Animals explore their environment through locomotion. Motion is largely generated by the activity of neurons in the spinal cord. Genes expressed during embryonic development are essential for the formation of connections between spinal cord neurons that are responsible for producing movement. The murine Gbx genes, Gbx1 and Gbx2, are transcription factors widely involved in central nervous system development. In the spinal cord, they are expressed in unique populations of developing spinal cord neurons. To examine the role of Gbx1 in the developing spinal cord, we generated mice carrying a loss-of-function allele for Gbx1 (Gbx1<sup>−/−</sup>). By postnatal day 15, Gbx1<sup>−/−</sup> mice begin to display a gross locomotive defect that specifically affects hindlimb walking gait. Molecular analysis of mutant embryos revealed premature termination of proprioceptive sensory afferents (1a afferents) in the dorsal spinal cord. In addition, a subset of ISL+ spinal motor neurons is eliminated by apoptosis, at mid-embryonic stages. Both observations persisted at postnatal stages. This study demonstrated a requirement for Gbx1 for normal locomotor output. We also briefly assessed possible interaction of Gbx1 with its family member Gbx2 in spinal neuron development using Gbx1<sup>−/−</sup>; Gbx2<sup>−/−</sup> double knockout mutant embryos. Data from this study suggest that Gbx family members are required to maintain PAX2 interneuron cell fate in the dorsal spinal cord, but not functionally redundant for this shared function.