Translation elongation factor eEF1A exists as two variants in mammals which are 98% similar and 92% identical. Whereas eEF1A1 is almost ubiquitously expressed eEF1A2 shows a very restricted pattern of expression. Wasted is a spontaneous mouse mutation: homozygous wst/wst mice develop normally until shortly after weaning, but then lose muscle bulk, acquire tremors and gait abnormalities and die by four weeks. This mutation has been shown to be a deletion of 15kb upstream of the gene encoding eEF1A2. The reciprocal pattern of expression of eEF1A1 and eEF1A2 in muscle coincides with the timing of the onset of the phenotype in wasted mice. No other gene is present in the wasted deletion, and transgenic studies have shown that the phenotype is due to loss of eEF1A2. We have shown that eEF1A2, but not eEF1A1, is also expressed at high levels in motor neurons in the spinal cord. Wasted mice develop pathological features of motor neuron degeneration and may represent a good model for early onset motor neuron disease. Further transgenic studies are being carried out to establish the relative contributions of muscle and neuronal expression of eEF1A2 to the wasted phenotype. Molecular modelling of the eEF1A1 protein structures reveals differences between the two variants that may be critical for functional differences.