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Copy number variation and mutation BRIAN CLARK, JACOB WEIDNER, KEVIN WABICK, Illinois State University — Until very recently, the standard model of DNA included two genes for each trait. This dated model has given way to a model that includes copies of some genes well in excess of the canonical two. Copy number variations in the human genome play critical roles in causing or aggravating a number of syndromes and diseases while providing increased resistance to others. We explore the role of mutation, crossover, inversion, and reproduction in determining copy number variations in a numerical simulation of a population. The numerical model consists of a population of individuals, where each individual is represented by a single strand of DNA with the same number of genes. Each gene is initially assigned to one of two traits. Fitness of the individual is determined by the two most fit genes for trait one, and trait two genetic material is treated as a reservoir of junk DNA. After a sufficient number of generations, during which the genetic distribution is allowed to reach a steady-state, the mean numberof genes per trait and the copy number variation are recorded. Here, we focus on the role of mutation and compare simulation results to theory.

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