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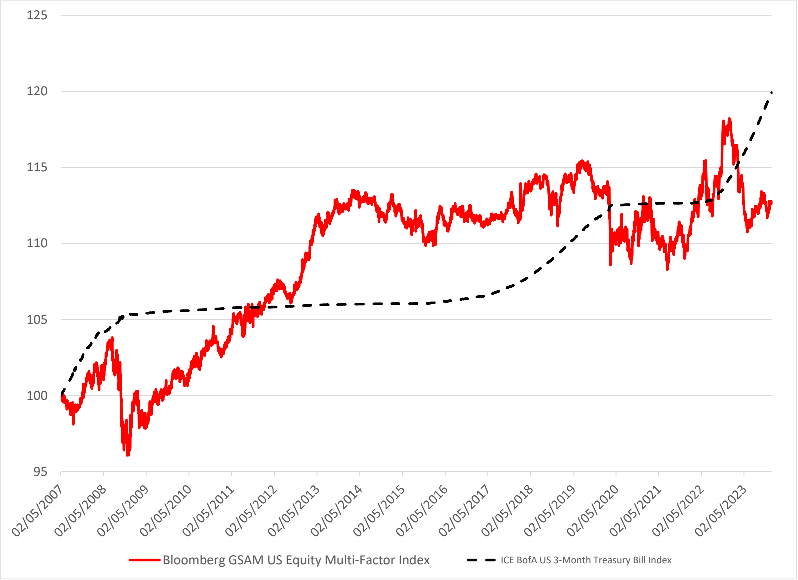
ADIA ¦ Lab

Can Factor Investing Become Scientific?

Marcos López de Prado

Performance of Factor Investing

- Bloomberg – Goldman Sachs Asset Management US Equity Multi-Factor Index (BBG code: BGSUSEMF <Index>)
  - It tracks the long/short performance of the momentum, value, quality, and low-risk factors in U.S. stocks
  - Annualized Sharpe ratio: -0.08 (t-stat=-0.33, *p*-value=0.63)
  - Average annualized return has been -0.30%
- This performance does not include:
  - transaction costs
  - market impact of order execution
  - cost of borrowing stocks for shorting positions
  - management and incentive fees



Before including transaction costs, borrowing costs, and fees, factor investing strategies deliver an annual return of 0.72%, thus underperforming 3-Month Treasury Bills.

Seminar's Objective

- Every student of statistics learns that correlation does not imply causation
  - Association is an observational property
  - Causation is an interventional concept
- Causality plays a fundamental role in the scientific method
  - Scientific theories are falsifiable statements of the form "X causes Y through mechanism M"
- Factor investing models remain at a pre-scientific stage
  - Authors have failed to formulate falsifiable theories
- The "factor zoo" is a prime example of rampant spuriosity in investing:
  - **Type-A: Statistical flukes**
  - **Type-B: Non-causal association**
- This seminar proposes ways to solve the **replication crisis** that afflicts the factor investing literature
  - To read the full manuscript, visit: http://ssrn.com/abstract_id=4205613

My Background & Experience

Science

BERKELEY LAB

ADIA ⁞ Lab

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Industry

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GUGGENHEIM

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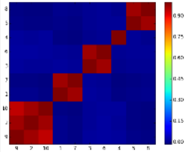
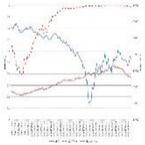
Innovations

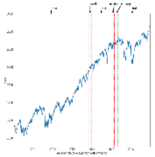


HRP|NCO



VPIN|OEH

Corrective AI



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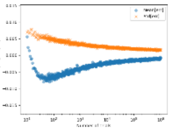
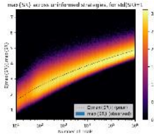


False Strat Theorem

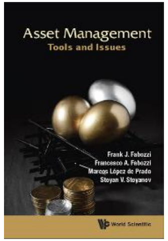


PSR|DSR

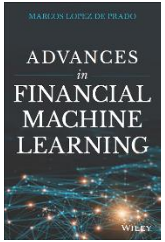
Publications

Cambridge Elements
Quantitative Finance
Causal Factor Investing
Marcos M. López de Prado

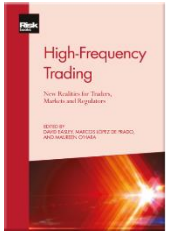
Cambridge Elements
Quantitative Finance
Machine Learning for Asset Managers
Marcos M. López de Prado



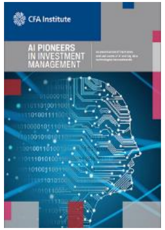
Asset Management
Tools and Issues
Frank J. Fabozzi
Francesco A. Fabozzi
Marcos López de Prado
Stoyan V. Stoyanov
World Scientific



MARCOS LOPEZ DE PRADO
ADVANCES *in* FINANCIAL MACHINE LEARNING
WILEY



Risk Books
High-Frequency Trading
New Realities for Traders, Markets and Regulators
Edited by David Easley, Marcos Lopez de Prado, and Maureen O'Hara



CFA Institute
AI Pioneers in Investment Management

Govt Policy

UNITED STATES CONGRESS

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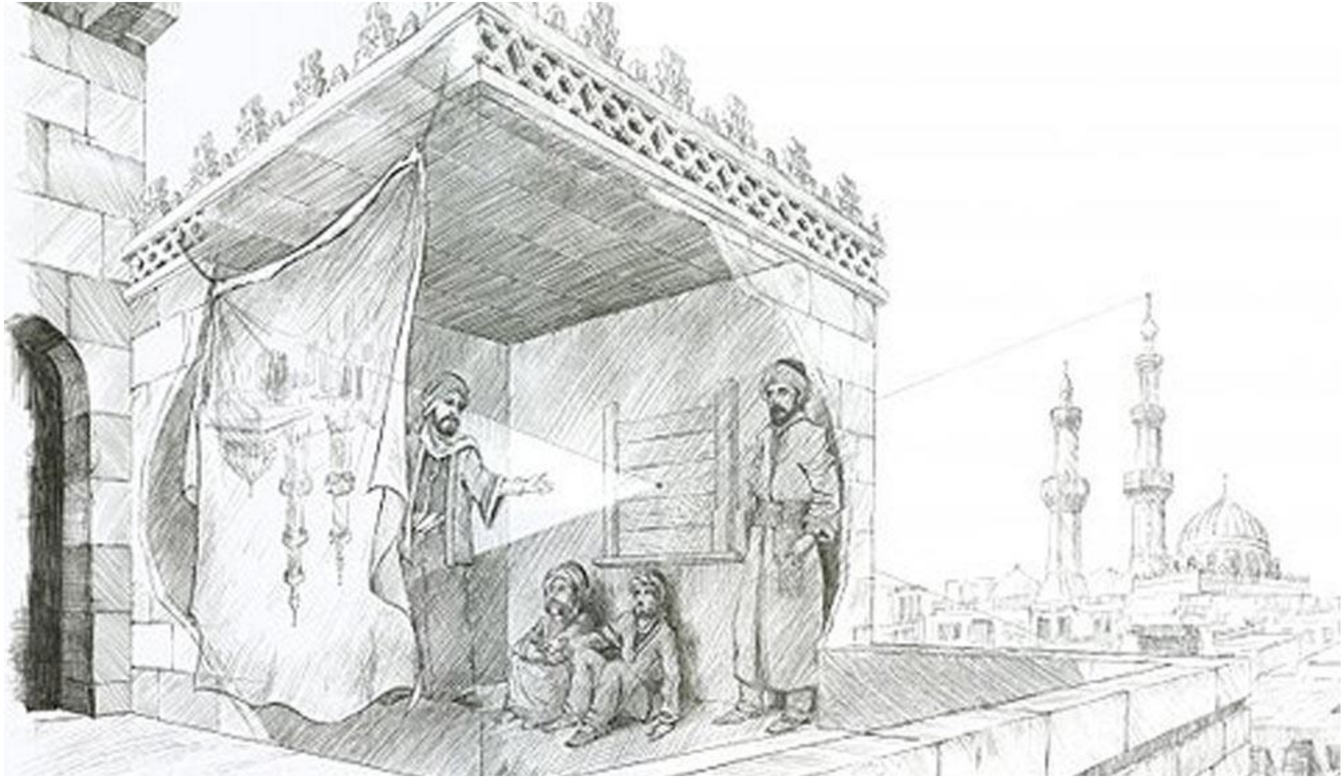
Why Study Cause and Effect?

“Happy the man, who,
studying Nature’s laws,
thro’ known effects can
trace the secret cause”

The Second Book of the Georgics
Publius Vergilius Maro, “Virgil” (70 – 19 BC)

Around the year 1011 that Arab mathematician Hasan Ibn Al Haytham (965 - 1040) proposed a scientific method for deducing causal mechanisms.



David Hume defined a cause-effect (causal) relation as that “where, if the first object had not been, the second never had existed.”

An Enquiry concerning Human Understanding. Sec. VII. (1748)
David Hume (1711 - 1776)

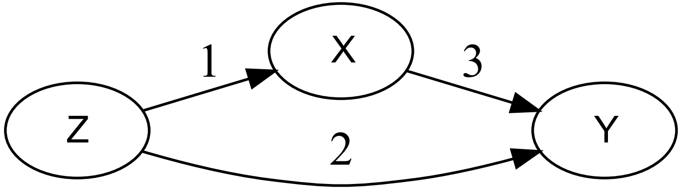
The Three Stages of the Scientific Method

Stage	Statement	Example
Phenomenological (induction)	X is associated with Y	Smoking is associated with lung cancer
Theoretical (abduction)	X causes Y through mechanism M	Smoking causes lung cancer through chemicals that mutate the DNA of lung cells, inducing uncontrolled cell growth
Falsification Refutation (deduction)	Refutation attempts: • X does not cause Y • X causes Y, but not through mechanism M	• Controlled experiments, e.g. animal lab studies • Natural experiments, e.g. regression discontinuity

Association vs. Causation

Paths

- A **data-generating process** can be represented as a directed acyclic graph (DAG)
  - Nodes are variables
  - Arrows indicate the direction of dependence
- A **path** is a sequence of arrows and nodes that connect two variables X and Y, regardless of the direction of causation
- A **directed path** is a path where all arrows point in the same direction
- In a directed path that starts in X and ends in Y
  - X is an **ancestor** of Y, and
  - Y is a **descendant** of X



A DAG is a directed graph with no directed cycles.

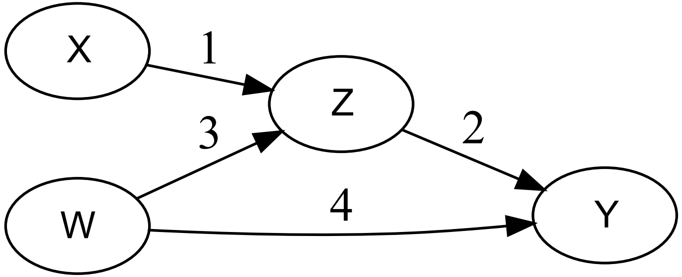
In the DAG above, there are two paths between X and Y:

a) A directed path: $X \rightarrow Y$
b) A non-directed path: $X \leftarrow Z \rightarrow Y$

In the DAG above, X is a descendant of Z, and Y a descendant of X and Z.

Blocked Paths

- In a DAG with three variables $\{X, Y, Z\}$, a variable Z is
  - a **confounder** when the causal relationships include a structure $X \leftarrow Z \rightarrow Y$
  - a **collider** when the causal relationships are reversed, i.e. $X \rightarrow Z \leftarrow Y$
  - a **mediator** when the causal relationships include a structure $X \rightarrow Z \rightarrow Y$



In the above DAG:

- W is a confounder to Z and Y
- Z is a collider to X and W
- Z is a mediator between X and Y

The path $X \rightarrow Z \leftarrow W \rightarrow Y$ is blocked by Z.

The only unblocked path between X and Y is the causal path, $X \rightarrow Z \rightarrow Y$.

A path between X and Y is **blocked** if either:

a) the path traverses a collider, and the researcher has not conditioned on that collider or its descendants; or

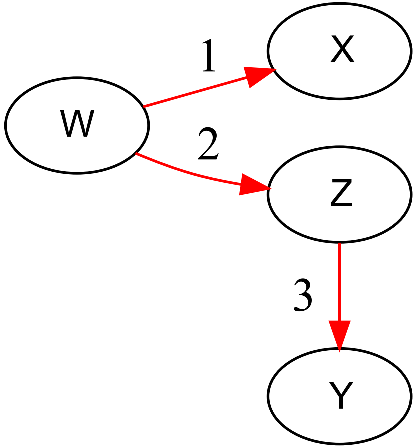
b) the researcher conditions on a variable in the path between X and Y, where the conditioned variable is not a collider

What is Association?

- **Association** flows along an unblocked path between X and Y
  - Association is *symmetric* because paths do not follow the direction of causation
- Probabilistically, two variables X and Y are associated when knowing the value of one conveys information about the value of the other

$$\exists \boldsymbol{x}, \boldsymbol{y} | \boldsymbol{P}[\boldsymbol{Y} = \boldsymbol{y} | \boldsymbol{X} = \boldsymbol{x}] \neq \boldsymbol{P}[\boldsymbol{Y} = \boldsymbol{y}]$$

- Statistical association is merely an observational statement on the joint distribution of probability
  - $P[Y = y | X = x]$ does not measure the *effect* of X on Y

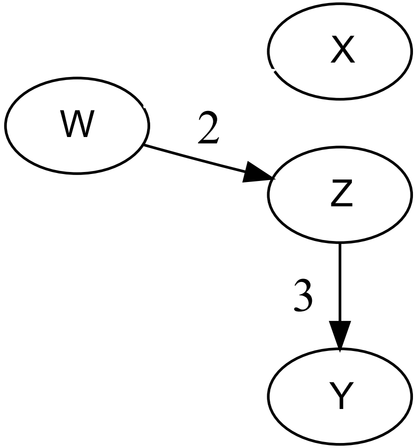


Weather (W) influences ice cream sales (X) and the number of swimmers (Z), hence the number of drownings (Y).

There is no directed path between X and Y, however X and Y are associated (red-dashed undirected edge), because of the unblocked path $X \leftarrow W \rightarrow Z \rightarrow Y$.

What is Causation?

- **Causal association** flows along an unblocked *directed* path that starts in treatment X and ends in outcome Y, denoted the causal path
- Let $do[X = x]$ represent the do-operator on X
  - This is an intervention that sets the value of X to x, hence X is not influenced by any other variable
- **Definition: X causes Y iff $P\left[Y|do[X]\right] > P[Y]$**
- Association implies causation only if all non-causal paths are blocked
- Causality is
  - an interventional (beyond observational) concept
  - asymmetric (directional)
  - sequential: X happens first, and then Y adapts



A do-operation on X removes arrow (1), because X is no longer a function of W, while keeping all other things equal ("ceteris paribus"). As a result, there is no unblocked path between X and Y, and $P\left[Y|do[X]\right] = P[Y]$.

Blocking Non-Causal Paths

Interventional Studies

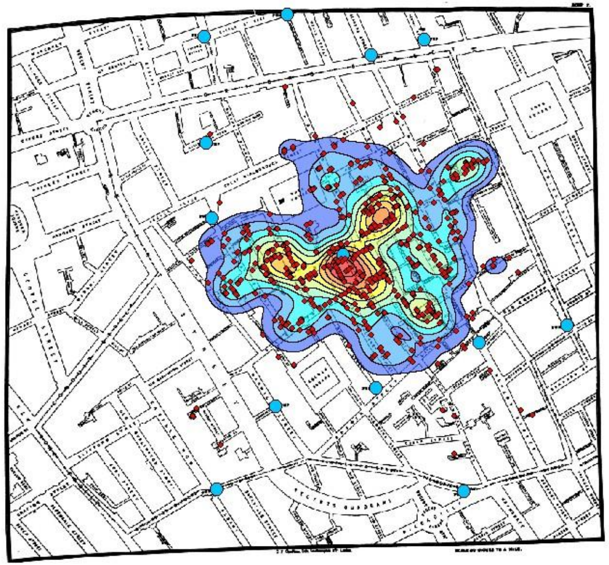
- In a **controlled experiment**, scientists assess causality by observing the effect on Y of changing the values of X, while keeping constant all other variables
  - E.g., Ohm's law of current, Newton's law of gravitation, etc.
- When some of the variables are not under direct experimental control, scientists may execute a **randomized controlled trial** (RCT)
  - E.g., Effectiveness of Pfizer's COVID-19 vaccine
- Under random assignment, subjects in the treatment group ($X = x_1$) are assumed to be indistinguishable from subjects in the control group ($X = x_0$)
  - Thanks to this assumption, the difference in outcomes can be attributed to the treatment

In the 1930s, Ronald Fisher popularized randomized experiments as a way to de-confound variables.

The first published RCT appeared in 1948. Today, well-blinded RCTs are considered the gold standard in experimental research.

Natural Experiments

- Sometimes interventional studies are not possible, because they are unfeasible, unethical, or prohibitively expensive
- In a natural experiment, subjects are assigned to the treatment and control groups determined randomly by Nature or by other factors not controlled by scientists
- Examples of natural experiments include
  - **Regression discontinuity design (RDD)**: When treatment and control groups are comparable in everything but the slight difference in the assignment variable, attributed to noise
  - **Crossover studies (COS)**: When the effect of confounders does not change per subject over time
  - **Difference-in-differences studies (DID)**: When factors other than the treatment influence the outcome over time



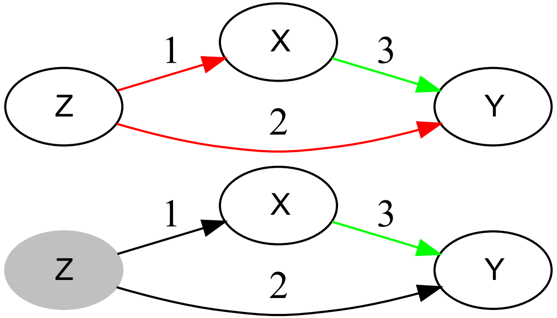
In 1854, Dr. John Snow found that exposure to contaminated water causes cholera. Sick and healthy neighbors of London's Soho district were comparable in all respects, except by their use of different water pumps.

Simulated Intervention: Backdoor Adjustment

- Under some conditions, we can simulate an intervention
- A **backdoor path** between X and Y is an unblocked non-causal path that connects those two variables
- A set of variables S satisfies the **backdoor criterion** if the following two conditions are true:
  - conditioning on S blocks all backdoor paths between X and Y
  - S does not contain any descendants of X
- Then, S is a sufficient adjustment set, and the causal effect of X on Y can be estimated as:

$$P[Y=y|do[X=x]] = \sum_{s} P[Y=y|X=x, S=s]P[S=s]$$

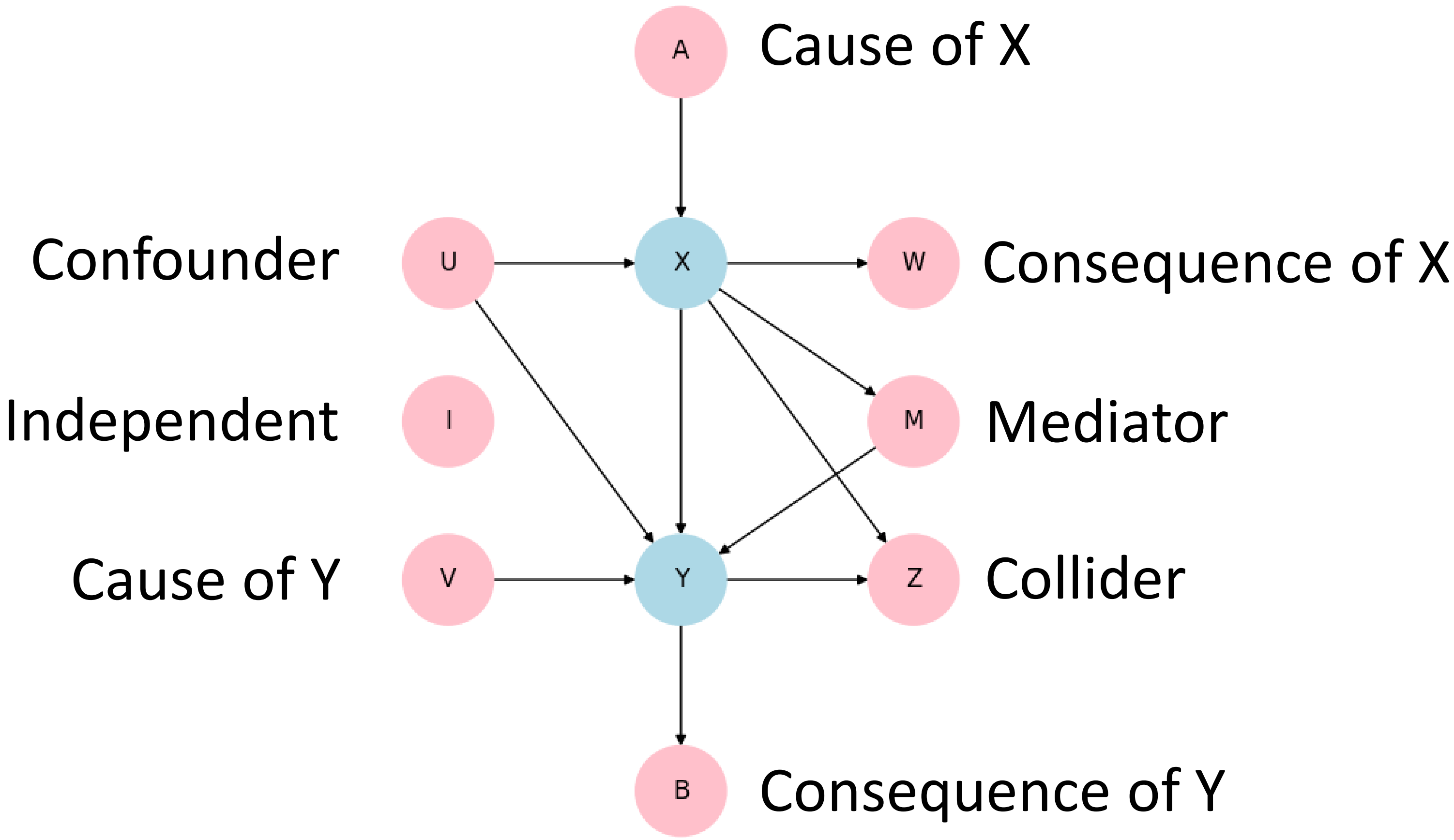
- Examples of other adjustments: Front-door, IV, etc.



Conditioning on confounder Z (shaded node) blocks the path $X \leftarrow Z \rightarrow Y$, leaving the causal path $X \rightarrow Y$ as the only unblocked path.

Under those circumstances, association does imply causation, and we can simulate the outcome of a do-operation through conditional probabilities.

Classification: Public



Cause of X

Confounder

Consequence of X

Independent

Mediator

Cause of Y

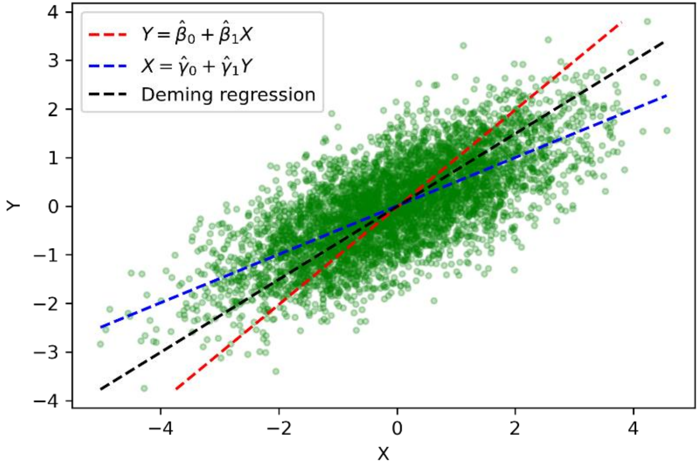
Collider

Consequence of Y

Causality in Econometrics

Authors Mistake Causation for Association

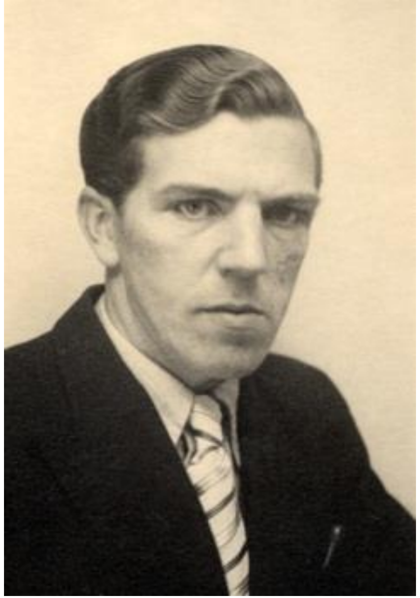
- On the joint distribution of (X, Y), a researcher can fit the linear model $Y_t = \beta_0 + \beta_1 X_t + \varepsilon_t$
- Alternatively, one could fit $X_t = \gamma_0 + \gamma_1 Y_t + \zeta_t$
- In general, for least-squares (LS) estimates:
  - $\hat{\gamma}_0 \neq -\hat{\beta}_0/\hat{\beta}_1, \hat{\gamma}_1 \neq 1/\hat{\beta}_1$, and $\hat{\zeta} \neq -\hat{\varepsilon}/\hat{\beta}_1$
- The reason for this asymmetry is, each model defines the error as the portion of the *effect* that cannot be adjudicated to the chosen *cause*
  - $Y_t = \beta_0 + \beta_1 X_t + \varepsilon_t$ implies a causal graph $X \rightarrow Y$
  - $X_t = \gamma_0 + \gamma_1 Y_t + \zeta_t$ implies a causal graph $Y \rightarrow X$
- Econometric models rely on LS estimators, hence implying *causal* relationships, not associational relationships



By computing LS estimates on a particular specification, an econometrician injects extra-statistical information consistent with a particular causal graph. Alternatively, econometricians could have used a Deming (or orthogonal) regression, a type of errors-in-variables model that attributes errors to both X and Y (a non-causal association).

Authors Misunderstand the Meaning of β

- For the LS estimate to be unbiased ($E\left[\hat{\beta}|X\right]=\beta$), it must occur that $E[\varepsilon|X]=0$ (**exogeneity condition**)
- Econometricians typically address this requirement by defining $\varepsilon \equiv Y - E[Y|X]$, hence $E[Y|X]=X\beta$
  - β receives a distributional interpretation, as the slope of a line
  - This associational interpretation of ε is inconsistent with the causal meaning of LS
- The correct (causal) interpretation of ε is "all causes of Y that are uncorrelated to X"
  - This is a consequence of random assignment (e.g., in an RCT)
  - In purely observational studies, exogeneity is contingent on correct model specification
- Then, the correct meaning of β is: $E\left[Y|\boldsymbol{do}[\boldsymbol{X}]\right]=X\beta$



Economist Trygve Haavelmo was among the first to recognize that β has causal meaning in Economics. Unfortunately, his point was ignored.

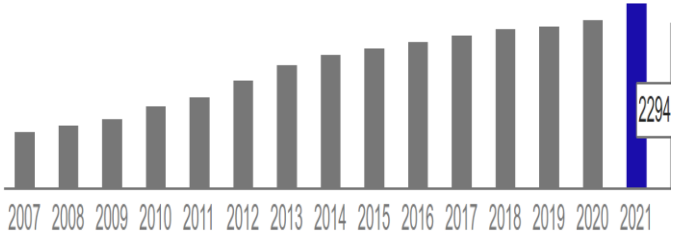
Authors Mistake Association for Causation

- For stationary $\{X_t\}$ and $\{Y_t\}$, Granger [1969] proposed an econometric test for (linear) causality

$$Y_t = \beta_0 + \sum_{i=p}^{I} \beta_i X_{t-p} + \sum_{j=1}^{J} \gamma_j Y_{t-j} + \varepsilon_t$$

- According to Granger, X causes Y iif at least one of the estimated $\{\hat{\beta}_i\}$ is statistically significant
  - This approach was later expanded to multivariate systems, in the form of a VAR specification
- Granger causality is a misnomer
  - For example, if X and Y are caused by Z (a confounder), Granger's test will still falsely conclude that X causes Y
- The test itself is susceptible to selection bias
  - The specification search requires multiple testing

Number of citations > 31,037



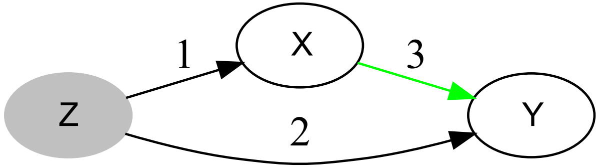
Granger attempted to define causality in terms of predictability (a characteristic of the joint distribution of probability).

Granger [1969] remains one of the most highly cited articles in the econometrics literature, with thousands of new citations each year. This abuse of the term causality has led to numerous false claims in the factor literature.

Causality in Factor Investing

Causal Content

- The objective of a factor model is not to use X to predict Y, but to determine *rewarded risk exposures*
- A researcher who just wanted to predict Y would have
  - used more powerful techniques than a linear model
  - minimized the mean-squared error, instead of using a MVUE
  - used MDA or Shapley values, instead of *p*-values
- Factor investors build portfolios that
  - overweight stocks with a high exposure to X, and
  - underweight stocks with a low exposure to X,
  - at the tune of one separate portfolio for each factor,
  - with no regard for the value of ε
- By making these modelling decisions, researchers have injected causal assumptions into their analyses



The objective of a factor model such as $Y = X\beta + Z\gamma + \varepsilon$ is *not* to predict Y conditioned on X and Z ($E[Y|X,Z]$), but to estimate the causal effect of X on Y ($E\big[Y|do[X]\big]$), which requires adjusting for the confounding effect of Z.

The model specification $Y = X\beta + Z\gamma + \varepsilon$ is consistent with a particular causal graph, **of which the above is just one possibility among several**.

Omitted Mediation Analysis

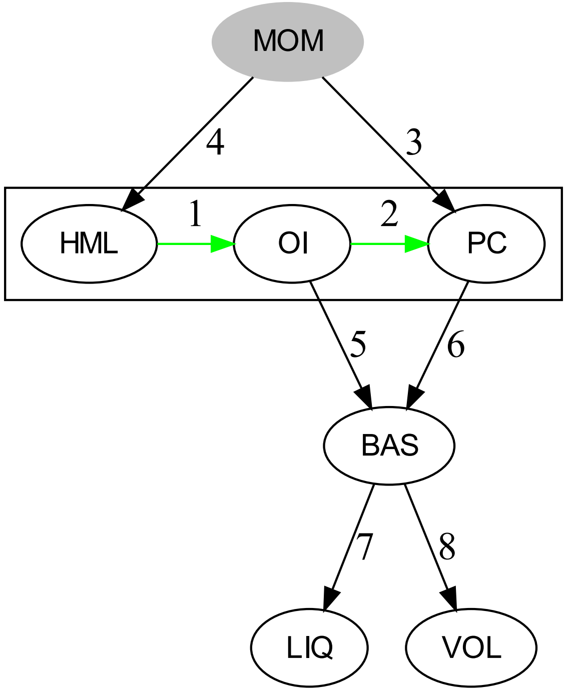
- Factors are often justified with economic rationales
  - E.g., reward for accepting a natural undiversifiable risk
- An economic rationale does not raise to the level of scientific theory
  - *"X causes Y through a falsifiable mechanism M"*
- An example of a causal theory would be the SEM

$$OI_t \coloneqq f_1 \underbrace{[p_t - v_t]}_{HML_t} + \varepsilon_{1,t}$$

$$\underbrace{p_{t+h} - v_t}_{PC_{t+h}} \coloneqq f_2[OI_t] + f_3[MOM_t] + \varepsilon_{2,t+h}$$

$$HML_t \coloneqq f_4[MOM_t] + \varepsilon_{3,t}$$

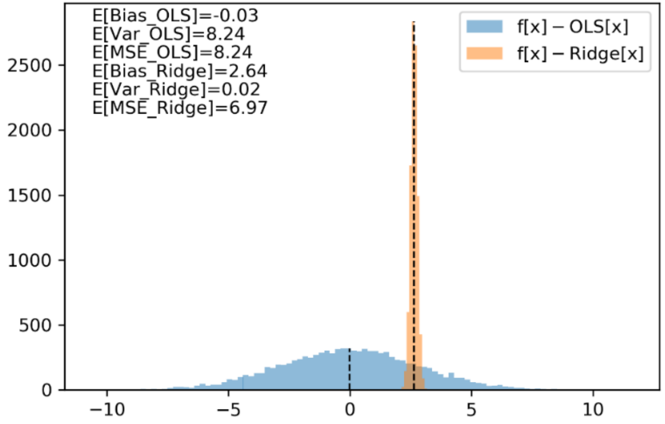
- Absent a hypothesized causal mechanism, it is not possible to design an experiment to assess the validity of factor investing claims



Hypothetical causal mechanism for value, controlling for momentum.

Causal Denial

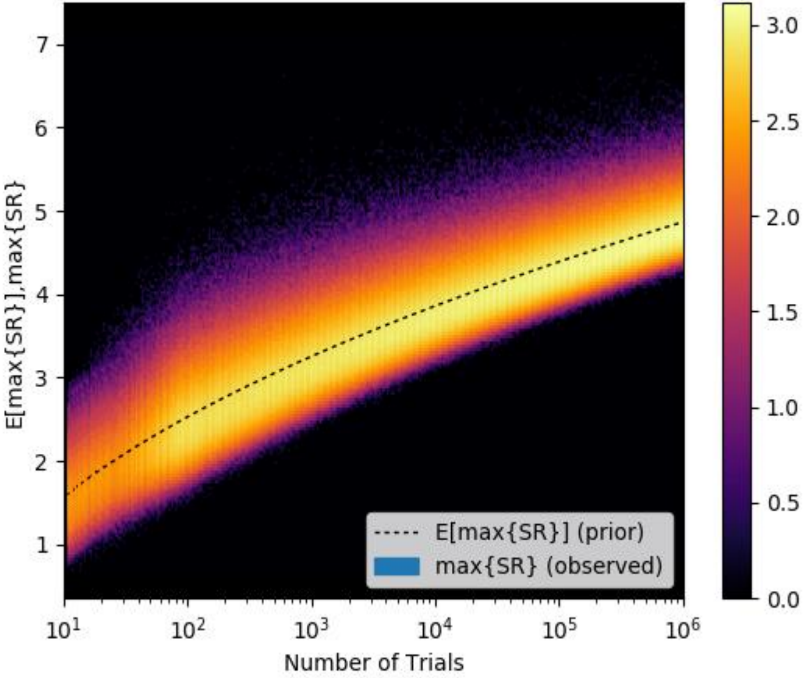
- Despite of the causal content of factor investing strategies, authors almost never
  - Declare a causal mechanism, or
  - Justify their model specification with a causal graph
- Without a declared causal structure,
  - the estimated β loses its causal meaning (the effect on Y of an intervention on X), and
  - *p*-values merely convey the strength of associations of unknown origin (causal and non-causal combined).
- The consequence of factor investing's causal denial
  - without a causal mechanism, there is no investment theory;
  - without investment theory, there is no falsification;
  - without falsification, investing cannot be scientific



If factor researchers only cared about prediction, then they would minimize the overall mean squared error, not just the variance among unbiased estimators (BLUE). The use of BLUE and *p*-values implies a causal interpretation of β.

Type-A Spuriosity

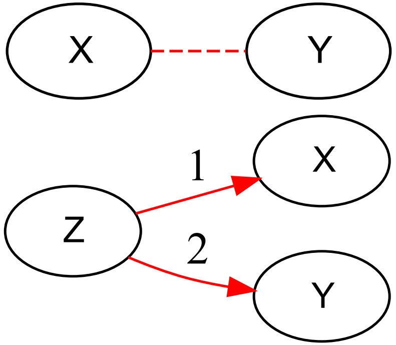
- Type-A spuriosity occurs when a researcher mistakes random variability (noise) for signal, resulting in a *false association*
- Type-A spuriosity has several attributes:
  a) it results in type-1 errors (false positives)
  b) for the same number of trials, it has a lower probability to take place as the sample size grows
  c) it can be corrected through multiple-testing adjustments
- Two main reasons for Type-A spuriosity
  – *p*-hacking, e.g., Hochberg [1988]
  – Backtest overfitting, Bailey and López de Prado [2014]



Distribution of the maximum Sharpe ratio as a function of the number of trials, where the true Sharpe ratio is zero. See "The False Strategy Theorem."

Type-B Spuriosity

- Type-B spuriosity occurs when a researcher mistakes association for causation (e.g., due to misspecification)
- Type-B spuriosity has several attributes:
  a) it results in type-1 errors and type-2 errors (false positives and false negatives);
  b) it can occur with a single trial;
  c) it has a greater probability to take place as the sample size grows, because the non-causal association can be estimated with lower error; and
  d) it cannot be corrected through multiple-testing adjustments. Its correction requires the injection of extra-statistical information, in the form of a causal theory
- Type-B spurious factors exhibit
  - misattributed risk and returns
  - time-varying risk premia

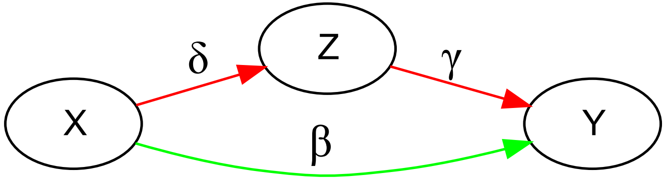


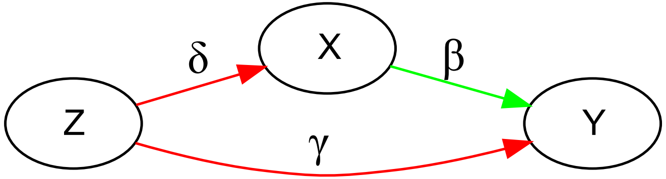
The top graph is an example of false association (type-A spuriosity). The bottom graph is an example of association mistaken for causation (type-B spuriosity).

Type-A and type-B spuriosity are mutually exclusive. For type-B spuriosity to take place, the association must be non-causal but true, which precludes that association from being type-A spurious.

Type-B(1) Spuriosity: Under-Controlling

- Consider a researcher who fits $Y = X\beta + \varepsilon$ on data generated by $Y := X\beta + Z\gamma + u$, where $\gamma \neq 0$ and u is white noise
  - As a consequence, $E[\varepsilon|X] = \gamma E[Z|X]$
  - $E[Z|X] \neq 0 \Rightarrow E[\varepsilon|X] \neq 0$ (exogeneity is not satisfied)
- Case 1: Z is a mediator ($Z \coloneqq X\delta + v$, with $\delta \neq 0$)
  - the chosen specification biases $\hat{\beta}$
  - however $\hat{\beta}$ can still be interpreted as a total causal effect
- Case 2: Z is a confounder ($X \coloneqq Z\delta + v$, with $\delta \neq 0$)
  - the chosen specification biases $\hat{\beta}$
  - $\hat{\beta}$ cannot be interpreted as a causal effect (direct or total)
- Wrong risk attribution means wrong allocations!





Econometric textbooks treat all missing variables as equal. **This is a mistake.**

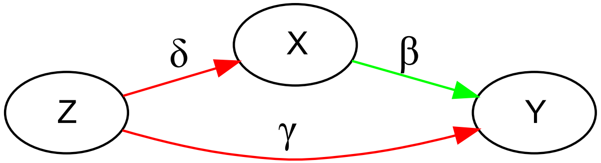
In the top graph, Z is a mediator, and missing Z has mild consequences. In the bottom graph, Z is a confounder, and missing Z will likely lead to false positives or false negatives.

A Mystery Solved: Time-Varying Risk Premia

- The time-varying nature of risk premia has puzzled researchers for 3+ decades. Explanations include:
  - changes in expected market returns
  - temporary changes in investor or market behavior
- There is an easier explanation: A missing confounder Z

$$E\left[\hat{\beta}|X\right] = \beta + \gamma\delta(1+\delta^2)^{-1}$$

- Consider the case where the market rewards exposure to X and Z ($\beta > 0, \gamma > 0$)
  - Even if the two risk premia remain constant, changes over time in δ will change $\hat{\beta}$
  - In particular, for a sufficiently negative value of δ, then $\hat{\beta} < 0$
- Time-varying risk premia is due to **Type-B(1)** spuriosity



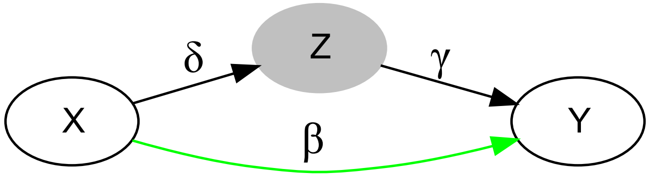
A leading explanation for the time-varying nature of risk premia is that β changes in response to changes in investors' expectations or behavior.

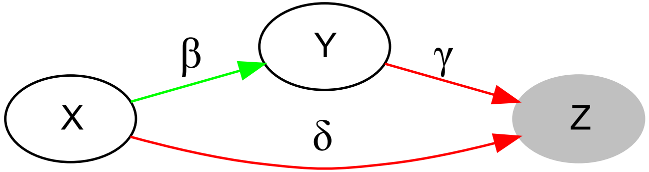
A more likely explanation is that $\hat{\beta}$ changes (but not β) due to changes in γ or even worse, changes in δ.

Time-varying risk premia is not a feature of the markets, it is a bug in factor investing.

Type-B(2) Spuriosity: Over-Controlling

- Statisticians have been trained for decades to control for any variable Z associated with Y that is not X
  - Econometrics textbooks dismiss as a harmless error the inclusion of an irrelevant variable, regardless of the variable's role in the causal graph
- Case 1: Z is a mediator
  - Controlling for a mediator interferes with the mediated effect and the total effect, which the researcher may wish to assess
  - $\hat{\beta}$ measures only the direct effect
- Case 2: Z is a collider
  - Controlling for a collider opens a backdoor path, $X \rightarrow Z \leftarrow Y$ (**Berkson's fallacy**)



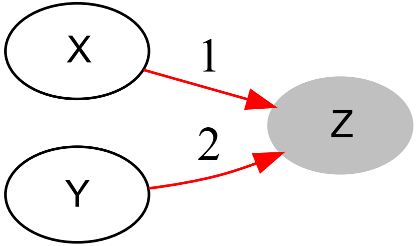


Greene [2012, section 4.3.3] states that the only downside to adding superfluous variables is a reduction in the precision of the estimates. **This is a mistake.**

Over-controlling for a collider has the same consequences as under-controlling for a confounder: **an open backdoor**.

Type-B(3) Spuriosity: Specification-Searching

- The use of explanatory power (an associational, non-causal concept) for selecting the specification of a factor model is inconsistent with that model's causal content
- Specification-searching commingles two separate and sequential stages of the causal analysis:
  1) **Causal discovery**: Finding the causal graph
  2) **Control**: Use the graph to determine the correct specification
- Stage (2) should be informed by stage (1), not the other way around
  - A researcher may achieve higher explanatory power by combining multiple causes of Y, at the expense of biasing the multiple parameters' estimates due to multicollinearity or over-controlling for a collider



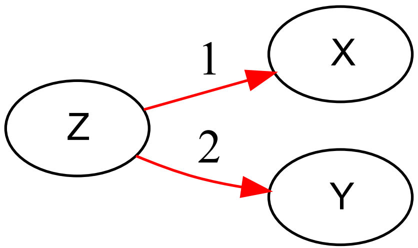
Econometric studies often justify the chosen specification in terms of explanatory power. This comingles causal discovery with controlling, and all but ensures that the regressors will include colliders (Berkson's fallacy).

Monte Carlo Experiments

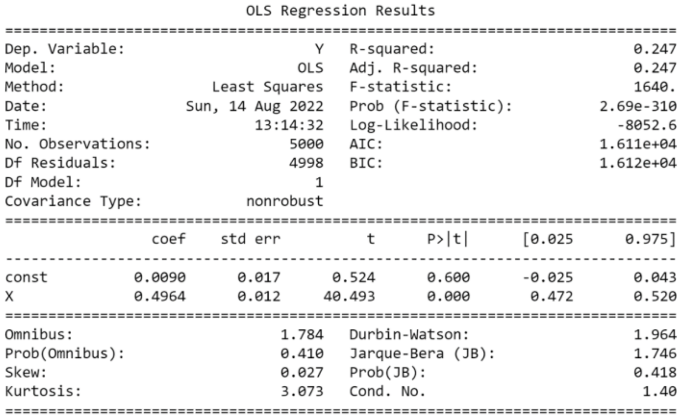
Type-B(1) Spuriosity :
Forks

Confounders

- Consider the fork structure in the right graph
- Applying Bayesian network factorization
$$P[X,Y,Z] = P[Z]P[X|Z]P[Y|Z]$$
- X and Y are associated, since
$$P[X,Y] = \sum_{Z} P[Z]P[X|Z]P[Y|Z] \neq P[X]P[Y]$$
- This is an example of non-causal association
  - X and Y are associated through the backdoor path
$$Y \leftarrow Z \rightarrow X$$
- Given the causal content of the factor model, a statistically significant $\hat{\beta}$ implies that X causes Y
  - This claim of statistical significance is type-B spurious



$$Y_t = \alpha + \beta X_t + \varepsilon_t$$



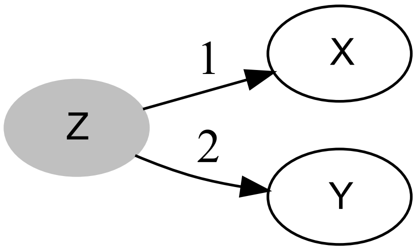
```
                            OLS Regression Results
==============================================================================
Dep. Variable:                      Y   R-squared:                       0.247
Model:                            OLS   Adj. R-squared:                  0.247
Method:                 Least Squares   F-statistic:                     1640.
Date:                Sun, 14 Aug 2022   Prob (F-statistic):          2.69e-310
Time:                        13:14:32   Log-Likelihood:                -8052.6
No. Observations:                5000   AIC:                         1.611e+04
Df Residuals:                    4998   BIC:                         1.612e+04
Df Model:                           1
Covariance Type:            nonrobust
==============================================================================
                 coef    std err          t      P>|t|      [0.025      0.975]
------------------------------------------------------------------------------
const          0.0090      0.017      0.524      0.600      -0.025       0.043
X              0.4964      0.012     40.493      0.000       0.472       0.520
==============================================================================
Omnibus:                        1.784   Durbin-Watson:                   1.964
Prob(Omnibus):                  0.410   Jarque-Bera (JB):                1.746
Skew:                           0.027   Prob(JB):                        0.418
Kurtosis:                       3.073   Cond. No.                         1.40
==============================================================================
```

The Backdoor Adjustment

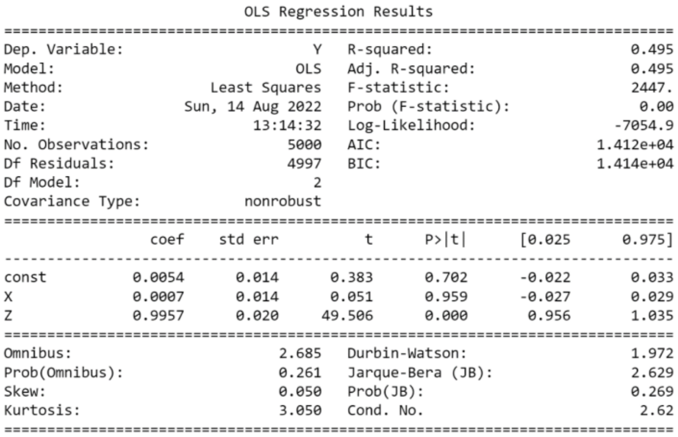
- The effect of conditioning by Z is equivalent to simulating a do-operation (an intervention)
  - It blocks the backdoor path, resulting in the conditional independence of X and Y,

$$P[X,Y|Z] = \frac{P[X,Y,Z]}{P[Z]} = P[X|Z]P[Y|Z]$$

- It is possible to remove the confounder-induced bias by adding Z as a regressor (the partial correlations method)
- With the correct model specification, the researcher concludes that X does not cause Y



$$Y_t = \alpha + \beta X_t + \gamma Z_t + \varepsilon_t$$

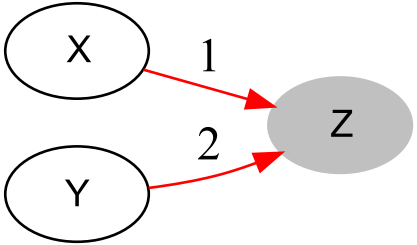


```
                            OLS Regression Results
==============================================================================
Dep. Variable:                      Y   R-squared:                       0.495
Model:                            OLS   Adj. R-squared:                  0.495
Method:                 Least Squares   F-statistic:                     2447.
Date:                Sun, 14 Aug 2022   Prob (F-statistic):               0.00
Time:                        13:14:32   Log-Likelihood:                -7054.9
No. Observations:                5000   AIC:                         1.412e+04
Df Residuals:                    4997   BIC:                         1.414e+04
Df Model:                           2
Covariance Type:            nonrobust
==============================================================================
                 coef    std err          t      P>|t|      [0.025      0.975]
------------------------------------------------------------------------------
const          0.0054      0.014      0.383      0.702      -0.022       0.033
X              0.0007      0.014      0.051      0.959      -0.027       0.029
Z              0.9957      0.020     49.506      0.000       0.956       1.035
==============================================================================
Omnibus:                        2.685   Durbin-Watson:                   1.972
Prob(Omnibus):                  0.261   Jarque-Bera (JB):                2.629
Skew:                           0.050   Prob(JB):                        0.269
Kurtosis:                       3.050   Cond. No.                         2.62
==============================================================================
```

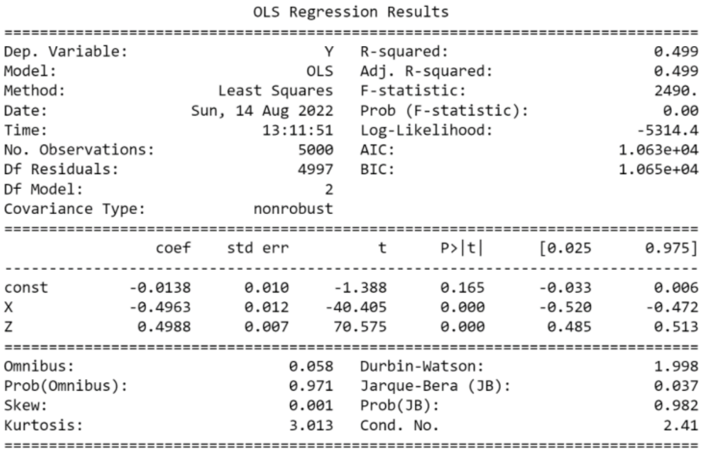
Type-B(2) Spuriosity : Immoralities

Colliders

- This causal graph shows a collider:
  - Variable Z is influenced by both, the treatment X and the outcome Y
- If a researcher controls for Z, the result is a false positive (bottom table)
- Compare the fork structure with the immorality structure
  - When the direction of causality is reversed, a confounder becomes a collider
  - The direction of causality is critical for specification
- One problem is, the direction of causality cannot always be determined from data
  - Causal graphs incorporates extra-statistical (beyond observational) information



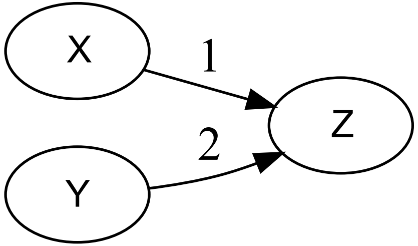
$$Y_t = \alpha + \beta X_t + \gamma Z_t + \varepsilon_t$$



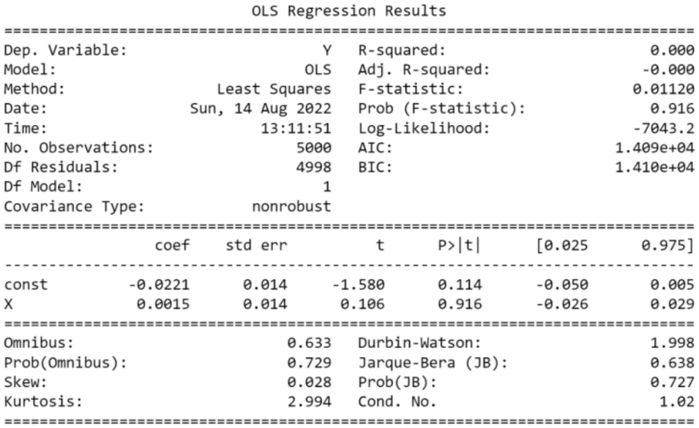
```
                            OLS Regression Results
==============================================================================
Dep. Variable:                      Y   R-squared:                       0.499
Model:                            OLS   Adj. R-squared:                  0.499
Method:                 Least Squares   F-statistic:                     2490.
Date:                Sun, 14 Aug 2022   Prob (F-statistic):               0.00
Time:                        13:11:51   Log-Likelihood:                -5314.4
No. Observations:                5000   AIC:                         1.063e+04
Df Residuals:                    4997   BIC:                         1.065e+04
Df Model:                           2
Covariance Type:            nonrobust
==============================================================================
                 coef    std err          t      P>|t|      [0.025      0.975]
------------------------------------------------------------------------------
const         -0.0138      0.010     -1.388      0.165      -0.033       0.006
X             -0.4963      0.012    -40.405      0.000      -0.520      -0.472
Z              0.4988      0.007     70.575      0.000       0.485       0.513
==============================================================================
Omnibus:                        0.058   Durbin-Watson:                   1.998
Prob(Omnibus):                  0.971   Jarque-Bera (JB):                0.037
Skew:                           0.001   Prob(JB):                        0.982
Kurtosis:                       3.013   Cond. No.                         2.41
==============================================================================
```

Berkson’s Fallacy

- Berkson’s fallacy occurs when a spurious association is observed between two independent variables, as a result of conditioning on a collider
- With a careful selection of colliders, a researcher can present evidence in support of any spurious investment factor
- The correct causal treatment of a collider is to indicate its presence, and justify why researchers should not control for it
- Over-controlling leads to
  - false positives, in the presence of colliders
  - false negatives, in the presence of mediators
    - E.g., controlling for Z in $X \rightarrow Z \rightarrow Y$



$$Y_t = \alpha + \beta X_t + \varepsilon_t$$

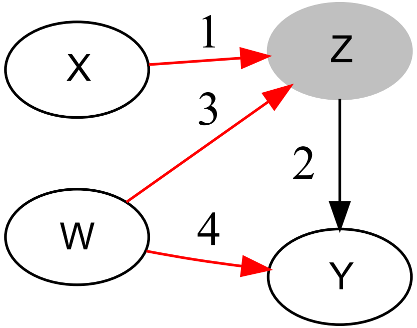


```
                            OLS Regression Results
==============================================================================
Dep. Variable:                      Y   R-squared:                       0.000
Model:                            OLS   Adj. R-squared:                 -0.000
Method:                 Least Squares   F-statistic:                   0.01120
Date:                Sun, 14 Aug 2022   Prob (F-statistic):              0.916
Time:                        13:11:51   Log-Likelihood:                -7043.2
No. Observations:                5000   AIC:                         1.409e+04
Df Residuals:                    4998   BIC:                         1.410e+04
Df Model:                           1
Covariance Type:            nonrobust
==============================================================================
                 coef    std err          t      P>|t|      [0.025      0.975]
------------------------------------------------------------------------------
const         -0.0221      0.014     -1.580      0.114      -0.050       0.005
X              0.0015      0.014      0.106      0.916      -0.026       0.029
==============================================================================
Omnibus:                        0.633   Durbin-Watson:                   1.998
Prob(Omnibus):                  0.729   Jarque-Bera (JB):                0.638
Skew:                           0.028   Prob(JB):                        0.727
Kurtosis:                       2.994   Cond. No.                         1.02
==============================================================================
```

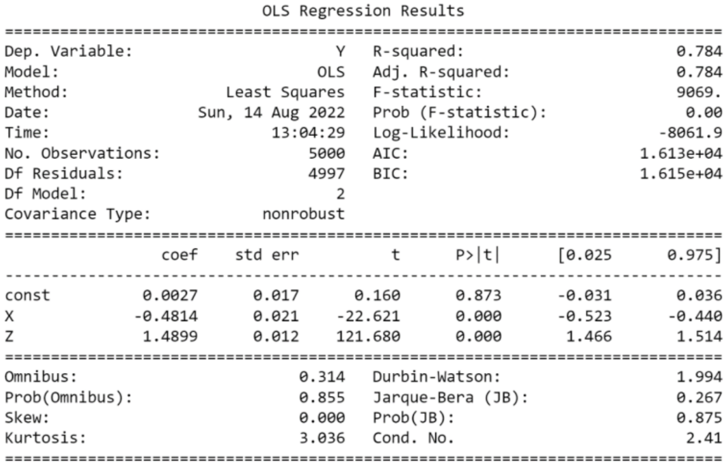
Type-B(3) Spuriosity : Chains

Confounded Mediators

- This causal graph shows a mediator and a confounder:
  - Variable Z mediates the causal flow from the treatment X to the outcome Y
  - Variable W confounds Z and Y
- If a researcher controls for Z, the outcome is a false positive (bottom table)
  - The reason is that Z also operates as a collider to X and W
    - Controlling for Z opens a backdoor path $X \rightarrow Z \leftarrow W \rightarrow Y$
  - While it is true that X causes Y (through Z), the collider's bias is so strong that the sign of the relationship is reversed ($\hat{\beta} \ll 0$)
- In the absence of link 3, controlling for Z would have led to a false negative



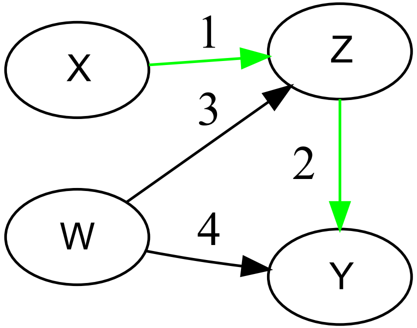
$$Y_t = \alpha + \beta X_t + \gamma Z_t + \varepsilon_t$$



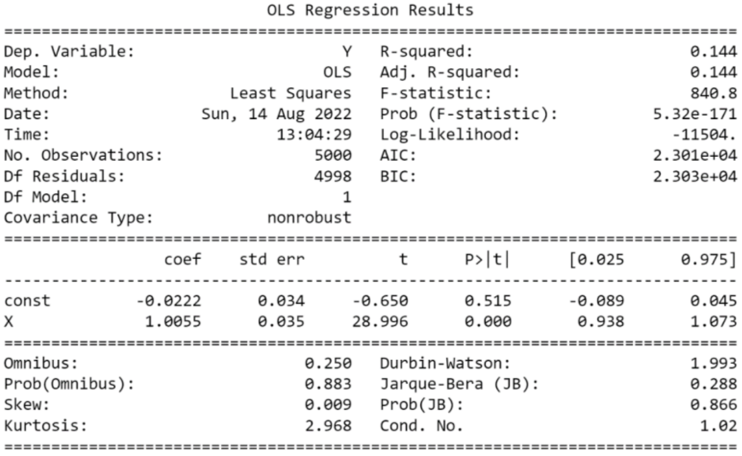
```
                            OLS Regression Results                            
==============================================================================
Dep. Variable:                      Y   R-squared:                       0.784
Model:                            OLS   Adj. R-squared:                  0.784
Method:                 Least Squares   F-statistic:                     9069.
Date:                Sun, 14 Aug 2022   Prob (F-statistic):               0.00
Time:                        13:04:29   Log-Likelihood:                -8061.9
No. Observations:                5000   AIC:                         1.613e+04
Df Residuals:                    4997   BIC:                         1.615e+04
Df Model:                           2                                         
Covariance Type:            nonrobust                                         
==============================================================================
                 coef    std err          t      P>|t|      [0.025      0.975]
------------------------------------------------------------------------------
const          0.0027      0.017      0.160      0.873      -0.031       0.036
X             -0.4814      0.021    -22.621      0.000      -0.523      -0.440
Z              1.4899      0.012    121.680      0.000       1.466       1.514
==============================================================================
Omnibus:                        0.314   Durbin-Watson:                   1.994
Prob(Omnibus):                  0.855   Jarque-Bera (JB):                0.267
Skew:                           0.000   Prob(JB):                        0.875
Kurtosis:                       3.036   Cond. No.                         2.41
==============================================================================
```

Mediation Fallacy & Simpson’s Paradox

- The Mediation Fallacy involves conditioning on the mediator when the mediator and the outcome are confounded
- Simpson’s paradox occurs when there is an association in several groups, but it disappears or reverses when the groups are combined
- The solution to Simpson’s paradox is to inject extra-statistical information in the form of a causal graph
- We can estimate the unbiased effect ($\hat{\beta} \gg 0$)
  - Specification-searching would have returned a misspecified model (R^2 drops from 0.78 to 0.14!)
  - Adding W increases R^2 to 0.71 (still below 0.78)



$$Y_t = \alpha + \beta X_t + \varepsilon_t$$

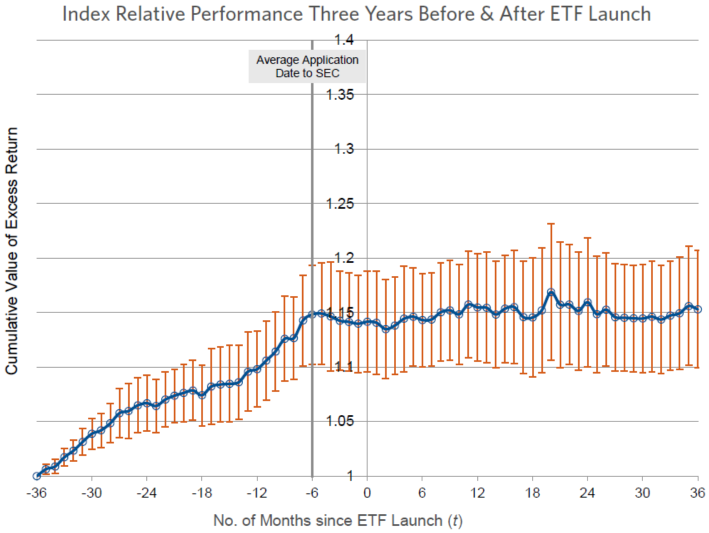


```
                            OLS Regression Results
==============================================================================
Dep. Variable:                      Y   R-squared:                       0.144
Model:                            OLS   Adj. R-squared:                  0.144
Method:                 Least Squares   F-statistic:                     840.8
Date:                Sun, 14 Aug 2022   Prob (F-statistic):          5.32e-171
Time:                        13:04:29   Log-Likelihood:                -11504.
No. Observations:                5000   AIC:                         2.301e+04
Df Residuals:                    4998   BIC:                         2.303e+04
Df Model:                           1
Covariance Type:            nonrobust
==============================================================================
                 coef    std err          t      P>|t|      [0.025      0.975]
------------------------------------------------------------------------------
const         -0.0222      0.034     -0.650      0.515      -0.089       0.045
X              1.0055      0.035     28.996      0.000       0.938       1.073
==============================================================================
Omnibus:                        0.250   Durbin-Watson:                   1.993
Prob(Omnibus):                  0.883   Jarque-Bera (JB):                0.288
Skew:                           0.009   Prob(JB):                        0.866
Kurtosis:                       2.968   Cond. No.                         1.02
==============================================================================
```

Conclusions

Spurious Investment Factors (1/2)

- Consider the influential three-factor (FF93) and five-factor (FF15) models proposed by Fama and French
- Type-A Spuriosity: *p*-hacking
  - These authors do not adjust *p*-values for multiple trials
  - In the year 2023, it is unfortunate that financial econometricians still deny the need to adjust for selection bias under multiple testing
- Type-B(1) Spuriosity: Under-controlling
  - Missing confounders (e.g., momentum, macro variables) may explain the puzzle of time-varying risk premia
  - Carhart (C97) added momentum to FF93, however his rationale was greater explanatory power (specification-searching), not de-confounding

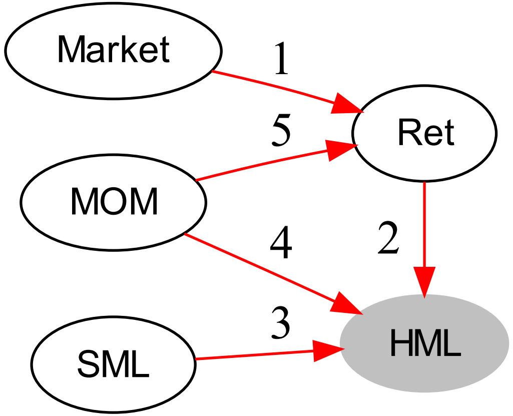


Source: Research Affiliates, LLC, using data from Bloomberg.

Performance of active ETFs launched in the U.S. between 1993 and 2014. Cumulative returns flatten 6 months before launch, coinciding with the end of the in-sample period. This points to backtest overfitting.

Spurious Investment Factors (2/2)

- Type-B(2) Spuriosity: Over-controlling
  - Controlled colliders (e.g., HML) may explain false associations, or true associations with wrong signs
- Type-B(3) Spuriosity: Specification-searching
  - A correctly specified model can deliver lower explanatory power than a misspecified model
  - Improving on FF93's explanatory power does not make C97's model better specified, or its estimates less biased
- To summarize, the findings in FF93, FF15 and C97 are likely type-A or type-B spurious
- To address these issues, factor researchers must
  - adjust for selection bias under multiple testing
  - justify their specification choices through a causal graph
  - propose a falsifiable causal mechanism



Under this graph, the estimates in FF93, FF15 and C97 are biased. This particular graph may be incorrect, however the burden of the proving it wrong belongs to the authors claiming the existence of investment factors.

The Dawn of Causal Factor Investing

- A scientific theory is a falsifiable statement of the form “X causes Y through mechanism M”
- Scientific theories matter to investors because
  - causality is a necessary condition for investment efficiency
    - associational models misattribute risks and performance, thus preventing investors from building efficient portfolios
  - causal models enable counterfactual reasoning, hence the stress-testing of investment portfolios in a coherent and forward-looking manner
    - associational models cannot answer counterfactual questions, such as what would be the effect of Y on a not-yet-observed scenario X, thus exposing those relying on associations to black-swan events
- **Financial economists’ adoption of causal inference has the potential to transform investing into a truly scientific discipline**

Type	Rigor	Example
Randomized controlled trials	Very high	Algo-wheel experiments
Natural experiments	High	Market-maker reaction to random spikes in order imbalance
Simulated interventions	Medium	Estimate effect of HML using a causal graph
Econometric (observational) studies	Low	Factor investing literature; backtested investment strategies
Case studies	Very low	Broker report / analysis
Expert opinion	Anecdotal	Investment guru’s prediction

Hierarchy of evidence: Pre-scientific vs. scientific evidence in financial research.

Call For Papers

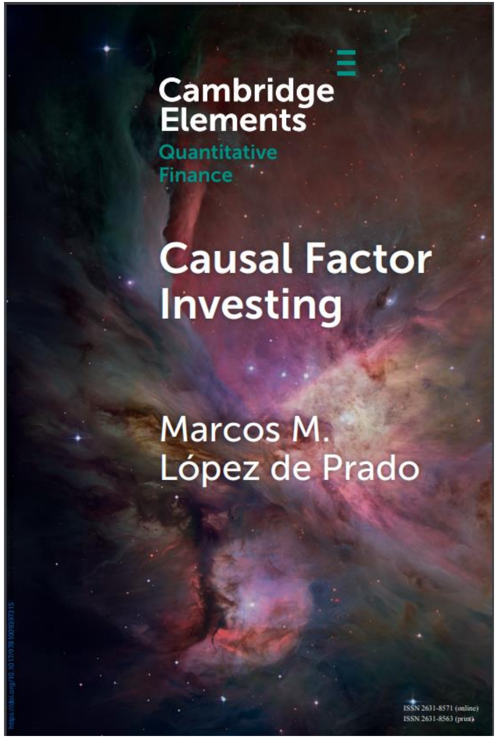
ADIA Lab encourages researchers to move factor investing beyond its current pre-scientific stage.

To help dawn the discipline of Causal Factor Investing, ADIA Lab has called for papers that promote the use of the formal language of causal inference in investing.

To learn more, visit:
https://www.adialab.ae/call-for-papers

We look forward to your papers!

For More Information



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Quantitative Finance

Causal Factor Investing

Marcos M. López de Prado

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