Title:	Increased dopaminergic neurotransmission results in ethanol dependent sedative behaviors in Caenorhabditis elegans
Authors:	Pandey, Pratima (/jspui/browse?type=author&value=Pandey%2C+Pratima) Singh, Anuradha (/jspui/browse?type=author&value=Singh%2C+Anuradha) Babu, Kavita (/jspui/browse?type=author&value=Babu%2C+Kavita)
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Abstract:	Ethanol is a widely used drug, excessive consumption of which could lead to medical conditions with diverse symptoms. Ethanol abuse causes dysfunction of memory, attention, speech and locomotion across species. Dopamine signaling plays an essential role in ethanol dependent behaviors in animals ranging from C. elegans to humans. We devised an ethanol dependent assay in which mutants in the dopamine autoreceptor, dop-2, displayed a unique sedative locomotory behavior causing the animals to move in circles while dragging the posterior half of their body. Here, we identify the posterior dopaminergic sensory neuron as being essential to modulate this behavior. We further demonstrate that in dop-2 mutants, ethanol exposure increases dopamine secretion and functions in a DVA interneuron dependent manner. DVA releases the neuropeptide NLP-12 that is known to function through cholinergic motor neurons and affect movement. Thus, DOP-2 modulates dopamine levels at the synapse and regulates alcohol induced movement through NLP-12.
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