A nonlinear intervention analysis model for treatment B TEXAS Education reversal single-case designs.

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Introduction

Recently, most research in parametric models for single-case designs (SCDs) has focused on multiple baseline designs, while treatment reversal designs have received considerably less attention. In part this can be attributed to the relatively simple models required, as multiple baseline designs require only a single baseline phase and a single treatment phase. Well designed treatment reversals require more than two phases. The What Works Clearinghouse guidelines (Kratochwill et al., 2010) require that a treatment reversal design have at minimum two baseline and two treatment phases to meet standards.

Moreover, treatment reversal designs assume that the treatment is something that can be withdrawn and treatment effects will decay. In order to develop a model that is credible and casually interpretable, the effect of treatment must be modeled as a consequence of the repeated or constant application of treatment. Case-level models for SCDs have focused on linear change within phases (Gorsuch 1983; Center, Skiba, and Casey, 1985; Maggin et al. 2011), or models for nonlinear growth after treatment that do not also model decay after withdrawal (Moeyaert et al., 2014; Shadish, Kyse, Rindskopf, 2013; Hembry et al., 2015; Rindskopf 2013).

In this study we extend work from Pustejovksy (2013) that suggested a particular form of the set of models for the analysis of interventions in time series proposed by Box and Tiao (1976). We propose a simple nonlinear model that allows for both non-linear growth due to treatment as well as non-linear decay after its withdrawal. In addition, we examine the performance of the model using both normally-distributed errors and quasi-Poisson errors.

The nonlinear model

Let μ_t represent the mean behavioral outcome μ , which occurs across equally space time points t = 1, ..., n. Let Trt_t act as an indicator, where $Trt_t = 0$ when treatment does not occur during time t and $Trt_t = 1$ if treatment occurs during time t. Given some link function g(x), where $g(\mu_t)$ $= \eta_t$ then,

 $\eta_t = \beta_0 + \beta_1 (1 - \omega) / (1 - \omega^m) \sum_{t=1}^i \omega^{i-t} Trt_i.$

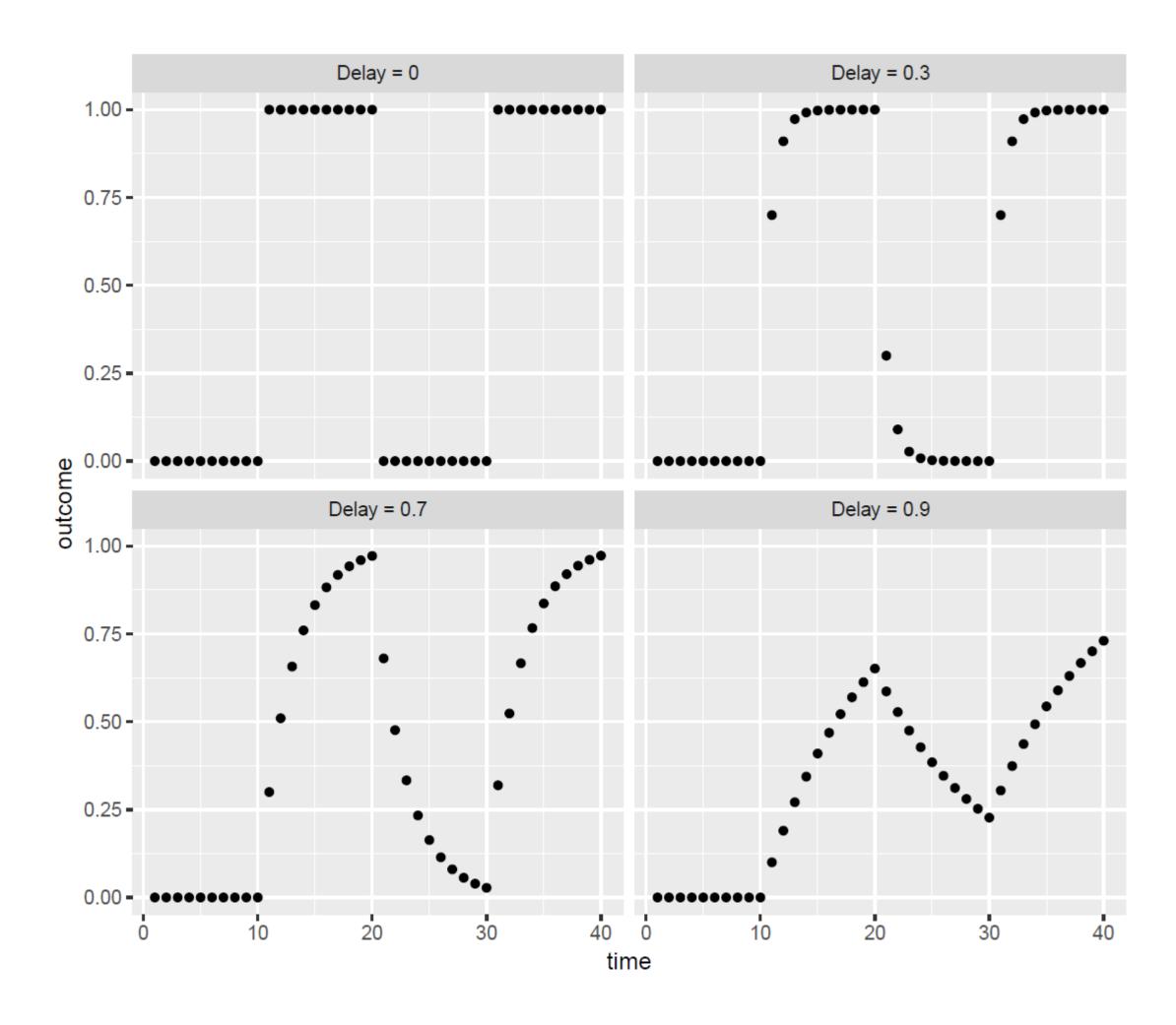
Under the identity link (normally-distributed errors):

- β_0 is the mean outcome in the absence of treatment.
- β_1 is the additive effect of treatment at the time point *m*.

Under the log link (quasi-Poisson errors):

- $\exp(\beta_0)$ is the mean outcome in the absence of treatment.
- $\exp(\beta_1)$ the multiplicative effect of treatment at time point *m*. Under either link:
- ω represents the delay of the effect of treatment, where $\omega \in [0,1)$.
- When $\omega = 0$, there is no delay the effect of treatment is immediate.
- As ω increases towards 1, full effect of treatment is increasingly delayed. A delay of 1 would represent full or infinite delay.

Figure 1. Example functional forms where $\beta_0 = 0$, $\beta_1 = 1$, across several values of ω .



The error structure

- We chose to examine the model using normally-distributed errors to match common modeling assumptions.
- We chose to examine the model using quasi-Poisson errors for two reasons:
- Quasi-Poisson errors more closely match the distribution of counttype measurements often used in the direct observation of behaviors in SCDs.
- Work by Rogosa and Ghandour (1991) using the alternating renewal process to model the direct observation of behavior suggests that, when frequency counts are used to directly observed behavior, there may be over- or under-dispersion in the variance with respect to the Poisson distribution. Quasi-Poisson errors operate under less restrictive assumptions than Poisson-distributed errors.
- Under both types of error structures, we assume that the errors are independent. This is a somewhat unusual assumption. However, Huitema and McKean (1998) pointed out that that many estimates of autocorrelation in the data may arise when the models do not fit the data well. If there are trends in the data that have not been modeled appropriately, the errors may be serially correlated.

Example: Thorne (2005)

Thorne (2008) examined the effects of a group contingency intervention on academic engagement and problem behaviors of at-risk students. We applied both forms of our model to the problem behavior data from the first four cases. We compared estimates from the normally-distributed errors model to a simple change-in-levels model. We compared the estimates from the quasi-Poisson errors model to the R_1 log-response ratio from Pustejovsky (2015). Our investigation focuses primarily on the estimates of treatment effects.

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Table 1. Estimates and standard errors for problem behavior from Thorne
 (2005) from the normally-distributed errors models.

Case	Nonlinear Treatment Effect	se	Change in Levels Treatment Effect	se
Participant 1	-18.97	2.32	-12.50	2.42
Participant 2	-17.39	2.37	-15.48	2.13
Participant 3	-6.17	1.35	-5.55	1.17
Participant 4	-20.48	2.72	-17.70	2.52

Table 2. Estimates and standard errors for problem behavior from Thorne
 (2005) from the quasi-Poisson errors model and the R_1 log-response ratio.

Case	Nonlinear Treatment Effect	se	R 1	Se	exp(Trt)	exp(<i>R</i> 1)
Participant 1	-1.91	0.21	-1.22	0.25	0.15	0.30
Participant 2	-2.25	0.22	-1.91	0.24	0.11	0.15
Participant 3	-0.74	0.15	-0.65	0.14	0.48	0.52
Participant 4	-1.38	0.18	-1.17	0.18	0.25	0.31

• In both cases, the treatment effects estimated by our nonlinear model are more extreme than the treatment effects estimated by the comparable model. This is expected, because the comparison models treatment effects are based on the treatment phase mean, whereas our model focuses on the treatment effects at or near the end of treatment.

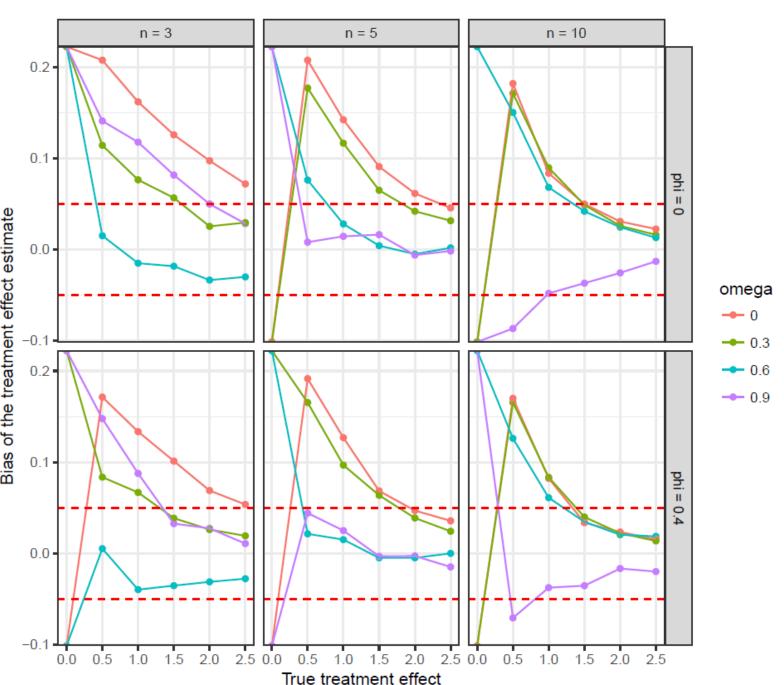
Simulation study

In order to investigate the properties of the model under conditions similar those seen in applied studies, we performed a simulation study. Since we cannot assume a lack of autocorrelated data, we examined both independent and serially correlated data. Serially correlated data was generated with an AR(1) structure. We generated normally-distributed AR(1) data using arima.sim in R and Poisson-distributed AR(1) data using binomial thinning (McKenzie, 1988), matching generated data to the model with the closely related error structure. All of the data were for ABAB designs. For the normally-distributed errors the total variance was fixed to 1 and the value of the variance of the innovations was calculated from the autocorrelation. We focused on the bias of the treatment effect and accompanying variance estimates.

Factor	Normally-distributed errors	Quasi-Poisson errors
Baseline	B ₀ : 0	exp(β ₀): 5, 15, 35
Treatment Effect	B ₁ : 0.5 to 2.50 in steps of 0.50	$exp(\beta_1)$: 0.20, and 0.50 to 2.50 in steps of 0.50
Delay Parameter	0 to 0.90 in steps of 0.30	0 to 0.90 in steps of 0.30
Points Per Phase	3,5,10	3,5,10

Table 3. Simulation Conditions

Figure 2 Plot of the relative bias of the treatment effect for the normally-distributed errors model.



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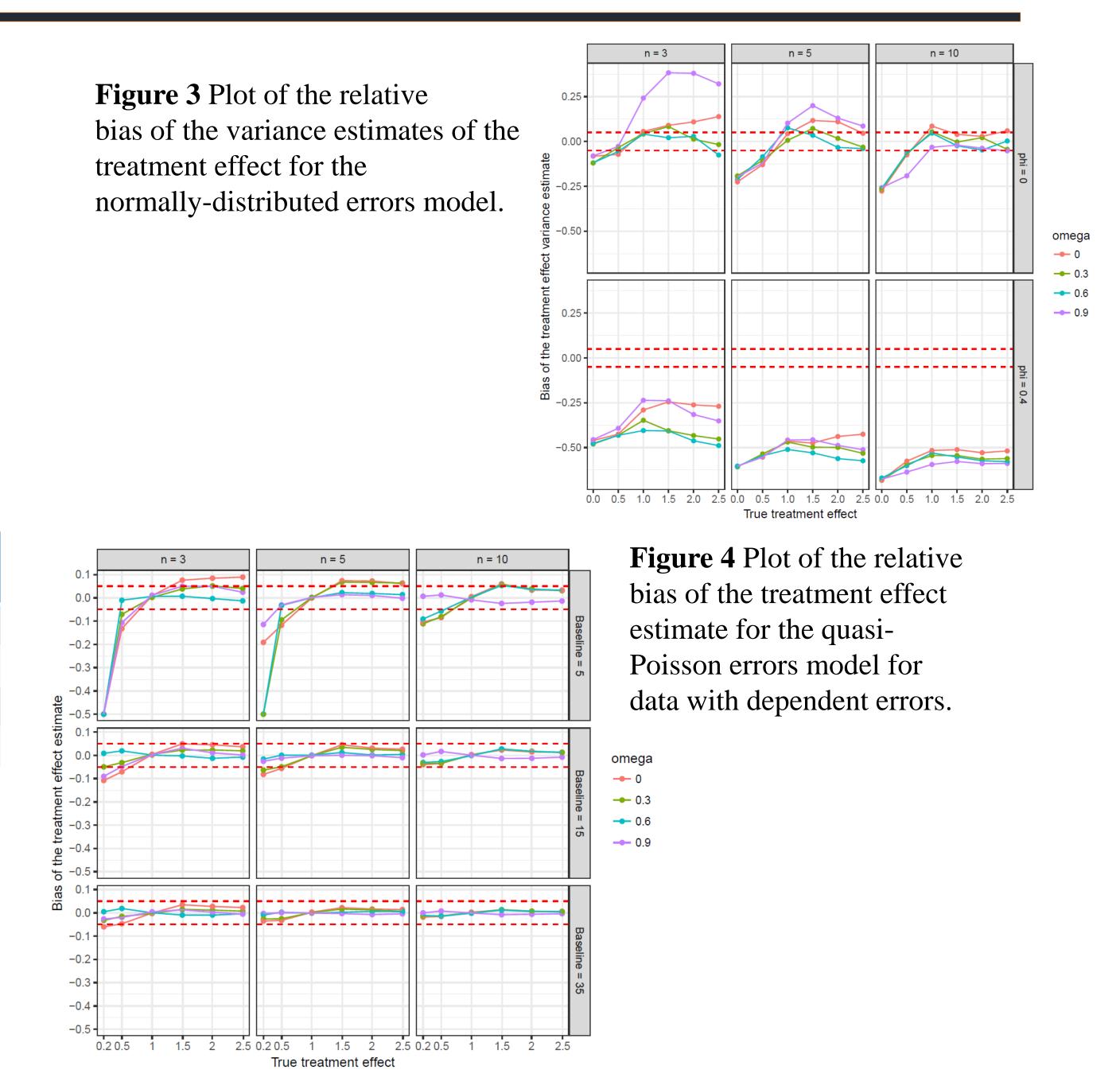
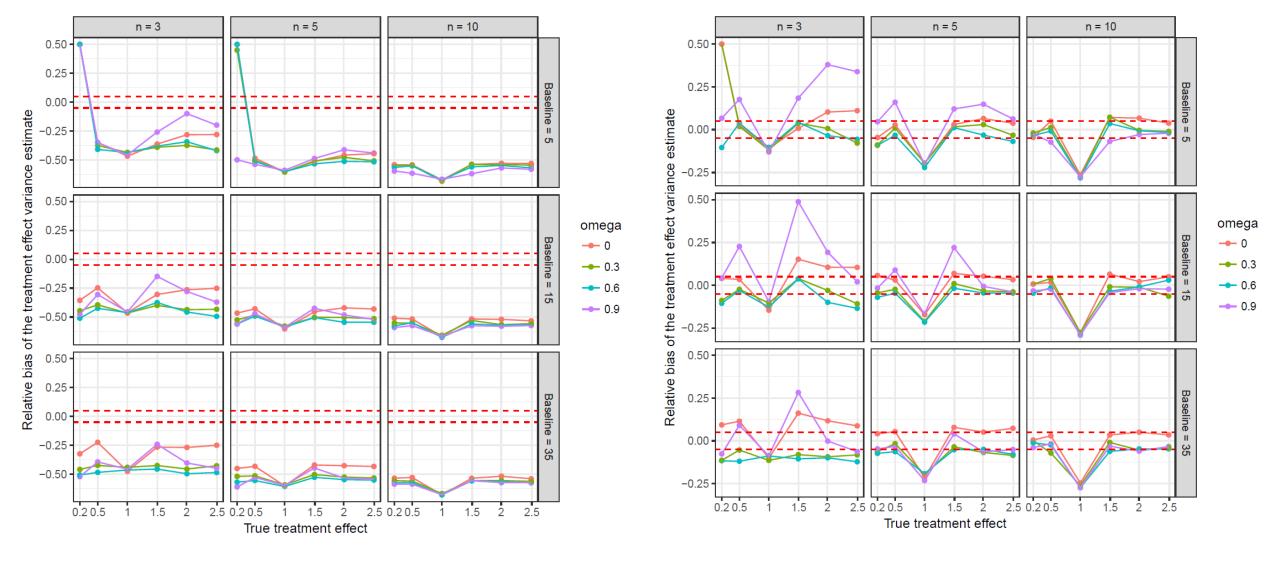


Figure 5 Plot of the relative bias of the variance estimate for data with dependent errors (left) and independent errors (right) under the quasi-Poisson errors model.



- In the normal errors model, when the treatment effect is sufficiently large, the estimates of the treatment effect are unbiased.
- Under the quasi-Poisson model, when the baseline is sufficiently large or the treatment effect is sufficiently large, the treatment effects are unbiased. When the errors are independent there is slightly less bias.
- When there is no autocorrelation and the treatment effect estimates are unbiased, the variance estimates are unbiased or close to unbiased. The quasi-Poisson model is potentially concerning, even under independence.
- When autocorrelation is present, the variance estimates are all biased. However, the use of meta-analytic methods with robust variance estimation proposed by Pustejovsky (2017) could be used to find the common effects within studies and still obtain unbiased variance estimates.