MISCLASSIFICATION OF EXPOSURE pdf

1: Information bias (epidemiology) - Wikipedia

Differential Misclassification of Exposure If errors in classification of exposure status occur more frequently in one of the groups being compared, then differential misclassification will occur, and the estimate of association can be over estimated or under estimated.

You will learn about common errors in epidemiological measurements. All measurements are prone to error. Understanding common errors and the means to reduce them improves the precision of estimates. Read the resource text below. Resource text Random error chance Chance is a random error appearing to cause an association between an exposure and an outcome. A principal assumption in epidemiology is that we can draw an inference about the experience of the entire population based on the evaluation of a sample of the population. However a problem with drawing such an inference is that the play of chance may affect the results of an epidemiological study because of the effects of random variation from sample to sample [1]. The effect of random error may produce an estimate that is different from the true underlying value. Note that the effect of random error may result in either an underestimation or overestimation of the true value. Sampling Error Because of chance, different samples will produce different results and therefore must be taken into account when using a sample to make inferences about a population [2]. This difference is referred to as the sampling error and its variability is measured by the standard error. Sampling error may result in A Type I error - Rejecting the null hypothesis when it is true A Type II error - Accepting the null hypothesis when it is false For example, when comparing the mean weights of primary class students in a government school and private school, it is generally assumed that students in government schools have a poorer nutrition and less weight hypothesis. To prove the same, the null hypothesis is stated, no difference in the weights of students in either schools. However, because of sampling errors, there is a statistical probability of identifying a difference when truly there is no difference. On the other hand, a type 2 error is when we fail to observe a difference when there is a difference due to say, inadequate sample. Reducing sampling error Sampling error cannot be eliminated but with an appropriate study design can be reduced to an acceptable level. One of the major determinants to the degree to which chance affects the findings in a study is sample size [2]. In general, sampling error decreases as the sample size increases. Therefore, use of an appropriate sample size will reduce the degree to which chance variability may account for the results observed in a study. The role of chance can be assessed by performing appropriate statistical tests and by calculation of confidence intervals. Note that the value of p will depend on both the magnitude of the association and on the study size. Confidence intervals are more informative than p values because they provide a range of values, which is likely to include the true population effect. They also indicate whether a non-significant result is or is not compatible with a true effect that was not detected because the sample size was too small. Measurement error reliability and validity All epidemiological investigations involve the measurement of exposures, outcomes and other characteristics of interest e. Types of measures may include: Responses to self-administered questionnaires.

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2: FEM - Information (Measurement) Bias

It has traditionally been assumed that in the case of binary or dichotomous variables nondifferential misclassification would result in an 'underestimation' of the hypothesized relationship between exposure and outcome.

Selected References These references are in PubMed. This may not be the complete list of references from this article. Misclassification of smoking habits as a source of bias in the study of environmental tobacco smoke and lung cancer. Lung cancer and passive smoking: Validity of claims to lifelong non-smoking at age 36 in a longitudinal study. Misclassification of smoking status and lung cancer risk from environmental tobacco smoke in never-smokers. Passive smoking and lung cancer among Japanese women. Reliability of surrogate information on cigarette smoking by type of informant. The accumulated evidence on lung cancer and environmental tobacco smoke. Exposure of nonsmoking women to environmental tobacco smoke: Nicotine concentrations in urine and saliva of smokers and non-smokers. Validation of studies on lung cancer in non-smokers married to smokers. Carboxyhemoglobin, cotinine, and thiocyanate assay compared for distinguishing smokers from non-smokers. Use of serum cotinine to assess the accuracy of self reported non-smoking. Passive smoking and lung cancer association: Discrepancies between self-reported and validated cigarette smoking in a community survey of New Mexico Hispanics. Am Rev Respir Dis. Measurement of current exposure to environmental tobacco smoke. Cotinine validation of self-reported smoking in commercially run community surveys. Cotinine as a biomarker of environmental tobacco smoke exposure. Environmental tobacco smoke and lung cancer in nonsmoking women. A European validation study of smoking and environmental tobacco smoke exposure in nonsmoking lung cancer cases and controls. Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe. J Natl Cancer Inst. Exposure of the US population to environmental tobacco smoke: Smoking and other risk factors for lung cancer in women. Lung cancer among women in north-east China. Passive smoking and lung cancer in nonsmoking women. Am J Public Health. Lung cancer in nonsmokers. Passive smoking and diet in the etiology of lung cancer among non-smokers. A case-control study of lung cancer in nonsmoking women. Tohoku J Exp Med. Involuntary smoking and lung cancer: Lung cancer and exposure to tobacco smoke in the household. N Engl J Med. Relation between exposure to environmental tobacco smoke and lung cancer in lifetime nonsmokers. Risk factors of lung cancer by histological category in Taiwan. Relationship of passive smoking to risk of lung cancer and other smoking-associated diseases. Risk factors for primary lung cancer among non-smoking women in Taiwan. Measurements of passive smoking and estimates of lung cancer risk among non-smoking Chinese females. Environmental tobacco smoke and lung cancer risk in nonsmoking women. Familial risk of lung cancer among nonsmokers and their relatives. Exposure to environmental tobacco smoke and risk of lung cancer in non-smoking women from Moscow, Russia. Differential exposure misclassification in case-control studies of environmental tobacco smoke and lung cancer. The reliability of passive smoking histories reported in a case-control study of lung cancer. Reliability of passive smoke exposure histories in a case-control study of lung cancer. Questionnaire assessment of lifetime and recent exposure to environmental tobacco smoke. Mathematical models for predicting indoor air quality from smoking activity.

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3: Epiville: Bias -- Data Analysis Questions

The effects of exposure misclassification on odds ratios estimation are shown in table www.enganchecubano.com we set the true odds ratios to for all exposure levels of daily intake and used the nonexposed group as the referent, the resulting misclassified odds ratio estimates were for the total sample at all levels of exposure and ranged from to by age-sex stratum.

All Modules Differential Misclassification of Exposure If errors in classification of exposure status occur more frequently in one of the groups being compared, then differential misclassification will occur, and the estimate of association can be over estimated or under estimated. There are several mechanisms by which differential misclassification of exposure can occur. Recall bias Recall bias occurs when there are systematic differences in the way subjects remember or report exposures or outcomes. Recall bias can occur in either case-control studies or retrospective cohort studies. In a case-control study: Mothers of children with birth defects are likely to remember drugs they took during pregnancy differently than mothers of normal children. In this particular situation the bias is sometimes referred to as maternal recall bias. Mothers of the affected infants are likely to have thought about their drug use and other exposures during pregnancy to a much greater extent than the mothers of normal children. The primary difference arises more from under reporting of exposures in the control group rather than over reporting in the case group. However, it is also possible for the mothers in the case group to under report their past exposures. For example, mothers of infants who died from SIDS may be inclined to under report their use of alcohol or recreational drugs during pregnancy. Recall bias occurs most often in case-control studies, but it can also occur in retrospective cohort studies. For example, those who have been exposed to a potentially harmful agent in the past may remember their subsequent outcomes with a different degree of completeness or accuracy. In the retrospective portion of the Ranch Hand Study which looked at effects of exposure to Agent Orange dioxin. Pilots who had been exposed may have had a greater tendency to remember skin rashes that occurred during the year following exposure. In a case-control study, if both cases and controls have more or less equal difficulty in remembering past exposures accurately, it is nondifferential, and it is a form of nondifferential misclassification. In contrast, if one group remembers past exposures more accurately than the other, then it is called "recall bias" which is a differential type of misclassification. Ways to Reduce Recall Bias Use a control group that has a different disease that is unrelated to the disease under study. Use questionnaires that are carefully constructed in order to maximize accuracy and completeness. For socially sensitive questions, such as alcohol and drug use or sexual behaviors, use a self-administered questionnaire instead of an interviewer. If possible, assess past exposures from biomarkers or from pre-existing records. Interviewer Bias Also Recorder Bias Differential bias can be introduced into a study when there are systematic differences in soliciting, recording, or interpreting information on exposure in a case-control study or outcome in retrospective and prospective cohort studies and in intervention studies [clinical trials]. This type of bias can also occur when data is collected by review of medical records if the reviewer abstractor interprets or records information differently for one group or if the reviewer searches for information more diligently for one group. Since this is introduces a differential misclassification, it can cause bias either toward or away from the null, depending on the circumstances. Ways to Reduce Interviewer Bias Use standardized questionnaires consisting of closed-end, easy to understand questions with appropriate response options. Train all interviewers to adhere to the question and answer format strictly, with the same degree of questioning for both cases and controls. Obtain data or verify data by examining pre-existing records e. Differences in the Quality of Information Obviously, if the data for each of the groups being compared comes from different sources, the accuracy of the data may be better in one group, and this will introduce differential misclassification. For example, if exposure data for a case group were obtained from a facility specializing in the care of that condition and data from the comparison group were obtained from another source, there might be significant differences in the completeness and accuracy of the exposure data.

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4: CTSPedia: www.enganchecubano.comlassNondiffOutcome

Misclassification of exposure status is more of a problem than misclassification of outcome (as explained on page 6), but a study may be biased by misclassification of either exposure status, or outcome status, or both.

Advanced Search Abstract This study was conducted to assess the effect of exposure misclassification when coffee is used as a surrogate measure of caffeine exposure. Subjects were randomly selected from the telephone directories of four regional municipalities in southern Ontario, Canada. Data on daily caffeine intake from foods, beverages, and medications were collected from June to November through self-administered, mailed questionnaires from men and women aged 30â€”75 years. Although coffee was the main source of caffeine, cross-tabulations of exposure to coffee by total caffeine intake showed that assessment of coffee alone severely underestimated caffeine intake by at least one exposure level. A hypothetical fold increase in risk was completely obscured when only coffee was used to estimate total caffeine intake. The results of this study suggest that measuring coffee instead of caffeine intake may contribute to a lack of positive findings in studies of coffee as a risk factor for disease occurrence, if in fact caffeine is the exposure of interest. On the other hand, measurement of coffee, tea, and cola soft drink intake in the present study appeared to approximate caffeine intake sufficiently and not affect risk estimates adversely. In some areas of research, coffee is the focus of interest e. The literature suggests that although coffee contains many different chemical compounds, no certainty exists as to which ones may be associated with disease risk. Caffeine, the main psychoactive ingredient in coffee, has been the substance studied most often. Perhaps the lack of certainty about which exposure should be measured, combined with the fact that coffee is the main source of caffeine, has led to a tendency to equate the two exposures. The confusion in distinguishing between the effects of coffee and caffeine is reflected in literature reviews that often report results from coffee and caffeine studies together 20 , 23 , Review articles on the health effects of caffeine and caffeine-related compounds either use evidence or draw conclusions from studies that measured coffee consumption alone 8 , 13 , 25 â€” This lack of distinction has resulted in incomplete caffeine measurement; that is, only coffee intake is examined and other significant sources of caffeine are not included 13 , 23 , 28 , To our knowledge, only one study 30 to date has examined this type of misclassification of exposures, and no study has yet examined the effects this misclassification might have on effect estimates. The objectives of this study were to assess the extent of underestimation when coffee is used as a surrogate for total caffeine intake and the effects on odds ratio estimation for multiple levels of exposure. Eligible subjects were men and women aged 30â€”75 years residing in Ontario. The sample was stratified by sex and age i. Subjects were recruited from June to November People were initially contacted by telephone to determine whether they would participate in this study by completing a mailed questionnaire. During this call, they were asked to provide their age to ensure that they were eligible for the study. Of the 2, telephone numbers called, numbers were nonresidential; respondents were ineligible according to the age criterion or to age-sex-filled quotas; and, for numbers, there was no answer or no one who understood English. Thus, 1, respondents remained, and Of the people who consented, Six questionnaires were excluded from the analysis: Thus, questionnaires were retained for analysis. The questionnaire elicited demographic and anthropometric data, a selected medical history, exposure to tobacco, reproductive history women only , and a brief dietary history. It was designed to collect complete information on caffeine intake from foods, beverages, and medications. Subjects were asked to report when they began consuming a caffeine-containing substance, the usual number of servings or dose per day, and, if applicable, when they stopped doing so. Serving size was not specified on the questionnaire. Daily caffeine intake was calculated by including only current intake of the different caffeine sources; lifetime measures not reported in this paper added the dimension of length of intake to each caffeine source. Total caffeine intake was categorized by using the same caffeine cutpoints as those for coffee. Telephone calls were made to respondents for clarification or to obtain additional information if any caffeine data were missing. Information was collected on 23 caffeine-containing medications, including analgesic, diet, migraine, and menstrual. The per-tablet caffeine content of these medications ranges from 15 to mg 34 , Estimates of

consumption of each measure were calculated for each of the six age-sex-specific strata and for all strata combined. Caffeine intake mg from regular coffee was classified into approximate quartiles. Total caffeine intake was then categorized by using the same cutpoints, and cross-tabulations were constructed. Calculations were performed on estimates of daily intake within age and sex strata and for all strata combined. The cross-tabulations, which led to misclassification estimates, provided the basis for studying the effects of underestimating risk when regular coffee is used to approximate total caffeine intake. A hypothetical case-control distribution was constructed to provide various odds ratio estimates reflecting both threshold and dose-response associations between total caffeine intake and disease occurrence. Subjects were then reclassified into the caffeine exposure level based on caffeine estimates from coffee only, assuming nondifferential misclassification for cases and controls Once again, calculations were performed for all age-sex strata separately, in which misclassification estimates for each stratum were used, and then for the combined sample. RESULTS In all strata, the four main sources of caffeine were regular coffee, instant coffee, regular tea, and cola soft drinks tables 1 and 2. These sources accounted for 90â€”98 percent of total daily caffeine intake. Males table 1 had a higher caffeine intake than females table 2 , and respondents aged 45â€”59 years had a higher caffeine intake than the other two age groups.

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5: PPT - Why Misclassification of Exposure Status? PowerPoint Presentation - ID

We review some of the literature on the effects of exposure misclassification on the statistical analysis of case-control studies. In particular, we focus on evidence for exposure misclassification which may be different for cases and controls in studies of environmental tobacco smoke (ETS).

All Modules Information Bias Observation Bias From the previous section it should be clear that, even if the categorization of subjects regarding exposure and outcome is perfectly accurate, bias can be introduced differential selection or retention in a study. The converse is also true: These errors are often referred to as misclassification, and the mechanism that produces these errors can result in either non-differential or differential misclassification. Ken Rothman distinguishes these as follows: Similarly, misclassification of disease [outcome] is nondifferential if it is unrelated to the exposure; otherwise, it is differential. Misclassification of exposure status is more of a problem than misclassification of outcome as explained on page 6 , but a study may be biased by misclassification of either exposure status, or outcome status, or both. Nondifferential misclassification of a dichotomous exposure occurs when errors in classification occur to the same degree regardless of outcome. Nondifferential misclassification of exposure is a much more pervasive problem than differential misclassification in which errors occur with greater frequency in one of the study groups. Subjects with heart disease and controls without heart disease might be recruited and asked to complete questionnaires about their dietary habits in order to categorize them as having diets with high fat content or not. It is difficult to assess dietary fat content accurately from questionnaires, so it would not be surprising if there were errors in classification of exposure. However, it is likely that in this scenario the misclassification would occur with more or less equal frequency regardless of the eventual disease status. Nondifferential misclassification of a dichotomous exposure always biases toward the null. In other words, if there is an association, it tends to minimize it regardless of whether it is a positive or a negative association. The figure above depicts a scenario in which disease status is correctly classified, but some of the exposed subjects are incorrectly classified as non-exposed. This would result in bias toward the null. Rothman gives a hypothetical example in which the true odds ratio for the association between a high fat diet and coronary heart disease is 5. In other words, it resulted in bias toward the null. This additional nondifferential misclassification would result in even more severe bias toward the null, giving an odds ratio of perhaps 2. Note that If there are multiple exposure categories, i. Mechanisms for Nondifferential Misclassification Nondifferential misclassification can occur in a number of ways. Records may be incomplete, e. Subjects completing questionnaires or being interviewed may have difficulty in remembering past exposures. Note that if difficulty in remembering past exposures occurs to the same extent in both groups being compared, then there is nondifferential misclassification, which will bias toward the null. However, if one outcome group in a case-control study remembers better than the other, then there is a differential misclassification which is called "recall bias.

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6: Differential Misclassification of Exposure

It happens when exposure is unrelated to other variables (including disease), or when the disease is unrelated to other variables (including exposure). Bias introduced by non-differential misclassification is usually predictable (it goes towards the null value), but this isn't always the case.

Example of Confounding Hypothesis: Diabetes is a positive risk factor for coronary heart disease We survey patients as a part of the cross-sectional study asking whether they have coronary heart disease and if they are diabetic. The prevalence of coronary heart disease among people without diabetes is 91 divided by , or 3. Similarly the prevalence among those with diabetes is Our prevalence ratio, considering whether diabetes is a risk factor for coronary heart disease is The prevalence of coronary heart disease in people with diabetes is 3. We can also use the 2 x 2 table to calculate an odds ratio as shown above: Which of these do you use? They come up with slightly different estimates. It depends upon your primary purpose. Is your purpose to compare prevalences? Or, do you wish to address the odds of dibetes as related to coronary health status? We are evaluating the relationship of CHD to hypertension in non-diabetics. You can calculate the prevalence ratios and odds ratios as suits your purpose. Again, the results are highly significant! Therefore, our first two criteria have been met for hypertension as a confounder in the relationship between diabetes and coronary heart disease. Based on the biology, that is not the case. Diabetes in and of itself can cause coronary heart disease. Using the data and our prior knowledge, we conclude that hypertension is a major confounder in the diabetes-CHD relationship. What do we do now that we know that hypertension is a confounder? A cross-sectional study - Example Earlier we arrived at a crude odds ratio of 3. The Mantel-Haenszel method takes into account the effect of the strata, presence or absence of hypertension. If we limit the analysis to normotensives we get an odds ratio of 2. Among hypertensives we get an odds ratio of 3. Both estimates of the odds ratio are lower than the odds ratio based on the entire sample. This is an example of confounding - the stratified results are both on the same side of the crude odds ratio. This is positive confounding because the unstratified estimate is biased away from the null hypothesis. The null is 1. The true odds ratio, accounting for the effect of hypertension, is 2. The crude odds ratio of 3. In some studies you are looking for a positive association; in others, a negative association, a protective effect; either way, differing from the null of 1. You may have a priori knowledge of confounded effects, or you may examine the data and determine whether confounding exists. The question is not so much the statistical significance, but the amount the confounding variable changes the effect. We will talk more about this later, but briefly here are some methods to control for a confounding variable known a priori: Effect Modification interaction Effect modification: We see evidence of this when the crude estimate of the association odds ratio, rate ratio, risk ratio is very close to a weighted average of group-specific estimates of the association. Effect modification is similar to statistical interaction, but in epidemiology, effect modification is related to the biology of disease, not just a data observation. In the previous example we saw both stratum-specific estimates of the odds ratio went to one side of the crude odds ratio. With effect modification, we expect the crude odds ratio to be between the estimates of the odds ratio for the stratum-specific estimates. Consider the following examples: Breast cancer occurs in men at approximately a rate of 1. Breast cancer occurs in women at approximately a rate of This is about an fold difference. We can build a statistical model that shows that gender interacts with other risk factors for breast cancer, but why is this the case? Obviously, there are many biological reasons why this interaction should be present. This is the part that we want to look at from an epidemiological perspective. Consider whether the biology supports a statistical interaction that you might observe. Why study effect modification? Why do we care? The incorrect crude estimator e. If you do not sort out the stratum-specific results, you miss an opportunity to understand the biologic or psychosocial nature of the relationship between risk factor and outcome. To consider effect modification in the design and conduct of a study: Collect information on potential effect modifiers. Power the study to test potential effect modifiers - if a priori you think that the effect may differ depending on the stratum, power the study to detect a difference. To consider effect modification in the analysis of data: Again, consider what potential effect modifiers might be. Stratify the data

by potential effect modifiers and calculate stratum-specific estimates of the effect of the risk on the outcome; determine if effect modification is present. If so, Present stratum-specific estimates. Use Breslow-Day Test for Homogeneity of the odds ratios, from Extended Mantel-Haenszel method, or -2 log likelihood test from logistic regression to test the statistical significance of potential effect modifiers and to calculate the estimators of exposure-disease association according to the levels of significant effect modifiers. Stratifying by gender, we can calculate different measures. Look at the odds ratios above. The odds ratio for women is 6. Therefore, women are at much greater risk of diabetes leading to the incident coronary heart disease. For men, the odds ratio is 2. Is diabetes a risk for incident heart disease in men and in women? Is it the same level of risk? For men the OR is 2. The overall estimate is closer to a weighted average of the two stratum specific estimates. Gender modifies the effect of diabetes on incident heart disease. We can see that numerically because the crude odds ratio is more representative of a weighted average of the two groups. What is the most informative estimate of the risk of diabetes for heart disease? What is much more informative is to present the stratum specific analysis. During data analysis, major confounders and effect modifiers can be identified by comparing stratified results to overall results. In summary, the process is as follows: Estimate a crude unadjusted estimate between exposure and disease. Stratify the analysis by any potential major confounders to produce stratum-specific estimates. Compare the crude estimator with stratum-specific estimates and examine the kind of relationships exhibited. RR, OR is closer to a weighted average of the stratum-specific estimators; the two stratum-specific estimators differ from each other Report separate stratified models or report an interaction term. To review, confounders mask a true effect and effect modifiers means that there is a different effect for different groups. You have reached the end of the reading material for Week 3!!!

7: - Bias, Confounding and Effect Modification | STAT

2 Misclassification bias â€¢ Bias in informationleads to misclassification of exposure and/outcome status (maybe the interviewer or the responder that.

8: Exposure misclassification bias in studies of environmental tobacco smoke and lung cancer.

It is now recognized that exposure to environmental tobacco smoke (ETS) in the workplace and other settings outside the home may be equally as important as residential ETS exposure. This review examines the sources of misclassification in the assessment of workplace ETS exposure in questionnaire-based epidemiologic studies.

9: Information Bias (Observation Bias)

Bias arising from misclassification of exposure or disease status in an exposure-disease association study is often thought to be intractable and can go in any direction. We assessed the effect of disease misclassification on the OR estimation, by assuming that the exposure status was correctly measured.

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