Sexually dimorphic behaviors are widespread throughout the animal kingdom. Approaches to understand how these differences in behavior arise have focused primarily on the roles of gonad secreted hormones that infiltrate the nervous system during development to orchestrate sexually dimorphic structural remodeling. But an additional mechanism, controlled by the sex chromosomal content of the nervous system itself, has recently emerged as a contributor to sex differences in behavior. Specifically, the genetic sex of the nervous system is able to autonomously alter functional and structural properties of neural circuits present in both sexes. However, the mechanisms by which this occurs is poorly understood. To better understand how genetic sex modulates shared circuitry to elicit sex differences in behavior, I examined the role of genetic sex in sexually dimorphic attraction to ascaroside sex pheromones in the nematode C. elegans. Previous work demonstrates that hermaphrodite sex pheromone is a complex chemical mixture that can be broadly categorized into two classes of chemicals whose synthesis either requires the enzyme daf-22 (daf-22-dependent) or does not (daf-22-independent). The ascarosides ascr#2, #3, and #8 are daf-22-dependent chemicals and elicit strong attraction in males but weakly repel hermaphrodites. By studying sexually mosaic animals, I discovered that the circuitry eliciting this attraction is present in both sexes but is functionally silent in hermaphrodites. That is, switching the sexual state of shared circuits is sufficient to switch the sexual phenotype of ascaroside attraction behavior. Moreover, I found that sexual state is particularly important in the sensory neuron ADF. Feminizing ADF alone causes a complete loss of male attraction, while masculinizing it is sufficient to generate attraction in hermaphrodites. Consistent with this, ablation of ADF in males eliminates attraction, suggesting that the male state of ADF promotes attraction to ascarosides. Finally, I showed that the inability to detect daf-22-dependent pheromones impairs male mating efficiency. Together, these experiments demonstrate that modulation of sensory function by genetic sex plays a key role in the generation of sexually dimorphic behaviors.