THE CYCLE OF VIOLENCE IN THE SECOND INTIFADA: CAUSALITY IN NONLINEAR VECTOR AUTOGRESSIVE MODELS

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ABSTRACT

Using daily fatalities data during the Second Intifada, we show that Israelis and Palestinians were engaged in tit-for-tat violence. However, this mutual violence was asymmetric: Israel reacted more rapidly and aggressively with a kill-ratio several times larger than that of the Palestinians. These results refute the claims of Jaeger and Paserman (2008) that, whereas Israelis reacted to Palestinian aggression, Palestinians did not react to Israeli aggression but randomized their violence instead. Our different conclusions stem from the fact that we (i) address the fundamental differences between the two sides in terms of patterns, timing and intensity of violence; (ii) apply nonlinear VAR models that are suitable for analyzing fatalities data when the linear VAR residuals are not normally distributed; (iii) identify causal effects using the principle of weak exogeneity rather than Granger-causality, and (iv) introduce the "kill-ratio" as a concept for testing hypotheses about the cycle of violence.

Keywords: The Israeli-Palestinian Conflict, tit-for-tat, nonlinear VARs
JEL-Classification: D74, H56

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1. INTRODUCTION

Jaeger and Paserman (2008) (JP, henceforth) examined the dynamics of the Palestinian-Israeli conflict during the Second Intifada (September 2000 - January 2005) along the lines of Schelling (1960). In particular, they aimed to determine whether the violent actions of one side lead the other side to retaliate or not. Using daily data on the number of fatalities of both sides to estimate their reaction functions in a linear VAR setting, they rejected the view that Palestinians and Israelis are engaged in "*tit-for-tat*" violence. Specifically, they argued that although Israel reacted to Palestinian violence, Palestinians did not react to violence committed by Israel.

Haushofer, Biletzki, and Kanwisher (2010) (HBK henceforth) have argued that JP's findings are overturned when Qassam rocket attacks from Gaza, in addition to Israeli fatalities, are included as part of the Palestinian reaction. Our purpose is to show that JP's results are not robust with respect to their choice of econometric methodology, even using their definition of violence. We draw attention to several econometric problems with JP's analysis and show that their results are overturned once these issues are addressed. Specifically, we show that Israelis and Palestinians were locked into tit-for-tat violence, that Israel reacted more quickly than the Palestinians, that the Israeli "long-run kill-ratio" (Palestinian deaths per Israeli death) is many times larger than the Palestinian's, and that the detected Granger causality is genuinely causal.

There are several aspects to our study. First, we show that JP's results are sensitive to the specification of the lag structure in their linear VAR model. Second, we show, as do Golan and Rosenblatt (2011), that because the VAR innovations are not normally distributed, chi-square and related tests may be misleading for purposes of inference and hypothesis testing. Whereas Golan and Rosenblatt (2011) proposed using a square-root variance stabilizing transformation to overcome the problem of non-normality of the VAR innovations, we suggest that the innovations are not normally distributed because the

dependent variables shown in Figure 1 are "limited" in the sense of Maddala (1983). Daily data on fatalities are discontinuous and the number of fatalities is mostly zero. Therefore, as a remedy we suggest estimating nonlinear VAR models that are designed for such limited dependent variables. We show that nonlinear VAR models that handle the discrete nature of the data reverse JP's results and indicate that both sides were engaged in a cycle of mutual violence. Third, we emphasize the difference between testing for Granger causality and genuine causality in the context of mutual violence.

Fourth, we introduce the "kill-ratio" as a metric for decomposing the cycle-of-violence. In the cycle-of-violence A attacks B, after which B attacks A, after which A attacks B again, and so on. JP calculate cumulative impulse responses generated by the VAR to quantify the cycle-of-violence, which measure the effect of A's initial attack on B on the eventual number of victims in A, i.e. after the cycle-of-violence has worked through. By contrast, the kill-ratio measures the eventual number of victims in A assuming that A does not respond to B's reprisals, i.e. the cycle-of-violence is defused. The kill-ratio measures the strength of B's reaction to A whereas the cumulative impulse response depends on the mutual reactions of A and B. We argue that the kill-ratio is a more accurate measure of B's response to A.

The paper is organized as follows. The remaining subsection of the introduction provides a brief geopolitical background of the Israeli-Palestinian conflict. Section 2 addresses the choice of the lag order of the linear VAR and suggests a suitable lag structure that is compatible with the dynamics of the conflict. Section 3 relates to the non-normality of the residuals of the linear VAR and advocates the usage of nonlinear VAR models. Section 4 concludes.

1.1 Geopolitical Background

On June 5 1967 the Six Days War broke out when Israel carried out a pre-emptive attack on Egypt after which Jordan and then Syria attacked Israel. As a result of the war Israel captured the Sinai Peninsula and the Gaza Strip from Egypt, the Golan Heights was captured from Syria, and the West Bank was captured from Jordan. Following the 1978 peace agreement

with Egypt, Israel returned the Sinai Peninsula to Egypt. In 1980 Israel annexed the Golan Heights. Prior to the 1993 peace agreement between Jordan and Israel, Jordan gave up territorial claims to the West Bank. The First Intifada (Palestinian popular uprising) which began in December 1987 and ended in 1990 paved the way to the Oslo Accords in September 1993 between Israel and the Palestine Liberation Organization, which established a Palestinian Authority with autonomy over large sections of the West Bank and Gaza. The Oslo Accords never led to a full and permanent peace agreement between Israel and the Palestinians despite numerous diplomatic attempts.

The Second Intifada (also known as Al-Aqsa Intifada), erupted in September 2000 following a provocative visit by Israel's then opposition leader Ariel Sharon to the Temple Mount (Al-Aqsa) considered as the third holiest site of Islam. This wave of violence was more violent and protracted than the First Intifada; Palestinians' violent actions/reactions included demonstrations, stone throwing, gun firing and most distinctly suicide bombings against Israeli civilian and military targets. Israel's actions/reactions included using rubber and live ammunition, house demolitions, closures and curfews, and targeted killing of Palestinian leaders and activists. This episode (September 2000 to January 2005) of intense violence have claimed the lives of more than 3200 Palestinians and about 1000 Israelis.

In July 2005 Israel withdrew unilaterally from the Gaza Strip and in June 2007 Hamas overthrew the Palestinian Authority in the Gaza Strip. Following the firing of rockets on Israel by Hamas, Israel retaliated by military incursions into the Gaza Strip. There were several such incursions since 2006, the last occurring in July 2014 (Operation "Protective Edge"). These incursions are not intifadas.

2. LINEAR VECTOR AUTOREGRESSIONS

JP employed a linear VAR framework to estimate the Israeli and Palestinian empirical reaction functions. Specifically, they estimated the following linear VAR using daily data on Israeli (ISR) and Palestinian (PAL) fatalities:

$$PAL_t = \alpha_i + \sum_{j=1}^{q} \beta_{ij} ISR_{t-j} + \sum_{j=1}^{q} \gamma_{ij} PAL_{t-j} + \delta_i X_t + u_t \quad (1)$$

$$ISR_t = \alpha_p + \sum_{j=1}^{q} \beta_{pj} PAL_{t-j} + \sum_{j=1}^{q} \gamma_{pj} ISR_{t-j} + \delta_p X_t + v_t \quad (2)$$

Equation (1), in which the number of Palestinian fatalities resulting from Israeli attacks serves as the dependent variable, represents the Israeli reaction function. The coefficients β_i denote Israel's reaction to Israeli fatalities resulting from Palestinian attacks. *X* is a vector of geopolitical variables that may shift the reaction function and includes the length in kilometers of the completed Separation Barrier between the West Bank and Israel, the day of the week, and dummies for seven political milestones during Intifada 2.[1] Finally, *u* is an innovation induced by Israeli violence, assumed to be asymptotically normally distributed, which is independent but not necessarily identically distributed. Equation (2) represents the Palestinian reaction function, in which the parameters of interest are denoted by β_p, and *v* is an innovation induced by Palestinian violence, assumed to have a similar distribution to *u*, but is independent of *u*. Notice that a common VAR order (q = 14 days) is assumed by JP to apply in both equations.

In each of the reaction functions JP were interested in the joint significance of own fatalities, i.e. the β coefficients. Thus, in Equation (1) if the estimated β_i coefficients are jointly significant then, according to JP, Israelis are said to react to Palestinian-inflicted violence. Similarly, if in Equation (2) the estimated β_p coefficients are jointly significant, then Palestinians are said to react to Israeli-inflicted fatalities.

The data in VAR models should be stationary. JP assumed by default that these fatality data are stationary. Not surprisingly, as Intifada 2 intensified (Figure 2) the data appear to be nonstationary. However, the ADF and ADF-GLS statistics reported in Table 1

[1] Definitions of the variables, data sources and some summary statistics are available in JP (2008).

clearly reject the unit root hypothesis for ISR and PAL. In finite samples, however, these results may be indicative only because the data are discontinuous[2].

Column 1 in Tables 2 and 3 replicate JP's estimated reaction functions for lag order (q) of 14 days for both variables. We also replicate their chi square Granger-causality tests, which show that lagged Israeli fatalities in the Israeli reaction function are jointly significant at the 4% level, whereas lagged Palestinian fatalities in the Palestinian reaction function are not statistically significant at conventional levels (p-value = 0.23). JP also conducted several robustness checks, including alternative number of lags and using weekly, biweekly and monthly data,[3] the results of which led them to conclude that "the Israelis react in a significant and predictable way to Palestinian violence against them, but no evidence that the Palestinians react to Israeli violence."

JP did not report tests for serial correlation in the VAR innovations. Significant serial correlation might indicate that their lag order of $q = 14$ is too restrictive. We therefore report robust lagrange multiplier (LM) tests for up to 6th order serial correlation which allow for the fact that the VAR innovations are clearly heteroskedastic according to White's LM test.[4] It turns out that we cannot reject the hypothesis that the VAR residuals are serially independent, suggesting that JP's 14 day lag length is sufficiently long. Since the VAR residuals are heteroskedastic, OLS standard errors are incorrect. We therefore report robust standard errors for the estimates as JP did. Had the residuals also been serially correlated it would have been appropriate to calculate HAC standard errors as suggested by Newey and West (1987).

[2] The critical values for DF are generated by assuming that the data generating process is a random walk with drift, i.e. the data are continuous (Dickey and Fuller 1979). If the data are not continuous e.g. because they are binary or because they are counts, ADF may nonetheless be valid if they tend asymptotically to continuous Brownian motion.

[3] Although the problems associated with daily observations might be mitigated, these frequencies are not compatible with the observed dynamics of the conflict. Indeed their results at these frequencies are weak and contradict with their baseline; Israel is found to react in the case of 4 weekly lags (but not the two weekly lags=14 daily lags), and in the case of one monthly lag, and is not reacting at any of their biweekly lag orders (including one and two lags). Palestinians are not found to react in any of their specifications.

[4] Also known as the robust Durbin's alternative test. By contrast, the standard LM test misleadingly implies that the VAR innovations are serially correlated. We note that many investigators use the standard LM test when its robust alternative is appropriate.

JP conclude by using their VAR model to calculate cumulative impulse responses (*CIR*) for Palestinians and Israelis: "We find that one Palestinian fatality raises the cumulative number of Israeli fatalities by 0.25 (standard error 0.15) in the long-run. In contrast, one Israeli fatality raises the number of Palestinian fatalities by 2.19 (standard error 1.15), nearly a factor of ten greater than that caused by a Palestinian fatality." (JP, p 1603). In column 1 of Tables 2 and 3 we report our replications of these CIRs, which turn out to be slightly lower[5] than JP's, although the ratio between them is approximately ten as claimed by JP.

CIR measures the strength of the cycle-of-violence because it allows for both sides to react to the violence committed by the other. We introduce the "kill-ratio" ratio (*k*) as an additional metric, which calculates the cumulative number of fatalities suffered by the belligerent party assuming that it practices restraint by turning the other cheek when counter-attacked by the injured party. Whereas *CIR* is bilateral, *k* is unilateral because the belligerent party does not respond to the reaction of the injured party. In summary, *k* measures the long-run number of fatalities that would occur if the cycle-of-violence was defused. *CIR* is naturally larger than *k*; the difference between them measures the reduction in violence that would have been achieved had the belligerents practiced restraint. Both *CIR* and *k* are legitimate metrics that are salient to the study of the cycle-of-violence.

The Israeli kill-ratio, k_i, denoting the number of Palestinians eventually killed by Israel for every Israeli fatality (reported at the bottom of Table 2) is calculated from the coefficients of Equation 1 as:

$$k_i = \frac{\beta_i}{1-\gamma_i}$$

where $\beta_i = \sum_{j=1}^{q} \beta_{ij}$ and $\gamma_i = \sum_{j=1}^{q} \gamma_{ij}$.

Similarly, the long-run Palestinian kill-ratio, k_p, (reported at the bottom of Table 3) is calculated from the coefficients of Equation 2 as:

$$k_p = \frac{\beta_p}{1-\gamma_p}$$

[5] This difference and the difference between standard errors may be due to the fact that we calculate CIR analytically whereas JP calculated it numerically over 60 periods.

where $\beta_p = \sum_{j=1}^{q} \beta_{pj}$ and $\gamma_p = \sum_{j=1}^{q} \gamma_{pj}$.

Appendix I shows that the cumulative impulse responses for Israel (CIR_i) and Palestine (CIR_p) are respectively:

$$CIR_i = \frac{\beta_i}{(1-\gamma_i)(1-\gamma_p)-\beta_i\beta_p}$$

$$CIR_p = \frac{\beta_p}{(1-\gamma_i)(1-\gamma_p)-\beta_i\beta_p}$$

Therefore, the kill-ratios are special cases of the cumulative impulse responses when the coefficients of the other equation are ignored ($\beta_p = \gamma_p = 0$ for the Israeli *CIR* and $\beta_i = \gamma_i = 0$ for the Palestinian *CIR*). As noted in Appendix 1, the relative kill-ratio may be greater or smaller than the relative *CIR* since:

$$\frac{CIR_i}{CIR_p} = \frac{(1-\gamma_i)k_i}{(1-\gamma_p)k_p}$$

JP focus on the statistical significance of β. However, *k* might be statistically significant despite the fact that β is not statistically significant because *k* also depends on γ. The same applies a fortiori to *CIR*, which depends on β and γ for both sides. Notice, however, that relative *CIR* depends on relative β; so γ does not matter, whereas relative *k* depends on β and γ for both parties. Notice also, that relative *CIR* may be greater or smaller than the relative kill-ratio.

In column 1 of Table 2 (JP's original specification) the Israeli kill-ratio is 1.32 while *CIR* is 1.62, implying that bilateral violence increases the number of Palestinian fatalities by 0.3. The standard deviations of *k* and *CIR* are calculated using the delta method which accounts for the covariance between the estimates of β and γ. The p-values of Israeli *k* and *CIR* suggest that these metrics are statistically significant. The Palestinian kill-ratio (column 1 of Table 3) is 0.094 and its p-value (0.028) indicates that it is statistically significant at conventional levels. This means that Palestinians do react to Israeli violence, but they only manage to kill 0.094 Israelis for each Palestinian fatality. By contrast CIR_p is 0.179 (p-value = 0.038) which means that the cycle of violence leads to almost doubling the number of Israeli fatalities for each Palestinian fatality. Relative *CIR* is 9.05 in Israel's favor, whereas

the relative kill-ratio is 14.04, so *CIR* understates the degree of asymmetry in the cycle-of-violence.

The kill-ratios appear to contradict the claim of JP about Granger-non-causality for Palestinians. Whereas the kill-ratio test concerns the joint statistical significance of the estimates of β and γ, Granger causality is only concerned with the former. If the covariance between the estimates of β and γ happened to be zero, Granger non-causality would imply that the kill-ratio is not statistically significant. In general, however, the absence of Granger causality does not necessarily imply that the kill-ratio is zero. Therefore, we conclude from the kill-ratios and *CIR*s that there was two-way tit-for-tat violence, but its intensity is asymmetrical in that the Israeli reaction is 14 times greater than the Palestinians'.

Note also that Granger causality does not necessarily imply that β_p (sum of coefficients of own fatalities in Palestinian reaction function) is positive. Alternatively, if $\beta_p = 0$ Palestinian fatalities would Granger cause Israeli fatalities if positive and negative terms in β_{pj} happened to cancel each other out. However, if $\beta_p = 0$ the overall Palestinian response is zero despite Granger causality. Therefore, what matters for the cycle of violence is not Granger causality, but the statistical significance of β_p and β_i, which are included in the kill-ratios. Hypothetically, there would be no cycle of violence if β_p and β_i were zero despite evidence of mutual Granger causality.

2.1 Lag Order in Jaeger and Paserman's Linear VAR

We agree with HBK that there is no reason why the lag orders have to be the same for Palestinians and Israelis,[6] and suggest applying the general-to-specific (GTS) dynamic specification search methodology of Hendry (1995) by initially setting the lag order to be sufficiently large to ensure that the innovations are serially uncorrelated. Typically, serial correlation may be induced if the lag order is too short. This initial model, or unrestricted model, avoids pre-test bias because the lag length is sufficiently long to capture protracted

[6] HBK optimized *q* at 5 days for Palestinian fatalities, 4 for Israeli fatalities and 22 days for Qassam rocket attacks.

dynamic effects. We continue to use the robust Durbin's alternative test to detect serial correlation in the VAR residuals.

The asymmetric dynamic specification of the VAR recognizes the underlying differences in the ability of Israelis and Palestinians to attack each other. While Israel can react rapidly to Palestinian attacks, Palestinians faced severe movement restrictions within the Occupied Territories and are generally denied access to Israel. Therefore, they took longer to retaliate. Furthermore, Palestinian reactions might not materialize due to Israeli security forces' success in thwarting attacks, last minute regrets by Palestinian suicide bombers, or premature detonations as a result of "industrial accidents." Moreover, whereas Israel has a unified command, the Palestinian command is decentralized and many acts of violence are undertaken by individuals.

The VARs in JP's specification (column 1 of Tables 2 and 3) are clearly over-parameterized because numerous lag coefficients are not statistically significant. Over-parameterization may induce pre-test bias in favor of falsely rejecting Granger causality. Suppose, for example, that the true VAR order (q) is one, and that X Granger-causes Y. However, a second order VAR is estimated. The second lag of X is not statistically significant and a joint test for the significance of both lags may falsely reject the hypothesis of Granger-causality.

Just as setting the lag order too small may induce pre-test bias, so may setting it too large. The natural and most common way to determine the number of lags in the VAR system, rather than setting it arbitrarily as is the case in JP, is to utilize goodness-of-fit and information criteria tests. Restricting both variables (Israeli and Palestinian fatalities) in the two reaction functions (equations 1 and 2) to be the same and setting the maximum lag to be 14 resulted in an optimal lag order of 8 based on Likelihood Ratio (LR), Final Prediction Error (FPE), and Akaike Information Criteria (AIC),[7] as reported in column 2 of Tables 2 and 3. The innovations in this VAR model continue to be serially uncorrelated, and the lags of both the Israeli and Palestinian fatalities in their own reaction functions are jointly

[7] Other tests indicated even lower lag orders.

significant at p-values 0.027 and 0.072 respectively. Thus, switching from an arbitrary symmetric lag order of 14 to an optimal symmetric lag order of 8 overturns JP's conclusions and indicates that both sides react to each other's violence. This conclusion is derived from Granger-causality tests as well as from the significance of the kill-ratios (0.006 and 0.017, respectively) as shown at the bottom of column 2 of Tables 2 and 3.

In order to deal with the problem of over-parameterization and allow for a flexible setting in which Israelis and Palestinians can react asymmetrically we advocate applying the GTS method. GTS involves estimating a restricted or parsimonious VAR model which retains the long-run properties of the unrestricted JP model (column 1 of Tables 2 and 3), and which does not induce serial correlation resulting from dynamic misspecification. Since the restricted model may depend on the order of the restrictions imposed, we apply two restriction strategies. In the first we drop all the lags with t-statistics less than 1 in absolute value,[8] until the restricted model has no such lagged variables (equivalently, until adjusted R^2 is maximized). In the second, we sequentially drop the lag with the smallest absolute t-statistics that is less than one, and continue until the lowest t-statistic is at least 1. Both strategies produced identical results (that is, arrived at the same final specification) for each reaction function, the Israeli and the Palestinian, as reported in column 3 of Tables 2 and 3.

The results in Table 3 show that Palestinian fatalities Granger-cause Israeli fatalities, overturning the results of JP. This happens simply because JP's model was over-parameterized[9]. The LM test statistics for serial correlation reported in column 3 in Tables 2 and 3 indicate that the VAR innovations remain serially uncorrelated within equations. We also checked for (up to 14^{th} order) serial correlation between the innovations (results not shown), which show that the innovations are serially uncorrelated between and within equations. The largest of these cross autocorrelations is only 0.0097. By contrast, the

[8] Since according to Haitovsky (1969) adjusted R^2 remains unchanged when a variable with t-statistic = 1is dropped.

[9] Ironically, JP mention that over or under-parametrization may affect Granger causality tests, but they do not check for this.

contemporaneous innovations are slightly correlated at 0.06. However, consistency of the estimates of the VAR parameters does not require these innovations to be independent.

The absence of serial correlation within equations, e.g. equation (1), means that omitted variables cannot be correlated with lagged values of ISR, for otherwise these omitted variables would have to be serially correlated, which would contradict the result that u is serially uncorrelated. The absence of serial correlation between equations, i.e. u_t is not correlated with lags of v, and v_t is not correlated with lags of u, means that omitted variables in equation (2) cannot be correlated with lagged values of ISR in equation (1), and omitted variables in equation (1) cannot be correlated with lagged values of PAL in equation (2). This also means that lagged values of ISR are weakly exogenous for β_i in equation (1) and lagged values of PAL are weakly exogenous for β_p in equation (2). Therefore, the parameter estimates of this GTS-based restricted VAR model have a causal interpretation rather than being merely Granger-causal, because the VAR innovations are serially uncorrelated both within and between equations.

2.2 Robustness Checks of the Linear VAR Model

As expected AIC and BIC are smallest in column 3 of Tables 2 and 3 and are significantly smaller than their counterparts in columns 1 and 2. On the whole the restricted GTS model is not sensitive to choice of model selection criteria despite the fact that BIC penalizes model complexity more heavily.

We have also experimented with an alternative threshold for GTS lag elimination of $|t| < 1.5$. The results of the two elimination schemes that we outlined above are similar to what we reported for the case of $|t| < 1$; the lagged Israeli fatalities in the Israeli reaction function are jointly significant at the 3.5% level when all lags with $|t| < 1.5$ are dropped at once and at the 0.8% level when we only drop the lag with the smallest absolute t-statistic that is less than 1.5. Moreover, lagged own fatalities in the Palestinian reaction function are jointly significant at the 2% level for both elimination strategies. Thus, our conclusions are kept intact under the alternative t-statistic threshold.

In addition to using GTS to allow for a flexible lag structure that accords with the conflict dynamics, we have estimated another form of flexible VAR in which the lag

structures of Israeli and Palestinian fatalities were allowed to differ, and were based on minimal information criteria (like AIC) or maximal Adjusted R Squared. The results (not reported but available upon request) are similar to those obtained by GTS i.e. causality is bi-directional and the cycle of violence was mutual.

Although most,[10] including JP, consider that Intifada 2 ended by late 2004 or early 2005, some such as HBK believe it ended after 2005. Therefore, in Table 4 we extend the observation period to the end of 2007.[11] During the extended period there were no Israeli fatalities on 96% of the days (as opposed to 81% during the actual Intifada days) and no Palestinian fatalities on 63% of the days (compared to 39% during the Intifada). This extension exacerbates the problems associated with linear VARs because of the dominance of zeros. However, it supports JP's contention that when q=14 Palestinians did not react to their own fatalities. Nevertheless, the Palestinian kill-ratio is statistically significant (0.063 p-value 0.043) although this result is slightly weakened according to the second GTS restriction strategy. As in Table 3 the GTS model in Table 4 overturns the former result, but less strongly. The Israeli kill-ratio is 0.981 in the extended sample instead of 1.23, and its Palestinian counterpart is 0.053 instead of 0.094. In summary, extending the data to December 2007 does not change our qualitative criticism of JP's conclusions, but the cycle of violence is quantitatively less vicious. This is expected because Intifada 2 ended in early 2005 rather than late 2007.

2.3 Normality

Investigators typically rely on asymptotic theory in estimating parameters and in carrying out hypothesis tests. JP are no exception. By using chi-square tests for Granger causality they implicitly rely on asymptotic theory by assuming that their VAR innovations are approximately normal. It is well known that the exact distribution does not have to be normal under asymptotic normality. However, the deviation from the normal distribution is not expected to be serious. Therefore, asymptotic normality may be a safe assumption for

[10] See for example Wikipedia for "Second Intifada".
[11] We thank Johannes Haushofer for providing the extended sample used in HBK (2010).

hypothesis testing (Davidson and MacKinnon, 2009 p. 660). However, in finite samples the VAR innovations might not be even approximately normal, in which case the assumption of normality may induce size distortions in empirical tests.

In discontinuous autocorrelated time series convergence induced by the central limit theorem may be slow for two reasons. First, it is well-known that in continuous autocorrelated (but stationary) time series the central limit theorem applies more gradually as the sample size increases (Gordin's CLT). It is for this reason that e.g. Hendry (1995) among others tests for normality in the residuals of time series models. Second, when the data are discontinuous the OLS residuals are more likely to be non-normal.in finite samples. For example, when the dependent variable is binary the residuals have mass point at $y = 0$ and $y = 1$. Although these residuals maybe asymptotically normal, convergence is naturally slower the more they deviate from normality. In JP's data $y = 0$ in many cases, and if $y > 0$ the data are count-like. For both of these reasons, therefore, finite sample estimates of u and v in equations (1) and (2) may be quite different from the normal distribution.

We use the Jarque-Bera statistic (Jarque and Bera, 1987) to test whether the VAR innovations are normally distributed.[12] This statistic tests the joint hypothesis that the innovations are not skewed ($S = 0$) and are not fat or thin-tailed (kurtosis = 3), and it has a chi-square distribution with 2 degrees of freedom. Note that JB may exceed its critical value even for small deviations from the normal, simply because the number of observations is large. The JB statistics reported in Tables 2-4 are extremely large and easily reject the hypothesis that the innovations are normally distributed. More seriously, the estimates of skewness and kurtosis reported in Tables 2-4 are extremely large. The VAR innovations are heavy-tailed and severely skewed to the right. This means that the test statistics used by JP and HBK (and us so far) might be seriously distorted .

To quantify the size distortion in JP's model two nonparametric recursive bootstrapping exercises (detailed in Appendix 2) were carried out. In the first the size

[12] In contrast to the Kolmogorov – Smirnov test for normality, the JB test does not assume that the observations are independent. Therefore in autocorrelated time series the JB test is preferable.

distortion of the chi-square test for Granger causality in JP's model is calculated under the null hypothesis of no Granger causality, i.e. $\beta_{ij} = \beta_{pj} = 0$. For example, in JP's model the effective size for their Granger causality tests is 0.089 in the Israeli case and 0.106 in the Palestinian case when the nominal size is 0.05. Hence, the size distortions are large as might be expected, given positive skewness and fat-tails in BP's VAR innovations. In the second exercise the bootstrapped samples are generated under the null hypothesis that Granger causality is true. The bootstrapped distributions for β_i and β_p are reported in Appendix II. The p-value for $\beta_i > 0$ is zero and the p-value for $\beta_p > 0$ is 0.0052. Therefore, there is a 2-way Granger causality in JP's model. Their result that Palestinian fatalities do no Granger-cause Israeli fatalities was apparently induced by size distortion.

We also computed the bootstrapped means and p-values for the kill-ratios and the cumulative impulse responses. The bootstrapped mean (p-value) kill-ratios for Israel and the Palestinians are 1.2759 (0) and 0.0855 (0.0064), respectively. Because these bootstrapped estimates are smaller than their counterparts in Tables 2 and 3, there is evidence of finite sample bias in JP's model, quite apart from size distortions. The bootstrapped estimates (p-value) for the Israeli and the Palestinian cumulative impulse responses are 1.4455 (0.00) and 0.1534 (0.0051), respectively. According to these estimates relative *CIR* is 9.42 and the relative kill-ratio is 14.92. In summary, bootstrapping confirms the robustness of our main contention regarding the mutuality of the cycle-of-violence.

3. NONLINEAR VAR MODELS

The VAR innovations are not normally distributed for two reasons. First, because violence is sporadic and the dependent variables (number of daily fatalities) are zero for 81 percent of the time in the Israeli case and 39 percent in the Palestinian case. Secondly, when violence erupts the number of fatalities does not behave as a continuous random variable. Indeed, the data are highly dispersed; the conditional mean of Israeli fatalities is 0.63 and its variance is much higher at 5.31 while for Palestinian fatalities the conditional mean is 2.05 and its variance is 13.90. Such data necessitate other econometric methodologies that deal with over-dispersion and non-continuity.

We note that many investigators simply assume that the residuals are normally distributed according to asymptotic theory, and do not check whether in fact they are approximately normally distributed. One solution for this problem would be to block-bootstrap the VAR model to obtain data specific distributions of the parameter estimates. However, we think that the natural solution to this problem is to treat the dependent variables as "limited" and to estimate nonlinear VAR models (NLVAR).

3.1 Data Generating Processes for Discontinuous Time Series

Limited dependent variables have many different DGPs. There are several candidates. One is to treat fatalities as count data while another is to treat fatalities as resulting from violence as a latent variable. A third is to treat fatalities as censored as in the Tobit model. Both HBK and JP treat their dependent variables as count data. HBK estimated negative binomial (NB) models while JP estimated a Poisson model which assumes that the mean fatalities equals its variance, however, these models are not designed to address excessive zeros as in the case at hand. Should fatalities during the Second Intifada be regarded as counts? The answer would be yes if during the Second Intifada aggression was a continuous process which produced fatalities of 0,1,2 etc. However, there were periods of "Hudna" (truce in Arabic) during which the intifada was temporarily halted. During these hudnas, fatalities were zero. Since aggression was not a continuous process, it is questionable whether count data methods are appropriate. In any case, the preponderance of zeros in the data would require zero-inflating these count data methods.

A possible model is the zero-inflated ordered probit model (ZIOP) suggested by Harris and Zhao (2007). The ordered probit (OP) model hypothesizes the existence of an unobservable latent variable "aggression" which expresses itself in the number of fatalities. The model assumes that, in general, fatalities vary directly with aggression. Thus, OP is conceptually different from count data methods because it does not require continuity. It simply assumes that there may be more or less aggression, or even no aggression at all, which gives rise to different numbers of fatalities.

Ordered probit and logit have been applied by political scientists in other contexts of conflict. For example, Esteban et al. (2012) address the impact of ethnic divisions on conflict

intensity, and Besley and Persson (2009) study repression and civil war and link them to economic and political factors. Bagozzi et al. (2015) use Monte Carlo experiments and replications of published work to advocate the use of ZIOP rather than OP in conflict event counts.

In the end, the choice of the appropriate model is an empirical issue. Thus, we report several NLVARs estimated using ZIOP, ZIP (zero-inflated Poisson) and ZINB (zero-inflated negative binomial). All these zero-inflated models are designed to address the excess of zeros in fatalities data by assuming that zero fatalities originate from two distinct processes and thus estimate two models; first, a logit/probit model that models the probability of zero fatalities, and second, a count/ordinal model. Although ZIP is nested in ZINB because the former restricts the mean and variance to be equal while the latter does not, ZIOP and ZINB are non-nested since neither is a restricted version of the other. Therefore choosing between ZIP and ZINB is straightforward whereas choosing between ZIOP and ZINB is not. Santos Silva (2001) has suggested a non-nested test that may be used to compare all these nonlinear VAR models based on their estimated likelihoods.

3.2 Results of Nonlinear Vector Autoregressions

Tables 5 and 6 report unrestricted and restricted (GTS) NLVAR models using the same 14 lags and controls in JP for the Israeli and Palestinian reaction functions, respectively. In Table 5 the unrestricted ZINB and ZIP models (columns 1 and 3) indicate that Israel reacted to fatalities inflicted by the Palestinians, but the ZIOP model (column 5) does not (p-value for chi square = 0.16).[13] However, in all GTS models we find that Israel consistently reacted to Palestinian inflicted casualties. The results in Table 6 (Palestinian reaction functions) are similar to those in Table 5 in that whereas the unrestricted ZINB and ZIP models (columns 1 and 3) show that Palestinians reacted to fatalities inflicted by Israel, it is less clear according to the ZIOP model (p-value for chi square = 0.081 in column 5). However, in the GTS models we consistently find that Palestinian reacted to Israeli violence. Thus, in contrast to JP's conclusions that are derived from an inappropriate and over parameterized econometric

[13] Hence, the ZIOP results contradict JP's findings when using their lag specifications.

model, our NLVARs reveal that Israelis and Palestinians were locked in a circle of violence during the Second Intifada.

Although they do not provide detailed results, JP state that ZIP yielded "no qualitative differences" to their VAR models reported in columns 1 of Tables 2 and 3. The evidence provided here, however, does not seem to support this conclusion. The JP-reported chi-square statistic for Granger causality in column 1 of Table 3 is 17.5 with p-value 0.23. By contrast the chi square statistic for the unrestricted ZIP model (which we report in column 3 of Table 6) is 28.97 with p-value 0.011. This is a qualitative difference. Our main result does not depend on the type of NLVAR. Furthermore, their generalized residuals are not serially correlated, suggesting genuine causality rather than merely Granger causality.

4. CONCLUSIONS

This paper addresses five methodological issues arising from JP's investigation of the Second Intifada. Naturally, these issues transcend the specifics of JP's investigation and are relevant more generally to the study of dynamic responses between combatants, economic agents and other parties when the data happen to be discontinuous. First, the estimated linear VAR innovations might not be normally distributed in finite samples especially when the data are discontinuous. Consequently, reliance on asymptotic theory may reject true hypotheses or fail to reject false ones. Second, nonlinear VAR methods are better suited to discontinuous data e.g. when there are excess zeros and over-dispersion as in the data for fatalities. Third, we address the asymmetric reactions of the adversaries by allowing a flexible specification of the lagged fatalities in the VAR system. Fourth, we distinguish between Granger causality and genuine causality by testing for weak exogeneity, which would be rejected if the VAR innovations were serially correlated. Fifth, we argue that the kill-ratios and cumulative impulse responses are conceptually more appropriate than Granger causality in evaluating the cycle of violence. The kill-ratio is the number of fatalities suffered by the belligerent party for each fatality it inflicts on the injured party, assuming the belligerent party does not respond to its fatalities. The cumulative impulse response is the number of fatalities suffered by the belligerent party for each fatality it inflicts on the injured party assuming the

belligerent party responds to its fatalities. We show that kill-ratios and *CIR*s may be statistically significant despite the absence of Granger causality.

We may summarize our findings as follows: first, JP's result that Palestinians did not react to their own fatalities stemmed from a pre-test bias due to excessive parametrization. This result is overturned when statistically insignificant lag terms are omitted from the VAR. Second, The Palestinian kill-ratio is statistically significant in JP's VAR model despite the absence of Granger causality. Therefore, even in their over-parametrized model there is evidence that Palestinians reacted to their own fatalities. Third, the innovations of JP's VAR model are heavily skewed and fat-tailed, which undermines the validity of their chi square tests for Granger causality. OLS estimates of innovations estimated using serially correlated discontinuous time series may not be normally distributed even in large finite samples. Estimates from nonlinear VAR models, which take account of the fact that fatalities are discontinuous, overturn JP's result that Palestinian fatalities do not Granger cause Israeli fatalities. Also, bootstrapping JP's VAR model overturns their results, which indicates the large size distortion induced by assuming asymptotic normality. Finally, because the innovations are not serially dependent both within and between the VAR equations, Palestinian fatalities are weakly exogenous for Israeli fatalities, and vice-versa. Therefore, the evidence in favor of mutual Granger causality is not merely predictive but it is also genuinely causal. This means that if either side becomes less or more aggressive, the other side would become less or more aggressive too.

We have shown that JP's claim that Israel reacted to Palestinian violence whereas Palestinians did not react to Israeli violence breaks down under alternative dynamic specifications, when nonlinear VAR methods are used instead of JP's linear VAR method, and when attention is paid to size distortions in asymptotic tests when the data are discontinuous. Specifically we find that during the Second Intifada the violence was mutual and causal, and the Israeli reaction was quicker and stronger than the Palestinian reaction. According to our bootsrapped estimates Israel's kill ratio was1.276 Palestinians for every Israeli fatality whereas Palestinians killed only about 0.086 Israelis for every Palestinian fatality. Therefore, the relative kill-ratio was 14.92. The cumulative impulse responses

measure the final body-counts after the cycle-of-violence has worked through. According to the bootstrapped estimates these responses are 1.446 Palestinians killed for every Israeli killed, and 0.153 Israelis killed for every Palestinian killed. Thus when compared to the kill ratios, the relative cumulative impulse response (9.42) understates the asymmetry in the Second Intifada. The differences between the cumulative impulse responses and kill-ratios shed quantitative light on how the cycle-of-violence exacerbates the number of fatalities. When violence is mutual or bilateral rather than unilateral, the additional numbers of Palestinian and Israeli victims are 0.16 and 0.068 respectively. This means that a policy of self-restraint would reduce the number of Israeli victims by 43 percent and the number of Palestinian victims by 11 percent.

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Figure 1 - The Distribution of Daily Fatalities during the Second Intifada

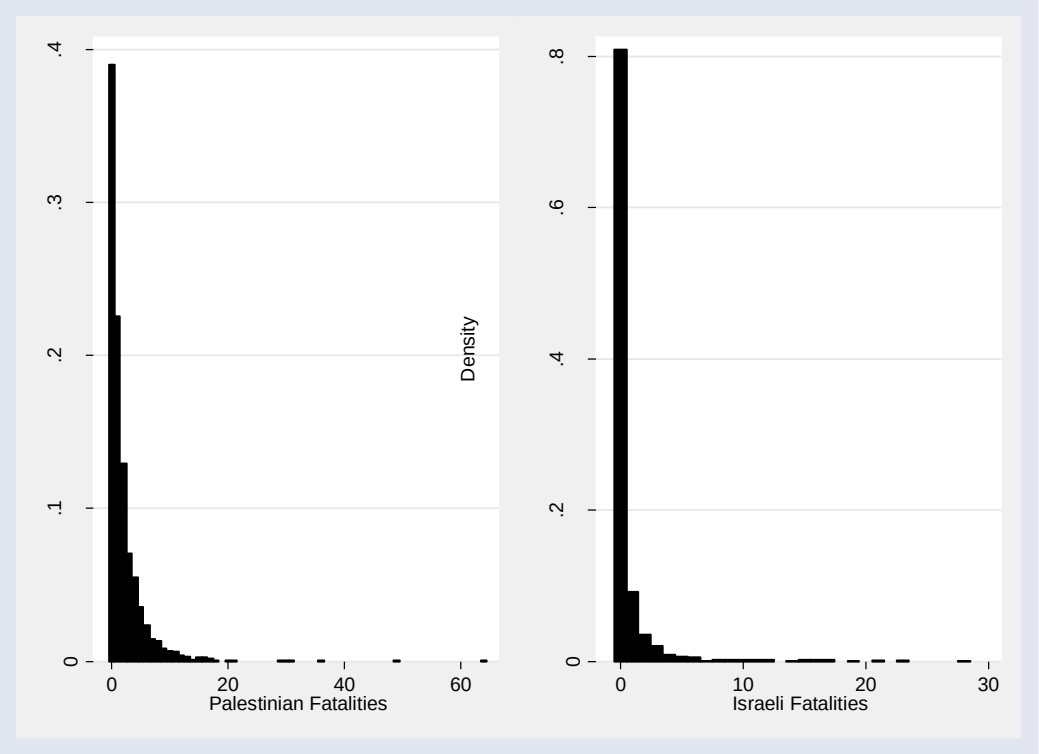


Figure 2 – Monthly Israeli and Palestinian Fatalities during Intifada 2

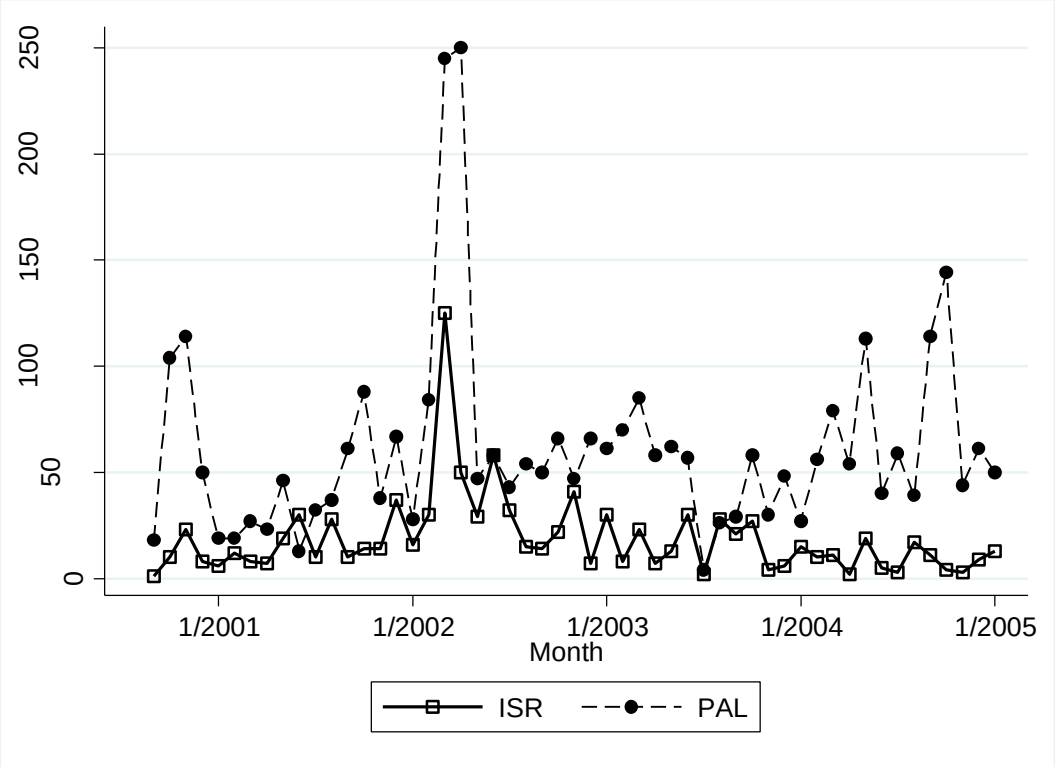


Table 1 – Unit Root Tests

	Palestinian Fatalities	Israeli Fatalities
ADF	-11.01*** (p = 5)	-15.76*** (p = 4)
ADF-GLS	-5.55*** (p =14)	-8.71*** (p = 12)

Notes:

In the case of ADF the number of augmentations (p) is determined by AIC and in the case of ADF-GLS p is determined by Ng and Perron's sequential t statistic.
ADF and ADF-GLS clearly reject the unit root hypothesis, and are robust with respect to p.
*** indicates significance at the 1% level.

Table 2 – Israeli Reaction Functions – Linear VAR

	(1)		(2)		(3)	
	14 lags		VAR System Optimal lag		General to Specific	
	Coeff.	**Robust t**	**Coeff.**	**Robust t**	**Coeff.**	**Robust t**
Israeli fatalities						
t-1	0.128	1.94	0.123	1.85	0.130	2.01
t-2	0.066	1.29	0.064	1.27	0.064	1.25
t-3	0.096	2.11	0.104	2.27	0.100	2.24
t-4	0.051	0.75	0.050	0.71		
t-5	0.223	1.73	0.218	1.74	0.225	1.79
t-6	0.050	1.12	0.055	1.25	0.048	1.10
t-7	0.054	1.18	0.052	1.12	0.055	1.20
t-8	0.138	1.03	0.134	0.99	0.137	1.03
t-9	-0.023	-0.49				
t-10	0.049	1.32			0.047	1.18
t-11	-0.070	-1.65			-0.074	-1.62
t-12	0.002	0.05				
t-13	0.024	0.65				
t-14	0.008	0.24				
Palestinian fatalities						
t-1	0.164	3.31	0.163	3.20	0.168	3.28
t-2	0.100	3.21	0.100	3.23	0.102	3.37
t-3	0.140	1.27	0.136	1.23	0.142	1.30
t-4	0.020	0.41	0.017	0.34		
t-5	0.043	1.25	0.045	1.26	0.043	1.13
t-6	-0.005	-0.13	-0.017	-0.45		
t-7	0.009	0.26	0.004	0.10		
t-8	-0.024	-0.73	-0.031	-0.95		
t-9	-0.050	-1.65			-0.051	-1.80
t-10	-0.019	-0.73				
t-11	0.035	1.51			0.030	1.41
t-12	0.011	0.37				
t-13	-0.027	-1.14			-0.028	-1.23
t-14	0.001	0.05				
Durbin's alternative test of serial correlation (p-value)						
AR(1)	1.505 (0.220)		0.007 (0.932)		0141 (0.707)	
AR(2)	1.376 (0.253)		0.049 (0.952)		0.292 (0.747)	
AR(3)	1.436 (0.230)		1.698 (0.166)		0.209 (0.890)	
AR(4)	1.828 (0.121)		1.365 (0.244)		0.380 (0.823)	
AR(5)	1.508 (0.184)		1.309 (0.257)		0.329 (0.896)	
AR(6)	1.277 (0.265)		1.330 (0.240)		0.279 (0.947)	
Jarque-Bera normality test	119.7		124.0		124.8	
(x1,000) (*p*-value)	(0.0000)		(0.0000)		(0.0000)	
S skewness	4.45		4.48		4.49	
k kurtosis	44.84		45.60		45.74	
χ^2 for joint significance of	24.30		17.33		21.12	
own fatalities (*p*-value)	(0.042)		(0.027)		(0.012)	
Kill-ratio (p-value)	1.32 (0.003)		1.37 (0.006)		1.23 (0.003)	
CIR (p-value)	1.62 (0.000)		1.60 (0.000)		1.53 (0.000)	
AIC	8264.6		8253.4		8245.7	
BIC	8489.7		8414.2		8406.5	
White χ^2 test for generalized	1436.2		1349.0		1338.5	
heteroscedasticity (p-value)	(0.0000)		(0.0000)		(0.0000)	

Notes:
Dependent variable is the daily number of Palestinian fatalities.
The coefficients of the exogenous variables used by JP (period dummies, length of completed security barrier, and days of the week) are not presented to conserve space.
Serial correlation test statistics allow for heteroskedasticity.

Table 3 – Palestinian Reaction Functions – Linear VAR

	(1)		(2)		(3)	
	14 lags		VAR system optimal lag		General to specific	
	Coeff.	**Robust t**	**Coeff.**	**Robust t**	**Coeff.**	**Robust t**
Israeli fatalities						
t-1	0.072	2.18	0.072	2.17	0.073	2.21
t-2	-0.012	-0.59	-0.013	-0.68		
t-3	0.008	0.39	0.010	0.48		
t-4	0.026	0.60	0.023	0.53		
t-5	-0.013	-0.80	-0.016	-0.94		
t-6	-0.021	-0.79	-0.016	-0.64		
t-7	-0.013	-0.46	-0.016	-0.55		
t-8	-0.024	-1.51	-0.027	-1.66	-0.024	-1.41
t-9	-0.006	-0.25				
t-10	0.010	0.40				
t-11	-0.001	-0.05				
t-12	-0.007	-0.41				
t-13	0.046	1.04			0.049	1.06
t-14	0.002	0.06				
Palestinian fatalities						
t-1	0.026	1.33	0.024	1.27	0.021	1.15
t-2	0.027	1.08	0.025	1.04	0.026	1.19
t-3	0.000	0.01	0.003	0.21		
t-4	-0.009	-0.55	-0.009	-0.54		
t-5	0.014	0.47	0.016	0.53		
t-6	-0.011	-0.54	-0.01	-0.45		
t-7	-0.029	-1.81	-0.027	-1.78	-0.030	-2.09
t-8	0.064	2.73	0.070	2.91	0.068	2.65
t-9	0.005	0.24				
t-10	0.009	0.44				
t-11	0.012	0.69				
t-12	-0.026	-1.96			-0.020	-1.57
t-13	-0.020	-1.14			-0.017	-1.07
t-14	0.027	1.13			0.029	1.21
Durbin's alternative test of serial correlation (p-value)						
AR(1)	0.118 (0.731)		0.156 (0.693)		0.010 (0.919)	
AR(2)	1.104 (0.332)		0.149 (0.862)		0.083 (0.920)	
AR(3)	0.850 (0.467)		0.176 (0.913)		0.157 (0.925)	
AR(4)	0.877 (0.477)		1.186 (0.315)		0.294 (0.882)	
AR(5)	0.718 (0.610)		0.956 (0.444)		0.303 (0.912)	
AR(6)	0.630 (0.706)		1.035 (0.401)		0.325 (0.924)	
Jarque-Bera normality test	159.3		161.4		158.2	
(x1,000) (*p*-value)	(0.0000)		(0.0000)		(0.0000)	
S skewness	6.11		6.16		6.11	
k kurtosis	50.82		51.13		50.63	
χ^2 for joint significance of	17.50		14.41		13.14	
own fatalities (*p*-value)	(0.230)		(0.072)		(0.069)	
Kill-ratio (p-value)	0.094 (0.028)		0.094 (0.017)		0.084 (0.043)	
CIR (p-value)	0.179 (0.038)		0.185 (0.006)		0.158 (0.033)	
AIC	7070.6		7055.8		7039.6	
BIC	7295.6		7216.6		7168.2	
White χ^2 test for generalized	959.1		398.5		283.7	
heteroscedasticity (p-value)	(0.008)		(0.746)		(0.090)	

Notes:
Dependent variable is the daily number of Israeli fatalities.
See notes to Table 2.

Table 4 – Linear VAR, Extended Sample 1/2000-12/2007

	Israeli Reaction Function				Palestinian Reaction Function			
	14 lags		GTS		14 lags		GTS	
	(1)		(2)		(3)		(4)	
Durbin's alternative test of serial correlation								
AR(1)	0.058	(0.81)	1.439	(0.23)	0.446	(0.50)	0.067	(0.80)
AR(2)	0.375	(0.69)	0.741	(0.48)	1.045	(0.35)	0.099	(0.91)
AR(3)	0.856	(0.46)	0.508	(0.68)	0.839	(0.47)	0.250	(0.86)
AR(4)	0.874	(0.48)	0.525	(0.72)	0.927	(0.45)	0.354	(0.84)
AR(5)	0.702	(0.62)	0.453	(0.81)	0.768	(0.57)	0.313	(0.91)
AR(6)	1.063	(0.38)	0.402	(0.88)	0.718	(0.64)	0.287	(0.94)
Jarque-Bera normality test (x1,000)	277.6	(0.000)	356.4	(0.000)	695.2	(0.000)	693.4	(0.00)
S skewness	4.94		5.23		7.69		7.71	
K kurtosis	52.16		58.84		80.84		80.73	
χ^2 for joint significance of own fatalities	22.45	(0.070)	16.16	(0.013)	14.19	(0.435)	9.64	(0.086)
White χ^2 test for heteroscedasticity	2191.6	(0.000)	1958.6	(0.000)	1407.3	(0.000)	353.4	(0.000)
RMSE	3.004		3.010		1.771		1.769	
Long-run kill ratio	1.31	(0.013)	0.981	(0.021)	0.063	(0.043)	0.053	(0.063)
AIC	13393.3		13388.2		10593.5		10565.1	
BIC	13646.2		13552.9		10846.4		10700.4	
White test for generalized heteroskedasticity	2191.7	(0.0000)	1958.6	(0.0000)	1407.1	(0.000)	353.4	(0.0000)

Notes:
Dependent variable in columns 1 and 2 is the daily number of Israeli fatalities. It is the daily number of Palestinian fatalities in columns 3 and 4.
P-values in parentheses.
Coefficients and their t-stats were omitted to preserve space and are available upon request.
See notes to Table 1.

Table 5 — Israeli Reaction Functions: Non-Linear VAR

	Zero Inflated Negative Binomial				Zero Inflated Poisson				Zero Inflated Ordered Probit			
	(1)		(2)		(3)		(4)		(5)		(6)	
	Unrestricted		**GTS**		**Unrestricted**		**GTS**		**Unrestricted**		**GTS**	
	Coeff.	z	Coeff.	z	Coeff.	z	Coeff.	z	Coeff.	z	Coeff.	z
Israeli fatalities												
t-1	0.022	1.87	0.027	2.28	0.023	2.12	0.021	2.00	0.024	0.77		
t-2	0.027	2.13	0.014	1.09	0.021	1.30	0.020	1.30	0.010	0.58		
t-3	0.017	1.64	0.017	1.66	0.023	1.74	0.023	1.63	0.015	1.16	0.021	1.60
t-4	0.008	0.54			0.020	1.50	0.020	1.53	-0.011	-0.61		
t-5	0.031	2.06	0.041	2.86	0.053	3.89	0.052	3.95	0.006	0.39		
t-6	0.018	1.11			-0.000	0.00			0.025	1.1		
t-7	0.021	1.45	0.025	1.72	0.008	0.53			0.008	0.46		
t-8	0.007	0.55			0.021	1.89	0.020	1.81	-0.006	-0.46		
t-9	-0.008	-0.55			-0.034	-2.26	-0.033	-2.01	-0.006	-0.4		
t-10	0.015	1.24	0.016	1.32	0.011	0.74			0.036	1.54	0.033	1.83
t-11	-0.036	-2.22	-0.035	-2.31	-0.044	-2.54	-0.035	-2.55	-0.022	-1.36		
t-12	-0.007	-0.47			-0.001	-0.09			-0.005	-0.27		
t-13	0.018	1.16	0.020	1.33	0.004	0.27			0.032	1.73	0.036	2.03
t-14	-0.005	-0.32			-0.000	0.00			-0.028	-1.89	-0.017	-1.19
Palestinian fatalities												
t-1	0.051	4.13	0.058	4.61	0.028	3.48	0.028	4.08	0.059	4.22	0.058	4.56
t-2	0.028	2.91	0.031	3.22	0.01	1.28	0.013	2.31	0.049	3.64	0.049	4.02
t-3	0.008	0.9			0.023	3.43	0.022	3.56	-0.017	-1.64	-0.016	-1.43
t-4	0.010	1.11	0.011	1.31	-0.000	-0.05			0.021	1.63	0.029	2.32
t-5	0.010	1.15	0.012	1.40	0.013	1.86	0.016	2.21	0.007	0.64		
t-6	0.003	0.4			-0.000	-0.06			0.021	1.67	0.021	1.73
t-7	0.006	0.69			0.005	0.62			0.006	0.59		
t-8	-0.014	-1.45	-0.016	-1.68	-0.001	-0.11			-0.018	-1.75	-0.014	-1.52
t-9	-0.009	-0.91			-0.013	-1.43	-0.012	-1.43	0.003	0.26		
t-10	0.005	0.58			-0.005	-0.59			0.009	0.85		
t-11	0.013	1.61	0.012	1.89	0.007	1.08	0.008	1.33	0.019	1.8	0.021	2.33
t-12	-0.004	-0.53			0.007	0.84			-0.004	-0.34		
t-13	-0.005	-0.57			-0.016	-1.84	-0.015	-1.87	0.001	0.08		
t-14	0.006	0.71			0.008	1.13	0.009	1.24	0.010	0.78		
AR(6) p-value	0.057		0.592		0.932		0.881		0.449		0.467	
χ^2 for joint significance of own fatalities (p-value)	28.36 (0.013)		29.27 (0.0003)		46.71 (0.0000)		43.97 (0.0000)		19.13 (0.160)		12.08 (0.017)	
Log likelihood	-2809.72		-2816.77		-3219.64		-3231.88		-1539.44		-1558.99	

Notes:
Dependent variable in columns 1-4 is the daily number of Palestinian fatalities. Dependent variable in columns 5-6 is an ordered mapping of Palestinian fatalities, that takes on the value 0 if there were zero Palestinian fatalities, 1 if there was one fatality, and 2 if there were two or more fatalities.
The coefficients of the exogenous variables (dummies for the discussed periods, length of completed barrier, and days of the week) are not presented to conserve space.
The specifications in columns 1, 3, and 5 are the unrestricted forms. Those in columns 2, 4, and 6 are the respective General-to-Specific specifications.
The "inflation" equations include lags of own fatalities.
Reported standard errors are heteroskedasticity-robust.
AR(6) p-value of Durbin's alternative test for up to 6th order serial correlation in the generalized residuals.

Table 6 — Palestinian Reaction Functions: Non-Linear VAR

	Zero Inflated Negative Binomial				Zero Inflated Poisson				Zero Inflated Ordered Probit			
	(1)		(2)		(3)		(4)		(5)		(6)	
	Unrestricted		**GTS**		**Unrestricted**		**GTS**		**Unrestricted**		**GTS**	
	Coeff.	z	Coeff.	z	Coeff.	z	Coeff.	z	Coeff.	z	Coeff.	z
Israeli fatalities												
t-1	0.049	1.84	0.045	1.76	0.014	0.72			0.042	1.51	0.037	1.64
t-2	-0.015	-0.55			0.020	1.09			0.014	0.49		
t-3	0.018	0.59			-0.000	-0.02			-0.009	-0.38		
t-4	-0.014	-0.47			0.031	1.68	0.038	2.90	-0.019	-0.68		
t-5	-0.011	-0.37			-0.065	-2.19	-0.064	-2.44	0.034	1.46	0.023	1.16
t-6	-0.023	-0.57			0.009	0.19			0.012	0.33		
t-7	-0.037	-1.14			-0.019	-0.56			-0.018	-0.91		
t-8	-0.046	-1.88	-0.048	-2.00	-0.053	-1.84	-0.057	-2.06	-0.005	-0.26		
t-9	0.034	0.56			0.058	1.75	0.062	2.64	-0.028	-1.05	-0.022	-1.00
t-10	0.020	0.63			-0.026	-1.42	-0.024	-1.34	0.022	0.89		
t-11	-0.012	-0.35			0.012	0.43			-0.030	-1.28	-0.026	-1.18
t-12	-0.042	-1.26	-0.034	-1.21	-0.067	-1.52	-0.071	-1.92	0.026	0.66		
t-13	0.020	0.71			0.048	2.06	0.042	1.94	-0.017	-0.66		
t-14	-0.025	-0.7			0.003	0.09			0.000	0.00		
Palestinian fatalities												
t-1	0.030	1.04	0.031	1.38	0.006	0.29			0.026	1.35	0.028	2.34
t-2	0.042	1.68	0.025	1.31	0.006	0.33			-0.003	-0.16		
t-3	0.023	1.06	0.024	1.27	0.023	1.29	0.023	1.34	-0.007	-0.24		
t-4	0.026	1.2			0.006	0.21			0.018	0.76		
t-5	-0.028	-1.6	-0.022	-1.62	-0.039	-3.41	-0.037	-3.36	-0.008	-0.47		
t-6	-0.049	-2.65	-0.043	-2.65	-0.013	-0.89			0.009	0.61		
t-7	-0.023	-0.9			-0.016	-0.6			-0.047	-2.16	-0.022	-1.24
t-8	0.022	1.33	0.031	2.18	0.027	1.95	0.021	2.45	0.007	0.35		
t-9	0.053	2.07	0.039	1.75	0.019	0.83			-0.022	-1.25		
t-10	-0.015	-0.6			0.041	1.72	0.047	2.08	0.028	1.10		
t-11	0.009	0.32			-0.010	-0.47			0.007	0.22		
t-12	0.036	1.28			-0.042	-2	-0.034	-2.54	0.019	0.69		
t-13	-0.051	-1.97	-0.038	-1.69	0.008	0.29			-0.029	-0.57		
t-14	0.076	2.39	0.062	1.95	0.032	1.18	0.037	1.72	0.042	1.81	0.038	2.04
AR(6) p-value	0.468		0..561		0.151		0.275		0.349		0.339	
χ^2 for joint significance of own fatalities (p-value)	37.11 (0.0007)		27.38 (0.001)		28.97 (0.011)		28.20 (0.0001)		21.88 (0.081)		11.59 (0.009)	
Log likelihood	-1278.08		-1281.97		-1481.79		-1491.69		-903.67		-911.24	

Notes: Dependent variable is number of Israeli fatalities.
See notes to Table 5.

Appendix I – Impulse Responses and Long Run Kill Ratios

Suppose the VAR is:

$y_{1t} = \gamma_1 y_{1t-1} + \beta_1 y_{2t-1} + \varepsilon_{1t}$

$y_{2t} = \gamma_2 y_{2t-1} + \beta_2 y_{1t-1} + \varepsilon_{2t}$

where y_1 and y_2 denote two outcomes and the innovations (ε) are *iid* and independent.

The general solution to the VAR is:

$$y_{1t} = \frac{1}{\rho_1 - \rho_2}\left[\sum_{i=0}^{\infty}(\rho_1^{i+1} - \rho_2^{i+1})(\varepsilon_{1t-i} - \gamma_2\varepsilon_{1t-1-i} + \beta_1\varepsilon_{2t-1-i})\right] + A_1\rho_1^t + A_2\rho_2^t$$

$$y_{2t} = \frac{1}{\rho_1 - \rho_2}\left[\sum_{i=0}^{\infty}(\rho_1^{i+1} - \rho_2^{i+1})(\varepsilon_{2t-i} - \gamma_1\varepsilon_{2t-1-i} + \beta_2\varepsilon_{1t-1-i})\right] + B_1\rho_1^t + B_2\rho_2^t$$

where *A* and *B* are arbitrary constants, the roots ρ_1 and ρ_2 are real, less than one in absolute value, and $(1-\rho_1 L)(1-\rho_2 L) = (1-\gamma_1 L)(1-\gamma_2 L) - \beta_1\beta_2 L^2$ is the determinant of the VAR where *L* denotes the lag operator.

The impulse response of e.g. y_{1t} with respect to ε_{2t-i} equals:

$$\frac{\partial y_{1t}}{\partial \varepsilon_{2t-i}} = \frac{\beta_1(\rho_1^i - \rho_2^i)}{\rho_1 - \rho_2} = \frac{\partial y_{1t+i}}{\partial \varepsilon_{2t}}$$

Cumulative Impulse Response

The cumulative impulse response of $y_1$with respect to ε_{2t} is:

$$\sum_{i=0}^{\infty}\frac{\partial y_{1t+i}}{\partial \varepsilon_{2t}} = \frac{\beta_1}{\rho_1 - \rho_2}\left[\frac{1}{1-\rho_1} - \frac{1}{1-\rho_2}\right] = \frac{\beta_1}{(1-\gamma_1)(1-\gamma_2) - \beta_1\beta_2} = CIR_{12}$$

Similarly, the cumulative impulse response of $y_2$with respect to $\varepsilon_1$is:

$$CIR_{21} = \frac{\beta_2}{(1-\gamma_1)(1-\gamma_2) - \beta_1\beta_2}$$

which is equivalent to setting *L = 1* in the determinant of the VAR.

Note that $CIR_{12}/CIR_{21} = \beta_1/\beta_2$ does not depend on γ_1 and γ_2, and $CIR_{12} = CIR_{21}$ if $\beta_2 = \beta_1$.

More generally, in q-order VARs there are 2q roots, and its determinant is:

$$\prod_{s=1}^{2q}(1-\rho_s L) = \left(1-\sum_{s=1}^{q}\gamma_{1s}L^s\right)\left(1-\sum_{s=1}^{q}\gamma_{2s}L^s\right) - \sum_{s=1}^{q}\beta_{1s}L^s\sum_{s=1}^{q}\beta_{2s}\,L^s$$

Setting $L = 1$ gives:

$$CIR_{12} = \frac{\sum\beta_{1s}}{(1-\sum\gamma_{1s})(1-\sum\gamma_{2s}) - \sum\beta_{1s}\sum\beta_{2s}}$$

$$CIR_{21} = \frac{\sum\beta_{2s}}{(1-\sum\gamma_{1s})(1-\sum\gamma_{2s}) - \sum\beta_{1s}\sum\beta_{2s}}$$

Kill-Ratios

Let k_{1j} denote the conditional expectation of the cumulative change in y_{1t+j} given a unit change in $y_{2t}$when $\Delta y_{1t} = 0$. That is:

$$k_{1j} = E(y_{1t+j} - y_{1t}/\,\Delta y_{2t}\,, \Delta y_{1t} = 0)$$

Differencing the first equation in the VAR and using the chain-rule to project y_{1t+j} gives:

$k_{1j} = \beta_1 \sum_{s=0}^{j}\gamma_1^s$

as the cumulative response of $y_1$to a unilateral increase in y_2 after j periods. As j tends to infinity the long-run cumulative response is:

$$k_1 = \frac{\beta_1}{1-\gamma_1}$$

The counterpart for y_2 is:

$$k_2 = \frac{\beta_2}{1-\gamma_2}$$

In contrast to *CIR*, *k* ignores feedback between y_1 and y_2. Therefore, CIR_{12} is generally larger than k_1 and CIR_{21} is generally larger than k_2. Note that:

$$\frac{CIR_{12}}{CIR_{21}} = \frac{(1-\gamma_1)k_1}{(1-\gamma_2)k_2}$$

Therefore, if $\gamma_1 < \gamma_2$ the relative CIR is larger than the relative kill-ratio.

Appendix II – Bootstrapping

Exercise 1: Null hypothesis Granger causality is false

JP's model (columns 1 in Tables 2 and 3) is bootstrapped recursively and nonparametrically by resampling with replacement from the estimated innovations (Davidson and MacKinnon 2009, pp 160-3) under the null hypothesis of no Granger causality, i.e. $\beta_{ij} = \beta_{pj} = 0$. We demonstrate that the asymptotic distributions of the parameters are bad proxies of their finite sample distributions, and induce considerable size distortion in carrying out hypothesis tests regarding Granger causality.

The exercise was conducted according to the following steps:

1. Estimate JP's model under the null hypothesis of no Granger causality (coefficients of own fatalities equaling zero) and obtain the coefficients (14 γ_i and γ_ps) as well as the two sets of residuals.
2. Bootstrap the values of Israeli and Palestinian fatalities using the estimated coefficients from step 1 and error terms drawn from the empirical distribution of the innovations estimated in step 1, and using recursively the 14 initial conditions.
3. For observation i draw an error term $\hat{u}_{id}$ with replacement where $id = int(1570 \times U)$, $U \sim U[0,1]$ is a uniformly distributed random variable, and 1570 is the sample size. This ensures that each residual has an equal probability of being chosen. Since the innovations are independent, the draws from the two sets of innovations is independent
4. Use the bootstrapped data from steps 2 and 3 to estimate the unrestricted VAR (i.e. with the coefficients β_i and β_p). Save the Chi-squared test statistic for Granger causality.
5. Repeat steps 2 - 4 1000 times, with new random draws from the innovations estimated in step 1. This step produces the empirical distribution of Chi-squared under the null hypothesis of zero coefficients of joint fatalities.

The results in the following table show the size distortions of JP's tests for Granger causality. For example, when the nominal size is 5% the actual size is 8.9% for Israel and 10.6% for Palestinians. The size distortion is more severe in the Palestinian case.

Nominal vs. Actual Sizes for Granger Causality in JP's Model

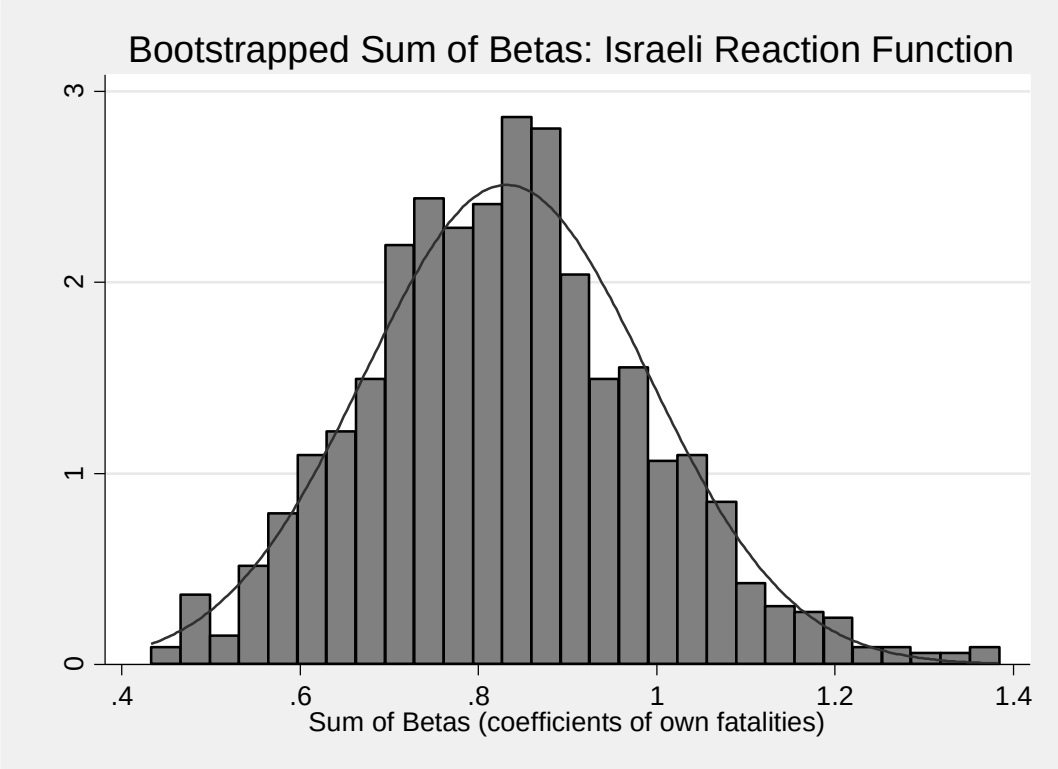
	Actual Significance Levels	
Nominal	**Israeli Reaction Function**	**Palestinian Reaction Function**
10%	13.9%	16.0%
5%	8.9%	10.6%
2.5%	6.8%	8.0%
1%	4.1%	5.1%
0.5%	2.5%	4.0%
0.1%	1.3%	2.1%

Exercise 2: Null hypothesis- Granger causality is true

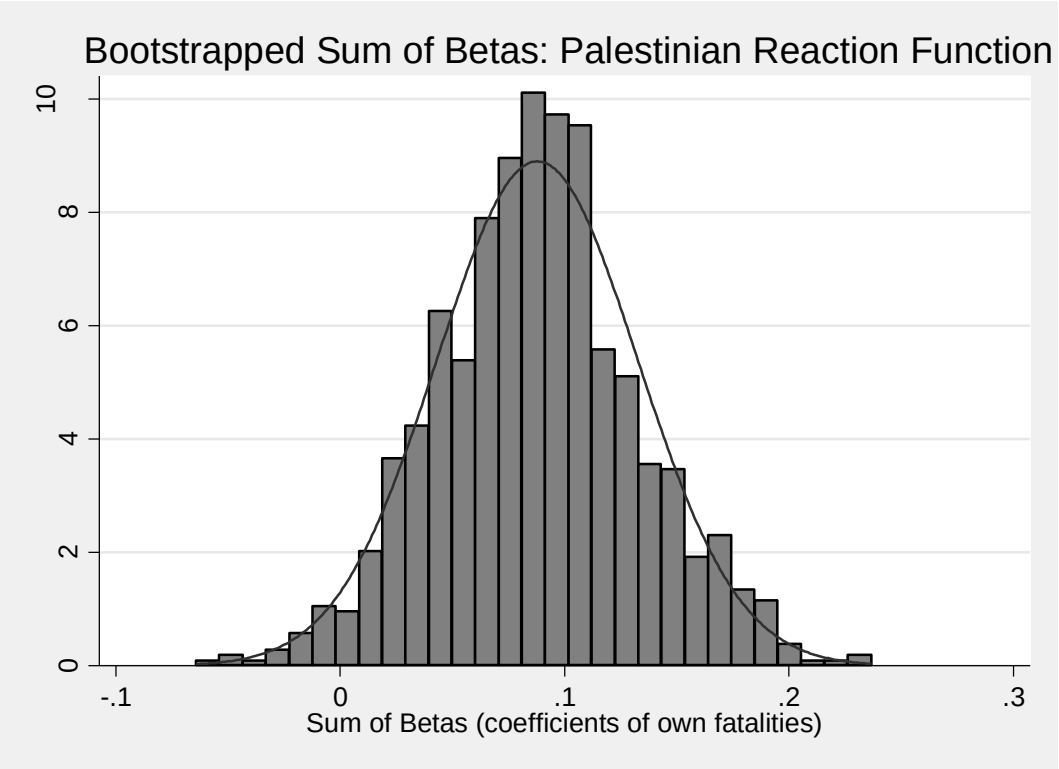
This exercise follows Freedman and Peters (1984) who proposed the recursive bootstrap to estimate the empirical distribution of parameters in unrestricted VAR models. Exercise 2 has the following steps.

1. Save the parameters (β_{ij}, β_{pj}, γ_{ij}, γ_{pj}) and innovations (u_t, v_t) of JP's model.
2. As in exercise 1.
3. As in exercise 1.
4. Use the bootstrapped data to estimate the unrestricted VAR model of step 1. Save the estimates of β_i (sum of β_{ij}) and β_p (sum of β_{pj}).
5. Repeat steps 2-4 1000 times.

The bootstrapped distributions for the sum of β_i and β_p are respectively:



Mean sum (β_i) = 0.83156



Mean sum of (β_p) = 0.08786

The bootstrapped mean for β_i is larger than JP's estimate of 0.796, and the mean for β_p is smaller than JP's estimate of 0.109, suggesting that apart from size distortions JP's estimates are biased in finite samples.