Psychology 434 Mid-Term Test Answers

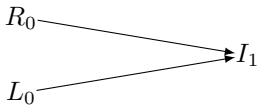
Part 1: Multiple Choice Answer Key

1. b
2. b
3. b
4. a
5. a
6. b
7. c
8. d
9. d
10. c
11. a
12. b
13. a and d
14. c
15. b
16. a
17. b
18. c
19. d
20. c

Part 2: Model Answers

1. Religiosity, life satisfaction, and income

This finding could be misleading if income is a collider. Suppose religiosity affects income and life satisfaction also affects income, but religiosity does not itself lower life satisfaction. Conditioning on income then opens a non-causal path and can induce a spurious negative association between religiosity and life satisfaction.



The key point is that the negative association appears after conditioning, not because there is a real negative causal effect. This is collider bias.

2. Internal and external validity

Internal validity concerns whether a study identifies the causal effect of an exposure on an outcome without bias from uncontrolled confounding, collider adjustment, or other design problems. External validity concerns whether that effect generalises to other people, settings, or times.

For example, a study of social media use and self-esteem could have high internal validity if it measures and adjusts for the right confounders. It could still have weak external validity if the sample is drawn only from one university or one cultural setting and the effect differs elsewhere.

3. Workflow for studying social media and self-esteem

A sensible workflow begins by defining the exposure, outcome, population, and time order. For example, let exposure be daily social media use at time t_1 and let outcome be self-esteem at time t_2. Then draw a causal diagram, identify plausible confounders such as age, socioeconomic status, prior mental health, and baseline self-esteem, and decide what needs to be measured before exposure.

After that, measure the variables in a way that preserves temporal order, assess whether the adjustment set is sufficient, and estimate the causal effect using a method that matches the design. Finally, check assumptions about consistency, exchangeability, positivity, and measurement quality, and interpret the estimate as causal only if those assumptions remain plausible.

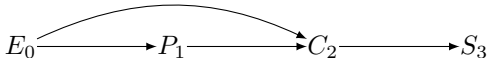
4. Proxy rule

The proxy rule says that conditioning on a descendant is akin to conditioning on its parent. This can help when the true confounder is unmeasured but a descendant carries some information about it. For example, if childhood cultural environment is unmeasured, then childhood religion or country of birth may partly proxy that common cause and reduce bias.

The same logic can introduce bias if the parent is a collider or mediator. If wealth is a collider of marriage and happiness, then conditioning on home ownership, as a descendant of wealth, can open the same non-causal path and create spurious association.

5. Personality and life satisfaction

One reasonable diagram is shown below.



To estimate the total effect of personality on later life satisfaction, investigators must measure variables that block backdoor paths into personality and life satisfaction. In this example, early childhood experiences matter because they shape personality and later career choice. Investigators should also be

clear about the causal question, because conditioning on career choice would block part of the effect of personality on life satisfaction if career is a mediator.

6. Exercise, longevity, and cardiovascular health

If cardiovascular health lies on the causal pathway from exercise to longevity, then controlling for it can bias the total effect estimate. A simple diagram is:

$$E_0 \longrightarrow C_1 \longrightarrow L_2$$

To estimate the causal effect correctly, investigators must measure confounders of exercise and longevity, such as age, baseline health, smoking, or socioeconomic position. They should then decide whether they want the total effect of exercise or only a direct effect not operating through cardiovascular health. If the target is the total effect, they should not condition on the mediator.

7. Social media use and depression at three time points

A causal effect cannot be identified from a variable list alone. Investigators must first specify the exposure, outcome, and timing. They then need a diagram over the three waves showing which variables are pre-exposure confounders, which are mediators, and which may be time-varying confounders affected by prior exposure.

A practical workflow is to define the treatment regime, draw the longitudinal DAG, measure all common causes that precede each treatment decision, and then use a longitudinal causal method such as g-computation, inverse probability weighting, or the parametric g-formula. Investigators should avoid conditioning mechanically on post-exposure variables such as later online friends or later screen time unless the scientific target requires it and the method handles that structure correctly.

8. D-separation

D-separation is the graphical rule that tells us whether information flows between variables, given a set of conditioned variables. If a set of variables blocks every path between exposure and outcome, then those variables d-separate the two nodes and imply conditional independence in the graph.

For example, in the fork $A \leftarrow C \rightarrow B$, the path is open unless we condition on C. Once we condition on C, the path is blocked and $A \coprod B \mid C$. This matters for causal inference because it helps us identify which variables must be conditioned on to block backdoor paths and which variables should not be conditioned on because they are mediators or colliders.

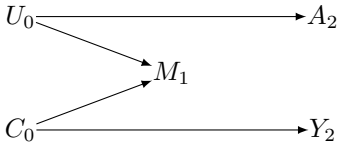
9. Collider bias

A collider is a variable caused by two otherwise independent variables. In the structure $A \rightarrow C \leftarrow B$, the variables A and B are marginally independent. Once we condition on C, they become associated because fixing the common effect makes information about one parent informative about the other.

An example is publication bias. Suppose trustworthiness and newsworthiness both affect whether a study is selected for publication. Among published studies only, low trustworthiness can appear associated with high newsworthiness because selection has conditioned on a collider.

10. M-bias

M-bias is a more complex form of collider bias. One standard structure is:



There is no open backdoor path from A_2 to Y_2 until we condition on the collider M_1. Once we do that, we open the path $A_2 \leftarrow U_0 \rightarrow M_1 \leftarrow C_0 \rightarrow Y_2$. Researchers can introduce M-bias when they adjust for variables simply because they are measured or improve model fit, rather than because the DAG shows they are valid adjustment variables.

11. Selection bias and transportability

Selection bias arises when inclusion in the analysed sample depends on variables related to exposure and outcome. Transportability asks whether a causal effect estimated in one setting carries over to another setting. The two are linked because a selected sample can distort the distribution of effect modifiers and confounders.

In cross-cultural research this matters because participation mechanisms often differ across settings. If one cultural group is overrepresented and the exposure effect varies by culture, then even a well-estimated sample effect may fail to transport to the target population.

12. Average treatment effect

The average treatment effect is $E[Y(1) - Y(0)]$. It is the mean difference between the potential outcome under treatment and the potential outcome under control across the population of interest.

It relates to individual causal effects by averaging them. The individual effect for person i is $Y_i(1) - Y_i(0)$, but we cannot observe both values for the same person. The ATE is often the focus because it is identifiable under the standard assumptions, it is estimable from population data, and it is directly relevant for many policy and scientific questions.

13. Positivity

Positivity requires that each treatment level has non-zero probability within every relevant confounder stratum. Formally, for all l with positive probability, $0 < P(A = a \mid L = l) < 1$.

In cross-cultural research this can fail when some exposures are rare or absent in particular populations, when certain combinations of culture and treatment are structurally impossible, or when data become sparse after stratifying by many confounders. Without positivity, causal inference requires extrapolation beyond the observed data and becomes much less credible.

14. Individual effects and CATEs

A true individual causal effect is $Y_i(1) - Y_i(0)$. It is defined for one person, but it is not directly observable because we never see both counterfactual outcomes for the same individual.

The conditional average treatment effect is $E[Y(1) - Y(0) \mid X = x]$. It is an average for a subgroup defined by observed characteristics. We can estimate the latter because we can compare treated and untreated people within strata of measured covariates, but we cannot recover the exact unobservable counterfactual contrast for a single person.

Part 3: Conceptual Question

The regression criteria capture part of the causal story, but the modern workflow begins earlier and proceeds in a stricter sequence. First we define a causal estimand. That means stating a contrast between at least two interventions to which an entire population, or a well-defined subpopulation, could in principle be exposed. In potential-outcomes language, we do not begin with a regression coefficient. We begin with a contrast such as the average treatment effect, $\mathbb{E}[Y(1) - Y(0)]$, or in Week 6 a subgroup contrast such as the conditional average treatment effect, $\mathbb{E}[Y(1) - Y(0) \mid X = x]$. This step forces us to define the intervention, the comparison condition, the target population, the outcome, and time zero.

Only after the estimand is clear do we move to identification. Here the three regression criteria overlap partly with the potential-outcomes framework, but they are not enough on their own. Temporal order maps onto the requirement that causes precede effects, but identification also requires consistency, exchangeability, and positivity. Consistency requires a well-defined intervention, so that $Y(a)$ refers to something coherent. Exchangeability requires that treated and untreated units be comparable, at least after conditioning on pre-treatment covariates. Positivity requires that both sides of the intervention contrast occur with non-zero probability in the strata needed for comparison. DAGs help here because they do not define the estimand, but they do help us assess whether exchangeability is plausible, which variables belong in L, and whether conditioning decisions are defensible.

This is where the first regression criterion becomes especially weak. The claim that A and Y must be correlated is not a general causal requirement. A causal effect can exist even when the marginal association is near zero, for example when effects differ across subgroups and cancel in the aggregate, when confounding masks the effect, or when measurement error attenuates the observed association. Conversely, a strong association can easily be non-causal if it is produced by confounding, selection

bias, or conditioning on a collider. So correlation is neither sufficient nor always necessary as a practical screening rule for causation.

After identification comes estimation. Week 5 makes the point that design comes before estimation. Once we have stated the estimand and argued that the identification assumptions are plausible, we then choose an estimator that matches the design and the data, such as standardisation, inverse probability weighting, or regression adjustment. Week 6 extends this logic by distinguishing the causal estimand from the statistical estimand. A regression interaction term is just a model parameter. It does not by itself establish causal interaction or effect modification unless the underlying causal estimand and identification assumptions have already been made clear.

So the three regression criteria are not sufficient to establish causality in practice. What needs clarification is the intervention contrast, the target population, the timing of treatment and outcome, the identification assumptions that connect the estimand to observed data, and the estimation procedure used once those assumptions are defended. A better summary is: first state the causal question as an intervention contrast, then assess identification through consistency, exchangeability, and positivity, and only then estimate the contrast from observed data. Without that workflow, the three criteria remain too vague to justify a causal claim.