Randomised experiments

- Have n experimental units (for measurements);
- For ith unit observe (pre-treatment) covariates X_i; treatment $Z \in \mathcal{Z}$ (usually $Z = (Z_1, \ldots, Z_n)$); (after treatment) outcome Y_i;
- Exposure for unit i is $A_i = A_i(Z) \in \mathcal{A}$ (usually $A_i = Z_i$ and for most of the course $\mathcal{A} = \{0, 1\}$).

Definition. The *treatment assignment mechanism* or *randomisation scheme* refers to the probability distribution

$$\pi(z|x) = \mathbb{P}(Z = z|X = x).$$

Examples.

- Bernoulli trials:

$$\pi(z|x) = \prod_{i=1}^{n} (\pi(X_i))^{Z_i} (1 - \pi(X_i))^{1-Z_i}$$

  (in simple Bernoulli trials $\pi(x_i) = \pi$).

- Sampling without replacement:

$$\pi(z|x) = \begin{cases} \binom{n}{n_1}^{-1} & \text{if } \sum_{i=1}^{n} Z_i = n_1 \\ 0 & \text{otherwise} \end{cases}.$$

- Randomised complete block design: have k kinds of treatment/exposure $\mathcal{A} = \{0, 1 \ldots, k-1\}$ and B blocks of units. Each block has k units. Here $Z = (Z_{ij})_{i \in [B], j \in [k]}$ and

$$\pi(z|x) = \begin{cases} (k!)^{-B} & \text{if } (Z_{ij})_{j \in [k]} \text{ is a permuation of } \{0, \ldots, k-1\} \\ 0 & \text{otherwise} \end{cases}.$$

  For example if $k = 3$ we could have

	1	2	3
1	1	0	2
2	0	2	1
3	0	1	2
⋮	⋮	⋮	⋮
B	0	2	1

.

"Implicit" causal inference

Often, experimental data are analysed using linear regression:

$$Y_i = \alpha + \beta A_i + \gamma^T X_i + \varepsilon_i.$$

- Does $\beta \neq 0$ mean there is a causal effect?
- Or $\mathbb{E}[Y|A = 1] \neq \mathbb{E}[Y|A = 0]$?

Formal theory: Neyman-Rubin causal model

We assume potential outcomes $(Y_i(Z))_{i\in[n],z\in\mathcal{Z}}$. We also assume *consistency of potential outcomes*, that is $Y = (Y_1, \dots, Y_n) = Y(Z) = (Y_1(Z), \dots, Y_n(Z))$, i.e only one potential outcome is observed.

Fundamental problem: causal effect is conceptualised as contrasts of potential outcomes under different z. But by consistency only one potential outcome is observed.

Definition. We say an *exposure mapping* $A : \mathcal{Z} \to A^n$, $z \mapsto (a_1, \dots, a_n)$ is *valid* if

$$Y_i(z) = U_i(z') \text{ for all } i, z, z' \text{ such that } A_i(z) = A_i(z').$$

In this case we write the potential outcome as $Y_i(a)$. When $\mathcal{A} = \{0, 1\}$, we define the *individual treatment effect* $Y_i(1) - Y_i(0)$.

We will almost always assume the identity exposure mapping is valid - but this may not be true in many problems: vaccine studies, experients on social networks, etc.

We may have interference via networks, suppose $G = (V = [n], \Sigma \subseteq [n] \times [n])$. An example of an exposure mapping may be $A_i = \left(Z_i, \sum_{(i,j)\in\Sigma} Z_j\right) \in \{0, 1\} \times \{0, 1, \dots, n-1\}$.

Example (Lady tasting tea)**.** A young lady claimed that she is able to tell whether the tea or milk was added first to a cup. The experiment provides the lady with eight randomly ordered cups of tea - four prepared by pouring milk then tea, four by pouring tea and then milk. The lady attempts to select the four cups prepared by one method or the other, and may compare cups directly against each other as desired. The method employed in the experiment is fully disclosed to the lady.

We write 0 for milk then tea and 1 for tea then milk. The results were

i	$Z_i = A_i$	Y_i	$Y_i(0)$	$Y_1(1)$	$Y_i(1) - Y_i(0)$
1	1	1	?	1	?
2	1	1	?	1	?
3	0	0	0	?	?
4	0	1	0	?	?
5	1	1	?	1	?
6	0	0	0	?	?
7	1	0	?	0	?
8	0	0	0	?	?

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