SAGE reference

Encyclopedia of Research Design

U-Shaped Curve

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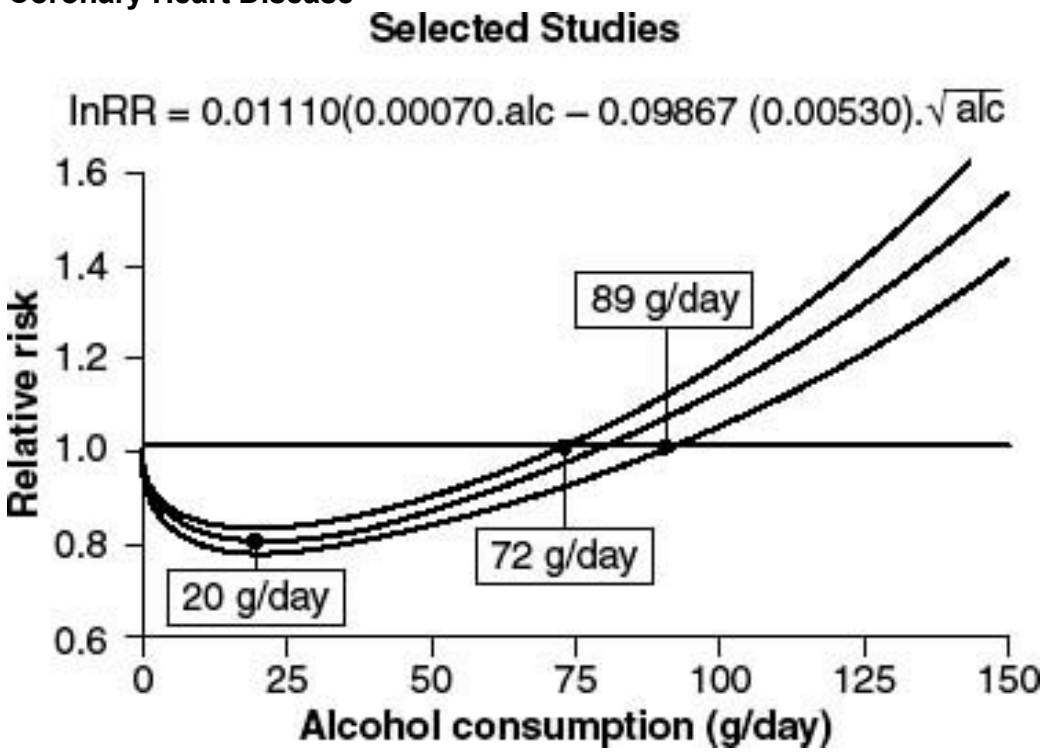
Book Title: Encyclopedia of Research Design
Chapter Title: "U-Shaped Curve"
Pub. Date: 2010
Access Date: September 8, 2018
Publishing Company: SAGE Publications, Inc.
City: Thousand Oaks
Print ISBN: 9781412961271
Online ISBN: 9781412961288
DOI: http://dx.doi.org/10.4135/9781412961288.n485
Print pages: 1587-1588

The U-shaped curve usually refers to the nonlinear relationship between two variables, in particular, a dependent and an independent variable. Because many analytic methods assume an underlying linear relationship, systematic deviation from linearity can lead to bias in estimation. Meaningful U-shaped relationships can be found in epidemiology (e.g., between risk factor and disease outcome or mortality), psychology (often age-related developments, such as delinquency or marital happiness), and economics (e.g., short-run cost curves between the variate cost and quantity).

In medicine, U-shaped risk curves have been found for risk factors such as cholesterol level, diastolic blood pressure, work stress, and alcohol use. Of these factors, the alleged U-shape relationship between alcohol use and disease risk has been the most controversial. By the 1920s, a U.S. study by Raymond Pearl already showed a depressed longevity for abstainers. At that time, with alcohol prohibition in effect, this was not a politically correct message. Many years later, better controlled cohort studies looking into what Alvan R. Feinstein in his Science article called the "menace of daily life" have also reported lowest risk estimates for light or moderate drinkers of alcoholic beverages. Heavier drinkers are at highest risk, as could be expected. However, abstainers or non-drinkers in general also are found to have a higher risk for several negative health outcomes. This effect has been observed for overall mortality and specific categories such as cardiovascular diseases. For the latter, some studies report a J-shape rather than a U-shape, with little increased risk at higher consumption levels. Generally, the risk is estimated to be approximately 20% higher for abstainers, as shown in Figure 1 by Giovanni Corrao and colleagues.

Figure 1 Example of U- or J-Shaped Curve Between Alcohol Intake and Risk for Coronary Heart Disease



Source: Corrao, G., Rubbiati, L., Bagnardi, V., Zambon, A., & Poikolainen, K. (2000). Alcohol and coronary heart disease: A meta-analysis. Addiction, 95, 1505 −1523.

During the last 20 years, as results from more and more cohort studies have been

accumulating, the J-shaped risk curve has been considered to be the aggregate result of several biological processes underlying the most prevalent of pathologies in the Western world, coronary heart disease. For some diseases or bodily processes, any alcohol has an outright negative effect. Alcohol has been found to raise blood pressure even in small amounts, which in turn is a risk factor for cardiovascular disease. However, alcohol has a proven negative effect on the formation of thrombi or blood clots, which in itself is considered a risk factor for ischemic diseases (heart attack, brain infarctions). A major third process is the positive effect of alcohol use on the high-density cholesterol (HDL) level in the blood, which is considered to be a protective factor in the genesis of arterial plaques, eventually obstructing blood flow to vital tissues of heart or brain. Across the years, several other biological processes and genetic vulnerability factors have been suggested as potential candidates for the explanation of the lower risk for moderate drinkers of alcohol. The message of a potential beneficial health effect of alcohol use has caused considerable debate, as alcohol use at higher intake levels may be considered a serious health hazard. The detrimental effects of alcohol are less disputed, with monotonically increasing risk for outcomes such as injuries, liver functions, liver cirrhosis, and certain forms of cancer (e.g., breast cancer).

Next to the biological explanations of the J-shape, suggesting several direct (e.g., clotting) or indirect (high density cholesterol level) biological effects, some have provided alternative explanations, stemming from methodological flaws or specific design features. For example, with evidence from the British Regional Heart Study (BRHS), A. Gerald Shaper suggested that the lower effect could be the result of a mixture of nondrinkers with heterogeneous risk profiles. At the time, not all studies made a difference among lifetime abstainers, teetotalers, and ex-drinkers. From the BRHS cohort, it was obvious that the ex-drinkers were at the highest risk and that the risk of British teetotalers could not be distinguished from that of light drinkers. However, heterogeneity of risk in the nondrinker category cannot explain the U-shape at the lower end of the drinking scale.

Another explanation for the U- or J-shape at the lower end is the selection of high-risk individuals in the abstainer category, leading to what in epidemiology has been termed by Feinstein as *susceptibility bias*. Susceptibility bias would occur when persons more vulnerable to the disease would refrain from engaging in a drinking career. There is some evidence from a Dutch cross-sectional population study of a selection of young people with poorer health into the category of teetotalers, when health complaints among teetotalers increased with age as number of teetotalers in the population decreased with age. Solid empirical evidence for this phenomenon is difficult as it requires a follow-up of long duration of adolescent cohorts. In one such rare study of Bostonian teens, George Eman Vaillant indeed presented evidence for selection leading to an overrepresentation of people with a higher disease burden among abstainers. A variant of this is termed *protopathic bias*, when people with preexisting disease or with a high-risk profile are overrepresented among the nondrinkers. Empirical evidence for these phenomena remains inconclusive, however.

Three other design features have been brought forward as potentially creating a bias in estimates of risk at the lower end of the alcohol consumption curve. In all epidemiologic studies, alcohol use (risk factor) is measured close to the endpoint (disease outcome) in cohorts that usually have passed middle age because incidence and mortality in younger age groups is quite low. Apart from a restricted time window of observation, this also means that risk assessments refer to recent alcohol intake at middle age, thereby disregarding the effects of drinking in the (average) first 20 to 30 years of life. Both aspects could produce a bias because changes in drinking status as a result of bad health would go unnoticed. Proof of such a bias was found in a New Zealand case-control study by Stijn Wouters and colleagues

who found that cases were significantly more likely than controls to report recent abstention from drinking because they felt unwell. The U- or J-shaped association between recent alcohol consumption and acute coronary heart disease seemed to be largely caused by the confounding effect of preclinical, usually not measured, symptoms on drinking. Third, it has been reported in recent reviews that the lower end of the J-shape becomes less pronounced as quality of the design increases.

Apart from the caveats in assessing and interpreting the J- or U-shaped curve, estimation of the exact shape of the curve might present problems. Els J. T. Goetghebeur and Stuart J. Pocock have warned against oversimplification with potential bias in estimating the nadir (lowest turning point) and upward slope on particularly the left side of a U-shape. The often sparse numbers of observations available make exact fitting difficult, and categorization leads to loss of information. Several approaches to (parametric) fitting are possible. The authors suggest sequential quadratic and linear tests for the downward trend from the left of the curve.

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http://dx.doi.org/10.4135/9781412961288.n485

See also

- Bias
- Cohort Design
- Confounding
- Odds Ratio
- Survival Analysis

Further Readings

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