We propose a novel mechanism for Turing pattern formation that provides a possible explanation for the regular spacing of synapses along the ventral cord of *C. elegans* during development. The model consists of two interacting chemical species, where one is passively diffusing and the other is actively trafficked by molecular motors; we identify the former as the kinase CaMKII and the latter as the glutamate receptor GLR-1. We use linear stability analysis to derive conditions on the associated nonlinear interaction functions for which a Turing instability can occur. We find that the dimensionless quantity $\gamma$, the ratio of switching rate and diffusion coefficient to motor transport velocity, must be sufficiently small for patterns to emerge. One consequence is that patterns emerge outside the parameter regime of fast switching where the model effectively reduces to a two component reaction-diffusion system. Furthermore, these patterns are also maintained during domain growth. We discuss selection and stability of patterns for this mechanism in both 1- and 2-dimensional domains. (Received September 25, 2017)