Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

From Exposure to Intervention: A Continuous Kinetic Model of Community Violence

Yassin Bahid
University of Colorado Boulder
Jeff Brantigham
University of California, Los Angeles
Nancy Rodriguez
rodrign@colorado.edu

University of Colorado Boulder

Research Article

Keywords: Mathematical models, Public health, Violence exposure

Posted Date: June 1st, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9752796/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

From Exposure to Intervention: A Continuous Kinetic Model of Community Violence

Yassin Bahid[1], Jeff Brantigham[1†], Nancy Rodríguez[2*†]

[1*]Applied Mathematics, University of Colorado, Boulder, 1111 Engineering Center, ECOT 225, Boulder, 80309, CO, USA.
[2]Anthropology, University of California, Los Angeles, 375 Portola Plaza, 341 Haines Hall, Los Angeles, 90095, CA, USA.

*Corresponding author(s). E-mail(s): rodrign@colorado.edu;
Contributing authors: Yassin.Bahid@colorado.edu; branting@g.ucla.edu;
†These authors contributed equally to this work.

Abstract

Background: Building on a decades-long effort to treat violence as a public health issue, researchers have started to use more formal epidemiological tools to understand the spread of violent behavior in communities. The work of Sisk et al. (2022) introduced a compartmental model inspired by the dynamics of infectious diseases to capture the transitions between susceptible, exposed, and violent individuals. While their approach provided useful insights, it used fixed exposure categories that may not capture the continuous nature of behavior change.

Methods: We extend the framework of Sisk et al. by creating kinetic models that treat violence as a contagion process evolving along a continuous scale. This scale represents either a person's propensity toward violence in the general population or their level of violence in an actively violent group. Instead of using discrete compartments, our model uses a density function to describe how individuals are distributed across different risk levels. The system is governed by integro-differential equations that include social influence, escalation, rehabilitation, and reintegration.

We introduce two models. The first allows only the general population to evolve continuously in the behavioral state, while the second extends the continuous framework to all groups and incorporates the influence of response management. For the first model, we derive a threshold quantity we refer to as the *violence persistence number* (VPN), which measures the extent to which interactions

within the population amplify or suppress violence at the collective level. Unlike the classical basic reproduction number in epidemiology, the VPN is not interpreted as the number of new violent individuals generated by one violent individual. Instead, it characterizes whether the combined effects of exposure, escalation, recovery, and intervention are sufficient for violence to persist within the modeled system. For the second model, we investigate how different management strategies influence long-term population dynamics.

Results: Simulations reveal rich population dynamics that are not captured by discrete compartmental models. Increasing exposure intensity or reducing de-escalation shifts the susceptible population toward higher propensities for violence, while enhanced recovery or reintegration moves the distribution toward lower-risk states. The VPN identifies threshold regimes in which modeled interaction mechanisms either sustain or suppress violence. Numerical results further suggest that relatively small changes in intervention or de-escalation mechanisms can substantially alter the long-term distribution of violent behavior. In the model with management strategies, different institutional response strategies produce qualitatively distinct outcomes in both the prevalence and severity of violence.

Conclusion: This work extends the framework of Sisk et al. by providing a more flexible and detailed description of how exposure, intervention, and institutional responses shape community violence. By representing exposure as a continuous behavioral variable, the kinetic framework captures cumulative and heterogeneous changes in violence propensity that are difficult to model using discrete compartments alone. More broadly, the framework connects epidemiological ideas with mesoscopic behavioral dynamics, providing a quantitative tool for studying how social interactions and interventions contribute to the persistence or suppression of violence in communities.

Keywords: Mathematical models, Public health, Violence exposure

1 Background

Exposure to violence is increasingly understood to be an important driver of future violence. Studies of gang retaliation show that shootings often trigger cycles of retaliatory violence [1, 2], while recent work suggests that online conflicts among gangs can spill over into offline shootings [3, 4]. At the social network level, violence has also been shown to cascade through interpersonal connections, increasing the risk of future victimization and offending [5, 6]. More broadly, public health studies have consistently demonstrated that exposure to violence during childhood and adolescence is associated with an increased likelihood of later violent behavior, victimization, trauma, and adverse health outcomes [7–9]. These observations support the idea that violence is not simply the result of isolated individual decisions but emerges through repeated social exposure and interaction within communities.

This exposure-driven perspective has motivated the use of epidemiological models for studying violence dynamics. Early work by Slutkin argued that violence exhibits several features commonly associated with infectious disease spread, including clustering, transmission through social contact, and epidemic-like outbreaks [10, 11]. These ideas motivated intervention programs such as Cure Violence, which apply public health strategies including interruption, outreach, and exposure reduction [12]. In parallel, mathematical models inspired by susceptible-infected-recovered (SIR) frameworks have been developed to describe how individuals transition between nonviolent, at-risk, and violent states through exposure to violence [13]. Related epidemiological and agent-based approaches have also been used to study riots, crime hotspots, and social contagion phenomena [14–16]. These models provide a useful mathematical framework for studying how violence can persist and spread through populations, while connecting individual experiences of exposure to broader community-level patterns.

Despite their usefulness, compartmental models necessarily reduce exposure into transitions between a small number of discrete classes. In reality, however, exposure to violence is not binary. Individuals experience different frequencies, intensities, and forms of exposure, and these experiences may gradually alter their propensity toward violence over time. Moreover, social and institutional factors, including policing strategies, peer networks, neighborhood conditions, and structural inequality, may continuously reinforce or suppress these behavioral tendencies [17, 18]. As a result, the effects of exposure are often cumulative, heterogeneous, and dynamic, making them difficult to capture through compartment changes alone.

In this work, we develop a mesoscopic kinetic framework that treats propensity toward violence as a continuous microstate variable. From this perspective, exposure does not simply move individuals from one compartment to another. Instead, repeated social interactions continuously reshape the distribution of propensities across the population. This approach allows exposure itself to be incorporated directly into the evolution of the microstate distribution, while freeing subsystem structure to model broader social and institutional mechanisms, including policing responses, escalation effects, and heterogeneous interaction patterns. In this way, the framework connects individual-level exposure dynamics with emergent community-level patterns of violence.

2 Methods

2.1 Building on the analogy between violence and infectious disease

Sisk and collaborators draw a compelling analogy between violence and infectious disease, highlighting how exposure can increase both a person's risk of victimization and their likelihood of committing violence. Similar to how a pathogen can weaken the body, exposure to violence can negatively affect mental health and increase behavioral risk. Conversely, programmed interventions, family support, and mentoring, can act like an immune system, disrupting the spread of violence and promoting resilience [19].

While this analogy provides important insight, traditional compartmental models treat individuals in discrete states (e.g., susceptible, exposed, violent), which are at

odds with the continuous nature of real-world behavior. Indeed, people may have varying propensities for violence depending on their age, experiences, resources, and social context [20, 21]. A kinetic framework naturally extends the epidemiological analogy. Kinetic equations originate from physics, where they describe the motion and interactions of large groups of particles. In public health, the same concept can be applied to explain how large groups of people modify their behaviors, risks, or health status over time. In the context of violence, we extend the analogy by allowing individuals to be distributed along a continuous "*propensity toward violence*" scale, much like a distribution of immune strength or viral load in models of disease dynamics. This approach enables us to model gradual behavioral change, cumulative exposure, and partial recovery in a way that discrete compartments cannot.

Moreover, kinetic frameworks capture interactions at multiple scales. Individual-level interactions (peer influence, family support) influence an individual's propensity, while community-level structures (policing, social services, neighborhood inequality) shape the overall distribution of risk. By tracking the full distribution of propensities over time, we can quantify not only average outcomes but also the emergence of clusters, hotspots, or high-risk subpopulations. This makes it easier to identify which interventions are most effective in reducing people's risk of violence and preventing its spread, much like public health models guide efforts to control disease outbreaks.

2.2 Overview and Modeling Approaches

2.3 The Sisk model

Our starting point is the model proposed by Sisk et al. [13], which divides individuals into several compartments: Susceptible (S), three Exposed states (E_1, E_2, E_3), and Infected (V). Note that "infected" is equivalent to "violent" in this context. The model proposed in [13] was a system of differential equations illustrated in the top part of Figure 1. The parameters μ_1, μ_2, and μ_3 represent the initial exposure rates, enabling individuals to adjust to a suitable exposure level based on the severity of the first exposure. The parameters ω_1, ω_2, and ω_3 describe additional exposure that occurs after the initial one. The parameter β_3 represents the rate at which individuals move from the violent state back to the exposed state. The parameters β_1 and β_2 represent processes that reduce exposure levels. More generally, the values β_i can be viewed as measures of efforts or interventions that help people reduce their likelihood of reoffending [13].

2.4 A hybrid mesoscopic-compartmental model of violence dynamics

We build on the above framework by extending it into a multiscale kinetic formulation that links individual-level interactions to population-scale social dynamics. Our mesoscopic model describes population distributions in terms of individual tendencies rather than simple compartmental counts. In this setting, each subpopulation is characterized by a probability density over behavioral states, allowing us to represent heterogeneity within groups and to model feedback between social and institutional factors.

We introduce and analyze two models. The first tracks two groups: the *general population* and the *actively violent individuals*. People in the general population can move toward higher levels of propensity towards violence if they interact with violent individuals. They can also move toward a lower propensity towards violence through positive influences, such as supportive relationships, education, or community programs. Those who reach a very high propensity may transition into the violent group, while some actively violent individuals can return to the general population through interventions or reintegration programs. In this model, the actively violent population does not exhibit a behavioral tendency. Rather, we only keep track of the violent compartmental count. The framework for this new model and its comparison to the ODE model introduced by Sisk and collaborators are illustrated in Figure 1, which highlights the generalization made by the kinetic framework. We refer to this new model as the Progressive-Risk Model (PRM).

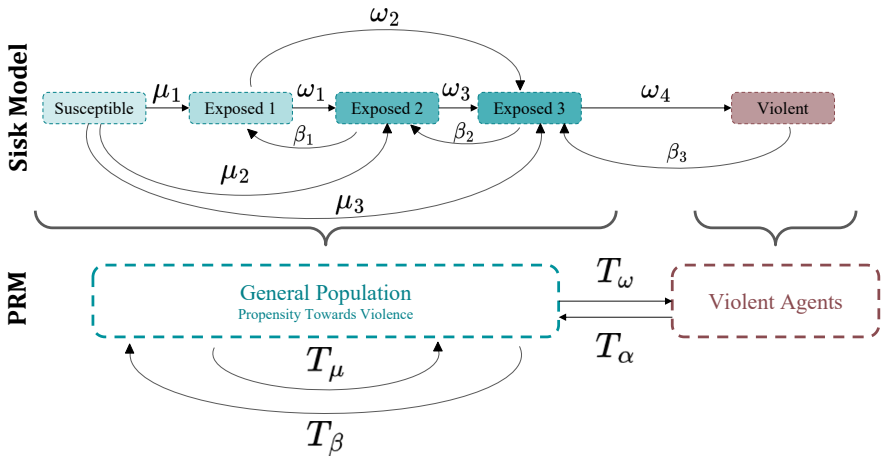


Fig. 1 Illustration of the transition from a compartmental model to a kinetic model. The Sisk model is illustrated at the top, showing the S, E_1, E_2, E_3 and V compartments and their transitions. The bottom model illustrates the kinetic framework for PRM. The compartments S, E_1, E_2, and E_3 are combined into the general population, denoted by S for simplicity, while the violent compartment remains unchanged.

The system of equations can be found in the Supplementary Material. Here, we summarize the factors modeled by this system.

- *Continuous susceptibility:* The sharp distinction between "susceptible" and "exposed" individuals is now replaced by a population subsystem wherein each individual has a continuous *propensity towards violence*, denoted by u. Larger values of u correspond to individuals who are more likely to transition into active violence and thus can be interpreted as having a higher susceptibility to violence. Conversely, smaller values of u indicate individuals with less propensity towards violence.
- *Exposure from violent interactions:* The interaction between an individual with a propensity towards violence u with an individual who is actively violent increases the propensity towards violence by some amount $(u \to u + \mu^*(1-u))$.

- *Recovery:* The impact of interventions or societal influences that reduce violent behavior. It models how individuals can gradually lower their exposure to violence over time, reflecting disengagement from violent activity.
- *Interactions among susceptibles:* Highly exposed susceptibles influence other less exposed individuals, increasing the latter's risk of becoming violent.
- *Movement to the violent population:* Interactions with violent individuals can drive highly exposed susceptibles into the violent population.
- *Violent population decay:* The recovery or exit from violence moving to the susceptible compartment with a high level of susceptibility.

We compare the parameters and variables of PRM and the Sisk model in Table 1.

2.5 A fully mesoscopic model for violence

To study the effect of institutional intervention forces, such as policing, crisis responders, mental health responders, and other response teams, we introduce a fully mesoscopic model in which all subpopulations, the general population (S), the violent subgroup (V), and the responder population (R), are represented by distributions over individual-level traits. The goal is to capture the feedback between violence and institutional response, and to illustrate how different response strategies can influence overall crime levels and the distribution of violence across different severity levels. We refer to this framework as the Mesoscopic Violence–Responder Model (MVRM). In MVRM, we consider three main subpopulations, each characterized by a specific behavioral trait. The subpopulations are as follows:

- $S(t, u)$: the *general population*, where $u \in [0, 1]$ represents an individual's propensity toward violence;
- $V(t, v)$: the *actively violent subgroup*, where $v \in [0, 1]$ indicates the current level of violent activity;
- $R(t, w)$: the *response group density*, where $w \in [0, 1]$ represents the response level.

This fully mesoscopic model extends PRM by introducing internal structure not only for the general population, but also for the violent subgroup and the responder population. In PRM, only the susceptible population evolves over a continuous exposure scale, while the violent population is treated as a single homogeneous compartment. In contrast, the fully mesoscopic model represents both violent individuals and responders through continuous trait distributions, allowing the intensity of violent behavior and the level of response activity to vary across individuals.

This added structure introduces additional feedback mechanisms: the evolution of violence depends on how responders are distributed across their behavioral states, while the effectiveness of the response depends on the distribution and severity of violent behavior. As a result, the MVRM captures richer bidirectional interactions between violence and institutional response, and can describe how targeted intervention strategies influence not only the total level of violence, but also its distribution across different severity levels. Figure 2 illustrates the model (the full system is provided in the supplemental material).

Table 1 Comparison of the parameters and dynamics modeled in the Progressive-Risk Model and the compartmental model of Sisk et al. [13].

Parameter / Term	**PRM Model**	**Sisk et al. Model**
Susceptible population	$S(t,u)$: density distributed across propensity towards violence $u \in [0,1]$	$S(t)$: single susceptible compartment
Violent population	$V(t)$: total violent population	$V(t)$: total violent population
Exposure mechanism	Violent interactions continuously increase exposure through $T_\mu : u \mapsto u + \mu^*(1-u)$	Exposure modeled through transitions from S into discrete exposed compartments E_1, E_2, E_3 with rates μ_i
Recovery / de-escalation	Exposure reduced continuously through $T_\beta : u \mapsto (1-\beta^*)u$ at rate β	Recovery modeled through transitions between exposed compartments using rates β_i, with β_3 governing exit from V
Movement into violent class	Individuals with sufficiently high exposure transition into V through $T_\omega = \chi_{[1-\epsilon,1]}$ at rate ω	No explicit threshold mechanism; violent behavior emerges through transitions between discrete exposure compartments and V
Movement out of violent class	Violent individuals return to highly exposed susceptible states through $T_\alpha(u) = \chi_{[1-\epsilon,1]}$ at rate α	Return dynamics incorporated implicitly through ω_i transitions between E_i and V
Exposure structure	Continuous exposure variable allowing heterogeneous and cumulative behavioral change	Finite number of discrete exposure states
Exposure threshold	ϵ: determines when individuals are sufficiently exposed to enter or leave V	No explicit threshold; exposure levels represented implicitly by compartments
Modeling perspective	Mesoscopic kinetic framework with continuous behavioral evolution	Compartmental epidemiological framework

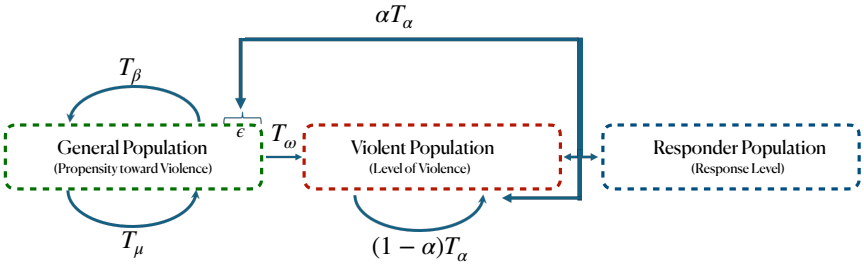


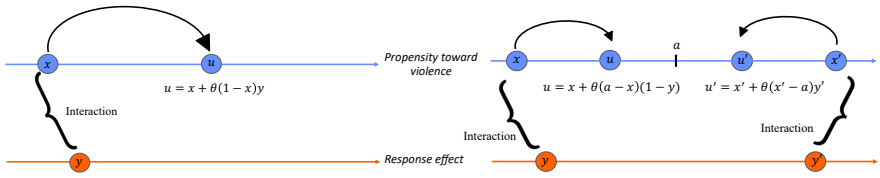
Fig. 2 Illustration of the Mesoscopic Violence–Response Model. In this system, violent agents have a microscale called the *level of violence*. They are interacting with the new subsystem "Response group" with the microscale *response level*. Upon interaction, violent agents either exit the subsystem to become part of the general population at a rate α or change microscale with rate $1-\alpha$. We illustrate an escalated response, where individuals level of violence increasing upon interacting with the response team.

The response effect. In the fully mesoscopic model, the effects of the response team on violence are captured through a *response effect function*. This function describes how an individual's level of violence changes after interacting with the response team. It tells us whether a person becomes more or less violent depending on the intensity of the specific situation they experience. We tested two types of functions relevant to fitting actual crime data in our preprint [22]. We consider two possible functions modeling either:

- **Escalation**: In this response strategy, interactions with responders increase an individual's level of violence, with the largest increases occurring among individuals who initially exhibit low levels of violent behavior, while highly violent individuals saturate near the maximal violence state. Such dynamics may arise in settings where responders employ escalated or aggressive intervention tactics, potentially amplifying hostility and retaliatory behavior. Related escalation mechanisms between institutional response and violence have been discussed in studies of conflict escalation and police use of force [23, 24].
- **Threshold**: In this response mechanism, individuals are driven toward a target violence level determined by a threshold parameter a. Individuals with violence levels below the threshold tend to escalate toward a, while individuals above the threshold are moderated toward it. As a result, the response strategy produces a stabilizing effect that limits runaway escalation and concentrates the violent population around an intermediate level of violence. Such behavior may arise from calibrated intervention strategies, including de-escalation tactics, crisis intervention, or graduated response protocols that aim to contain extreme violence while maintaining control of lower-level incidents.
- **De-escalation**: A de-escalation response strategy aims to reduce the intensity of violent behavior following interactions with responders. In this setting, interactions systematically shift individuals toward lower violence levels, with stronger effects occurring for highly violent individuals. Such mechanisms are consistent with

intervention strategies designed to reduce conflict intensity, including crisis intervention, procedural justice approaches, trauma-informed response practices, and communication-based de-escalation techniques. From a modeling perspective, these interactions produce a stabilizing effect by decreasing the proportion of individuals in high-violence states and redistributing the violent population toward lower levels of violence. Empirical studies suggest that de-escalation-focused response strategies can reduce the severity of violent encounters and lower the likelihood of escalation during high-conflict situations [25–27].

Figure 3 illustrates two of the three response effect functions. In Figure 3(a), interactions with response team increase an individual's propensity toward violence, representing escalation. In contrast, Figure 3(b) shows a threshold response, where interactions with the response team move an individual's propensity toward a target level. In both functions, the parameter θ measures the *violence adjustment rate*, that is, an increase in the propensity towards violence. The update rule for both functions ensures that the behavioral characteristics of each subpopulation remain within the interval $[0, 1]$. Table 2 lists all parameters with their physical interpretations.



(*a*) Escalation (*b*) Threshold

Fig. 3 Illustrations of how response team's interactions influence changes in violence levels of individuals. Each function represents how an individual's level of violence changes after encountering the response team with a given intensity. (a) Escalation effect: the presence of the response team leads to an increase in violence, with stronger response producing larger increases, especially for individuals who are not already highly violent. (b) Stabilizing (threshold) effect: interactions with the response team push individuals toward a target level of violence. Individuals below this level may become more violent, while those above it are de-escalated, resulting in an overall moderation of behavior.

Table 3 compares the three models in terms of their level of detail and the mechanisms they include. The model of Sisk et al. is the simplest, using a small system of ODEs with limited structure and no interactions within susceptibles or response team effects. The PRM adds more detail by modeling susceptibles as a continuous distribution, which allows for a clearer description of exposure. The MVRM is the most detailed, representing susceptibles, violence, and response team as continuous distributions and including feedback between violence and response. Overall, the models move from low to high resolution, with increasing ability to capture heterogeneity and interactions.

Parameter	Interpretation
μ	Rate at which susceptibles increase exposure due to interactions with violent individuals.
β	Reduction of propensity towards violence over time (disengagement).
α	Rate of exit from violent population into the highly exposed general population.
ϵ	Small parameter defining the interval of high exposure near one for the recruitment and interaction functions.
θ	Violenc adjustment rate.
a	Target violence level in individuals for threshold response strategy.
μ^*	Increase in propensity towards violence.
β^*	Decrease in propensity towards violence after positive interaction.

Table 2 Model parameters and interpretations.

2.6 Simulations

We run several simulations for our two models to understand the effect that parameters have on the behavior of the solution. For each experimental run, we initiate the general population to be 99% of the population and the initial violent population to be 1%. We normalize our densities to have a total mass of one for easy comparison.

3 Results

3.1 The Progressive Risk Model

Although our model is inspired by compartmental epidemic frameworks, the interpretation of a reproduction number in the context of violence must be approached with caution, as noted in [13]. Unlike infectious diseases, exposure to a single violent act does not generally produce a direct or immediate "transmission" event. Rather, violence emerges through a complex combination of repeated exposure, environmental conditions, social interactions, institutional responses, and individual predispositions. Consequently, the threshold quantity derived from our model should not be interpreted as the expected number of "new violent individuals" generated by one violent individual in the epidemiological sense. Instead, we interpret this quantity as a measure of how interaction mechanisms within the population amplify or suppress violent behavior at the collective level. In particular, it characterizes whether the combined effects of exposure, escalation, recovery, and intervention are sufficient for violence to persist or grow within the modeled system. For this reason, we refer to this quantity as the *violence persistence number* (VPN) rather than the classical basic reproduction number.

We note that because violent behavior may emerge from exogenous social, economic, or environmental factors not explicitly represented in exposure interactions, violence may still increase even when this threshold quantity is below one. Thus, the quantity should be viewed not as a strict predictor of violence emergence, but rather as a tool for quantifying the relative contribution of modeled interaction mechanisms to the persistence of violence.

Table 3 Comparison of the structural features and modeling resolution of the Sisk model, the Progressive-Risk Model (PRM), and the Multi-Variable Propensity Model (MVRM).

Feature	**Sisk et al.**	**PRM**	**MVRM**
Model type	Finite-dimensional ODE system	Mesoscopic model coupled with a scalar ODE	Fully mesoscopic kinetic system
Susceptible population	$S(t)$	$S(t,u)$ over $u \in [0,1]$	$S(t,u)$ over $u \in [0,1]$
Violent population	$V(t)$	$V(t)$	$V(t,v)$ over $v \in [0,1]$
Response team density	None	None	$P(t,w)$ over $w \in [0,1]$
Exposure increase	Discrete transitions with rates μ_i	Continuous exposure dynamics through $\mu\, T_\mu$	Continuous exposure dynamics through $\mu\, T_\mu$ coupled to $V(t,v)$
Recovery / de-escalation	Discrete recovery transitions with rates β_i	Continuous recovery through $\beta\, T_\beta$	Continuous recovery through $\beta\, T_\beta$
Susceptible–susceptible interactions	None	Modeled through $\omega\, T_\omega$	Modeled through $\omega\, T_\omega$
Movement into violent class	Through compartment transitions ω_i	Through $\alpha\, T_\alpha$	Through $\alpha\, T_\alpha$ with response influence
High-exposure threshold	Implicit through exposed compartments E_1, E_2, E_3	Explicit threshold ϵ	Explicit threshold ϵ
Violence–Response feedback	None	None	Two-way interaction and feedback
Model resolution	Low resolution (finite number of compartments)	Intermediate resolution (S continuous)	High resolution (S, V, and P continuous)

The violence persistence number for PRM is given by:

$$\mathcal{V}_0 = \begin{cases} \dfrac{\omega}{\beta+\omega}, & \mu^* \notin [1-\epsilon, 1], \\ \dfrac{\omega}{\beta+\omega}\left(1+\dfrac{\mu}{\alpha}\right), & \mu^* \in [1-\epsilon, 1]. \end{cases} \tag{1}$$

This reveals a threshold effect controlled by μ^*, which represents the increase in propensity toward violence after a violent interaction. Outside the interval $[1-\epsilon, 1]$, the dynamics are governed only by the balance between two processes: interactions among susceptibles that increase their propensity toward violence (at rate ω), and mechanisms that reduce exposure (at rate β). Within the interval $[1-\epsilon, 1]$, an additional amplification term proportional to μ/α becomes active. In this regime, increasing μ (the rate of interaction between susceptibles and the violent population) promotes the spread of violence, while increasing α (the rate of reintegration of the violent population to the general population) suppresses it. The parameter ϵ determines the size of this threshold region, and thus how easily the amplification mechanism is triggered.

Figure 4 presents heatmaps showing the maximum value of V as a function of different parameter pairs (β, μ) and (α, ω). The black line in each panel indicates the threshold where $\mathcal{V}_0 = 1$, with all other parameters held constant at $\alpha = 0.5$, $\omega = 0.05$, $\mu^* = 0.95$, $\beta^* = 0.1$, and $\epsilon = 0.1$ in (a), and $\mu^* = 0.95$, $\beta = 0.01$, $\mu = 0.1$, $\beta^* = 0.01$, and $\epsilon = 0.1$ in (b). Point (A) in panel (b) is chosen to have a high VPR, leading to sustained growth in violence, as shown in the top right panel. Point (B) lies near the threshold, with $\mathcal{V}_0$ slightly above one; here, violence exhibits a small initial increase but does not persist and eventually decays, as seen in the middle right panel. Finally, point (C) is selected with $\mathcal{V}_0 < 1$, resulting in an immediate decline in the violent population, as shown in the bottom right panel.

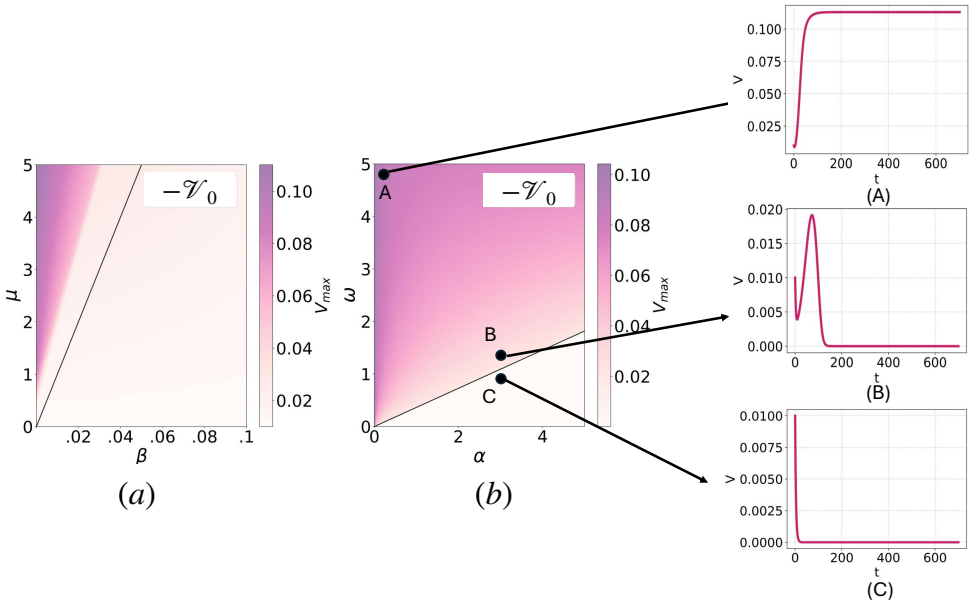


Fig. 4 Heatmaps of the maximum value of V over the time interval $t \in [0, 1000]$ for the hybrid system, initialized with a small perturbation $V = 0.01$ from the violence-free equilibrium $S(u) = \delta(u)$ and $V = 0$. The curve indicates $\mathcal{V}_0 = 1$, separating regions where violence decays from those where it grows. Panel (a) varies β and μ, with all other parameters held constant at $\alpha = 0.5$, $\omega = 0.05$, $\mu^* = 0.95$, $\beta^* = 0.1$, and $\epsilon = 0.1$. Panel (b) varies α and ω, with all other parameters fixed with $\mu^* = 0.95$, $\beta = 0.01$, $\mu = 0.1$, $\beta^* = 0.01$, and $\epsilon = 0.1$ in (b). The panel on the right show time series of V for representative parameter choices: (A) $\alpha = 0.1$, $\omega = 4.9$; (B) $\alpha = 2.2$, $\omega = 1.2$; and (C) $\alpha = 2.2$, $\omega = 0.8$, where $\mathcal{V}_0 < 1$.

Figure 5 shows the evolution of the PRM solution while varying three different parameters. The first row varies μ while keeping the other parameters fixed ($\mu^* = 0.4$, $\beta = 0.04$, $\beta^* = 0.1$, $\epsilon = 0.1$, $\omega = 0.01$, and $\alpha = 0.5$). The results show a clear transition as exposure increases. When exposure is low ($\mu = 0.1$), the violent population quickly dies out. At a moderate level ($\mu = 0.3$), there is a slight, short-lived increase, but violence still does not persist. In contrast, high exposure ($\mu = 0.6$) leads to sustained growth and the onset of a violence epidemic. Across all cases, the general population initially evolves similarly, reflecting the common starting condition. Over time, however, the trajectories separate: higher exposure steadily shifts the population toward greater propensity levels, which in turn supports the growth of violence.

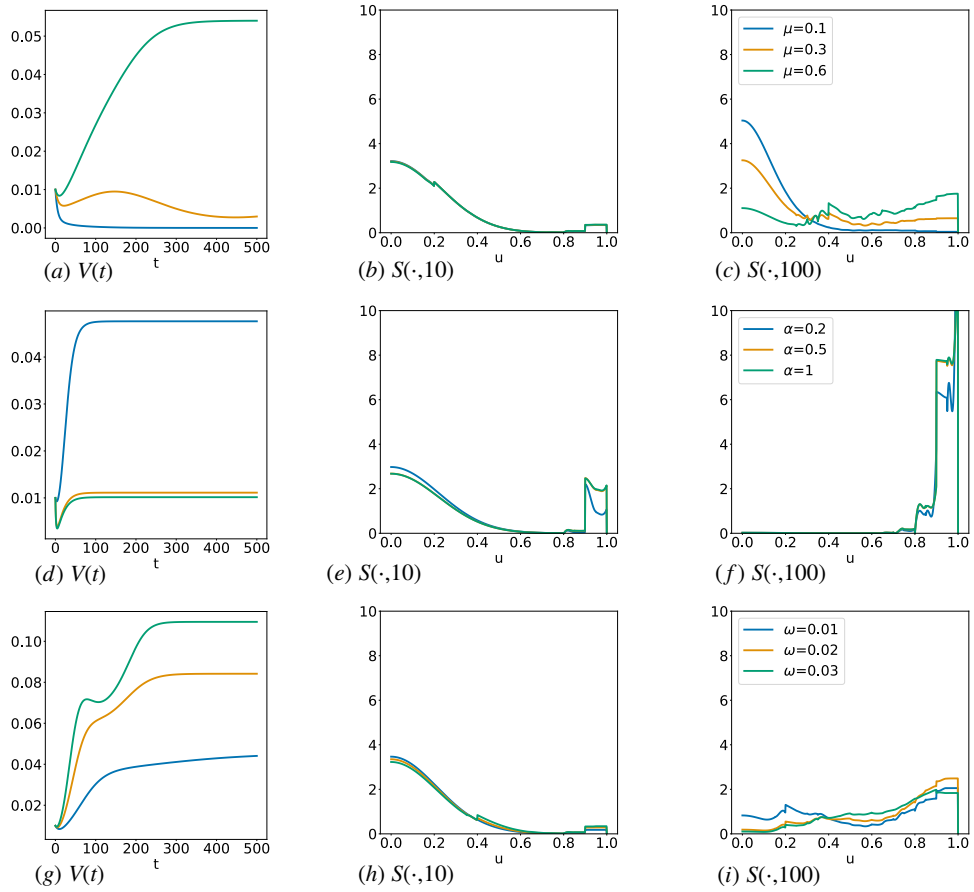


Fig. 5 Temporal evolution of solutions to PRM under different parameter variations, where each row varies a single parameter while the others are held fixed. The top row varies μ with $\mu^* = 0.4$, $\beta = 0.04$, $\omega = 0.01$, and $\alpha = 0.5$; the middle row varies α with $\mu = 0.1$, $\mu^* = 0.2$, $\beta = 0.04$, and $\omega = 0.01$; and the bottom row varies ω with $\mu = 0.1$, $\mu^* = 0.9$, $\beta = 0.04$, and $\alpha = 0.5$. All simulations use $\beta^* = 0.1$, $\epsilon = 0.1$. In each case, the first column shows the time evolution of V, while the second and third columns show the distribution of S over its microstate at $t = 10$ and $t = 100$, respectively.

In the middle row, we vary α while keeping $\mu = 0.1$, $\mu^* = 0.2$, $\beta = 0.04$, $\beta^* = 0.1$, $\epsilon = 0.1$, and $\omega = 0.01$ fixed. Recall that α represents the rate at which individuals leave the violent compartment due to interventions and positive societal influences. When α is small ($\alpha = 0.2$), the violent population increases. As α increases, the level of violence decreases. The difference in the time dynamics between $\alpha = 0.5$ and $\alpha = 1$ is relatively small, suggesting that even moderate increases in α can substantially reduce violence.

Finally, the bottom row varies ω while keeping $\mu = 0.1$, $\mu^* = 0.2$, $\beta = 0.04$, $\beta^* = 0.1$, $\epsilon = 0.1$, and $\alpha = 0.5$ fixed. Recall that ω is the rate at which highly susceptible individuals transition into the violent state. Small changes in ω lead to large differences in outcomes. For example, when $\omega = 0.01$, the final value of V is about

4%, while for $\omega = 0.03$, it exceeds 10%. This indicates that the dynamics are highly sensitive to ω, highlighting the importance of preventing individuals from reaching the violent state.

3.2 The Mesoscopic Violence–Response Model

In this section, we discuss the fully-mesoscopic model. We perform a comparison between the three response strategies. First, we consider similar initial conditions and parameters to see how the two strategies affect the evolution of S and V. This is illustrated in Figure 6, where S and V are in their respective microstates in MVRM with escalation, threshold, and de-escalation response strategies. The distribution of response teams is assumed to be normal with a mean of 0.5 and a standard deviation of 0.1. In terms of the microstate variable, this means that most individuals in the response population possess intermediate values of the modeled behavioral characteristic, while relatively few possess very low or very high values. Thus, the intervention population is concentrated around a moderate response level, with limited heterogeneity. The parameters used were $\mu = 0.8$, $\mu^* = 0.2$, $\beta = 0.01$, $\beta^* = 0.1$, $\epsilon = 0.1$, $\omega = 0.07$, $\alpha = 0.01$, $a = 0.6$, $\gamma = 0.4$.

We observe that the dynamics of the susceptible population are qualitatively similar under both response functions. Interestingly, the de-escalation response produces a higher average propensity toward violence within the susceptible population. This occurs because de-escalation increases the rate at which actively violent individuals return to the general population, while still retaining relatively high levels of exposure or propensity toward violence. As a result, the susceptible population contains a larger fraction of individuals near the transition threshold to violence.

In contrast, the dynamics of the violent population differ significantly across the response strategies. Under the escalation response, the average propensity toward violence increases rapidly and shifts toward higher violence levels over time, indicating strong amplification of violent behavior. Under the threshold-based response, violence levels grow more gradually, producing a stabilizing effect that limits extreme escalation. Under the de-escalation response, the violent population rapidly shifts toward lower violence levels, leading to an overall reduction in the severity of violent behavior.

Overall, the simulations suggest that different response strategies may produce similar short-term effects on the general population while generating substantially different long-term distributions of violence intensity. In particular, de-escalation strategies appear effective at reducing severe violence, although they may also increase the number of individuals in the general population with elevated susceptibility to future violent behavior.

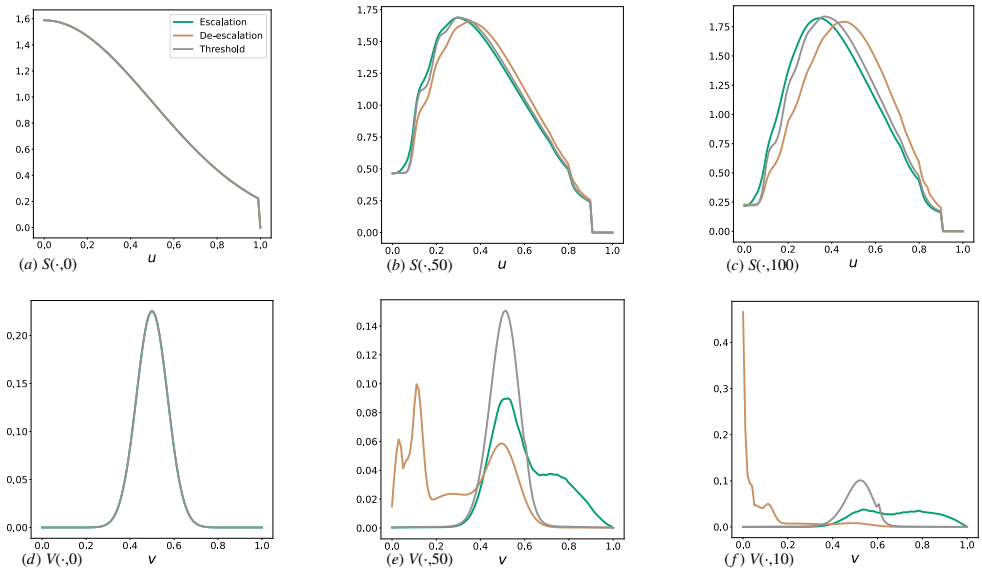


Fig. 6 Temporal evolution of the solutions to MVRM where the response effect is either of monotonic escalation or threshold type. The top row shows the evolution of S, while the bottom row illustrates the evolution of V. The simulation spans the time interval $[0, 100]$.

We also performed a systematic study of how the three response strategies affect the mean and mass of the subsystems S and V as the parameters θ and the response level vary. All other parameters are fixed at $\beta = 2$, $\mu = 0.3$, $\mu^* = 0.01$, $\beta^* = 0.3$, $\alpha = 0.05$, $\omega = 0.1$, and $\epsilon = 0.1$. The top row of Figure 7 shows the results for the escalation response, the middle shows the results for the threshold response with $a = 0.6$, and the bottom row illustrates the de-escalation response.

For the escalation response, increasing the response level leads to a decrease in the mass of the nonviolent population S and an increase in both the mass and the average violence level of the violent population V. From an application perspective, this suggests that stronger interactions move more individuals from the nonviolent subsystem into the violent subsystem, while also shifting violent individuals toward higher levels of violence. The combined effect is both a larger violent population and a higher average intensity of violence. For the threshold response, increasing the response level again increases the mass of the violent subsystem V, indicating that more individuals transition into violence. However, unlike the escalation case, the average violence level within V remains relatively unchanged as θ varies. For the chosen value of a, the response primarily affects the number of individuals entering the violent subsystem rather than the average severity of violence among those already in it.

Under de-escalation, the violent population remains extremely small across nearly all parameter values. This is visible in the very low mass of V, which is orders of magnitude smaller than in the other two response strategies. At the same time, the

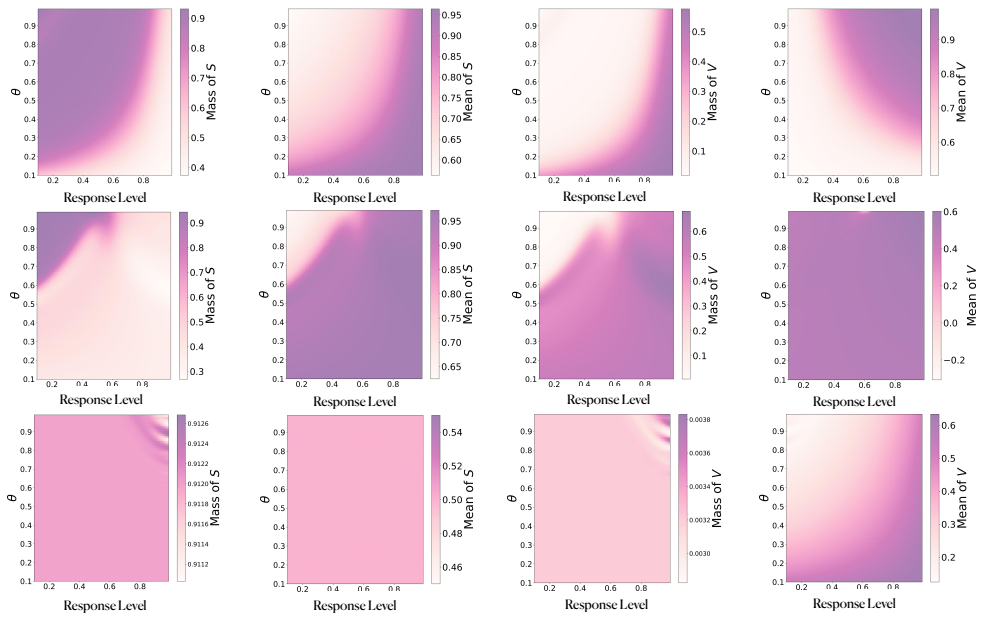


Fig. 7 Heatmaps showing the total mass and mean values of S (general population) and V (violent group) as functions of the average response rate and θ, computed from the fully mesoscopic model with the escalation function, in the first row, and the threshold response, in the second row. A normal distribution of response levels with varying means is compared across different θ values, representing changes in response effectiveness. All quantities are evaluated at $t = 100$, with parameters fixed at $\beta = 2$, $\mu = 0.3$, $\mu^* = 0.01$, $\beta^* = 0.3$, $\alpha = 0.05$, $\omega = 0.1$, and $\epsilon = 0.1$.

mass of S remains close to one, indicating that most individuals stay within the general population. However, the mean violence level within the violent subgroup can still increase moderately for large response levels. This suggests that de-escalation effectively suppresses widespread violence, but the small number of individuals who remain violent may still exhibit moderate or elevated violence levels. In particular, the mean propensity of the susceptible population remains near intermediate values. We can be interpreted as de-escalation returning individuals from the violent population back into the general population with high values of propensity before their underlying propensity toward violence has fully dissipated.

These results suggest that the qualitative structure of violence is strongly dependent on the institutional response. Escalation strategies tend to amplify both the prevalence and severity of violence, producing widespread recruitment into highly violent states. Threshold-based responses create a stabilizing effect that limits extreme escalation and concentrates the system near moderate violence levels. In contrast, de-escalation strategies are highly effective at suppressing the overall size of the violent population, although they may leave a larger fraction of susceptible individuals with elevated latent propensity toward violence.

4 Discussion

The results of the Progressive Risk Model show that the persistence of violence is highly sensitive to exposure and transition mechanisms. Increasing exposure to violence (μ) or increasing the rate of transition to violence (ω) can move the system from a state in which violence dies to a state in which violence persists. In contrast, increasing the intervention parameter α lowers violence levels and can prevent long-term persistence. These results suggest that reducing repeated exposure to violence and increasing pathways out of violence can strongly affect long-term outcomes.

From analysis of the Mesoscopic Violnece-Response Model we saw that different response strategies can lead to very different violence dynamics, even when they produce similar short-term effects on the general population. Under the escalation response, stronger response interactions increase both the number of violent individuals and the average level of violence. This suggests that stronger confrontational interactions can increase both participation in violence and violence severity. Under the threshold response, the violent population still increases, but the average violence level changes much less. In this case, the response mainly changes how many individuals enter the violent subsystem rather than the severity of violence itself.

The most important point we want to make is that looking only at total violence levels may miss important differences in the dynamics. Multiple response strategies can produce similar numbers of violent individuals while creating very different distributions of level of violence. The mesoscopic framework captures these effects by tracking how the full distribution of violence changes over time rather than only the total population sizes. More broadly, these results highlight the usefulness of kinetic and mesoscopic models for studying violence dynamics. Standard compartmental models track only the total number of individuals in each group, while the present framework also captures differences in individual behavior and violence severity. This makes it possible to study gradual behavioral changes, threshold effects, and more complicated interaction mechanisms.

From an application perspective, the results suggest that interventions may have very different long-term effects depending on how individuals respond to interactions with institutions. We do not aim to make normative claims about response strategies though our models, but it our results do show that different interaction mechanisms can lead to very different collective outcomes.

4.1 Limitations

While the proposed models provide a flexible framework for studying violence dynamics and intervention effects, several important limitations remain. First, the models are highly simplified representations of complex social systems. Violence is influenced by many interacting social, economic, political, and environmental factors that are not explicitly included in the framework. As a result, the models should not be interpreted as predictive tools for real-world violence outcomes, but rather as mechanisms for studying how different interaction structures can shape collective behavior.

A second limitation is the interpretation of the microstate variables. In the mesoscopic framework, the propensity toward violence, level violence, and response level are

all represented by continuous variables on $[0, 1]$. While this allows for a richer description of behavioral heterogeneity, these variables are abstract and difficult to measure directly from data. In practice, these quanitites are multi-dimensional. Moreover, calibrating these quantities to real populations would require significant assumptions and careful validation. The interaction rules used in the model are also simplified. The transition and response functions are stylized mechanisms designed to capture qualitative effects rather than exact behavioral processes. Real interactions between individuals, communities, and institutions are far more heterogeneous and context-dependent. For example, the same intervention may produce very different responses across communities or individuals depending on social conditions, prior experiences, and external influences.

Another limitation is that the current analysis focuses primarily on homogeneous mixing and non-spatial dynamics. Individuals are assumed to interact according to averaged encounter rates rather than through realistic social or geographic networks. In practice, violence often spreads through localized interactions, social ties, and spatial clustering. Incorporating network structure, mobility, and spatial heterogeneity would likely produce more complex dynamics. The response subsystem is also highly idealized. Response team interactions are represented through aggregate response mechanisms and continuous response levels, without modeling decision-making processes, institutional constraints, resource allocation, or feedback between communities and institutions. Consequently, the model should not be interpreted as a direct representation of real response practices.

Despite these limitations, the framework provides a useful starting point for studying how heterogeneous interactions and intervention mechanisms can shape violence dynamics at the population level. It also highlights several directions for future work, including data-informed calibration, network and spatial extensions, and more detailed representations of institutional and social processes.

5 Conclusion

This paper presented a mesoscopic framework for modeling violence, bridging individual-level behaviors with population-level dynamics, in line with the multiscale approach advocated by Sisk. Traditional compartmental models can show overall trends in violence, but cannot capture differences in how violent individuals are or how institutions like the response team affect these behaviors. By introducing a kinetic approach, we modeled distributions of violence within populations and analyzed how these distributions evolve under different response strategies. We developed both partial and fully mesoscopic models, capturing heterogeneity within the general and violent populations as well as the influence of response. This framework enables us to examine how individual propensities toward violence intersect with social and institutional factors, highlighting the complex feedback loops that Sisk emphasizes between agents and structures.

A key contribution of this work is the definition and application of a mesoscale violence persistence number, $(\mathcal{V}_0)$, which quantifies how social interactions and response influence the growth or decay of violent behavior. This idea puts Sisk's point into

practice: overall patterns in a system come from how individuals act and how institutions respond. Our simulations show that both the form of the interaction function and the response distribution critically determine whether the system stabilizes, escalates, or exhibits complex dynamics. Monotonic interaction functions with aggressive response can increase or decrease overall violence depending on the response, while threshold-based functions moderate violence when interventions remain proportionate, illustrating the delicate balance Sisk identifies between individual behavior and institutional policy.

Future work could extend these models to include network structures, adaptive response strategies, spatio-temporal dynamics, and more detailed empirical data to improve calibration and forecasting. The kinetic VPN ($\mathcal{V}_0$) offers a practical tool for designing targeted interventions that align with Sisk's multiscale perspective, allowing for effective management of violence at both individual and community levels.

From an analytical perspective, much of the study relies on numerical simulations and parameter exploration. Although the violence persistence number provides useful insight into threshold behavior in the reduced model, a complete mathematical understanding of the fully mesoscopic system remains open. In particular, questions related to global stability, long-term behavior, bifurcations, and sensitivity to the choice of interaction kernels require further study.

6 Data Availability and Materials

No datasets were used for this work. Access to the codes for the numerical simulation is available upon request.

7 Declarations

Ethics approval and consent to participate: Not applicable

Consent for publication: Not applicable

Availability of data and materials: No datasets were used, code is available upon request.

Competing interests: The authors declare that there are no competing interests.

Funding: NSF-DMS-2042413, AFOSR MURI FA9550-22-1-0380

Authors' contributions: NR conceived and lead the study and did most of the manuscript writing. YB ran simulations and perfomed the mathematical computations. JB help secure funding and with writing of the manuscript.

Acknowledgements: This work was partially funded by NSF-DMS-2042413, AFOSR MURI FA9550-22-1-0380.

References

[1] Decker, S.H.: Collective and normative features of gang violence. Justice Quarterly **13**, 243–264 (1996)

[2] Brantingham, P.J., Yuan, B., Herz, D.: Is gang violent crime more contagious than non-gang violent crime? Journal of quantitative criminology **37**(4), 953–977 (2021)

[3] Hsiao, Y., Leverso, J., Papachristos, A.V.: The corner, the crew, and the digital street: Multiplex networks of gang online-offline conflict dynamics in the digital age. American Sociological Review **88**(4), 709–741 (2023)

[4] Leverso, J., Diouane, Y., Mohler, G.: Measuring online–offline spillover of gang violence using bivariate hawkes processes. Journal of quantitative criminology **41**(1), 103–131 (2025)

[5] Papachristos, A.V., Braga, A.A., Hureau, D.M.: Social networks and the risk of gunshot injury. Journal of Urban Health **89**(6), 992–1003 (2012)

[6] Sanders, B., Monk, K.: Interrupting gang homicide. Deviant Behavior **45**(10), 1347–1365 (2024) https://doi.org/10.1080/01639625.2023.2294353 . doi: 10.1080/01639625.2023.2294353

[7] Widom, C.S.: The cycle of violence. Science **244**(4901), 160–166 (1989) https://doi.org/10.1126/science.2704995

[8] Fowler, P.J., T., C.J., Braciszewski, J.M., Jacques-Tiura, A.J., Baltes, B.B.: Community violence: A meta-analysis on the effect of exposure and mental health outcomes of children and adolescents. Development and Psychopathology **21**(1), 227–259 (2009) https://doi.org/10.1017/S0954579409000145

[9] Baglivio, M.T., Epps, N., Swartz, K., Huq, M.S., Sheer, A., Hardt, N.S.: The prevalence of adverse childhood experiences (ace) in the lives of juvenile offenders. Journal of Juvenile Justice **3**(2), 1–17 (2014)

[10] Slutkin, G.: Violence is a contagious disease. Infectious Disease Clinics of North America **26**(4), 635–648 (2012) https://doi.org/10.1016/j.idc.2012.07.006

[11] Loftin, C.: Assaultive violence as a contagious social process. Bulletin of the New York Academy of Medicine **62**(5), 550 (1986)

[12] Butts, A. J, Roman, C.G., Bostwick, L., Porter, J.R.: Cure violence: A public health model to reduce gun violence. Annual Review of Public Health **36**, 39–53 (2015) https://doi.org/10.1146/annurev-publhealth-031914-122509

[13] Sisk, A., Bamwine, P., Day, J., Fefferman, N.: Linking immuno-epidemiology principles to violence. BMC public health **22**(1), 2118 (2022)

[14] Berestycki, H., Nadal, J.-P., Rodriguez, N., Ryzhik, L.: A mathematical model of riots dynamics: Shocks, diffusion and thresholds. Networks and Heterogeneous Media **10**(3), 443–475 (2015) https://doi.org/10.3934/nhm.2015.10.443

[15] Short, M.B., D'Orsogna, M.R., Pasour, V.B., Tita, G.E., Brantingham, P.J., Bertozzi, A.L., Chayes, L.B.: A statistical model of criminal behavior. Mathematical Models and Methods in Applied Sciences **18**(suppl.), 1249–1267 (2008) https://doi.org/10.1142/S0218202508003029

[16] Burbeck, S.L., Raine, W.J., Abudu Stark, M.J.: The dynamics of riot growth: An epidemiological approach. The Journal of Mathematical Sociology **6**(1), 1–22 (1978) https://doi.org/10.1080/0022250X.1978.9989878 . doi: 10.1080/0022250X.1978.9989878

[17] Maharaj, S., Seepersad, R., Sooknanan, J., Anderson, S., Franco, D., Ramdass, A., Seemungal, T.: A public health approach to reducing violence within the caricom region. Frontiers in public health **12**, 1344387 (2024)

[18] Ensign, D., Friedler, S.A., Neville, S., Scheidegger, C., Venkatasubramanian, S.: Runaway feedback loops in predictive policing. In: Conference on Fairness, Accountability and Transparency, pp. 160–171 (2018). PMLR

[19] Whitehill, J.M., Webster, D.W., Frattaroli, S., Parker, E.M.: Interrupting violence: how the ceasefire program prevents imminent gun violence through conflict mediation. Journal of Urban Health **91**(1), 84–95 (2014)

[20] Lynam, D.R., Piquero, A.R., Moffitt, T.E.: Specialization and the propensity to violence: Support from self-reports but not official records. Journal of Contemporary Criminal Justice **20**(2), 215–228 (2004)

[21] Nagin, D.S., Land, K.C.: Age, criminal careers, and population heterogeneity: Specification and estimation of a nonparametric, mixed poisson model. Criminology **31**(3), 327–362 (1993)

[22] Bahid, Y., Brantigham, J.P., Rodriguez, N.: Understanding urban violence through a mesoscopic framework. preprint, 13 (2026)

[23] Busher, J., *et al.*: The relational dynamics of violence escalation and de-escalation. American Behavioral Scientist (2025) https://doi.org/10.1177/00027642251377875

[24] Kenneth, L.: Disentangling the consequences of the 1033 program. American Political Science Review **114**(4), 1001–1014 (2020)

[25] Todak, N., White, J.J.: Expert officer perceptions of de-escalation in policing. Policing: An International Journal **40**(4), 867–881 (2017) https://doi.org/10.1108/PIJPSM-07-2016-0100

[26] Engel, R.S., *et al.*: The impact of de-escalation training on police behavior. Criminology & Public Policy **19**(3), 721–759 (2020) https://doi.org/10.1111/1745-9133.12467

[27] Police Executive Research Forum: An integrated approach to de-escalation and minimizing use of force. Technical report, Police Executive Research Forum (2012)

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SuppMaterial.tex