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Introduction to Innovative Approaches to Longitudinal Data Analyses

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Studying human development and modeling complex developmental processes over time requires well-planned studies to collect longitudinal data. To plan such studies and to analyze the resulting wealth of longitudinal data, however, often seems to be a challenge for developmental researchers. When conducting longitudinal studies, investigators must consider issues such as identification of sample participants, participant retention, and appropriate timing of data collections. Analyzing the resultant data sets, by statistical or other means, might become difficult when researchers are confronted with questions such as: Which analytic strategy will yield the best answers to my research questions (e.g., focusing on inter-individual differences in intra-individual change patterns)? What is the most appropriate software tool for my project? How do I most effectively demonstrate causality within my longitudinal study? This special section aims at bringing together outstanding experts to discuss innovative approaches to longitudinal data analyses. In addition, examples of excellence in longitudinal studies within behavioral science will be introduced.

Four feature articles focus on innovative approaches to longitudinal data analyses. The first one by Nesselroade and Molenaar stresses the importance of investigating smaller samples over a long period of time (and several measurement occasions) rather than investigating very large samples over the short term. Von Eye in the second feature article discusses the meaning of change and the usefulness of statistical tools (e.g., ANOVA) to analyze change across time. The third paper by Bergman and Nurmi highlights the importance of a person-oriented framework to investigate human development over time, and, finally, Steyer et al. discuss the opportunities of intervention studies to investigate causal effects. These essays are accompanied by a series of Reports from the Labs exemplifying successful strategies for conducting longitudinal studies. These studies cover different research topics, age groups, time spans, and cultural and historical contexts. To begin with, Bynner reports on the opportunities and challenges of birth cohort studies. In addition, famous longitudinal studies are introduced by Poulton and Moffitt (Dunedin Multidisciplinary Health and Development Study, New Zealand), Tremblay (Montreal Longitudinal and Experimental Study, Canada), and Schaie and Willis (Seattle Longitudinal Study of Adult Cognitive Development, USA). In these reports, such methodological issues as sample selection, retention strategies, and analytic procedures to evaluate the data will be discussed. Finally, Sharma and Verma focus in their lab report on the usefulness of event sampling techniques to collect time-series of data across a short period.

In addition, this issue of the Bulletin presents a new section (Country Focus), wherein we aim at introducing in each issue developmental research foci of different nations around the globe. We are happy to start this section with a report from Guatemala by Ureta, Batz and Grazioso. This Bulletin also includes the Young Scholars' Corner by Joche Gayles, a report by Paul Oburu from the ISSBD workshop in Kenya, and a tribute to Professor Xiaojia Ge by Run Jin. The News Section includes the notes from our president Anne Petersen—her very last missive as president of ISSBD. We would like to thank Anne for her support of the editorial team, in particular for her encouraging support in its transition to becoming the ISSBD Bulletin. In addition, we are looking forward to a fruitful collaboration with the president-elect, Wolfgang Schneider.

We thank all authors of this Bulletin for their excellent contributions and investment of effort and time, and also thanks to Matthias Reitzle for expert consultation. By tackling the topic of developmental methodology in the special section of this ISSBD Bulletin, we aimed at meeting the needs of the members of the society, because a considerable number of respondents to the membership survey identified methodological aspects as a topic they wanted to see in a special section of the Bulletin. We are happy to respond to this input and hope that the readers of the Bulletin will find the papers interesting and stimulating for their own empirical research.

Finally, we are sad to announce that Bonnie Barber has left the editorial team. A special thanks to her for her extremely valuable input to the numerous issues of the ISSBD Bulletin (formerly ISSBD Newsletter) that we have worked on together since 2006.

of the collection of data before 1996 was David Magnusson. The data collections and database were supported by grants from the Swedish National Board of Education, the Swedish Committee for the Planning and Coordination of Research, The Bank of Sweden Tercentenary Foundation, and the Swedish Social Research Council.

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Analyzing Total, Direct and Indirect Causal Effects in Intervention Studies

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In longitudinal studies we aim at describing certain attributes of a single individual or group of individuals over time, studying change between different time points. Sometimes there are interventions to which all individuals are exposed (e.g., schooling) and sometimes one group of individuals is exposed while another group is not. In both cases we often are interested in the effects of interventions to which individuals might ($X = 1$) or might not be exposed ($X = 0$) on one or several outcome variables Y.

At first sight, in randomized intervention studies, analyzing the effect of the intervention seems to be an easy task. In fact, if randomization does not fail (e.g., due to systematic attrition), we can estimate the *total effect* of the intervention on the outcome variable Y by the difference between the means of Y in the two groups represented by ($X = 1$) and ($X = 0$). Furthermore, we can test the hypothesis that the total effect of the treatment is zero via the t-test for independent groups. If we are interested in the *conditional effects* of the intervention — e.g., given high, medium, or low pre-test scores — in order to see if the intervention effects differ between these conditions, we still can use traditional techniques of regression and/or analysis of variance in order to estimate and test these conditional effects.

However, traditional statistical tools are not sufficient any more when it comes to analyzing *direct and indirect effects* of the treatment with respect to a mediator variable, say M, even if we presuppose a perfect randomized

experiment. Not only that, we need to know the distinction between total, direct and indirect effects; we also have to rely on certain assumptions that are not guaranteed to hold even in a randomized experiment! While randomization guarantees that the mean difference between the intervention groups is an unbiased estimate of the total effect of the intervention, *randomization does not insure* that the conditional mean differences between intervention groups given fixed values m of the mediator variable M are unbiased estimates of the direct effect of the intervention for that value m of the mediator M. However, this is what many authors seem to assume when analyzing direct and indirect intervention effects, e.g., using well-known path analysis techniques (see, e.g., MacKinnon, 2008, Ch. 3).

Biased estimates of intervention effects are often disastrous for our substantive conclusions. If there is bias, this can mean that the true effects are strongly positive while we have estimates indicating that they are strongly negative and vice versa. We may also have estimates indicating no intervention effects although the intervention effects are strongly positive or strongly negative. This can be disastrous in the analysis of undesirable side effects of interventions. Note that bias is not a phenomenon due to inevitable sampling error. Instead we are talking about *systematic bias* that would even occur in infinitely large samples. In other words, bias does not only pertain to sample means and their differences, but also to *population means* and their differences.

Beyond the randomized experiment, ordinary mean differences between intervention groups usually do not estimate any more the total treatment effect. Instead, there are two kinds of biases that distinguish this mean difference from an estimate of the total intervention effect: the baseline bias and the effect bias. The *baseline bias* is due to selection into the intervention groups determined by *severity of disorder* or by *motivation for treatment*, for instance. If those subjects with a severe disorder tend to be in the intervention group rather than in the untreated control group and treatment effects are only small or medium, then the mean differences in the outcome variable between treatment and control can still be negative — erroneously indicating a negative treatment effect — even though the treatment effect is positive for each and every individual. In contrast, the *effect bias* is due to selection into the intervention groups determined by the size of the expected treatment effect. If diagnostics suggest that the treatment considered would be beneficial for the subject, and if this determines treatment assignment, then the differences between treatment and control in the outcome variable will not unbiasedly estimate the effect of the intervention.

To conclude: Outside the randomized experiment, traditional statistical methods including regression, analysis of variance, and structural equation modeling, are not sufficient to estimate and test intervention effects. Within the perfect randomized experiment, these skills only suffice to estimate and test the total effect as well as the conditional effects given covariates such as pre-tests, gender, or educational status. The analysis of direct and indirect effects — even within the randomized experiment, just like the analysis of causal effects in quasi-experiments — inevitably involves the *theory of causal effects* and its implications for the *design of studies* and *techniques of data analysis* that aim at the analysis of causal effects. In the remainder of this paper, we will outline these points. This outline is a summary of *Probability and Causality* (Steyer, et al., in press). We refer the reader to this book, both for the details of the theory and for the references to the contributions of other authors on this topic.

The Theory of Causal Effects

The theory of causal effects is based on atomic stratification. As already noted above, mean differences in the total population and in subpopulations can be biased. Hence, the basic idea is to construct causal effects on the conditional expectations of Y given $X = x$ in the smallest subpopulations. Oftentimes, these smallest subpopulations are the observational units (e.g., students, clients with a particular disorder, etc.), and in this case we can built the theory on the conditional expectations $E_{X=x}(Y \mid U = u)$ of the outcome variable Y given the observational unit u in treatment condition x. However, since, in many cases, there is still systematic variability within the observational units, we base the general theory on the conditional expectations of Y given $X = x$ in the most fine-grained strata, the *atomic strata*. These atomic strata are obtained by conditioning on *all* potential confounders, i.e., on all those variables that are prior or simultaneous to the treatment variable X. Typical and important variables that are *prior* to treatment are pre-tests of the outcome variable (e.g., *achievement before treatment, aggressivity before treatment*, if the Y is achievement and aggressivity, respectively). An example for a variable that is *simultaneous* to the treatment variable X (e.g., child's training vs. no child's training) is a second treatment variable Z (e.g., mother's training vs. no mother's training) that varies simultaneously to X.

True Outcome Variables and True Total Effects

The random variable τ_1 whose values are these conditional expectations of Y in the atomic strata given treatment ($X = 1$) is called the true-outcome variable in treatment 1. A particular value of τ_1 is the expected value of the outcome variable Y under intervention ($X = 1$) given a particular combination of values on all potential confounders. Correspondingly, the random variable τ_0 whose values are the conditional expectations of Y in the atomic strata given control ($X = 0$) is called the true-outcome variable under control (see Steyer et al., in press, for mathematical details). The difference $\delta_{10} = \tau_1 - \tau_0$ is then called the *true total effect variable*. Its values are the *true total effects* in the most fine-grained strata of the potential confounders. By definition, these true effects are unbiased, because, defining them, we condition on *all* potential confounders that might induce bias.

Average Total Effect and Conditional Total Effects

The expectation $E(\delta_{10}) = E(\tau_1) - E(\tau_0)$ of these true total effect variables over the distribution of the strata defines the

average total effect of intervention 1 compared to intervention 0. Similarly, various kinds of conditional total effects are defined by the difference between various conditional expectations of the true-outcome variables. Each of these kinds of conditional total effects provides specific information that might be of interest in intervention studies. An example in case is the *conditional total effect* $E(\delta_{10} \mid Z = z) = E(\tau_1 \mid Z = z) - E(\tau_0 \mid Z = z)$ given the value z (e.g., *very severe*) of the covariate Z (e.g., *severity of symptoms*).

True Direct and True Indirect Effects. The definition of the *true direct effect variable* is very similar to the definition of the true total effect variable. The definition implies that the direct effect does not have to be a single value but can vary depending on other variables. However, this time we not only condition on *all* potential confounders, i.e., on all those variables that are prior or simultaneous to the treatment variable X, but also on a specified mediator variable M that is 'posterior' or 'subsequent' to the treatment and prior to the considered outcome Y. In the simplest case, M is univariate variable; however, in general it may also consist of several univariate variables, i.e., $M = (M_1, \ldots, M_K)$. Hence, the random variable whose values are the conditional expectations of Y given an atomic stratum, a value of M, and treatment $X = 1$ is denoted by $\tau_{1;\,M}$. Its counterpart for control ($X = 0$) is $\tau_{0;\,M}$. The difference $\delta_{10;\,M} = \tau_{1;\,M} - \tau_{0;\,M}$ is then defined to be the *true direct effect variable* with respect to M, and the *true indirect effect variable* is the difference $\delta_{10} - \delta_{10;\,M}$.

Average and Conditional Direct and Indirect Effects

Again, the expectation $E(\delta_{10;\,M}) = E(\tau_{1;\,M}) - E(\tau_{0;\,M})$ of these true direct effect variables over the joint distribution of the strata and the mediator defines the *average direct effect* of intervention 1 compared to intervention 0. If this average direct effect is not informative enough, we may also be interested in the *conditional direct effect* $E(\delta_{10;\,M} \mid M = m) = E(\tau_{1;\,M} \mid M = m) - E(\tau_{0;\,M} \mid M = m)$ given the value m of the mediator M, or in the conditional direct effect $E(\delta_{10;\,M} \mid Z = z) = E(\tau_{1;\,M} \mid Z = z) - E(\tau_{0;\,M} \mid Z = z)$ given the value z of the covariate Z. Comparing $E(\delta_{10;\,M} \mid M = m_1)$ to $E(\delta_{10;\,M} \mid M = m_2)$ informs us about the difference in the conditional direct intervention effects between mediator values m_1 (e.g., high motivation after treatment) and m_2 (e.g., low motivation after treatment). In this way we can study if and how the direct intervention effect differs depending on the values of the mediator variable M. Similarly, comparing $E(\delta_{10;\,M} \mid Z = z_1)$ to $E(\delta_{10;\,M} \mid Z = z_2)$ informs us about the difference in the conditional direct intervention effects between covariate values z_1 (e.g., severe symptoms before treatment) and z_2 (e.g., no severe symptoms before treatment). In this way we can study how the direct intervention effect is modified by the values of the covariate Z. Of course, we may also consider the conditional expectations of $\delta_{10;\,M}$ given both m and z.

Similarly, we may consider the *average indirect effect* $E(\delta_{10} - \delta_{10;\,M}) = E(\delta_{10}) - E(\delta_{10;\,M})$ and different kinds of *conditional indirect effects*, the *conditional indirect effects* $E(\delta_{10} - \delta_{10;\,M} \mid M = m) = E(\delta_{10} \mid M = m) - E(\delta_{10;\,M} \mid M = m)$ given a value m of the mediator M, and the *conditional indirect effects* $E(\delta_{10} - \delta_{10;\,M} \mid Z = z) = E(\delta_{10} \mid Z = z) - E(\delta_{10;\,M} \mid Z = z)$ given the value z of a covariate Z.

Unbiasedness

The various kinds of causal effects defined above are of a purely theoretical nature, because they involve the theoretical variables τ_0 and τ_1 and/or $\tau_{0;\,M}$ and $\tau_{1;\,M}$. Nevertheless, they define what we *would like* to estimate. Furthermore, the theory provides knowledge about conditions that allow for a causal interpretation of estimable parameters. What *can* be estimated by the corresponding sample means are the conditional expected values $E(Y \mid X = x)$ of the outcome variable Y in treatment conditions, the conditional expected values $E(Y \mid X = x, Z = z)$ of the outcome variable Y given treatment x and value z of the covariate Z, the conditional expected values $E(Y \mid X = x, M = m)$ of the outcome variable Y given treatment x and value m of mediator M, and the conditional expected values $E(Y \mid X = x, Z = z, M = m)$ of the outcome variable Y given treatment x, value z of covariate Z, and value m of mediator M. Under certain conditions — some of which can be created by random assignment of units to treatment conditions and/or by careful selection of the covariates in the (possibly multivariate) covariate $Z = (Z_1, \ldots, Z_Q)$ — these conditional expected values can be *unbiased*.

The conditional expected value $E(Y \mid X = x)$ of Y in treatment x is called *unbiased* if $E(Y \mid X = x) = E(\tau_x)$. Similarly, the conditional expected value $E(Y \mid X = x, Z = z)$ of Y given treatment x and value z of covariate Z is called *unbiased* if $E(Y \mid X = x, Z = z) = E(\tau_x \mid Z = z)$. Furthermore, the conditional expected value $E(Y \mid X = x, M = m)$ of Y given treatment x and value m of mediator M is called *unbiased* if $E(Y \mid X = x, M = m) = E(\tau_{x;\,M} \mid M = m)$, and finally, the conditional expected value $E(Y \mid X = x, Z = z, M = m)$ of Y given treatment x, value z of covariate Z, and value m of mediator M is called *unbiased* if $E(Y \mid X = x, Z = z, M = m) = E(\tau_{x;\,M} \mid Z = z, M = m)$.

Unbiasedness of the conditional expectations $E(Y \mid X = x)$ can be created by *random assignment* of the unit to one of the treatment conditions. Unbiasedness of the conditional expectations $E(Y \mid X = x, Z = z)$ can be created by *conditional random assignment* of the unit to one of the treatment conditions given value z of Z and/or the appropriate selection of the covariates in $Z = (Z_1, \ldots, Z_Q)$. In contrast, unbiasedness of the conditional expectations $E(Y \mid X = x, Z = z, M = m)$ can *only* be created by appropriate selection of the covariates in $Z = (Z_1, \ldots, Z_Q)$. *Selecting the appropriate covariates is the only option in quasi-experimental studies, but also in the analysis of direct and indirect effects in the randomized experiment.*

Identification of Causal Effects

If the conditional expected values $E(Y \mid X = x)$ are unbiased, then the *average total effect* of the treatment is $E(Y \mid X = 1) - E(Y \mid X = 0) = E(\tau_1) - E(\tau_0)$. It is this difference that we estimate by the mean difference between treatment groups in a randomized study.

Next, consider the equation

$$E(Y \mid X, Z) = g_0(Z) + g_1(Z)X, \qquad (1)$$

which always holds for $E(Y \mid X, Z)$ if X is dichotomous, where $g_0(Z)$ denotes the *intercept function* and $g_1(Z)$ the *effect function*. Both functions are usually unknown but estimable.

According to this equation, the regression of Y on X given value z of Z is linear with intercept $g_0(z)$ and slope $g_1(z)$, both being functions of Z. If the conditional expected values $E(Y \mid X = x, Z = z)$ are unbiased, then the values $g_1(z)$ are the *conditional total treatment effects* given the value z of the covariate Z, and the expectation $E[g_1(Z)]$ is the *average total treatment effect* $E(\tau_1) - E(\tau_0)$.

If M is a mediator, we may consider the equation

$$E(Y \mid X, Z, M) = h_0(Z, M) + h_1(Z, M)X, \qquad (2)$$

which always holds for the regression $E(Y \mid X, Z, M)$ if X is dichotomous. If the conditional expected values $E(Y \mid X = x, Z = z, M = m)$ are unbiased, then the values of $h_1(z, m)$ are the $(Z = z, M = m)$-conditional direct treatment effects, the conditional expectations $E[h_1(Z, M) \mid M = m]$ are the *conditional direct treatment effects* given value m of the mediator M, the conditional expectations $E[h_1(Z, M) \mid Z = z]$ are the *conditional direct treatment effects* given value z of the covariate Z and the expectation $E[h_1(Z, M)]$ is the *average direct treatment effect* $E(\tau_{1;\,M}) - E(\tau_{0;\,M})$.

The various kinds of *indirect effects* are obtained by taking the differences between the corresponding total and direct effects. For instance, if both $E(Y \mid X, Z)$ and $E(Y \mid X, Z, M)$ are unbiased, $E[g_1(Z)] - E[h_1(Z, M)]$ yields the average indirect effect, whereas $E[g_1(Z) \mid Z = z] - E[h_1(Z, M) \mid Z = z]$ is the $(Z = z)$-conditional indirect effect and $E[g_1(Z) \mid M = m] - E[h_1(Z, M) \mid M = m]$ is the $(M = m)$-conditional indirect effect intervention effect.

Conclusions

Within perfect randomized intervention studies, mean differences between treatment groups can be causally interpreted as the *average total treatment effect*. Similarly, conditional mean differences between treatment groups given the value z of a covariate Z can be causally interpreted as the *(Z=z)-conditional total treatment effect*. Whenever feasible, randomization should be used when the total effect of an intervention or conditional total effects given covariates are of interest. If we want to study average and/or conditional total effects *beyond the randomized experiment*, i.e., in quasi-experimental and observational studies, we can also estimate various total effects provided that: (a) we select the appropriate covariate vector Z, (b) adequately estimate the regression $E(Y \mid X, Z)$ – which is not always linear – and (c) estimate and adequately test the various conditional and unconditional expectations such as $E[g_1(Z)]$ and $E[g_1(Z) \mid Z = z]$.

If we want to study direct and indirect effects in the randomized experiment or in quasi-experimental and observational studies, we have to (a) select an appropriate covariate vector Z such that the regression $E(Y \mid X, Z, M)$ is unbiased, (b) adequately estimate the regressions $E(Y \mid X, Z, M)$ – which again might be nonlinear – and (c) estimate and adequately test the various conditional and unconditional expectations such as $E[h_1(Z, M)]$, $E[g_1(Z, M) \mid Z = z]$, $E[g_1(Z, M) \mid M = m]$ or $E[g_1(Z, M) \mid Z = z, M = m]$. For these analyses of causal effects we recommend the program *EffectLite* (Steyer & Partchev, 2008).

A careful selection of all relevant covariates is essential for estimating the various causal effects mentioned above. Otherwise we cannot hope for unbiasedness. This also applies to the analysis of direct and indirect effects *within the randomized experiment*. Whenever possible always *include the pre-tests of the outcome variable and the pre-test of the mediator as covariates* in the vector Z of covariates! In many applications this will already prevent large biases in the estimation of the various causal effects. In a quasi-experiment, never trust an analysis of conditional or average total effects that does not include the pre-test of the outcome variable and even in a randomized experiment, never trust an analysis of conditional or average direct effects that does not include the pre-test of the mediator variable as a covariate.

Of course, what has been presented in this paper is just a short note on what needs and deserves a much longer and more detailed presentation. Many important topics such as propensity scores (see, e.g., Rosenbaum, 2002), instrumental variables (Greenland, 2000), and models for the analysis of individual effects (see e.g., Steyer, 2005) were not presented at all. Again, for these and other important issues we refer the reader to Steyer et al. (in press).

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