Attention deficit hyperactivity disorder (ADHD) is a common, impairing and persistent disorder affecting children, adolescents and adults. Genetic factors play a substantial role in the etiology of ADHD as is evident in family and twin studies. Despite an active search, few replicable genetic risks have been found and those that have been found do not account for much of the variance in the disorder. Although there are many reasons for this impasse, it is widely hoped that elucidation of the molecular mechanisms underlying quantitative neurocognitive phenotypes associated with ADHD will facilitate discovery of genetic risk factors and will, in addition, advance diagnosis and treatment. Motor response inhibition, as measured in the stop-signal paradigm, is a signature deficit in ADHD and this deficit meets criteria for an endophenotype or genetic risk marker. Research using response inhibition measured in the stop task is beginning to elucidate the genetic architecture of ADHD as will be shown. Precision of genetic hypotheses also depend on our understanding of the neurobiology of response inhibition. Recent fMRI studies using the stop task in ADHD patients identify the neural architecture of response inhibition and begin to locate the deficit in ADHD.