**Claim Evidence Reasoning (C.E.R.) Worksheet Answer Key**

Create a C.E.R. to answer the following driving question. Write the name of your assigned genetic disorder on the line below.

**Is non-homologous end joining or homology-directed repair best suited to curing \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_?**

|  |
| --- |
| **Claim** |
| Your claim should answer the driving question.  Homology-directed repair is best suited to curing sickle cell anemia/cystic fibrosis/Duchenne muscular dystrophy/Leber congenital amaurosis.  Non-homologous end joining is best suited to curing Huntington’s disease. |
| **Evidence** |
| What evidence do you have to support your claim? Include the specific gene sequence and how it is mutated, whether your mutation is dominant or recessive, and how the resulting protein is affected by the mutation.  Sickle cell anemia/cystic fibrosis/Duchenne muscular dystrophy/Leber congenital amaurosis follows a recessive inheritance pattern.  Huntington’s disease follows a dominant inheritance pattern. |
| **Reasoning** |
| Explain **how** your evidence supports your claim. **Why** is your chosen mechanism the best option for your disorder?  Because sickle cell anemia/cystic fibrosis/Duchenne muscular dystrophy/Leber congenital amaurosis follows a recessive inheritance pattern, there is no functioning copy of the gene in affected individuals. To cure the disease, a working copy of the gene must be present in the patient’s cells. Homology-directed repair can edit the gene to correct the mutation, giving the patient a functioning copy of the gene.  Because Huntington’s disease follows a dominant inheritance pattern, an affected individual will usually have one healthy version of the gene and one mutated version of the gene. Non-homologous end joining allows us to turn off the mutated huntingtin gene, while the healthy version of the gene is still functioning in the cells. |