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.Although numerous pathways are necessary for cerebellar function, much is known about the molecular mechanisms underlying synaptic transmission in the cerebellum. Recent work suggests that two distinct classes of inhibitory synapses may exist. One class, the GABAA receptors, are normally tonically activated by the inhibitory neurotransmitter GABA. However, GABAA receptors can be inhibited by a second class of receptors which are activated by the inhibitory transmitter glycine. We have studied this so-called glycinergic system and demonstrated that glycine-gated chloride channels are important for the regulation of cerebellar granule cells by the neurotransmitter GABA. In addition to these mechanisms, we have recently demonstrated that "spontaneous" glycinergic transmission is present in the cerebellar cortex. This spontaneous glycinergic transmission can occur in either an inhibitory or excitatory manner. Furthermore, the glycinergic



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system can be activated by endogenous agents such as the acidic amino acid, aspartate. These observations have led to the present proposal.

We postulate that the glycinergic system is fundamentally different from the GABAergic system. The inhibitory glycinergic synapse is important for fine regulation of cerebellar function and the excitatory glycinergic synapse could be involved in activating the cerebellum during conditioning. In addition, the spontaneous glycinergic transmission may function to modulate the strength of synaptic transmission from the cerebellar cortex to the deep cerebellar nuclei. In contrast to the inhibitory GABAergic synapse