It's the year 1960, and you are a junior plant geneticist with the USDA Agriculture Research Service, a governmental organization tasked with conducting practical research that can quickly help farmers with problems they encounter with their crops. Your specialty is soybean genetics, which is great because soybeans are America's most economically important crop! Recently, farmers have been reporting large losses in yield, which plant pathologists attribute to parasitism of the roots by *Heterodera glycines*, the Soybean Cyst Nematode (SCN). This parasite can survive dormant in fields and re-infect crops even after multiple seasons of rotating out non-hosts. However, farmers have had some luck planting the resistant varieties Peking and PI 88788. Unfortunately, these varieties are less popular with American farmers than the susceptible varieties such as Hill, Lee, and Williams 82, which are better-adapted to the growing conditions of America's Midwest. It is your job to determine the genetic basis of this disease resistance and to help farmers utilize this resistance in the popular varieties of soybeans they prefer to grow.

The symptoms of SCN, including patches of yellowing and stunted growth in a field, as observed in North Dakota. (Photo: Berlin D. Nelson)

Part 1: Heritability

The first step in understanding how to utilize the SCN resistance found in Peking is to determine its heritability.

Use your understanding of Mendelian genetics and the information from Article 1 (Caldwell, *et al.*, Inheritance of resistance to soybean cyst nematode, Heterodera glycines. Agronomy Journal (1960), v.52, p.635-636.) to help you answer the questions below:

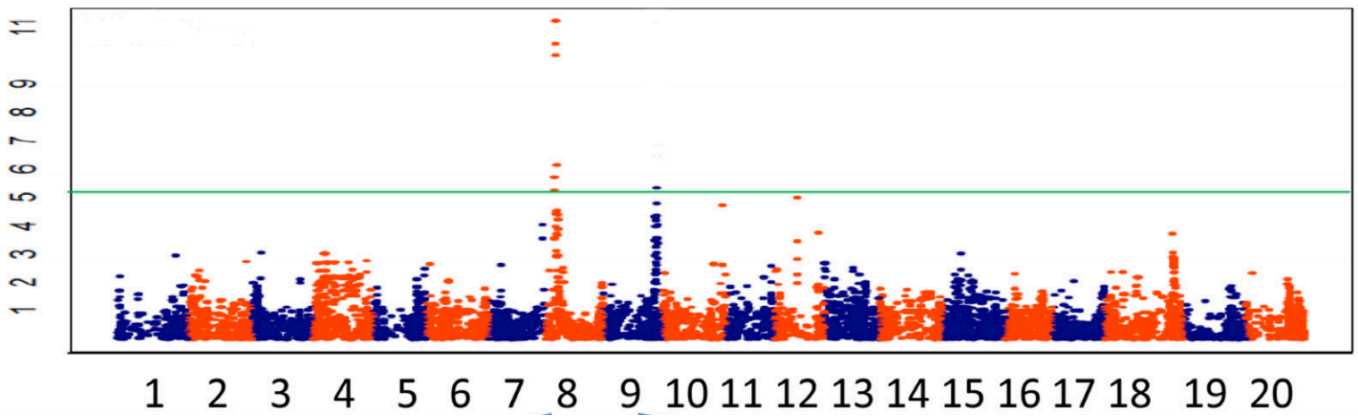
1. What is a test cross?

2. Can you draw a punnet square to explain the heritability observed in Article 1? What kind of trait is resistance to SCN?

Part 2: Mapping Quantitative Traits

It's now the year 1990, and we are poised on the edge of the genomic revolution! New tools have been developed to allow breeders to deliver crops with desirable traits much more quickly than they could when we first began studying SCN. Now that you have determined the heritability of resistance, it is useful to know where in the genome the genes encoding this resistance lie. Your job is to find molecular markers with good linkage to SCN resistance, so that breeders can easily introgress resistance into different soybean varieties.

To map SCN resistance, you create a mapping population of RILs. You then genotype the population and the parents and generate a QTL map based on this population, shown below:



This Manhattan plot shows the whole soybean genome stretched out on the x axis, and the $-\log_{10}P$ (negative logarithm of the P value) of each marker is a dot on the plot. This means that markers with highest association with the phenotype (smallest P values) will plot highest on the plot. A line is drawn across the plot at the P value cut off, which is established based on the number of lines used in the genotyping (n).

You're interested in more finely mapping the most important locus found in your QTL map, which you named Rgh1. You sequence this region in a number of resistant and susceptible lines to look for markers that will be most effective for fine mapping:

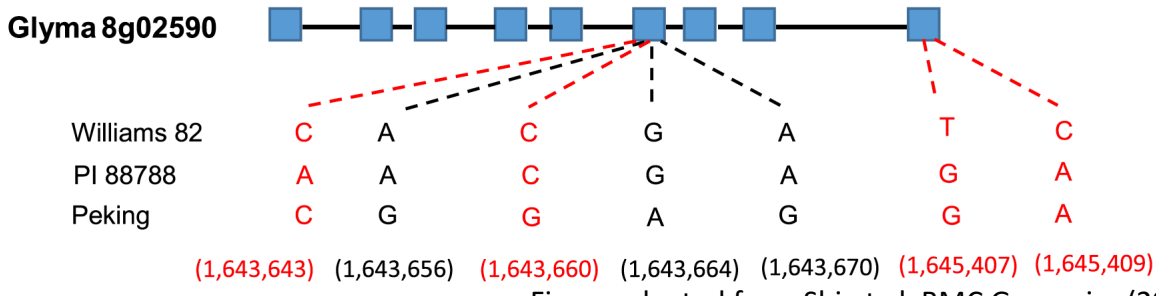


Figure adapted from Shi et al. BMC Genomics (2015) 16:314

Use the data presented here, as well as information from your reading, to answer the questions below:

1. What are the advantages and disadvantages of using a population of RILs to map a trait? Draw a diagram of another potential strategy for mapping.

2. What is the difference between a phenotypic marker and a molecular marker?

3. What types of molecular markers are available to geneticists? Which type are you using here to map SCN resistance?

4. What would be a fast, lower tech way for breeders to utilize the molecular markers you found to be closely linked with SCN resistance for breeding?

5. Based on the data, how many loci were you able to detect as contributing to SCN resistance? Where are these loci? Name them based on the degree of contribution to the quantitative trait (e.g. Rgh1= highest contributing locus). What do you think you could do to find more significant loci?

6. Which specific markers will likely be good tools to fine-map Rgh1, and under what conditions?

Part 3: Molecular Genetics

It's 2008 and you're almost there! Recently, researchers have fine-mapped Rgh1 to a locus containing only three genes! Strangely, it seems that in the whole genome sequences of both resistant PI 88788 and susceptible Williams 82 this locus has nearly 100% sequence similarity.

Questions:

1. What do you hypothesize is the basis for the molecular genetics of SCN resistance conferred by Rgh1? How would you test that hypothesis?

Homework:
What did the authors of Article 2 (Cook *et al.*, Copy number variation of multiple genes at Rhg1 mediates nematode resistance in soybean. Science (2012), v.338, p.1206-9.) find to be the basis of the SCN resistance conferred by Rgh1? How did they determine this?