About half of the human genome consists of mobile genetic elements that copy and insert themselves at different loci. The most common of these are Alu elements, which experienced rapid expansion during primate evolution. Recent studies have shown that Alus are a major source of new exons; since these elements have several sites resembling consensus splice sites, insertion of Alus into intronic regions may introduce new exons into existing genes. However aberrant inclusion of Alus can be deleterious to the organism. Using comparative iCLIP studies, RNA-seq and splicing reporter constructs we show how a combination of RNA-binding proteins can control the formation of new Alu exons. We find that recognition of Alu exons through the core splicing factor U2AF65 is suppressed by the abundant nuclear RNA-binding protein hnRNP C. This suppression is mediated through direct competition of hnRNP C and U2AF65 at the 3' splice site. Consistently, specific point mutations that are commonly observed in fully exonized Alus decrease hnRNP C’s ability to compete with U2AF65 binding. Thus, our study reveals a new mechanism for suppressing the expression of cryptic exons and maintaining splicing fidelity. The results have important implications for the evolution of the human genome and disease.