1. An EPSP stands for excitatory postsynaptic potential. It is a small depolarization in the postsynaptic cell (usually about 1-2mV) induced by excitation of receptors following release of neurotransmitter from the presynaptic cell. A unitary EPSP is the result of 1 synaptic event onto a postsynaptic cell. A compound EPSP is the results of multiple, simultaneous synaptic events onto a postsynaptic cell.

2. A CA3 neuron projects to about 15,000 different CA1 pyramidal neurons.

3. AMPA and NMDA receptors are both ionotropic glutamate receptors. This means they are both activated by glutamate and both contain a pore through which ions flow into and out of the cell. Both receptors are mixed cation channels, but NMDA is also permeable to Ca2+. Only AMPA receptors lacking the GluR2 subunit are permeable to Ca2+. NMDA receptors are blocked by Mg2+ at hyperpolarized potentials, so its IV curve is non-linear at hyperpolarized potentials. AMPA receptors are not blocked by Mg2+ and thus have a linear IV relationship at all membrane voltages. NMDA receptors have an IV relationship like that of the open circles, while the AMPA receptors are more like the closed circles.

Both NMDA and AMPA receptors are tetramers (consist of 4 subunits). AMPA receptors can contain any combination of GluR1, GluR2, GluR3, or GluR4 subunits and can also be homomers. If GluR2 is in the channel, it will not be permeable to Ca2+. NMDA receptors must have 1 NR1 subunit. The other subunits are some combination of NR2A and NR2B.

AMPA receptors are gated by glutamate binding alone. NMDA receptors require both glutamate and glycine to bind, but glycine is normally always bound to the channel due to the incredibly high affinity of the binding site. Because of the Mg2+ block at hyperpolarized potentials, NMDA receptors require both membrane depolarization and glutamate binding to open.

4. NMDAR are coincidence detectors because they only open in the presence of both glutamate and membrane depolarization. Thus they detect the coincidence of both pre and postsynaptic activity. Not only do presynaptic neurons need to release glutamate onto a cell, there need to be enough presynaptic neurons doing that simultaneously to get the membrane potential depolarized enough to relieve the Mg2+ block on NMDAR.
So stimuli that trigger the simultaneous release of glutamate from many neurons converging on the same postsynaptic cell, together with substantial depolarization of that cell will trigger the NMDA receptors. This coincidence detection makes NMDAR central to mechanisms of learning and plasticity at the cellular level.

5. Glutamate reaches a concentration of about 1mM when it is released into the presynaptic cleft. It is there for only a short time (0.5-1 ms) before it is taken up by glia. The glial convert glutamate to glutamine which is released and taken up again by neurons who convert it to glutamate to be used again in the cycle.