

Upon impact, glutamate and other excitatory neurotransmitters attach to N-methyl-D-aspartate (NMDA) receptors leading to a rapid ion shift across the cell membrane. Rapid loss of intercellular potassium and influx of calcium forces up-regulation of the sodium-potassium pumps in an attempt to restore normal resting membrane potential. As sodium-potassium pumps deplete cerebral stores of adenosine triphosphate (ATP) compensatory hyperglycolysis occurs

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