The thalamo-cortical circuit undergoes postnatal, activity-dependent synaptic refinement. An interruption of glutamate transmission during the maturation of the thalamocortical circuit could result in changes in neuronal connectivity and synaptic maturation. These changes could, in turn, alter thalamic functions such as attention, event-related potentials, and elements of the EEG, resulting in symptoms seen in many neurological disorders, ranging from schizophrenia to autism. Since this activity-dependent refinement is due to signaling through glutamate receptors, we investigated whether treatment with an N-methyl-D-aspartate (NMDA) receptor antagonist, MK-801, during the period of postnatal circuit refinement, would lead to the changes in dendritic arbor and complexity, an indicator of synaptic connectivity. Rats were injected daily with either MK-801 (1mg/kg) or saline from postnatal day (P)7-P14 or from P8-26. The morphology of dendritic arbor of neurons in the mediodorsal (MD) thalamic nucleus was quantitatively assessed using Golgi-Cox staining. Effects of treatment were found to be significant in each treatment group, although the opposite between treatment groups. MK-801 treatment from P8-P26 increased the total amount and complexity of dendritic arbor in MD thalamus neurons, whereas treatment from P7-P14 led to a significant decrease in dendritic arbor and complexity. These results suggest a critical role for glutamate signaling during the refinement of the thalamo-cortical circuit. The duration of glutamate disruption can also lead to differing effects on the maturing thalamic dendrite arbor, which may play a role in various neurological disorders.