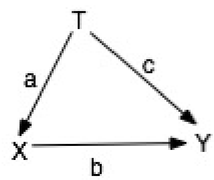
SCM with diff-in-diff design[1]

Difference-in-difference designs (d-i-d) are frequently employed to assess causal effects when there is an exogenous event that causes a potential change in outcome due to a change in perceived causal variable(s). Hence, the hypothesized causal effect occurs in the post intervention period. A simple case is represented by the DAG (directed acyclic graph) below where other than any interaction terms the structural causal model (SCM) is linear (functionally, rather than linear in the parameters) and the numbers on the arcs are the path coefficients. Y is outcome, X is the causal intervention (typically binary), and T is the exogenous intervention (binary).



There is a back-door path into X that connects with Y via T. Accordingly, identification of the causal effect of X on Y requires conditioning on T. The SCM for this DAG is typically (functionally) nonlinear due to the interaction term designed to account for occurrence of the causal effect only in the post intervention period.

$$M:$$

$$T = U_T$$

$$X = aT + U_X$$

$$Y = cT + bX + dX * T + U_Y$$

On the other hand, the intervention model where $X = x$ is

$$M_X:$$

$$T = U_T$$

$$X = x$$

$$Y = cT + bx + dx * T + U_Y$$

[1] SCM and ETT are adapted from Pearl, Glymour, and Jewell, 2016, *Causal Inference in Statistics: A Primer*, Wiley.

ETT for d-i-d

The quantity of interest is the causal effect of X on Y in the post-intervention period where the pre-intervention period serves as a control sample. Difference-in-difference is the expected treatment effect on the treated during the post-intervention period less the expected treatment effect on the treated during the pre-intervention period.

$$d - i - d = ETT\left(T = 1\right) - ETT\left(T = 0\right) = d$$

where

$$\begin{gathered} ETT\left(T = 1\right) = E\left[Y_{X=1} - Y_{X=0} \mid X = 1, T = 1\right] \\ = E\left[cT + b\left(1\right) + d\left(1\right) * T + U_Y \mid X = 1, T = 1\right] \\ -E\left[cT + b\left(0\right) + d\left(0\right) * T + U_Y \mid X = 1, T = 1\right] \\ ETT\left(T = 1\right) = \left\{c + b + d\right\} - \left\{c\right\} = b + d \end{gathered}$$

and

$$\begin{gathered} ETT\left(T = 0\right) = E\left[Y_{X=1} - Y_{X=0} \mid X = 1, T = 0\right] \\ = E\left[cT + b\left(1\right) + d\left(1\right) * T + U_Y \mid X = 1, T = 0\right] \\ -E\left[cT + b\left(0\right) + d\left(0\right) * T + U_Y \mid X = 1, T = 0\right] \\ ETT\left(T = 0\right) = \left\{b\right\} = b \end{gathered}$$

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Example

Consider a simple binary example to illustrate the above case. The joint probability distribution is

$\Pr\left(y, t, x\right)$	$X = 0$	$X = 1$
$T = 0, Y = 0$	0.125	0.1
$T = 0, Y = 1$	0.125	0.1
$T = 1, Y = 0$	0.125	0.125
$T = 1, Y = 1$	0.125	0.175

Marginal and conditional probabilities are

$$\begin{gathered} \Pr\left(Y = 1\right) = 0.525 \\ \Pr\left(T = 1\right) = 0.55 \\ \Pr\left(X = 1\right) = 0.5 \\ \Pr\left(X = 1 \mid T = 1\right) = 0.545 \\ \Pr\left(Y = 1 \mid T = 1\right) = 0.545 \end{gathered}$$

Conditional expectations are

$$\begin{gathered} E\left[Y \mid X = 1, T = 1\right] = 0.5833 \\ E\left[Y \mid X = 0, T = 1\right] = 0.5 \end{gathered}$$

The structural causal model is

$$Y = 0.5 + 0X + 0T + 0.0833X * T + U_Y$$

and counterfactual, causal analysis leading to the expected treatment effect on the treated agrees with observation.

$$ETT\left(T=1\right)=0.0833$$

$$E\left[Y\mid X=1,T=1\right]-\left[Y\mid X=0,T=1\right]=0.0833$$
$$ETT\left(T=0\right)=0.0$$
$$E\left[Y\mid X=1,T=0\right]-\left[Y\mid X=0,T=0\right]=0.0$$
$$d-i-d=ETT\left(T=1\right)-ETT\left(T=0\right)=0.0833$$

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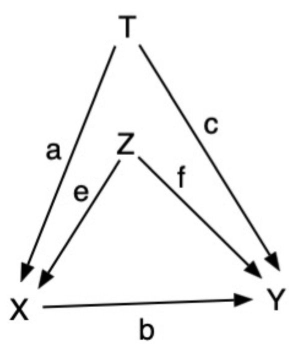
ETT for d-i-d with confounders

Frequently, the setting is not so clean but plagued with potential confounders denoted Z. One variation is a simple extension of the above where the causal effect of X on Y has a back-door through Z as well as T.



Below is one SCM for this setting.

$$M:$$

$$T=U_T$$
$$Z=U_Z$$
$$X=aT+eZ+U_X$$
$$Y=cT+fZ+bX+gX*T+hZ*T+kX*Z+dX*Z*T+U_Y$$

The causal effect involves intervention to set $X=x$.

$$M_X:$$

$$T=U_T$$
$$Z=U_Z$$

$$X = x$$
$$Y = cT + fZ + bx + gx * T + hZ * T + kx * Z + d * x * Z * T + U_Y$$

For this SCM, the d-i-d (post intervention) expected treatment effect on the treated involves the same estimand as above.

$$ETT\,(T=1) = E\left[Y_1 \mid X=1, T=1\right] - E\left[Y_0 \mid X=1, T=1\right]$$
$$\left\{ \begin{array}{c} c + fE\left[Z \mid X=1, T=1\right] + b + g + (h+k)\,E\left[Z \mid X=1, T=1\right] \\ +dE\left[Z \mid X=1, T=1\right] + E\left[U_Y \mid X=1, T=1\right] \end{array} \right\}$$
$$-\left\{c + (f+h)\,E\left[Z \mid X=1, T=1\right] + E\left[U_Y \mid X=1, T=1\right]\right\}$$
$$= b + g + kE\left[Z \mid X=1, T=1\right] + dE\left[Z \mid X=1, T=1\right]$$
$$ETT\,(T=0) = E\left[Y_1 \mid X=1, T=0\right] - E\left[Y_0 \mid X=1, T=0\right]$$
$$\left\{fE\left[Z \mid X=1, T=0\right] + b + E\left[U_Y \mid X=1, T=0\right] + kE\left[Z \mid X=1, T=0\right]\right\}$$
$$-\left\{fE\left[Z \mid X=1, T=0\right] + E\left[U_Y \mid X=1, T=0\right]\right\} = b + kE\left[Z \mid X=1, T=0\right]$$
$$d - i - d = ETT\,(T=1) - ETT(T=0)$$
$$= g + k\left\{E\left[Z \mid X=1, T=1\right] - E\left[Z \mid X=1, T=0\right]\right\} + dE\left[Z \mid X=1, T=1\right]$$

Example

A binary example illustrates the impact of the confounder Z where the treatment effect is homogeneous. The joint distribution is

$\Pr(y,t,x,z)$	$X=0, Z=0$	$X=0, Z=1$	$X=1, Z=0$	$X=1, Z=1$
$T=0, Y=0$	0.15025	0.0798	0.00075	0.0005
$T=0, Y=1$	0.15025	0.1197	0.00175	0.002
$T=1, Y=0$	0.031125	0.075	0.117375	0.0042
$T=1, Y=1$	0.031125	0.1125	0.117375	0.0063

Marginal and conditional probabilities are

$$\Pr(Y=1) = 0.541$$
$$\Pr(T=1) = 0.495$$
$$\Pr(X=1) = 0.25025$$
$$\Pr(X=1 \mid T=1) = 0.495454545$$
$$\Pr(X=1 \mid Z=1) = 0.0325$$
$$\Pr(Y=1 \mid T=1) = 0.54$$
$$\Pr(Y=1 \mid Z=1) = 0.60125$$
$$\Pr(Z=1) = \Pr(Z=1 \mid T) = 0.4$$
$$\Pr(Z=1 \mid X=1, T=1) = 0.042813456$$
$$\Pr(Z=1 \mid Y=1, T=1) = 0.827154047$$

Conditional expectations are

$$E\left[Y \mid X=1, T=1\right] = 0.504281346$$
$$E\left[Y \mid X=0, T=1\right] = 0.575075075$$
$$E\left[Y \mid X=1, T=0\right] = 0.75$$
$$E\left[Y \mid X=0, T=0\right] = 0.5399$$

The structural causal model is

$$Y = 0.5 + 0.2X + 0.1Z - 0.2X * T + U_Y$$

and the expected treatment effect on the treated is

$$ETT\,(T = 1) - ETT\,(T = 0) = 0.0 - 0.2 = -0.2$$

while the observed differences are $(0.504281346 - 0.575075075) - (0.75 - 0.5399) =$ -$0.07079373 - 0.2101 =$ -0.28089373.

Observable heterogeneity

The above SCM accommodates observable heterogeneity and is illustrated in the following example.

Example Heterogeneity as a function of the confounder Z is illustrated via the following binary example. The joint distribution is

$\Pr(y, t, x, z)$	$X = 0, Z = 0$	$X = 0, Z = 1$	$X = 1, Z = 0$	$X = 1, Z = 1$
$T = 0, Y = 0$	0.15025	0.0798	0.00075	0.001
$T = 0, Y = 1$	0.15025	0.1197	0.00175	0.0015
$T = 1, Y = 0$	0.031	0.075	0.0705	0.0021
$T = 1, Y = 1$	0.031	0.1125	0.1645	0.0084

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Marginal and conditional probabilities are

$$\begin{gathered}
\Pr(Y = 1) = 0.5896 \\
\Pr(T = 1) = 0.495 \\
\Pr(X = 1) = 0.2505 \\
\Pr(X = 1 \mid T = 1) = 0.495959596 \\
\Pr(X = 1 \mid Z = 1) = 0.0325 \\
\Pr(Y = 1 \mid T = 1) = 0.639191919 \\
\Pr(Y = 1 \mid Z = 1) = 0.60525 \\
\Pr(Z = 1) = \Pr(Z = 1 \mid T) = 0.4 \\
\Pr(Z = 1 \mid X = 1, T = 1) = 0.042769857 \\
\Pr(Z = 1 \mid Y = 1, T = 1) = 0.842508711
\end{gathered}$$

Conditional expectations are

$$\begin{gathered}
E[Y \mid X = 1, T = 1] = 0.704276986 \\
E[Y \mid X = 0, T = 1] = 0.575150301 \\
E[Y \mid X = 1, T = 0] = 0.65 \\
E[Y \mid X = 0, T = 0] = 0.5399
\end{gathered}$$

The structural causal model is

$$Y = 0.5 + 0.2x + 0.1Z - 0.2x * Z + 0.2x * Z * T + U_Y$$

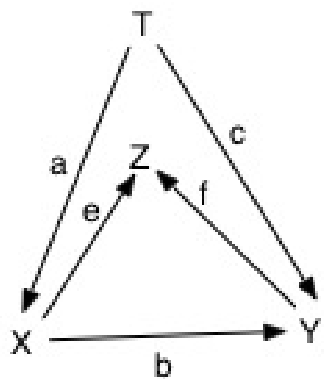
and the expected treatment effect on the treated is (the first line is $d - i - d$ from the data generating process while $d - i - d$ is recovered from the structural model in the second line)

$$d - i - d = ETT\,(T = 1) - ETT.\,(T = 0) = 0.2 - 0.1 = 0.1$$
$$d - i - d = -0.2 * (0.\,042769857 - 0.5) + 0.2 * 0.\,042769857 = 0.1$$

The observed differences are $(0.\,704276986 - 0.\,575150301) - (0.65 - 0.5399) = 0.\,129126685 - 0.\,1101 = 0.\,019026685$.

Collider

Rather than the above confounding back-door, suppose Z is a collider.



Adjusting by Z is confounding while omitting the collider identifies the causal effect of X on Y. The causal model is the same as the first (unconfounded) example.

$$Y = bx + cT + dx * T + U_Y$$

and

$$d - i - d = ETT\,(T = 1) - ETT\,(T = 0) = d$$

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Example

The above case is illustrated with the following binary example. The joint distribution is

$\Pr(y, t, x, z)$	$X = 0, Y = 0$	$X = 0, Y = 1$	$X = 1, Y = 0$	$X = 1, Y = 1$
$T = 0, Z = 0$	0.0432	0.1512	0.0432	0.0672
$T = 0, Z = 1$	0.0192	0.0432	0.0168	0.1568
$T = 1, Z = 0$	0.0648	0.1008	0.1008	0.0288
$T = 1, Z = 1$	0.0288	0.0288	0.0392	0.0672

$d - i - d = ETT\,(T = 1) - ETT\,(T = 0) = -0.1739 - 0.03172305 =$ -0.2055885

The unconfounded model above estimates

$$Y = 0.7570 + 0.0317\,x - 0.1764\,T - 0.2056x * T$$

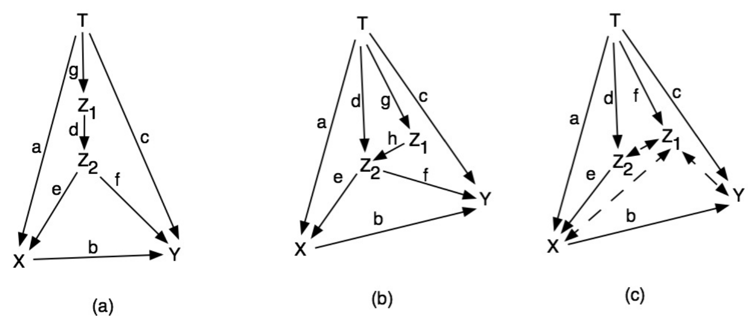
while the collider-confounded model accommodating heterogeneity estimates

$$Y = 0.7778 - 0.1681x - 0.1691T + 0.0855Z$$
$$-0.2174x * T - 0.0232Z * T + 0.3800X * Z + 0.1381x * Z * T$$

and incorrectly estimates the effect to be $-0.2174+0.3800*(0.4508-0.6113)+0.1381*0.4508=-0.2161$ which is equivalent to the bias induced from erroneous adjustment by the collider.

Nonconfounders

Now, we consider some DAGs in which at least some of the potential confounders are nonconfounding. Such variables may play a role in diagnosing the DAG via conditional independence tests of the data but play no direct role in identifying the causal effect and therefore no role in the experimental design. Importantly, colliders (Z_1 in DAG (c)) are not only nonconfounding but conditioning on them confounds the causal effect via Berkson's paradox.



In DAGs (a) and (b) back-door paths (into X connecting Y) are blocked by T and Z_2. Z_1 is conditionally independent of both X and Y given T and Z_2. Hence, Z_1 plays no direct role in identifying the causal effect of X on Y but it may play an indirect role in developing and diagnosing the DAG via conditional independence tests.

In DAG (c), the back-door path (into X connecting Y) is blocked by T, Z_2 is a nonconfounder, and importantly, Z_1 is a collider.[2] The causal effect of X on Y is identified by conditioning on T alone but conditioning on Z_1 opens a back-door path into X connecting Y confounding identification of the causal effect. In fact, conditioning on a collider like Z_1 typically biases every parameter in the model.

Conditional independence tests

Discovery and diagnostic testing of the DAGs is facilitated by conditional independence tests. In DAG (a) conditional independence tests include the following.

$$X, Y \perp Z_1 \mid T, Z_2$$

$$T \perp Z_2 \mid Z_1$$

$$T \sim\perp Z_2 \mid \varnothing$$

[2]The two-headed dashed arcs represent dependence between two observables through a set of unobservables.

where $\perp$ refers to independence and $\sim\perp$ refers to not independent. The first condition simply indicates paths from either X or Y to Z_1 are blocked by T and Z_2. The second condition reflects the idea that the direct path between T and Z_2 is blocked by Z_1 while back-door paths are blocked by X and Y which are colliders with respect to T and Z_2. If the direction of causality is reversed between Z_1 and Z_2, the latter two conditional independence conditions are reversed as Z_1 becomes a collider with the reversal and conditioning on a collider opens an otherwise blocked path.

DAG (b) is similar to DAG (a) except that T is a parent to both Z_1 and Z_2 with the same base conditional independence test.

$$X, Y \perp Z_1 \mid T, Z_2$$

If there were a direct path from Z_1 to Y the above test would fail for Y but the back-door path into X connecting Y is still blocked by T and Z_2.

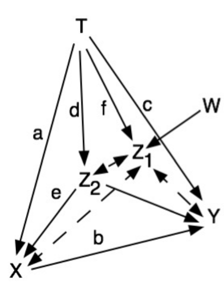
As discussed above, DAG (c) is more problematic as Z_1 is a collider. An analyst might be tempted to inappropriately treat it as a confounder by conditioning on it. Conditional independence tests include

$$Y \perp Z_2 \mid T, X$$

$$Y \sim\perp Z_2 \mid T, X, Z_1$$

The two conditions indicate that Z_1 is a collider with respect to Y and Z_2 — the key concern.

Suppose there is a direct path from Z_2 to Y, then the above tests fail (see DAG (c') below). However, if the analyst is able to measure a variable, say W, pointing into Z_1, then $W \perp X, Y, T, Z_2 \mid \varnothing$ but $W \sim\perp X, Y, T, Z_2 \mid Z_1$. This would be strong confirmation of Z_1 as a collider. While W is not employed directly in the empirical design to infer the causal effect of X on Y, W would nevertheless provide a useful diagnostic role in the DAG construction or, in other words, discovery of the causal structure supporting the empirical design.



(c')

Appendix

Counterfactual analysis leads to expected treatment effect on treated (ETT).

$$ETT = E[Y_1 \mid D = 1] - E[Y_0 \mid D = 1]$$

Iterated expectations/law of total probability implies

$$E[Y_x] = E[Y_x \mid D = 1] \Pr(D = 1) + E[Y_x \mid D = 0] \Pr(D = 0)$$

Consistency axiom says if desired action is observed then action is simply the observational probability

$$E[Y_1 \mid D = 1] = E[Y \mid D = 1]$$

Hence,

$$E[Y_1] = E[Y \mid D = 1] \Pr(D = 1) + E[Y_1 \mid D = 0] \Pr(D = 0)$$

or

$$E[Y_0] = E[Y_0 \mid D = 1] \Pr(D = 1) + E[Y \mid D = 0] \Pr(D = 0)$$

Rewriting the latter gives

$$E[Y_0 \mid D = 1] = \frac{E[Y_0] - E[Y \mid D = 0] \Pr(D = 0)}{\Pr(D = 1)}$$

All but $E[Y_0]$ is observed but this can be identified experimentally, or perhaps, by back-door adjustment from observational data.

$$\begin{aligned} E[Y_0] &= E[Y \mid do(D = 0)] \\ &= \sum_z E[Y \mid D = 0, Z = z] \Pr(Z = z) \end{aligned}$$

or the propensity score frame of the back-door adjustment

$$\begin{aligned} \Pr(Y = y \mid do(D = 0)) &= \sum_z \Pr(Y = y \mid D = 0, Z = z) \Pr(Z = z) \\ &= \sum_z \frac{\Pr(Y = y, D = 0, Z = z)}{\Pr(D = 0, Z = z)} \Pr(Z = z) \\ &= \sum_z \frac{\Pr(Y = y, D = 0, Z = z)}{\Pr(D = 0, Z = z) / \Pr(Z = z)} \\ &= \sum_z \frac{\Pr(Y = y, D = 0, Z = z)}{\Pr(D = 0 \mid Z = z)} \end{aligned}$$

Putting everything together we have

$$ETT = E[Y \mid D = 1] - \frac{E[Y_0] - E[Y \mid D = 0] \Pr(D = 0)}{\Pr(D = 1)}$$