These CGGs Are Making Me Fragile, Mentally That Is…

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Fragile X Syndrome is one of the most common forms of inherited mental retardation. Symptoms include learning disabilities, facial abnormalities, and social instability. The disease is caused by the expansion and hypermethylation of the CGG triplet in the fragile X mental retardation gene 1 (FMR1), leading to a loss of production of the fragile X mental retardation protein (FMRP), which is an RNA-binding protein. The mechanism of expansion and methlyation of the CGG triplet repeat, as well as the roles of FMRP have yet to be identified. Current research has shown that expansion of triplet repeats might be due to polymerase pausing during DNA replication as a result of formation of stable DNA secondary structures, such as hairpin loops and tetrahelices. Hypermethylation has been found to repress transcription either by recruiting histone deacetylase or by blocking transcription factor binding. Researchers have also found several methods for testing which RNAs are likely targeted by FMRP *in vivo*. These methods include mouse and fly models, microarray identification, and in vitro tests. There is no known cure for Fragile X syndrome but further research may help lead to treatments that can correct the FMR1 gene or replace the function of FMRP.

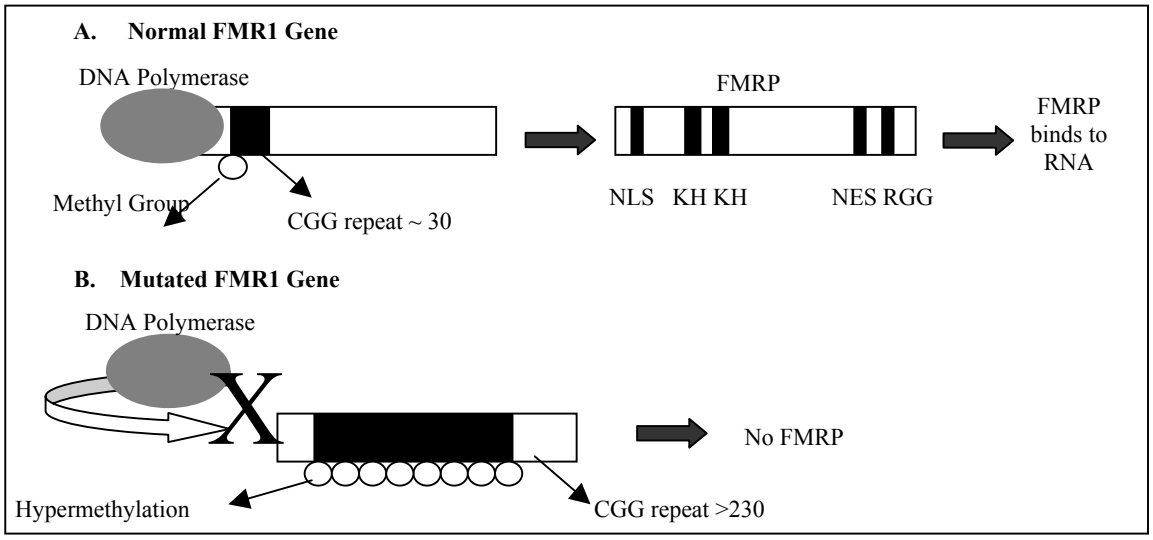

Figure 1: Activities of the FMR1 Gene and FMRP in Normal and Fragile X cells.
A. A normal FMR1 gene is transcribed and translated into FMRP.
B. DNA Polymerase cannot bind to FMR1 due to methylation and expansion. No FMRP is made.