Modelling the Spread of Terrorism Via Diffusion and Contagion

Gentry White and Fabrizio Ruggeri and Michael Porter

Summary. The spread of terrorism is a serious concern in national and international security, as its spread is seen as an existential threat to Western liberal democracies. Understanding and effectively modelling the spread of terrorism provides useful insight into formulating effective responses. A mathematical model capturing the theoretical constructs of contagion and diffusion is constructed for explaining the spread of terrorist activity and used to analyse data from the Global Terrorism Database from 2000–2016 for Afghanistan, Iraq, and Israel. Results show that the model identifies patterns in the diffusion and contagion processes that align with and provide insight into contemporary events.

Keywords: terrorism, contagion, diffusion, self-exciting process

1. Introduction

Terrorism is a complicated social phenomenon, and assessing the effectiveness of measures to prevent the spread or increase in the rate of terrorist events relies on a model that incorporates a qualitative and theoretical understanding of the process while maintaining fidelity to the data, hence accurately reflecting the effects of counter-measures. This paper presents such a model, it incorporates an accepted theoretical framework and refined elements of previous quantitative models and is parametrised to allow the extraction of meaningful insights from the results.

There are two aspects to acts of terrorism† that exacerbate their impact. The first is their relative rarity (in most contexts), the second is that the clustering of events in time 47, 48, 32, 49, 36, 71, 75, 43, 5 creates the impression of a sudden increase in the rate of terrorism. It is the uncertainty surrounding the permanence of this increase that causes additional anxiety 72, 38, 13, 14, 1. Determining whether the occurrence of a terrorist event is a transient anomaly or a signal of a change in the rate of events is of great interest and identifying the circumstances around these two possibilities is a feature of the model presented in this paper. Theoretical models posit the mechanisms of contagion and diffusion as explanations for these patterns of clustering and erratic activity. Each of these mechanisms has different implications for identifying whether events are part of an increase in the rate of events or are part of a transient anomaly.

Sociological theories to explain aberrant behaviour through contagion or diffusion‡ begin with 46, and appear in discussions of political violence and civil upheaval by the mid-twentieth century 37, enduring as a viable theoretical model for the spread of terrorism 48, 57, 49, 27, 33, 36, 77, 24, 7, 8, 6, 10. The mathematical and statistical concept of a “contagious” process is similarly well established 23, 54, 19 and the application of a model based approach to describing the theoretical dynamics of terrorism via social diffusion emerges in the social sciences with 12, followed by the development of theoretically based models 57, 16, 26, 27. These models build on theoretical constructs concerning social contagion, diffusion, and group learning demonstrate a sophisticated understanding of the theories and mathematical models but focus on a single mechanism to explain the spread of violence. Contemporary to these models the Hawkes self-exciting process model 30, 29 and its cluster process interpretation 31 are put forth with little initial attempt to apply them to terrorist activity, but more importantly they are stationary processes (stochastically), and accurately reflect the theoretical understanding of the role behavioural contagion plays in the rate of terrorist incidents.

Diffusion is distinguished from contagion in 48 by identifying it as the increase in the rate of events due to non-terrorist events that elicit a collective reaction among individuals in a population without observation or communication of others’ behaviour. This is distinct from contagion as it contains no element of imitation or

†The definition of terrorism is contested (see 67, 35, for an entrée into this discussion), in this paper the term “terrorist” and “terrorism” refer only to the fact that the events analysed in this paper are drawn from the Global Terrorism Database 53.

‡The distinction between diffusion and contagion can be difficult to discern in the sociological literature. In some cases the terms seem to be used interchangeably. This is specifically acknowledged in 48 which concludes by drawing a distinction between the two mechanisms.
the modelling of behaviour, rather it implies a shift in an individual’s personal beliefs and attitudes in reaction to external events. If these shifts in patterns of behaviour are enduring, then the mean of the process can shift, implying the possibility that diffusion can be a non-stationary process. Originally, [48] applied this theoretical model, in conjunction with contagion, to explain the spread of riots in US cities during the late 1960’s. But it is reasonable to extend this to terrorism more broadly, as diffusion in [48] is what [13] refers to as precipitants, or precipitating events, identified as an element in the spread of terrorism. The resulting theoretical model states that terrorism spreads through two mechanisms: contagion, which is a function of the influence that past terrorist events has on the future event rate, and diffusion, which is a function of exogenous events or processes. This serves as the basis for the model presented in this paper, which demonstrates a model for the combined effects of diffusion and contagion as a convolution of two process: a non-homogeneous Poisson process for diffusion (as originally proposed in [50]) and a negative-binomial Hawkes self-exciting process for contagion. Results show that under the cluster process representation the model can identify distinct behaviours in the two processes providing useful insight into the actual phenomenon and data.

The balance of this paper is structured as follows: in Section 2 the model is derived as a Bayesian hierarchical model for the convolution of a Poisson and negative-binomial processes. Section 3 presents details of the application of the model to multiple data sets, and in Section 4 results are presented. Section 5 contains a discussion of the results and their interpretation.

2. Model

The daily number of terrorist events can be described as the sum of events from two processes, a diffusion process and a contagion process. As proposed in [48] a Poisson distribution is used for the diffusion process and a negative-binomial distribution is used for the contagion process, resulting in a convolution model for the total number of daily events. A closed form for the likelihood of a Poisson-Negative-Binomial convolution is not available. This complication is addressed by noting that the total number of events on day \( t, Y_t = Y_{dt} + Y_{ct} \) is the sum of the number of events from the diffusion and contagion processes, respectively, and specifying a hierarchical convolution model,

\[
Y_{dt} \sim \text{Pois}(\lambda_{dt}^t) \quad (1)
\]
\[
Y_{ct} | \lambda_{ct}^t \sim \text{Pois}(\lambda_{ct}^t) \quad (2)
\]
\[
\lambda_{ct}^t \sim \text{Ga}(\sigma^2, \mu_{ct}^t \sigma^2) \quad (3)
\]

Note that (3) is parametrised so that \( E(\lambda_{ct}^t) = \mu_{ct}^t \), and that multiplying (2) and (3) and integrating out \( \lambda_{ct}^t \) yields

\[
Y_{ct} \sim \text{Neg} - \text{Binom}(\mu_{ct}^t, \sigma^2), \quad (4)
\]

where \( \mu_{ct}^t \) is parametrised as the expected value.

If the partitioning of \( Y_t \) into \( Y_{dt} \) and \( Y_{ct} \) were known, then the parameters for the two processes could be estimated using the (1) and (4). If \( Y_t | \lambda_{ct}^t \sim \text{Pois}(\lambda_{dt}^t + \lambda_{ct}^t) \) as implied by (1) and (2) then from [15]

\[
Y_{dt} | Y_t, \lambda_{ct}^t \sim \text{Binom} \left( Y_t, \frac{\lambda_{dt}^t}{\lambda_{dt}^t + \lambda_{ct}^t} \right), \quad (5)
\]

which can be used to stochastically attribute events to diffusion and contagion. These results can be used to construct a hierarchical model and an MCMC scheme that does not require explicit evaluation of the likelihood for
2.2. Incorporating Exogenous and Endogenous Effects

The model presented in (1)–(4) can be extended to allow variation in diffusion and contagion rates due to other factors. [26] divides these factors into endogenous, associated with the contagion process (e.g., timing, casualties, attack characteristics), and exogenous effects that influence the diffusion process (e.g., what [13] refers to as precipitating events or preconditions); thus exogenous effects are present in the diffusion process and endogenous effects in the contagion process.

Following the convention for generalised linear models, the rate of the diffusion process is therefore modelled

$$\lambda_t^d = \exp(x_t, \beta),$$

(6)

where the row vector $x_t$ contains the exogenous covariates at time $t$ and $\beta$ is a column vector of coefficients. The expected rate of the contagion process $\mu_t^c$ from (3) is

$$\mu_t^c = \sum_{s < t} \delta_s Y_s g(t - s; \phi).$$

(7)

The decay function $g(\cdot; \phi)$ is a non-negative function defined for the days $u \in \mathbb{N}$ such that $\sum_{u=1}^\infty g(u) = 1$, in this instance a shifted negative-binomial pmf parametrised in terms of mean and scale as in [58, 78] is used. The endogenous effects coefficient $\delta_s$ is equal to the expected number of additional events generated by each event on day $s$. As $\delta_s \geq 0$ in general the effects of covariates can be incorporated as in (6) defining

$$\delta_s = \exp(w_s, \eta),$$

(8)

where $w_s$ is a row vector of endogenous covariates at time $s$ and $\eta$ a column vector of coefficients; thus the contagion rate $\mu_t^c$ is the average of the number of events prior to time $t$ scaled by the endogenous effects coefficient and weighted by the decay function $g$.

It is useful to note that the conditional expectation of $Y_t$

$$\lambda_t | \lambda_t^d = \lambda_t^d + \lambda_t^c$$

$$= \lambda_t^d + \sum_{s < t} \delta_s Y_s g(t - s; \phi)$$

(9) (10)

is similar to the Hawkes self-exciting process intensity function [30, 29], but extends it by allowing $\lambda_t^d$ and $\delta_s$ to vary according to the exogenous and endogenous effects.

2.3. Selecting Endogenous and Exogenous Variables

The covariates $x_t$ for the endogenous effects can be defined based on known precipitating events or specified in order to allow a data-driven approximation, allowing the model to be used for exploratory purposes. In this paper penalised $b$-splines [45] are used for this purpose. Endogenous effects are based on characteristics of events that affect their “contagiousness”. Exploratory data analysis led to the selection of the number of fatalities as the variable describing the endogenous effects. The exact nature of the relationship between fatalities and contagiousness is not clear, therefore the covariates $w_s$ in (8) are defined as a penalised $b$-spline basis based on the natural log of the number of fatalities (plus 1), allowing a data-driven exploration of the relationship.

3. Computation and Implementation

As discussed in Section 2, the data can be partitioned stochastically using (5) at each iteration of an MCMC scheme to sample from the posterior distribution of the model parameters. The MCMC scheme can be completed given prior distributions for the parameters $\phi, \beta, \eta$ and $\sigma^2$. 
3.1. Priors

The likelihood for the partitioned data are derived from (1) and (4), and the Bayesian model is completed by specifying prior distributions for the parameters. The parameters $\beta$ and $\eta$ are given penalised first-order random walk priors as defined in [45] to encourage parsimony and discourage over-fitting.

$$
\pi(\beta | \rho) \propto \exp\left(-\frac{\rho}{2} \beta' K \beta\right)
$$

$$
\pi(\eta | \gamma) \propto \exp\left(-\frac{\gamma}{2} \eta' B \eta\right)
$$

where $K$ and $B$ are first-order random walk penalty matrices, e.g.

$$
K = \begin{pmatrix}
1 & -1 \\
-1 & 2 & -1 \\
& \ddots & \ddots & \ddots \\
& & -1 & 2 & -1 \\
& & & -1 & 1 \\
\end{pmatrix}
$$

Vague proper gamma distributions are specified as conjugate hyper-priors for $\rho$ and $\gamma$ as suggested in [45]. The remaining priors are

$$
\pi(\beta) \propto 1
$$

$$
\pi(\eta) \propto 1
$$

$$
\pi(\sigma^2) = \frac{1}{(1 + \sigma^2)^2}
$$

$$
\pi(\phi) = \prod_{i=1}^{2} \frac{1}{(1 + \phi_i)^2}.
$$

3.2. Computation

Mixture models can be difficult to evaluate as identifiability issues can cause poor mixing or convergence of MCMC chains [64]. This is addressed using additional re-sampling steps for $\beta$ and $\eta$ as suggested in [65]. For example, for $\beta$ at the $i$th iteration:

i) Sample $\beta^{(i)} \sim \pi(\beta | Y^d)$

ii) Sample $\beta^* \sim N(\beta^{(i)}, \alpha I)$

iii) Let $\beta^{(i)} = \beta^*$ w.p. $\min\left(1, \frac{\pi(Y^d | \beta^*)}{\pi(Y^d | \beta^{(i)})}\right)$.

The value $\alpha$ is chosen to set the acceptance rate to between 20% – 40%, as recommended in [65]. The rest of the implementation is a straightforward Gibbs’ MCMC scheme using a hit-and-run sampler with slice sampling [69] for those parameters without conjugate conditional posterior distributions.

4. Results

The Global Terrorism Database (GTD) is an open-source, publicly available dataset that contains records of terrorist events from 1970 [52], containing as at its most recent reporting year, 2016, over 170,000 cases each with up to 120 variables. GTD data from the period 2000–2016 from the countries of Afghanistan, Iraq, and Israel, shown in Figures [1][5] and [9] were analysed using the model derived in Sections [2] and [3]. Results indicate that the diffusion and contagion processes are identifiable and demonstrate distinct patterns that align with theoretical constructs and the historical narrative. Furthermore, the model suggests that the excitation effect varies significantly with the number of fatalities.
Modelling the Spread of Terrorism

Fig. 1. Events in Afghanistan 2000-2016

Fig. 2. $\mu_t^d$ for Afghanistan 2000-2016
4.1. Afghanistan

Figure 2 shows the median and 95% credible interval of the diffusion rate $\mu_d$ for Afghanistan from 2000 through 2016, with some key events annotated.

The diffusion rate is quite low in the period of de facto Taliban rule over a majority of Afghanistan from 1998 until the initial US-led invasion in October 2001 [60]. After the invasion, activity begins to slowly increase as the Taliban began re-organising and re-grouping, and beginning an insurgency and engaging in guerilla warfare [74, 21]. By 2006 US forces were replaced by NATO coalition forces in southern Afghanistan, with the goal of forming Provincial Reconstructions Teams to begin rebuilding Afghanistan and stabilise the political situation [2]. Multiple operations by US and NATO forces to push Taliban forces out of the provinces met with varying degrees of success over the next few years as the US increased troop levels by over 80% in an attempt to defeat the Taliban [55]. Despite these efforts, by the end of 2009 the Taliban’s strength had returned to near pre-invasion levels [56] and intelligence showed a steady increase in security incidents [4]. A slight decrease in the rate of incidents in 2010 coincides with the initiation of peace talks with the Haqqani network by Hamid Kharzai in March of 2010 and the Afghan Peace Jirga [61]. Increases in US troop levels continued in 2010 as part of a “surge” strategy with shift to target Taliban leadership resulting in the capture or killing of more than 900 low- to mid- level Taliban leaders [76]. There is a sharp upturn in $\mu_d$ starting in 2011 and coinciding with the death of Osama Bin Laden in May 2011 and the announcement of US troop withdrawals [44], followed by similar announcements and withdrawals by other coalition members. As the diffusion rate continued to increase, in May of 2012 NATO coalition members endorsed an exit strategy transferring responsibility for security to Afghan forces by mid-2013 [62, 18] which occurred on 18 June 2013 [34]. On 26 October 2014 Britain and the US formally ended their combat operations, handing over their last remaining bases to Afghan control [70, 40].

![Graph showing logarithmic scale and parameters](image)

Fig. 3. $\delta_s$ vs. Fatalities in Afghanistan 2000-2016

The contagion process is best explored in detail through the concepts of volatility and resilience as defined in [79]. Volatility is the expected number of events that occur via contagion after each event, this is parametrised as $\delta_s$. Figure 3 shows the median and 95% credible interval for the endogenous effects coefficient $\delta_s$ as a function of the number of fatalities due to events at time $s$. When the expected volatility is greater than 1, each event is expected to produce more than one subsequent event and the contagion process becomes non-stationary or “explosive”. The values for the median of $\delta_s$ provide evidence of a stationary contagion process (i.e. a volatility consistently less than 1). There is an upward trend in the excitation effect for events with greater than 50 fatalities, but the limited amount of data reflected in the large credible intervals at the limits, makes it difficult to verify. Considering the number of fatalities as a measure of attack size, and an indirect measure of the resources allocated towards an attack, $\delta_s$ becomes a measure of operational capability. Assuming that large attacks are a substantial drain on
resources to mount future attacks, then $\delta_s$ would be expected to decrease with the number of fatalities. The fact that it is approximately constant would seem to indicate that large attacks are not a significant drain on resources or capacity. The increase in frequency and lethality of attacks over time, indicated by the near constant values of $\delta_s$, reflects the Taliban’s resurgence during this time period. The parameters of the decay function $g(\cdot)$ (see 2.2)

\[
g(t) = g(t; \phi)
\]

Fig. 4. Estimated decay function, $g(\cdot; \phi)$, for Afghanistan 2000-2016.

describe resilience, or the duration and intensity of the contagion effect. The contagion effect can be explained using the language of [31] describing the self-exciting model as a cluster process where events serve as a “parent” producing “children” through the contagion process (which subsequently become “parents” have “children” of their own). The decay function for Afghanistan in Figure 4 (median with 95% credible interval) shows that the duration of the contagion effect of an event is limited to a few days. The expected time until a “child” event (i.e. an event attributed to the contagion effect of a previous, or “parent” event) is between 1.46 and 1.70 days after the “parent” event (95% credible interval) with an expected value of 1.6 days. The probability of a “child” event occurring more than 3 days after the originating event is less than 0.05.

4.2. Iraq

Figure 6 shows the posterior median and 95% credible interval for the diffusion rate $\mu^d_t$ in Iraq for the period 2000–2016.

Similar to Afghanistan there were few events in Iraq prior to the US-led invasion in 2003. The US-led invasion of Iraq began on 20 March 2003 and proceeded rapidly with the capital of Baghdad falling to US troops on 9 April 2003 and the declaration of the end of major combat operations on 1 May 2003 [41]. Saddam Hussein remained at large until 13 December 2003, and significant pockets of resistance remained despite the coalition’s efforts to establish a stable post-invasion democracy [28]. After the end of conventional fighting, an insurgency began. Initially fuelled by Ba’ath Party loyalists the insurgency soon drew religious radicals and regular citizens. The violence came to a head on 31 March 2004 when insurgents in Fallujah captured and killed four US private military contractors resulting in the First Battle of Fallujah from 1 April 2004 until 1 May 2004, a campaign to secure Fallujah and capture the insurgents responsible for the deaths of the US contractors [11]. The Second Battle of Fallujah from 7 November 2004 to 23 December 2004 followed as some of the bloodiest fighting in Iraq, and the first time US forces fought forces made up exclusively of insurgents and not the remnants of the Iraqi Republican Guard [63]. In 2005 the Iraqi Transitional Government was elected and charged with writing a new constitution. Despite this insurgent activity continued to escalate, including attacks on the Abu Ghraib prison and fighting around Baghdad and the Euphrates valley [63]. In January of 2007 President Bush announced an increase in both troop levels and reconstruction efforts, this “surge” strategy under the command of the newly appointed commander of
Fig. 5. Events in Iraq 2000-2016

Fig. 6. $\lambda_t^d$ for Iraq 2000-2016
the Iraq Multinational Force, Gen. David Petraeus [9]. In March of 2007 the Iraqi Parliament enacted legislation calling on the US to set a timetable for withdrawal of their forces [59]. By September of 2007 plans were in place to reduce US troop levels to pre-surge numbers [20]. On 4 December 2008, the Iraqi government approved the US-Iraqi Status of Forces Agreement requiring US forces withdraw from Iraqi cities by 30 June 2009 and all US forces be out of Iraq by 31 December 2011. US forces began their withdrawal at the end of June 2009, handing over 38 bases to Iraqi control and removing all forces from Baghdad. In October of 2011 the departure of the remaining US troops was announced, and on 18 December 2011 the last US troops left Iraq. After the withdrawal of US troops the insurgency increased dramatically as Sunni militant groups stepped up attacks of the Shia majority [42]. Between 2011 and 2013 the Arab Spring inspires uprisings across the Arab world, including Syria where it ignites a civil war and gives rise to the Islamic State [17, 66].

![Graph](https://example.com/graph.png)

Fig. 7. $\delta_s$ vs. Fatalities in Iraq 2000-2016

The variation in volatility as a function of fatalities, as shown in Figure 7, is pronounced. There is an obvious trend indicating that volatility decreases as the number of fatalities increases. The model suggests that events producing a large number of fatalities tend to generate almost no contagion effects. However, for events causing 0 or 1 fatalities the median value of $\delta_s$ is greater than 1 indicating that the contagion is potentially in a non-stationary, explosive state. The trend for the frequency of events and the number of fatalities is not as strong as it is in Afghanistan, and there are 38 events with over 100 fatalities, leading to a narrower credible interval at the extremes for $\delta_s$. This paints a different picture of the operational capacity. Rather than the steady increase as seen in Afghanistan by the resurgence of the Taliban, a power vacuum was created after the invasion. The coalition forces outlawed the ethnic minority Ba'ath party and the disbanded the Iraqi armed forces, many of whom were eventually integrated into the Islamic State [68]. Initially, the resistance to the occupation had no central unifying entity, and it wasn’t until the rise of the Islamic State post-2010 and their subsequent assimilation of other resistance groups that a similar unified force against the occupation existed [39]. This explains both the decrease in volatility with casualties, and the non-stationarity for events with 0 or 1 fatality. The resilience of activity in Iraq is described in the decay function $g(\cdot)$ shown in Figure 8 (median with 95% credible interval) and the parameters of the function $g(\cdot)$. The duration of contagion is limited to a few days, the expected time until a contagion event is 1.4 (1.31, 1.47) days and the probability of a contagion event occurring more than 3 days after the originating event is less than 0.05.

4.3. Israel

The history, context, and model results for Israel differ from those of Afghanistan and Iraq. This is reflected in Figure 10 showing the posterior median and 95% credible interval of $\mu_i^d$ for Israel from 2000 to 2016. There is
Fig. 8. Estimated decay function, $g(\phi)$, for Iraq 2000-2016.

Fig. 9. Events in Israel 2000-2016.
significant variation in the diffusion rate, but it never reaches the intensity of Afghanistan and Iraq. There are numerous events that could be identified as possible precipitants, a few have been noted here that align with the data to provide some reference to the results. There is a steep increase in $\mu dt$ following Prime Minister Sharon’s visit to the Temple Mount in Jerusalem [22]. Shortly after this visit the Second Intifada began, a period of increased Israeli-Palestinian violence, and the peak value of $\mu dt$, of approximately 0.15, is reached. After this peak there is a steady decrease surrounding a three-month cease fire announced by Fatah, Hamas and Islamic Jihad on 29 June 2003 [3]. There is a second peak in $\mu dt$ near the start of the 2006 Israel-Lebanon War [51]. The lowest value for $\mu dt$ is reached in September of 2010 around the time of the Sharm el-Sheikh peace summit [25]. After 2010 there is again an increase in $\mu dt$ culminating around the time Israel launched Operation Protective Edge, to stop the launch of rockets into Israel from Gaza [73].
The variation of the volatility as measured by $\delta_s$ for events in Israel follows a similar decreasing pattern as in Iraq and Afghanistan. The volatility never exceeds 1 indicating that the contagion process is stationary. While the number of fatalities per event tends to increase over time in Afghanistan, it is decreasing in Israel over the same period. The decay function in Figure 12 (median with 95% credible interval) shows that the duration of contagion in Israel is limited to a few days. Specifically, the expected time until a contagion event is around 1.6 (1.39, 1.82) days and the probability of a contagion event occurring more than 4 days after the originating event is less that 0.05.

5. Discussion

Results from the models provide two immediately evident results: in general, key shifts in the diffusion rate coincide with key events in the historical narrative; that is the notion of precipitants as exogenous factors is evident in the models, and endogenous excitation effects tends to decrease as the number of fatalities increase. Afghanistan, Iraq, and Israel each prevent different contemporary and historical contexts, which is reflected in in the analyses of their data from the GTD using the model presented here.

The results for all three countries agree with the existing narratives. Both Afghanistan and Iraq were under the rule of repressive regimes prior to invasion by US and Allied forces. Occupation rule in both countries resulted in increased violence which escalated rapidly after the withdrawal of forces and the ceding of authority for security to local forces. The difference between the two is that resistance in Afghanistan came primarily in the form of a reassertion of the deposed regime, the Taliban. In Iraq the regime of Saddam Hussein, including the Iraqi army and his Ba’ath party, were systematically dismantled in an effort to create a democratic secular government, creating a power vacuum and exacerbating sectarian tensions between the Shia majority and Sunni minority, contributing to the violence and unrest. This difference is manifest in the differences in the endogenous effects or volatility as measured by $\alpha_s$ in Figures 3 and 7. The volatility in Afghanistan is relatively constant, with little decrease or change with the number of fatalities, this indicates that there is likely no inhibitory or “blow-back” effects for large scale events. Coupled with the information that the fatalities and frequency of events both increase in time (as the Taliban consolidates and rebuilds post-invasion) the stable volatility also indicates there is no evidence that the execution of large-scale events is not an excessive drain on resources or capacity to attack. In Iraq the volatility shows more variation, as $\alpha_s$ decreases with the number of fatalities. The volatility in excess of 1 for events with 1 or 0 fatalities reflects that numerous groups or individuals were responsible for the events. The decrease in volatility with fatalities reflects either an inhibitory effect for large scale events, or the limited resources of groups.
or individuals to act. In either case the distinction between these patterns is informative.

The pattern in Israel reflects a relatively stable situation, where terrorist activity ebbs and flows. A precarious peace process exists between Israeli and Palestinian claims over the region. Describing the situation as complex is an understatement. In reality, the Israelis and Palestinians share not only claims of sovereignty over the region, but also cultural and economic ties. The ebb and flow of terrorist activity is a part of daily life and exists almost as an extension of political discourse. Increases in activity often result in new rounds of negotiation, or at least proportional responses rather than all-out offences. This is reflected in both the variation of the diffusion rate $\mu_d$, which does not reach the levels of intensity as in Afghanistan and Iraq, and the volatility which is consistently less than 1, indicating a stationary process, and the decrease in volatility as a function of fatalities. Given the relatively low diffusion rate, and the historical context, where violence is often used as a negotiating tool, it is more likely that the decrease in volatility as fatalities increase is likely due to an inhibitory effect.

The classification of mechanisms for the spread of terrorist activity into the categories of diffusion and contagion provides a useful framework for analysing activity. The intent in developing these mathematical models was to create a means of both analysing and classifying data, but also to test and measure the effectiveness of countermeasures. The correspondence between the model results and the existing narratives for the countries analysed validate the use of these models, both conceptually and mathematically. The validation of these models also has important implications for countermeasures. First is the distinction between endogenous and exogenous effects, and their roles in the contagion and diffusion processes. Second is the implication that the dominant mechanism should guide countermeasures, both counter-terrorism efforts and counter-radicalisation efforts. The contagion process is characterised by small-scale dynamics and is governed by endogenous effects, i.e. it is the characteristics of these events and immediate or tactical counter-measures that have influence over the contagion process. The diffusion process is associated with exogenous factors, or large-scale socio-economic and political factors, these are factors that take a different, more strategic set of countermeasures, in order to effect change. Both of these cases require different approaches, a more tactical approach to addressing factors that effect the contagion process, and a more strategic approach is required to address factors effecting the diffusion process. If, as is likely, real-world situations are a mixture of both diffusion and contagion then counter-terrorism and counter-radicalisation policies should be shaped to address the two processes and their particular balance in each setting. The models here provide a useful tool for analysis to both assess the mechanisms at work in any given context and to measure the effectiveness of enacted counter measures. In practice, counter-terrorism and counter-radicalisation activities typically have the possibility of negative consequences, an effective measure of both what measures are needed and their effectiveness is important to minimising potential negative effects. Thus the utility of the models proposed here is beyond academic and offer substantial benefit to real-world policy makers.

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