

Introduction to Fixed Effects Methods

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1.1 The Promise of Fixed Effects for Nonexperimental Research

Every empirical researcher knows that randomized experiments have major advantages over observational studies in making causal inferences. Randomization of subjects to different treatment conditions ensures that the treatment groups, on average, are identical with respect to all possible characteristics of the subjects, regardless of whether those characteristics can be measured or not. If the subjects are people, for example, the treatment groups produced by randomization will be approximately equal with respect to such easily measured variables as race, sex, and age, and also approximately equal for more problematic variables like intelligence, aggressiveness, and creativity.

In nonexperimental studies, researchers often try to approximate a randomized experiment by statistically controlling for other variables using methods such as linear regression, logistic regression, or propensity scores. While statistical control can certainly be a useful tactic, it has two major limitations. First, no matter how many variables you control for, someone can always criticize your study by suggesting that you left out some crucial variable. (Such critiques are more compelling when that crucial variable is named). As is well known, the omission of a key covariate can lead to severe bias in estimating the effects of the variables that are included. Second, to statistically control for a variable, you have to measure it and explicitly include it in some kind of model. The problem is that some variables are

notoriously difficult to measure. If the measurement is imperfect (and it usually is), this can also lead to biased estimates. So in practice, causal inference via statistical adjustment usually runs a poor second to the randomized experiment.

It turns out, however, that with certain kinds of nonexperimental data we can get much closer to the virtues of a randomized experiment. Specifically, by using the fixed effects methods discussed in this book, it is possible to control for all possible characteristics of the individuals in the study—even without measuring them—so long as those characteristics do not change over time. I realize that this is a powerful claim, and it is one that I will take pains to justify as we go along. What is also remarkable is that fixed effects methods have been lying under our noses for many years. If the dependent variable is quantitative, then fixed effects methods can be easily implemented using ordinary least squares linear regression. When the dependent variable is categorical, somewhat more sophisticated methods are necessary, but even then the fixed effects approach is a lot easier than many alternative methods.

There are two key data requirements for the application of a fixed effects method. First, each individual in the sample must have two or more measurements on the same dependent variable. Second, for at least some of the individuals in the sample, the values of the independent variable(s) of interest must be different on at least two of the measurement occasions.

1.2 The Paired-Comparisons *t***-Test as a Fixed Effects Method**

Perhaps the simplest design that meets these two requirements is a before-after study. Suppose, for example, that 100 people volunteer to participate in a weight loss program. They all get weighed when they enter the study, producing the variable W_1 . All 100 people are then given a new medication believed to facilitate weight loss. After two months on this medication, they are weighed again, producing the variable W_2 . So we have measurements of weight on two occasions for each participant. The participants are off the medication for the first measurement and are on the medication for the second.

How should such data be analyzed? Before answering that question, let's first concede that this is not an ideal study design, most importantly because there is no control group of people who don't receive the medication at either time. Nevertheless, the application of fixed effects methods has all the virtues that I claimed above. The objective here is to test the null hypothesis that mean weight at time 1 is the same as mean weight at time 2, against the alternative that mean weight is lower at time 2. In this case, an easily applied fixed effects method is one that is taught in most introductory statistics courses under the name of paired-comparisons *t*-test or paired-differences *t*-test. The steps are:

- 1. Form $D = W_2 W_1$.
- 2. Calculate \overline{D} , the mean of D.
- 3. Test whether \overline{D} is significantly less than 0.

The third step is accomplished by dividing \overline{D} by its estimated standard error s / \sqrt{n} , where s is the sample standard deviation of D, and n is the sample size. The resulting test statistic has

a *t* distribution with n - 1 degrees of freedom under the null hypothesis (assuming that *D* is normally distributed).

If \overline{D} is significantly less than 0, what can we conclude? Well, we can't be sure that the medication caused the weight loss, because it's possible that something else happened to these people between time 1 and time 2. However, we *can* be sure that the difference in average weight between the two time points is not explainable by stable characteristics of the people in the study. In other words, we can be quite confident that the weight loss was *not* produced by changes in race, gender, parental wealth, or intelligence.

While this conclusion may seem obvious, it's helpful to consider a mathematical formulation as a way of introducing some of the ideas that underlie the more complicated models considered later. Let

$$W_{i1} = \mu + \alpha_i + \varepsilon_{i1}$$
$$W_{i2} = \mu + \delta + \alpha_i + \varepsilon_{i2}$$

where W_{i1} is the weight of person *i* at time 1, and similarly W_{i2} is the weight at time 2. In this model, μ is the baseline average weight, and δ denotes the change in the average from time 1 to time 2. The disturbance terms ε_{i1} and ε_{i2} represent random variation that is specific to a particular individual at a particular point in time. As in other linear models, one might assume that ε_{i1} and ε_{i2} each have an expected value of 0. The term α_i represents all the person-specific variation that is *stable* over time. Thus α_i can be thought to include the effects of such variables as race, gender, parental wealth, and intelligence.

When we form the difference score, we get

$$D_i = \delta + \left(\varepsilon_{i2} - \varepsilon_{i1}\right)$$

which shows that both the baseline mean μ and the stable individual variation α_i disappear when we compute difference scores. Therefore, the stable individual differences can have no effect on our conclusions, even if α_i is correlated with ε_{i1} or ε_{i2} .

The essence of a fixed effects method is captured by saying that each individual serves as his or her own control. That is accomplished by making comparisons *within* individuals (hence the need for at least two measurements), and then averaging those differences across all the individuals in the sample. How this is accomplished depends greatly on the characteristics of the data and the design of the study.

1.3 Costs and Benefits of Fixed Effects Methods

As already noted, the major attraction of fixed effects methods in nonexperimental research is the ability to control for all stable characteristics of the individuals in the study, thereby eliminating potentially large sources of bias. Within-subject comparisons have also been popular in certain kinds of designed experiments known as changeover or crossover designs (Senn 1993). In these designs, subjects receive different treatments at different times, and a response variable is measured for each treatment. Ideally, the order in which the treatments are received is randomized. The objective of the crossover design is not primarily to reduce bias, but to reduce sampling variability and hence produce more powerful tests of hypotheses. The rationale is that by differencing out the individual variability across subjects, one can eliminate much of the error variance that is present with conventional experimental designs in which each subject receives only one treatment.

By contrast, when fixed effects methods are applied to nonexperimental data, there is often an *increase* in sampling variability relative to alternative methods of analysis. The reason is that in the typical observational study, the independent variables of interest vary both within and between subjects. Suppose, for example, that one of the independent variables is personal income, measured annually for five successive years. While there might be considerable within-person variation in income over time, the bulk of the variation is likely to be between persons.

Fixed effects methods completely ignore the between-person variation and focus only on the within-person variation. Unfortunately, discarding the between-person variation can yield standard errors that are considerably higher than those produced by methods that utilize both within- and between-person variation. So why do it? The answer is that the between-person variation is very likely to be contaminated by unmeasured personal characteristics that are correlated with income. By restricting ourselves to the within-person variation, we eliminate that contamination and are much more likely to get unbiased estimates.

So what we're dealing with is a trade-off between bias and sampling variability. For nonexperimental data, fixed effects methods tend to reduce bias at the expense of greater sampling variability. Given the many reasons for expecting bias in observational studies, I think this is usually an attractive bargain. Nevertheless, one crucial limitation to fixed effects methods arises when the ratio of within- to between-person variance declines to 0: *fixed effects methods cannot estimate coefficients for variables that have no within-subject variation*. Hence, a fixed effects method will not give you coefficients for race, sex, or region of birth. Among adults, it won't be very helpful in estimating effects regression, even if there are no measurements of them. In fact, the control is likely to be much more effective than in conventional regression. And as we'll see later, you *can* include interactions between stable variables such as sex and variables that vary over time. But for most observational studies, fixed effects methods are primarily useful for investigating the effects of variables that vary within subject.

For experimental data, the situation with respect to bias and sampling variability is exactly reversed. Bias is eliminated by giving the same set of treatments to all subjects and by randomizing the order in which the treatments are presented. The result is approximately zero correlation between treatment and stable characteristics of the subjects, which means that there is no need for fixed effects to reduce bias. On the other hand, by design, all the variation on the independent variables (the treatments) is within subjects. So no information is lost by restricting attention to the within-subject variation. Indeed, standard errors can be greatly reduced by fixed effects methods because the error term has smaller variance.

1.4 Why Are These Methods Called "Fixed Effects"?

The name "fixed effects" is a source of considerable confusion. As we shall see, the basic idea is very simple. Consider the linear model

$$Y_{ii} = \beta_0 + \beta_1 x_{ii} + \alpha_i + \varepsilon_{ii}$$

where the *i* subscript refers to different persons and *j* refers to different measurements within persons—i.e., the same variable measured at different points in time. In conventional linear model terminology, $\beta_1 x_{ij}$ is described as a fixed effect because the x_{ij} terms are all measured values and β_1 is a fixed parameter. On the other hand, ε_{ij} is regarded as a random variable with a probability distribution, and we make certain assumptions about this distribution. For example, we might assume that ε_{ij} has a normal distribution with mean 0 and variance σ^2 . So the typical linear model has both fixed components and random components.

What about the term α_i , which we use to represent all stable characteristics of persons? Here we have an important choice between treating α_i as either fixed or random. Some methods, such as the so-called mixed models estimated by PROC MIXED, treat α_i as a random variable with a specified probability distribution (usually normal, homoscedastic, and independent of all measured variables). In the econometric literature, these are called random effects models. In fixed effects models, however, the α_i term is treated as a set of fixed parameters, which may either be estimated directly or conditioned out of the estimation process. Hence the name, "fixed effects."

Which is better, fixed effects or random effects? That depends on your objectives. In the preceding section, I already described the advantages and disadvantages of fixed effects methods. The advantages and disadvantages of random effects methods are the mirror image. Random effects methods do *not* control for unmeasured, stable characteristics of the individuals. That's because the α_i terms are virtually always assumed to be uncorrelated with the measured variables that are included in the model. On the other hand, with random effects you can estimate the effects of stable covariates such as race and gender. And because they use variation both within and between individuals, random effects methods typically have less sampling variability than fixed effects methods. Although the primary focus of this book is on fixed effects methods, I will often contrast those methods with alternative random effects approaches.

In my view, then, the decision to treat the between-person variation as fixed or random should depend largely on

- whether it's important to control for unmeasured characteristics of individuals
- whether it's important to estimate the effects of stable covariates
- whether one can tolerate the substantial loss of information that comes from discarding the between-individual variation

In the literature on ANOVA and experimental design, however, the decision between fixed and random effects is often described in quite different terms. Consider the following characterization:

Common practice is to regard the treatment effects as fixed if those treatment levels used are the only ones about which inferences are sought If inferences are sought about a broader collection of treatment effects than those used in the experiment, or if the treatment levels are not selected purposefully . . . , it is common practice to regard the treatment effects as random (LaMotte 1983, pp. 138–139).

According to this view, it would nearly always make more sense to treat the between-person variation represented by α_i as random, because then one can make inferences to broader populations than the sample in hand. But for the kinds of applications considered in this book, I believe that argument is mistaken, for two reasons. First, the purpose of including α_i in the equation is usually *not* because we want to estimate or test hypotheses about the between-person effects. Instead, the goal is to estimate the coefficients of *other* variables while *controlling* for unmeasured covariates and adjusting for lack of independence among the multiple observations for each person. For these purposes, it's irrelevant whether inferences about α_i can or cannot be generalized to larger populations. Second, fixed effects models are generally much less restrictive than random effects models, and thus these models are more likely to represent the data in a realistic way. In fact, for linear models it has been shown that random effects estimators are a special case of fixed effects estimators (Mundlak 1978).

1.5 Fixed Effects Methods in SAS/STAT

Different kinds of dependent variables require different kinds of fixed effects methods. SAS has a variety of procedures that can be used to implement fixed effects methods, although the original programmers may not have had such applications in mind. For linear models, PROC GLM is probably the most convenient procedure for performing a fixed effects analysis, although PROC REG and PROC MIXED can also be used with a little effort. For fixed effects logistic regression, PROC LOGISTIC is the most convenient procedure, but PROC GENMOD can be used in applications where there are only two observations per individual. For fixed effects Poisson regression, PROC GENMOD appears to be the only choice. For fixed effects survival analysis, PROC PHREG is the procedure of choice, although PROC LOGISTIC can be used in some circumstances. Finally, PROC CALIS can be useful for fitting fixed effects linear models with lagged endogenous variables.

1.6 What You Need to Know

To understand this book, how much you should already know depends on how far you want to go. To read chapter 2 on linear models, you need to be familiar with multiple linear regression. That means that you should know something about the assumptions of the linear regression model and about estimation of the model via ordinary least squares. Ideally, you should have a substantial amount of practical experience using multiple regression on real data and should feel comfortable interpreting the output from a regression analysis. As part of this knowledge, you must certainly know the basic principles of statistical inference: standard errors, confidence intervals, hypothesis tests, *p*-values, bias, efficiency, and so on.

To read chapter 3, you should have, in addition to the requirements for chapter 2, a knowledge of logistic regression at about the level of the first three chapters of my 1999 book *Logistic Regression Using the SAS System: Theory and Application*. That is, you should

understand the basic model for binary logistic regression, how to estimate that model via maximum likelihood, and how to interpret the results in terms of odds ratios. Some familiarity with PROC LOGISTIC is helpful, but not essential.

For chapter 4 on fixed effects Poisson regression, you should have a basic familiarity with the Poisson regression model, discussed in chapter 9 of *Logistic Regression Using the SAS System: Theory and Application.* I'll use PROC GENMOD to estimate the model, so previous experience with this procedure will be helpful.

For chapter 5 on survival analysis, some basic knowledge of the Cox proportional hazards model and partial likelihood estimation is essential. These methods are described in my 1995 book *Survival Analysis Using SAS: A Practical Guide*, along with instruction on how to use the PHREG procedure.

Finally, to read chapter 6 you should have some knowledge of linear structural equation models (SEMs) that include both observed and latent variables. A good introduction to this topic for SAS users is *A Step-by-Step Approach to Using the SAS System for Factor Analysis and Structural Equation Modeling* (Hatcher 1994).

I have tried to keep the mathematics to a minimal level throughout the book. In particular, there is no calculus and little use of matrix notation. Nevertheless, to simplify the presentation of regression models, I frequently use the vector notation $\beta x = \beta_0 + \beta_1 x_1 + ... + \beta_k x_k$. While it would be helpful to have some knowledge of maximum likelihood estimation, it's hardly essential. With regard to SAS, the more experience you have with SAS/STAT and the SAS DATA step, the easier it will be to follow the presentation of SAS programs. On the other hand, most of the programs presented in this book are fairly simple and short, so don't be intimidated if you're just beginning to learn SAS.

1.7 Computing

All the computer input and output displayed in this book was produced by and for SAS 9.1 for Windows. Occasionally I point out differences between the syntax of SAS 9.1 and earlier releases. I use the following convention for presenting SAS programs: All words that are part of the SAS language are shown in upper case. All user-specified variable names and data set names are in lower case. In the main text itself, both SAS keywords and user-specified variables are in upper case.

The output displays were produced with the SAS Output Delivery System. To avoid unnecessary distraction, I do not include the ODS statements in the programs shown in the book. However, the reader who would like to duplicate those displays can do so with the following code before and after the procedure statements:

```
OPTIONS LS=75 PS=3000 NODATE NOCENTER NONUMBER;
ODS LISTING CLOSE;
ODS RTF FILE='c:\book.rtf' STYLE=JOURNAL BODYTITLE;
---PROC STATEMENTS HERE---
ODS RTF CLOSE;
ODS LISTING;
```

All the examples were run on a Dell Optiplex GX270 desktop computer running Windows XP at 3 gigahertz with 1 gibabyte of physical memory.