In lab, we were able to study gene mutations an patterns of inheritance using the model organisms, *Caenorgabditis elegans* (commonly referred to as "*C. elegans*"). *C. elegans* is a eukaryotic, multicellular organism that shares cellular and molecular structures with higher organisms and undergoes complex developmental processes, allowing the biological information we gather to be applied to more intricate organisms, such as humans (1). The sex of the *C. elegans* is determined by the ratio of sex chromosomes to autosomes. If the sixth chromosome is an XX combination, it is a hermaphrodite; if the worm possesses a XO combination, it is a male. Hermaphrodites are the most common sex of these worms and have the ability to either self-fertilize or mate with males (2). *C. elegans* worms are often used in genetic research because a single worm can produce about 300 offspring, making it easy to produce many genotypes and phenotypes. This combined with their short life cycle makes them convenient for research.

We chose to observe worms with the mutation ROL 6 and determine whether the mutation was recessive or dominant. We also planned to determine whether it was sex-linked or autosomal. We chose to observe this mutation because we deemed that it would be easy to notice, considering that worms with the ROL 6 mutation move in a circular formation unlike unaffected worms. In order to determine the traits of this mutation, we needed to produce offspring to observe. Knowing that if we only allowed a hermaphrodite to self-fertilize the offspring would be exact replicas, we chose to cross a homozygous ROL 6 mutated hermaphrodite and a homozygous wild-type (unaffected) male. This cross would allow us to determine whether the mutation was dominant or recessive by looking at the ratio of mutant to normal offspring; if the mutation was dominant, 100% of the offspring would be mutated and if it was recessive, 50% would be mutated. If the mutation is sex-linked, all of the males offspring would be mutated and all of the hermaphrodites would be normal.

Recessive/Sex Linked	X^a	X^a
X^A	X^AX^a	X^AX^a
O^A	O^AX^a	O^AX^a

Recessive/Autosomal	X^a	X^a
X^A	X^AX^a	X^AX^a
O^A	O^AX^a	O^AX^a

Dominant/Sex Linked	X^A	X^A
X^a	X^AX^a	X^AX^a
O^a	O^aX^A	O^aX^A

Dominant/Autosomal	X^A	X^A
X^a	X^AX^a	X^AX^a
O^a	O^aX^A	O^aX^A

We knew that by choosing the cross between the homozygous mutated hermaphrodite and the homozygous wild-type male, we would be able to tell if the mutation was dominant or recessive. After placing both *C. elegans* onto a petri dish using a sterilized worm spatula, and allowing them to reproduce for one week, we observed the offspring under a microscope. We expected to see hundreds of offspring in the dish, but unluckily, all of our worms died. We were able to look on with another group to observe what the more typical results should look like. Their dish did indeed contain hundreds of *C. elegans*, so we decided to divide the dish into four quadrants. We chose one quadrant to identify and count the number of males, hermaphrodites, and how many of which were mutated worms. Males and hermaphrodites were distinguished from each other by differentiating the shapes of their tails. Males possess a tail that has a rounded edge with a lip on the end, while hermaphrodites' tails are tapered and are much more skinny. Our results were as follows:

Wild type male	8
ROL 6 male	16
ROL 6 hermaphrodite	24

Because the offspring were not 100% mutated, we immediately know that the ROL 6 mutation is recessive. Looking at the punnett squares for the possibilities of a recessive mutation, the ratio of offspring could either turn out to be 100% unaffected or 50% mutated and 50% unaffected. Our experiment yielded neither of these outcomes, however these punnett squares do not take into consideration the offspring produced by the hermaphrodite through self-fertilization, which would produce mutated hermaphrodites. In order to decide whether the mutation was sex-linked or autosomal, we needed to analyze the gender of the worms and how many of each was mutated. The mutation seems to be sex-linked, considering that there are multiple normal males . Therefore, the mutation must be autosomal.

To prove our theory that the mutation is autosomal recessive, we decided to do another crossing of a wild-type hermaphrodite with a wild-type male. Assuming they are both F_1 offspring from the crossing of the homozygous mutated hermaphrodite and the unaffected male, they would both be heterozygous. Therefore, if we are correct, their offspring would be 75% normal and 25% mutated. We did two crossings to ensure accuracy in case we accidently misidentified a worm.

When we came back to the lab to observe the F_2 offspring produced by our second crossing, we were pleased to see that the offspring were conclusive with the results of our original observations. The ratios were not exact, but both normal and mutated worms were present, proving to us that our initial inference was indeed correct.

Works Cited

1. "*C.elegans* as a Model System." Waksman Student Scholars. 9 April 2013. <http://avery.rutgers.edu/WSSP/StudentScholars/project/introduction/worms.html
>.