacology, 12th edition:

10. **Classes of Neurotransmitters**
    a. **Amines:** acetylcholine, dopamine, norepinephrine, serotonin
    b. **Amino Acids:** neutral amino acids (GABA, glycine), glutamate/glutamic acid
    c. **Peptides:** opioids
11. **Drug targets in CNS disease:** Presynaptic neuron, neurotransmitter synthesis, storage, metabolism, release, uptake, and degradation, post-synaptic receptor, ionic conductance, etc.

CNS Diseases

1. **Drug Goals**
    a. **Depression:** Enhance serotonin and norepinephrine transmission
    b. **Pain:** μOR agonists
    c. **Psychosis/Schizophrenia:** Block dopamine receptor activity
2. **Pathogenesis of Depression**
    a. **Monoamine hypothesis:** Depression reflects deficiency in serotonin and norepinephrine transmission in CNS. Reserpine can chemically induce depression. All effective anti-depressants affect norepinephrine, serotonin and dopamine.
    b. **Neurotrophic hypothesis:** Depression reflects loss of synaptic connectivity. Depressed patients have a loss of volume in hippocampus and dendritic sprouting. If you increase BDNF, you can promote dendritic sprouting, synaptic connectivity, and neurogenesis. Anti-depressants and electroconvulsive treatment boost BDNF.
    c. **Neuroendocrine regulation:** Hypothalamic-pituitary-adrenal (HPA) axis of hormone signaling impacts depression. Severe MDD is associated with elevated cortisol and dysregulation of stress hormone responses. Glucocorticoids, hypothyroidism, and estrogen deficiency cause subtypes of depression.