EWRS Herbicide-Resistance Workshop

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Improving design of spray trials to estimate species propensity to evolve herbicide resistance

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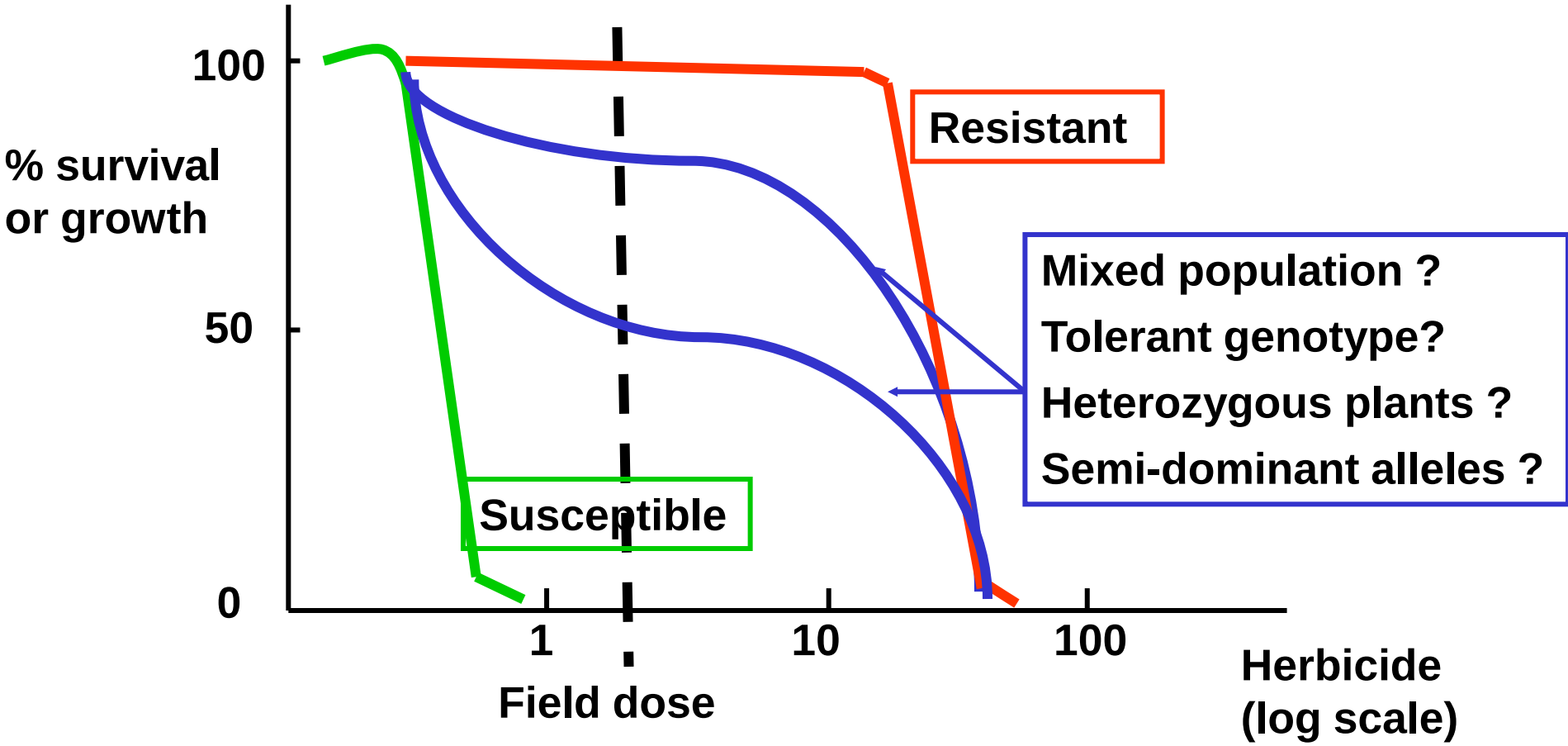
INRA SCIENCE & IMPACT

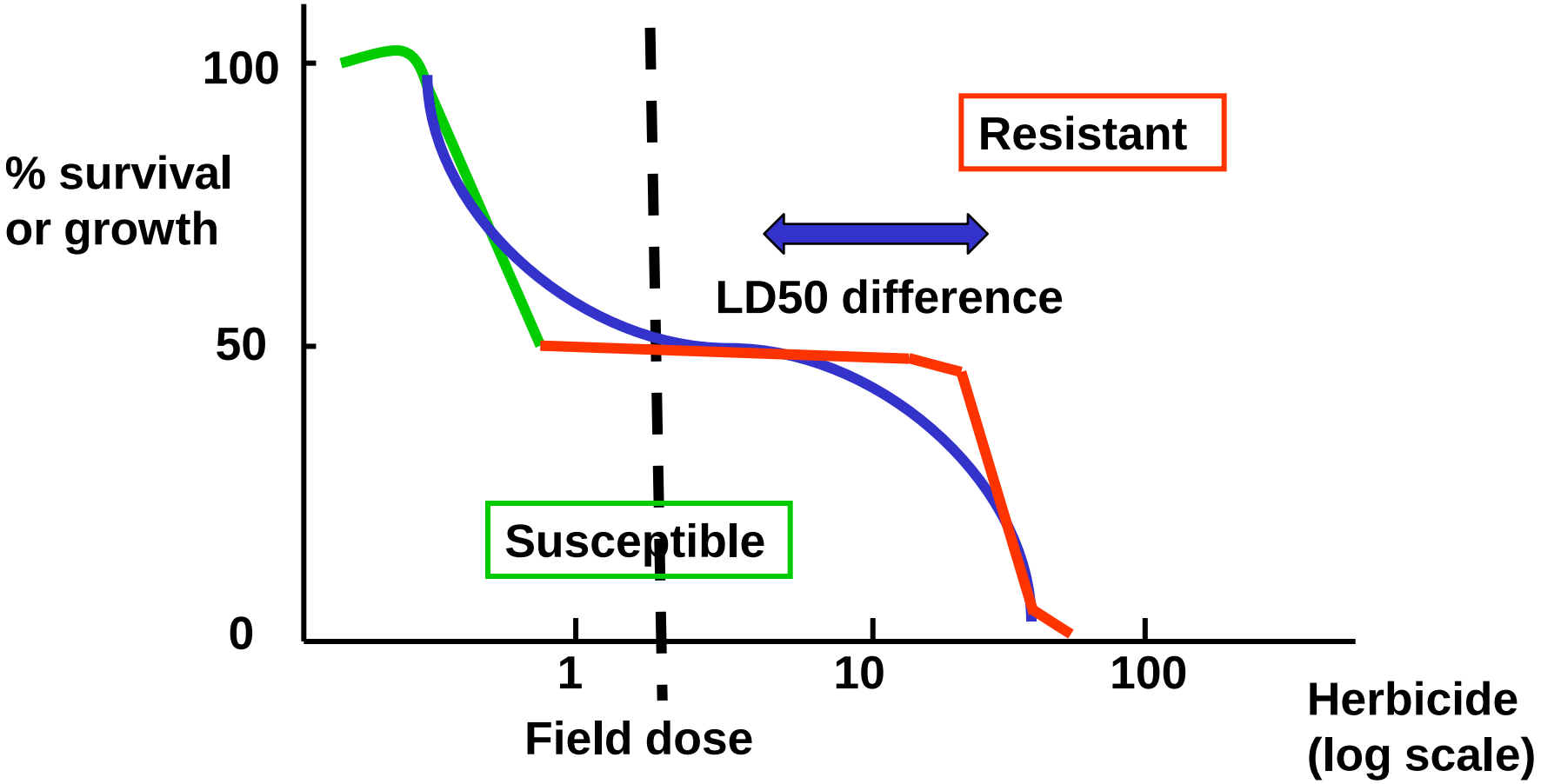
Interpreting the herbicide dose response curve



➔ **Different interpretations according the sample is a natural or a purified population**

Example: the sample is a field population

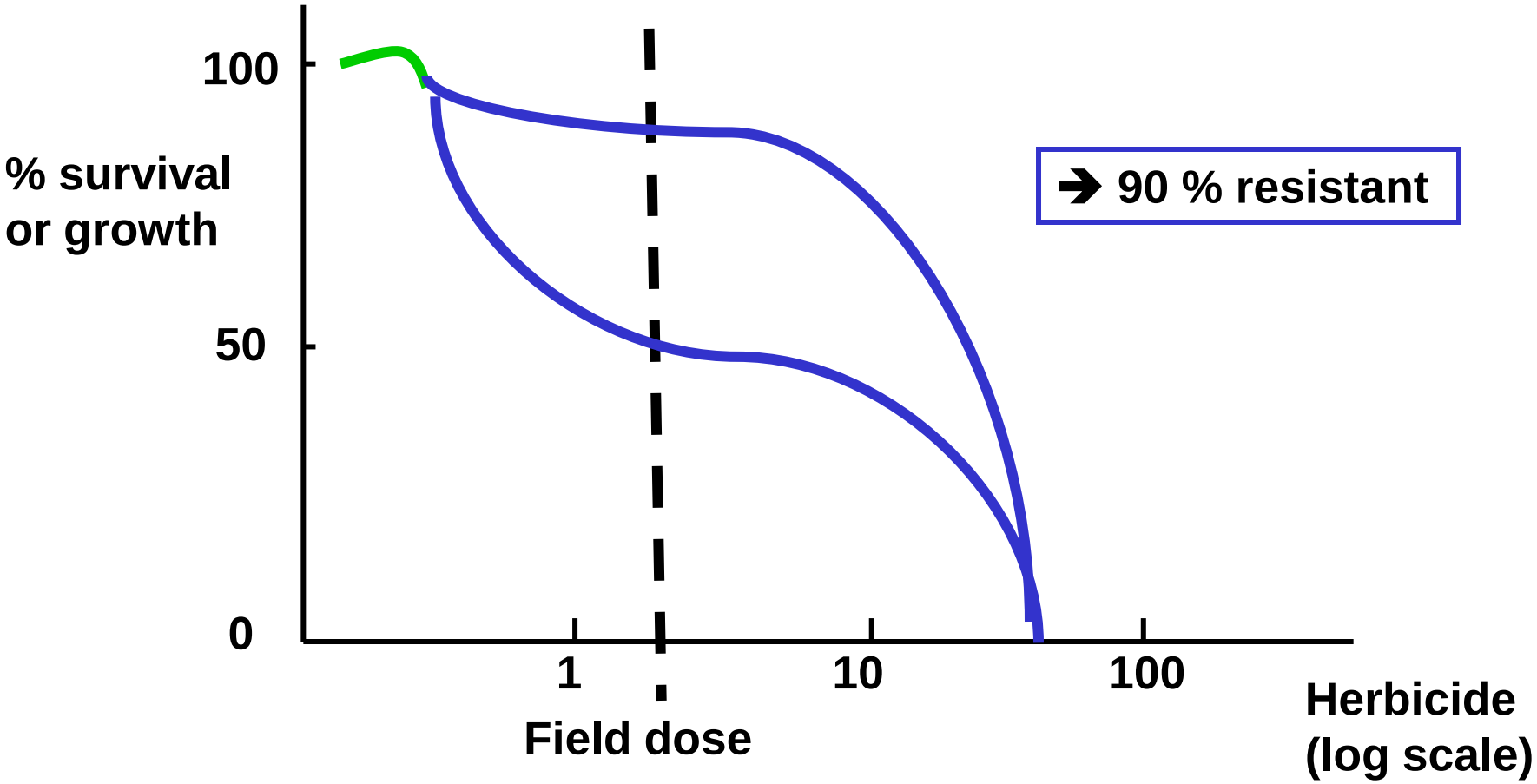
50 % S and 50 % R



➔ LD50 does not correspond to any genotype !

➔This is a photography of a population containing resistant plants that already causes troubles (= resistant population ?)

The mixed population can evolve with time and farmer's practices

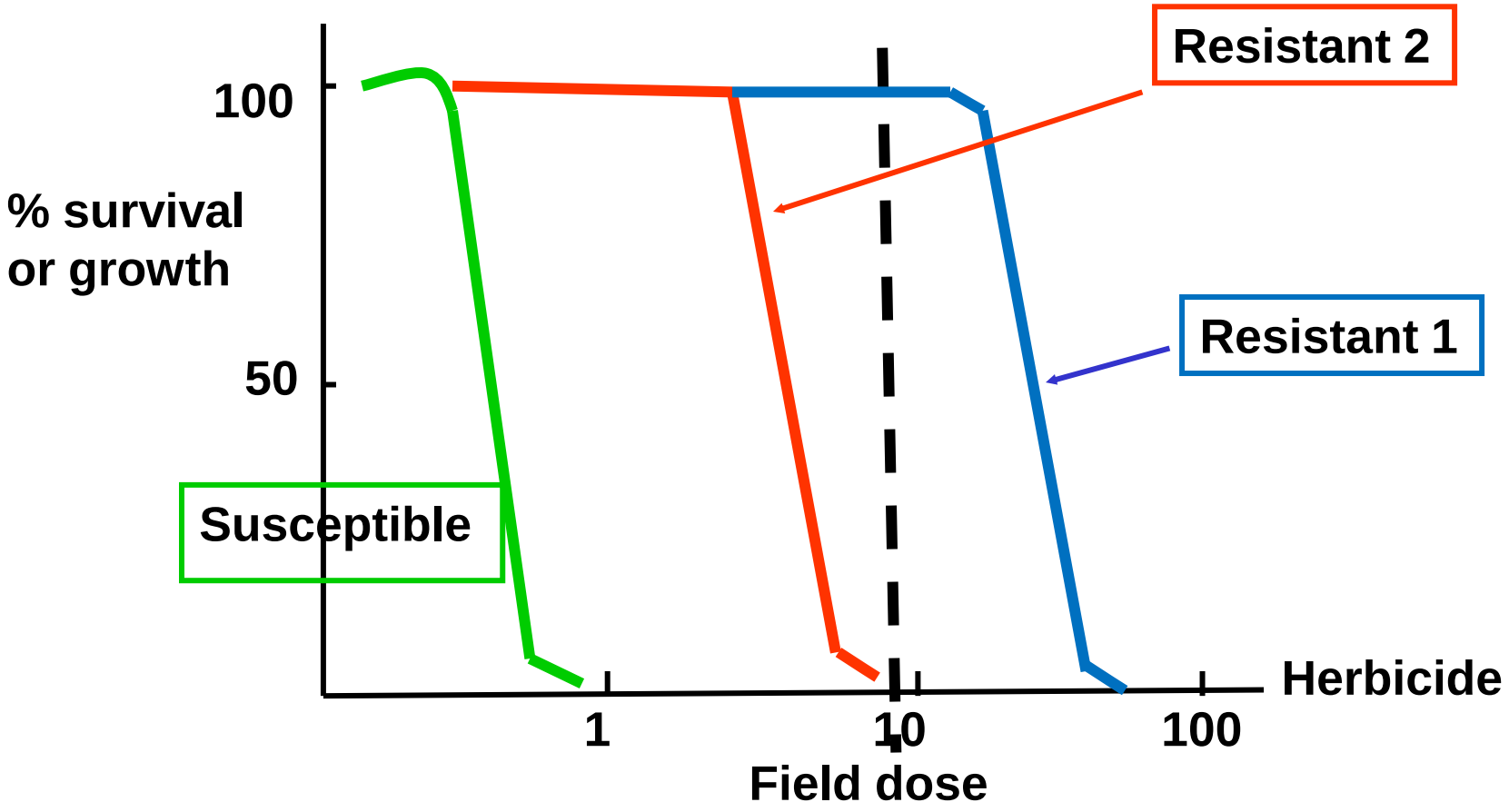


➔ Parameters are soil seed bank, fitness cost, repeated use of the same herbicide, segregation of dominant versus recessive gene

Sampled versus purified populations

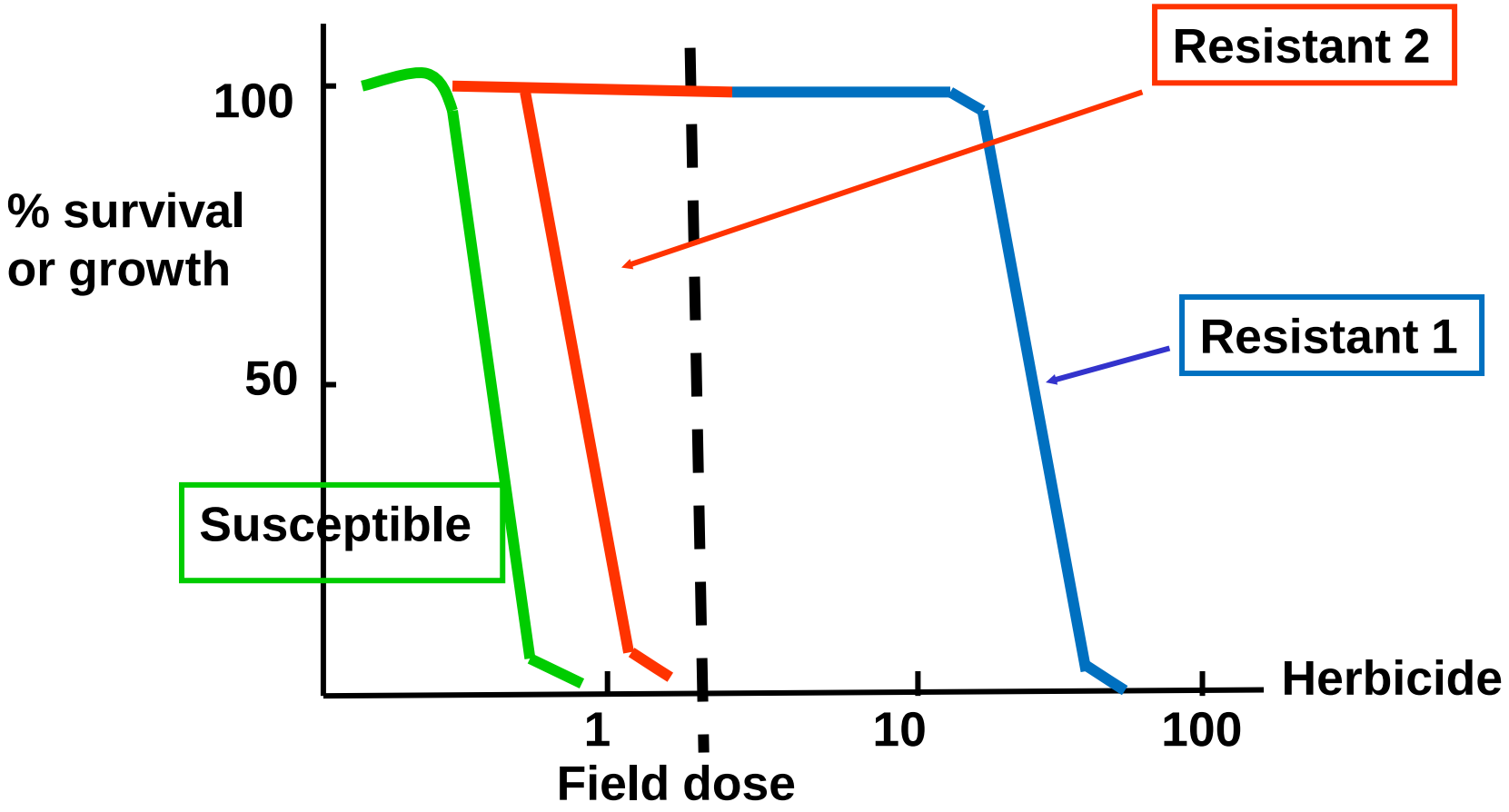
- **In-field or laboratory sampled (« resistant ») seed population always contain susceptible plants or mixture of resistance genes:**
  - **➔ R/S value is a simple indication of the resistance level**
  - **➔ No indication about the mechanism**
  - **➔ No indication about the future and evolutionary potential of the population**
- **Genetic studies allow selecting for purified genotype (crosses, backcrosses, segregation, several generations, clones, molecular markers):**
  - **➔ Accurate resistance level**
  - **➔ Accurate material for studying mechanisms**
  - **➔ Accurate material for fitness study**
  - **➔ But time consuming…**

The data we have = phenotype
= genome-environnement interaction



➔ **Same gene activity but different environment (here herbicide dose, but also temperature, humidity, ligth, plant development and physiology, etc)**

The data we have = phenotype
= genome-environnement interaction



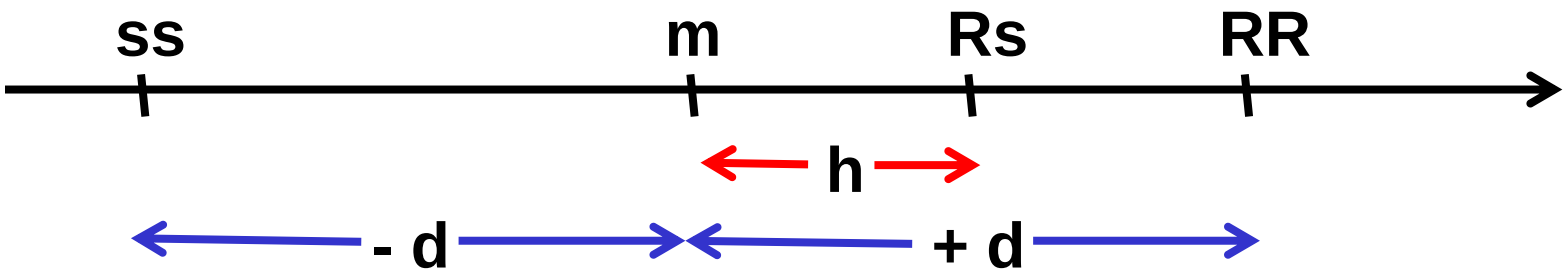
➔ **A modified environment lead to modified gene activity (plant development and physiology, stress, temperature, humidity, ligth,, etc**

How interpreting the data ?
Mendelian versus quantitative genetics

- **Mutations and molecular markers: data are genotypes (only for the marker itself)**
- **Experiments provide quantitative value: data are phenotypes**
- **Resistance is sometime complex with several genes/mechanisms involved: non target-site resistance**

➔ **Quantitative genetics models allow estimating minimum number of gene involved, gene additivity or interaction, genotype-environment interaction.**

Some old-fashion definitions: the additive-dominance model with interactions



- and interactions between genes: i, j and l, homo x homozygote, homo x heterozygote, and hetero x heterozygote;
- and parameters for interaction with and between environments, e and g;
- etc…

➔ and estimate these parameters

The additive-dominance model with allelic interaction

An example with four generations allowing estimates:

- **$m = ½ P_1 + ½ P_2 + 4 F_2 - 2 BC_1 – 2 BC_2$**
- **$[d] = ½ P_1 – ½ P_2$**
- **$[h] = 6 BC_1 + 6 BC_2 – 8 F_2 – F_1 – 1½ P_1 – 1½ P_2$**
- **$[i] = 2 BC_1 + 2 BC_2 – 4 F_2$**
- **$[j] = 2 BC_1 – P_1 – 2 BC_2 + P_2$**
- **$[l] = P_1 + P_2 + 2 F_1 + 4 F_2 – 4 BC_1 – 4 BC_2$**

\+ **their variance,**

\+ **their significance test and test of the model**

And then estimate the number of effective factors:

- **$k = (P_1 – P_2)^2 / 4 V_H$** **(with V_H the heritable variance)**
- **$k = (1/4\ d + 1/8\ h)^2 / V_H$**

How estimating the heritable variance ? Dose-response experiments with susceptible populations

t treatments, f families or populations, n plants each

Source of variation	df.	MS	Expected MS
Treatments	t-1	MS_T	$V_E + nV_G + nfV_T$
Families / populations	t(f-1)	MS_F	$V_E + nV_G$
Plants	tf(n-1)	MS_E	V_E

Broad sense Heritability: $H^2 = V_G / (V_G + V_E)$

H^2 is the genetic variance divided by the phenotypic variance

Dose-response experiments with susceptible populations

If families were structured progeny of crosses (e.g. F_2), additive and dominance variances can be separated ($V_G = V_A + V_{D)}$ and h^2 calculated as:

Narrow sense heritability $h^2 = V_A / (V_G + V_E)$

Speed and range of advance under selection is estimated as:

$$R = h^2S$$

with R the response to selection (difference between population means before and after one generation selection),

and S the selection coefficient (the efficacy of the herbicide)

Example with Fops in susceptible a wild oat population

- 10 seed families (= collected plant per plant)
- 5 blocks, 5 randomized germinated seeds each family (= 25 plants per family)
- Dry weight per plant

Herbicide	Dose	a.i. (g)	Significant groups	H^2
Control			4	0.35
Fluazifop-p-butyl	1/2X	94	3	0.09
Diclofop-methyl	1/4X	200	2	0.24
Fenoxaprop-p-ethyl	1/4X	21	2	0.07

Conclusion

Please be sure considering the genetic status of the plants to extract the very detailed information from your data