INCORPORATING TRAUMA PROPAGATION AND TRANSMISSION IN AGENT BASED MODELS: A PRELIMINARY FRAMEWORK

Nicholas Bishop^a and Hamdi Kavak^a

^aGeorge Mason University, United States {nbishop3,hkavak}@gmu.edu

ABSTRACT

Traumatic experiences, such as war, genocide, and famine, significantly influence human decision-making. Such traumas are passed down to newer generations genetically, making their long-lasting impact on populations. Agent-based modeling offers a unique avenue to study trauma, its communication within communities, and its transmission to future generations. Despite the scarcity of agent-based model (ABM) use in trauma research, it holds immense potential due to its relevance in understanding interpersonal interactions. To address this gap, we propose an accessible ABM framework tailored for researchers with limited ABM experience. Our framework is a simple, easy-to-understand model built with intuitive and transparent code. It facilitates the creation of generalizable functions and serves as a foundational design for researchers to copy, adapt, or build upon when constructing their own models. Our framework contains feature sets to aid in the creation of models addressing transgenerational traumas passed down genetically, through epigenetics, and community propagation of collective trauma.

Keywords: Epigenetics, Trauma, Agent-Based Model, Framework, Simulation.

1 INTRODUCTION

Besides logic, there are other factors that can affect our decision-making, whether we are aware of these factors or not. One of these factors is trauma, which can be an unknown and major driving force behind our decisions. Trauma can result from different mechanisms, including personal experience, epigenetic mechanisms, or behavioral [1-3]. Personal experiences include an individual's direct experience of some traumatic event [4] while epigenetics and behavioral mechanisms are transmitted or propagated. Trauma research is typically done in the context of psychology or sociology. Since the traumas people experience influence their interactions with others, and agent-based models (ABMs) focus on simulating interactions between people, agent-based models have the potential to provide a great deal of value to the research area [5-7]. Several literature searches within the field of trauma research have found only three publications utilizing ABMs [5,6]. Furthermore, there is agreement in the literature that ABMs in the field of trauma research are severely under-utilized, with several challenges contributing to the current state of ABMs These challenges include a lack of researcher expertise with tools for creating ABMs, a lack of general knowledge regarding designing ABMs to gain insights into the real world, and the challenges of performing proper verification and validation of ABMs [5,6]. If researchers utilize the full potential of ABMs to create plausible models of people and traumas comprising communities, it will be the first step towards modeling the policy impacts on communities, which cannot be directly observed or occur generations later.

Traditional trauma research intersects between psychology, sociology, and biology, with epigenetics being at the forefront of the connection between genetics and psychology in trauma research [8]. Moreover, the ability of a researcher to model epigenetic expressions resulting from ancestral trauma holds the potential to advance the fields of sociology, psychology, and genetics. Identifying the role that the biological and inheritable effects of trauma play in the collective propagation of trauma is vital for predicting and understanding the effects that large-scale traumatic events will have on societies in the coming years. These large-scale traumatic events may include famine, genocide, war, or terrorist attacks [1,9-15].

With computational tools designed for a population of researchers, unprecedented advancements could be made in the field of trauma research if they are utilized. As it stands, the currently available ABM simulation frameworks, although comprehensive, are difficult to understand and operate, especially for a researcher who might only have basic proficiency in a coding language like Python. The goal of creating our framework is to create a tool for researchers that is transparent, intuitive, easy to use, easy to operate, and easy to extend for specific use cases. This is done by creating a model framework in Python using the MESA package which is more specifically an ABM typification [16]. This model framework can act as a foundational base for researchers attempting to create models that link the science of inherited trauma, the psychology of individual direct trauma, and interactions between traumatized people in a population. The model framework has several feature sets that are designed to be easy to understand and to be expanded on for a researcher's unique needs with just a basic understanding of Python, matplotlib, and pandas. It should be noted that the MESA package replicates the capabilities of NetLogo, which is often seen as the introductory software and programming language for agent-based models.

This framework is significant because of its novelty in a field that is not a popular focus point for modelers and because it provides a method of simulation necessary to address questions in the field of epigenetics and trauma. Other work focusing on modeling epigenetic transmission and expression demonstrate it with regards to optimization of behaviors in non-human agents [16,17]; moreover, no other works attempt to address the more complex methods of epigenetic transmission and the variable influences on human agent behavior. Analytic mathematical models, such as ODE models, are unable to accurately capture the specificity of certain epigenetic mutation transmission; consequently, this underscores the necessity of agent-based models when simulating the intricate dynamics of epigenetic transmission.

2 BACKGROUND

People are most familiar with the type of trauma we will refer to as direct trauma: "an emotional response to a terrible event" with long-term psychological effects [4]; however, it is not just direct trauma through personal experiences that can shape how we make decisions and influence our emotions. Psychologists and geneticists have established that direct trauma, and many of the effects of direct trauma, can be inherited from one generation to another, up to third or fourth generations [2,15]. Furthermore, the behaviors of trauma response can be socialized [2,3,13,19]. The effects of all trauma can include depression, anxiety, an increased probability of violent or aggressive behaviors, unpredictable emotions, and changes in how we view ourselves and the world [20,21]. Understanding the effects of trauma, whether direct, inherited, or socially taught, is vital to pre-emptively remedy any negative influences on public mental health crises, violent crime rates, or the physical health of individuals that would be unknown without this knowledge [22-24].

As mentioned above, there are several sources by which one can receive trauma. The first of which is personal experience, which includes an individual's direct experience of some traumatic event [4]. The second is through an individual's family or ancestral line, and within this source of trauma, an individual can inherit trauma and its effects either through epigenetic mechanisms or behaviorally [1-3]. Epigenetic mechanisms work in a physical manner, biologically affecting how genes are expressed in a person and generations after an individual's traumatic experience [21]. On the other hand, trauma can be passed to future generations behaviorally, where the parent's behavior changes as a result of trauma, which then affects how the offspring behave and interact with the world [2,13,19]. A third source of trauma is the social interactions within a society, culture, or community that has experienced a collective trauma, which is propagated to future generations through social mechanisms [10]. When a group of people experiences a collective trauma, it can affect the behaviors and outlooks of many of the members, causing these actions to play into each other to create cyclical trauma [12,14,25]. Although sources of trauma can be organized into distinct categories, trauma itself can manifest from any combination of sources [26].

Epigenetics is the term used to refer to the inherited effects of trauma and experiences from parents and ancestors through changes in gene expression. The primary biological mechanisms in the realm of

epigenetics are DNA methylation, histone modification, and non-coding RNA, which all turn the expression of different genes on and off [27]. Unlike DNA mutations, epigenetic mutations are reversible and can change over a lifetime or several family generations [27]. There have been many studies done on animals regarding epigenetics showing that trauma of one or more parents of the offspring directly affects the offspring through inherited effects [1,2,28,29]. One study showed this is true by instilling a fear in parent mice of a particular scent; once these mice had offspring, they were separated and exposed to the same scent and exhibited a similar fear response [29]. Epigenetics in humans began to become more seriously studied after several studies were done on the descendants of holocaust survivors [2,13]. One finding is that the trauma of the parents affects the balance of cortisol creation and metabolism in their children through epigenetic mechanisms [13]. Many other case studies have been performed in the last several decades. These studies examine the descendants of those who experienced the Dutch Famine, the traumas of Native Americans, and the Suihua Chinese famine as some examples, but more have been studied [9,30-34]. Epigenetics and its effects can be shown in controlled studies with hard science.

The second source of trauma propagation is behaviorally through an individual's interactions with their family [13]. In many of the epigenetic human case studies, researchers state that the behaviors and influences of transgenerational trauma cannot be wholly attributed to epigenetic inheritance [2,13]. This is the case because it is very likely that the traumas of parents or grandparents influenced how they handled their role as parents, which would then affect the behavior and outlook of their children [13]. One of many cases in the literature where this was studied in animals is mentioned in Krippner and Barrett's work, where mother rats with varying degrees of trauma had the affectionate grooming of their offspring observed [1]. The offspring that experienced less affectionate grooming had higher anxiety levels and were less likely to explore their environment [1]. Barrett theorizes that in a more traumatic or harmful environment, it would be beneficial for offspring not to be overly curious; moreover, it would likely be beneficial to have a raised level of awareness through anxiety from a survival standpoint [35]. Although the mother rat was likely not consciously considering whether to affectionately groom her offspring to influence their behavior, the fact that rat behavior was influenced by trauma, which then affected the offspring through a type of social and behavioral means, shows that trauma can propagate in this way through family interactions [1]. The work by Scharf and Mayseless shows an example of this occurring in humans, specifically the children of holocaust survivors [3]. The trauma caused to the parents seemed to cause many cases of overprotection, lack of emotional capacity, and role reversal in the parents [3]. Trauma propagated through family behavioral interactions can be studied in controlled settings for animals, but it relies on understanding the basic psychology of the study specimen; therefore, most of the literature approaches the study of this trauma source from a soft science perspective.

A third medium by which trauma can propagate through several generations is that of the social interactions between members of a group or community that have experienced a collective trauma [12,14]. This concept is less linked with epigenetics and more closely associated with anthropology and sociology. The concept of the social propagation of trauma revolves around the idea that a traumatized group of people have their behaviors influenced in such a way that the community members all play into keeping the community from healing, and instead often create more net trauma [12]. One example of this is shown in many black communities in the United States, where a large group of people in many communities experienced similar multi-generational traumas through Jim Crow. The prosperity of their communities was systematically strangled, and the traumas of the community continued to propagate through the "bias of achievement" into new generations [25]. This has led many predominately low-income black communities to perpetuate urban racial inequality [25]. Although there are many factors external to these communities that also perpetuate racial inequality and the propagation of trauma, this example of low-income black communities serves to demonstrate the existence and effects of community propagation of trauma.

It is of crucial importance for medical doctors, sociologists, and other researchers to understand how trauma affects human health. An understanding of the causes of trauma, as well as its physical and mental manifestations, can allow policymakers to better address causes of violent crime and community mental health crises, as well as individual physical symptoms. It is more likely that one would have the ability to

identify a trauma that occurred within the capacity of their own personal experience; however, it is less likely that the trauma would be consciously identified if received through a more passive or seemingly indirect avenue such as through family or community. The processes through which transgenerational trauma are studied attain quantifiable details regarding the heritability of the trauma, and there are fewer studies with hard-science quantified results regarding community propagation of trauma through generations. To contribute to the usage of ABMs in the field, we propose the idea that ABMs should be used by subject matter experts studying community propagation of trauma in connection with the lower-level mechanisms of trauma transmission to further the field of study. One inhibiting factor of the usage of ABMs for this purpose is the lack of widespread expertise in the fields contributing to trauma research pertaining to modeling and simulation [7]. Many of the existing ABM frameworks designed for this field, though comprehensive in nature, keep the barrier-to-entry high for researchers with a desire to make their first attempts at incorporating ABMs into their work.

Our model framework is not serving to recreate the traumas of a specific community; but is being proposed as a novel, introductory code framework that researchers can use as a base-model to design an ABM to simulate community propagation of transgenerational trauma influenced by the epigenetic effects caused by the trauma. This framework aims to encourage the use of ABMs by anthropologists, sociologists, psychologists, and geneticists by lowering the barrier-to-entry of researchers learning how to utilize MESA to create and verify ABMs. Our transparent framework includes cookie-cutter functions for the common, core epigenetic mechanisms, with an easy-to-use functionality for adding their own unique epigenetic mechanisms, triggers, and symptoms, as well as model verification methods. Through this, we aim to encourage and inspire researchers to utilize the power of ABMs and decrease the time it would take these researchers to understand how to design an ABM and to create their own models.

3 MODEL FRAMEWORK

The model used to build the framework is one showing the effects of a collective trauma famine event on agents in a Sugarscape world, as seen in the official MESA examples repo [36]. Using a simple Sugarscape model allows for easy-to-understand implementations of different aspects of epigenetic mechanisms and diversity of trauma effects on agents. Agents experience a period of starvation if they run out of sugar, and each timestep without obtaining more sugar results in an increased level of trauma. Depending on the user-defined constraints of the timing and the type of a trauma in a specific agent, the trauma will directly affect the agent experiencing it or will affect later generations.

Our model framework can be divided into several key feature sets. These sets include <u>trauma definitions</u>, <u>epigenetic mechanisms</u>, <u>traumatic events</u>, and <u>verification and analysis tools</u>. All parts and subparts of our framework are generalizable to meet the custom needs of any researcher. The source code for the framework can be found in Appendix A.

3.1 Model Overview

To better understand the feature sets within the model framework, we describe how the model operates. Below, Figure 1 shows the process flow of the model framework.

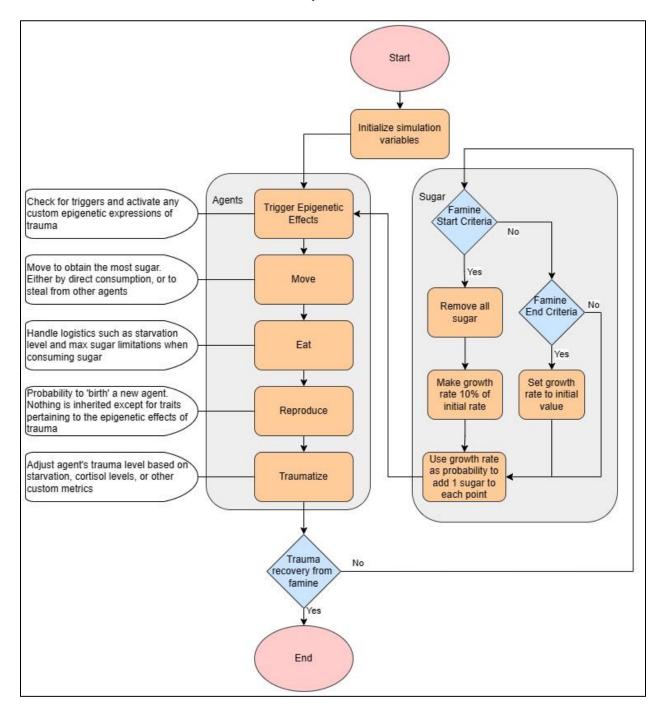


Figure 1: Model framework overview flow diagram.

This model framework is built off of the Sugarscape Constant Growth model within the official MESA set of examples [36]. At each timestep, agents first have any epigenetic effects triggered. This step is further explained within the <u>Epigenetic mechanisms</u> feature set section. Each timestep will also add 1 to the age of each agent, and when the age of an agent reaches its predetermined age of death, the agent dies. An agent's death will remove it from the canvas and the MESA agent schedule.

Then, similar to the original Sugarscape model, agents will move to the grid point on the canvas within their vision that has the maximum amount of sugar. If an agent is starving, the move behavior becomes more complicated, where behaviors rooted in trauma and fear of resource scarcity can be implemented.

Currently, depending on an agent's trauma and starvation levels, an agent will choose to rob, murder, or cannibalize another agent as a last resort to potentially obtain sugar.

The <u>eat</u> step includes processing the metabolism of the agent's current sugar stores, recording the amount of excess sugar, and tracking the starvation level of the agent. An agent's starvation level increases by 1 while the agent has no sugar and will reset to 0 when the agent is able to find sugar to consume. If the agent's starvation reaches a predefined level of 30, the agent dies.

Agents will reproduce periodically throughout their lifetime. In order to reproduce, an agent's age must be greater than the predefined puberty age of 20, at which point a probability is drawn to determine if the agent will reproduce. The probability of this occurrence linearly correlates with the amount of sugar an agent is holding relative to the maximum possible sugar it can hold. Only the traits related to epigenetic changes due to trauma are inherited. All other traits are created randomly for the new agent created by reproduction.

Finally, the <u>traumatize</u> phase is reached. At this point, the trauma levels of the agents are adjusted. If an agent is starving, the trauma level increases by a set constant, and when agents are not starving, the trauma level exponentially decays. The rate of decay is determined by an agent's <u>cortisol</u> levels, which have been shown in the literature to be affected in the offspring of people who experience extreme trauma [2,13]. The trauma level of each agent lies between 0 and 1 inclusively. The trauma level of an agent determines the severity of its behavior towards other agents when faced with increasing starvation levels, as is seen in the literature [37-41].

Once the population of agents is instantiated, the simulation is run until a steady state of the average trauma level among agents is found, which is determined using the Conway rule [42]. The Conway rule refers to counting the number of N consecutive instances the most recent timestep stays within the minimum and maximum values of the latest M steps; once N reaches a specified value, the system is considered to be in a steady state [42]. This triggers a collective trauma event which is simulated as a famine, where 90% of all sugar is erased from the Sugarscape canvas, and the growth rate is set to 10% of the initial growth rate for 100 steps. Once 100 steps have been completed, the growth rate is returned to the initial value, and the simulation is run for 800 steps after the collective trauma event has ended. Determining a steady state of the model is important for analyzing the trauma impacts on a representative population that is not influenced by the bias of initialization [43]

3.2 Trauma Definitions

Our framework includes general functions for prenatal, prepubescent, and other/uncategorized trauma, as well as modeling the effects of trauma internally or behaviorally on an agent. The current framework model implements several effects of direct experience trauma on agents through the influence of cortisol levels in cases of extreme trauma [2] and increasing the probability of agents taking increasingly violent actions against other agents to ensure their survival [37,38,41]. Through the current framework, researchers would be able to expand on trauma effects on an agent's internal mechanisms as well as an agent's external behaviors.

In addition to the general trauma a person may experience, many human epigenetic studies emphasize the effect of prenatal and prepubescent trauma on future generations [15,30-33], so it is anticipated that many researchers could benefit from this being a core part of the framework. Currently, the framework follows much of the literature in defining prenatal and prepubescent traumas as trauma types that only affect later, gapped generations [15]. This example allows researchers to understand how to add different trauma types and gives a general process for coding specific instances in which these different trauma types can be applied to an agent.

3.3 Epigenetic Mechanisms

In the context of our framework, <u>epigenetic mechanisms</u> are defined as any inherited effect of trauma influencing an agent's characteristics or behaviors. The current framework introduces simple and complex

generalizable methods to simulate the inheritance of trauma. An example of a simple trauma inheritance is recording the maximum lifetime trauma level of a parent, then using a percentage of that trauma level to be a baseline trauma level for the offspring. This example requires a very simple understanding of Python objects to execute. We also implement very simple effects of extreme trauma on the cortisol levels of an agent's offspring, both direct and several generations removed, as seen in epigenetic studies on families of holocaust survivors and other extreme traumas [2,13].

An example of a complex trauma inheritance would be a decrease in life expectancy of a grandchild if the grandchild is the same sex as the grandparent and the grandparent experienced prenatal famine trauma, as seen in González-Rodríguez, Füllgrabe, and Joseph's work [15]. This example requires tracking several agent attributes across several generations with several intersecting triggers for the epigenetic symptom to be expressed; consequently, we developed a feature that allows for general and arbitrary triggers and epigenetic symptom expression. The user can specify arbitrary agent attributes in a dictionary to create a set of intersecting trigger conditions and specify the function to use for the epigenetic symptom expression in the agent. All specified trigger and expression information is appended to an agent's list of epigenetic gene information until the generation of the agent is greater than the generation of the trigger. This feature allows epigenetic inheritance that skips any number of generations and allows for epigenetic expression under specific circumstances.

3.4 Traumatic Events

This model framework uses a collective trauma that is easily implemented with a Sugarscape agent-based model; famine. Because modeling a trauma event is situation-dependent and varies widely on the specific details at the code and model level, it would be extremely challenging to generalize this aspect of the framework. Although the traumatic event feature of our framework is not generalized, researchers attempting to pick up ABMs may still find this intuitive example helpful in digesting the other feature sets of the framework model.

When a famine is triggered, 90% of all sugar values on the Sugarscape board are set to zero, with the growth rate set to 10% of the initial growth rate for 100 steps, after which the growth rate returns to the initial value. A famine is triggered when the average trauma value of the agents in the population reaches a steady state, as determined by the Conway Rule [42]. The famine trigger and definition in the code are easy to understand and change, presenting a sandbox to researchers who are attempting to understand MESA and ABMs in general.

3.5 Verification and Analysis Tools

Our model framework contains several forms of verification tasks that can be easily understood and built off of by researchers. The first of these are examples of model and agent reports, which are a part of the MESA API. Having these examples will allow researchers to understand how to create MESA outputs for any model variable they would like to analyze.

There are some built-in framework agent features that allow for deeper analysis and verification by researchers. The family line and generation number are recorded in new agents as they are created through the reproduce function. This feature allows researchers to easily trace trauma levels and survivability of different family lines, track metrics on the number of generations it requires for a collective trauma to recede, add MESA reporters to specific family lines, or apply trauma to only specific family lines. The granularity provided by family and generational markers within agents will allow researchers to create specific and unique scenarios for debugging and verify that newly implemented features work as intended.

In addition to this, the ability to set the random seed of each model run adds another tool to perform verification and debugging of the model researchers create. Researchers can also easily use MESA to visualize their model as it runs as another powerful verification method.

3.6 Example Usage

Here, we show the usage of the framework with a very basic sensitivity analysis in Figures 2-5, demonstrating the basic implementation of transgenerational epigenetic mutations and the influence of agent behaviors on trauma. For this example framework usage, ten Monte-Carlo runs were performed for each example, each with the same random seed as the iteration number in the loop. Figures 2-5 show verification of the model. The purpose of this example is to demonstrate that our framework model works regarding the inheritance of trauma and its effects on agent behavior.

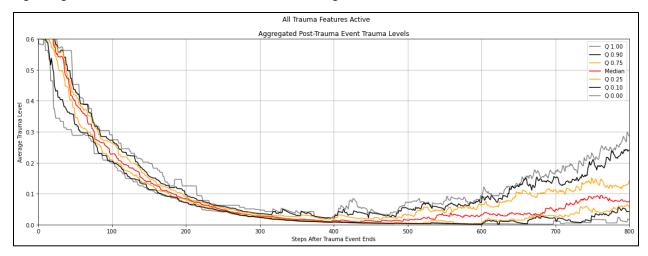


Figure 2: Aggregated trauma level statistics after a simulated collective trauma event with all trauma features active in the model.

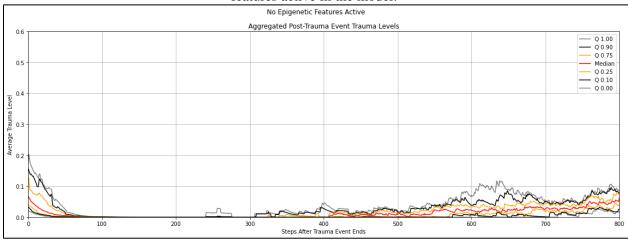


Figure 3: Aggregated trauma level statistics after a simulated collective trauma event with all trauma features active in the model except epigenetic inheritance.

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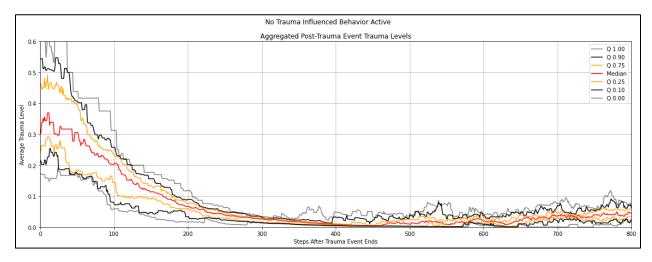


Figure 4: Aggregated trauma level statistics after a simulated collective trauma event with all trauma features active in the model except trauma influence behavior.

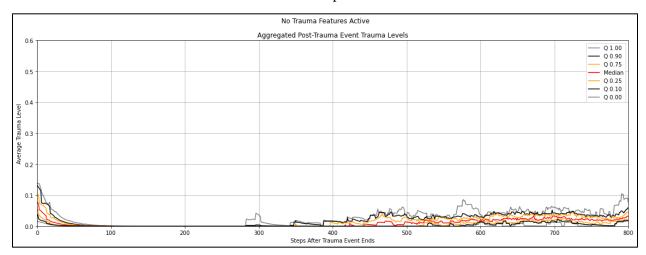


Figure 5: Aggregated trauma level statistics after a simulated collective trauma event with no trauma features active in the model.

The graphs of the post-famine average trauma level are shown in Figures 2-5. Each graph shows a different combination of trauma features being active in the simulation, such as all features, no epigenetic trauma inheritance, no influence of trauma on behavior, and no trauma features active at all, respectively. With this data graphed, it can be observed that the trauma features implemented in the framework do have a noticeable effect on the trauma levels of the agents in the simulation; moreover, this also acts as fundamental verification that the features implemented in the model framework have a non-trivial effect on the overall simulation.

Since this framework model is based on an example utilizing a theoretical community population, the model as it appears in this framework cannot be validated. Validation is left to the researchers who choose to apply this framework to model specific communities within the realm of their expertise. Validation of each community model may be different depending on the behavioral effects of trauma. For example, if there is a model made on community trauma propagation via theft, then the researchers can potentially validate the model BY matching the rates of theft to that of the real-life data.

4 DISCUSSION AND CONCLUSION

Although we provide a core framework model to model trauma, the model and framework have limitations. Our framework model only includes individual trauma experience, community propagation of trauma, and the biological inheritance of trauma through physical action. Moreover, this leaves trauma propagation modeled through psychological means, such as through a family unit or social interactions within the community. If trauma were to be modeled in the most accurate manner, all aspects, including biological, social, and individual psychological elements, would be required for such a model.

The next steps for future work involve adding social trauma propagation, which may be implemented as social interactions within the community. Adding trauma mitigation features may be another aspect to continue this work, such as the existence of altruistic actions of individuals, which act to mitigate the effects of community propagation of trauma [44].

Agent-based models are underutilized in the context of trauma research. Since trauma affects many aspects of society, including violent crime rates, community mental health, and individual physical health, it is important that researchers have all the possible tools at their disposal to advance this field [5]. Our model framework serves as a novel steppingstone for researchers with little coding and no ABM experience to understand how ABM models can be designed for trauma research; furthermore, this provides cookie-cutter, transparent feature sets to allow researchers to easily copy the methodology or the raw code directly into their own models. This is achieved by having feature sets that are generalizable to any unique epigenetic or direct trauma symptom that a researcher would want to simulate and by providing access to the source code of the framework.

We present this framework as a tool for researchers who wish to use their expertise to advance the field of trauma research or drive policy changes that positively impact communities. Such advancements in the trauma field may also inspire other expert modelers to consider incorporating trauma influence on behavior, since it has its own unique effect on individuals, much like education level, social interactions, etc.

A SOURCE CODE

The source code and example code explanations for this model framework can be found at this Github repo: https://github.com/nsbishop3/trauma_model_framework.

REFERENCES

- [1] S. Krippner and D. Barrett, "Transgenerational Trauma: The Role of Epigenetics," *The Journal of Mind and Behavior*, vol. 40, issue 1, pp. 53–62, 2023.
- J. Švorcová, "Transgenerational Epigenetic Inheritance of Traumatic Experience in Mammals," *Genes*, vol. 14, issue 1, p. 120, Jan. 2023, doi: 10.3390/genes14010120.
- [3] M. Scharf and O. Mayseless, "Disorganizing Experiences in Second- and Third-Generation Holocaust Survivors," *Qual Health Res*, vol. 21, issue 11, pp. 1539–1553, Nov. 2011, doi: 10.1177/1049732310393747.
- [4] "Trauma," https://www.apa.org. Accessed: Oct. 24, 2023. [Online]. Available: https://www.apa.org/topics/trauma
- J. E. Goldstick and J. Jay, "Agent-Based Modeling: an Underutilized Tool in Community Violence Research," *Curr Epidemiol Rep*, vol. 9, issue 3, pp. 135–141, Jul. 2022, doi: 10.1007/s40471-022-00292-x.

- [6] M. Tracy, M. Cerdá, and K. M. Keyes, "Agent-Based Modeling in Public Health: Current Applications and Future Directions," *Annu. Rev. Public Health*, vol. 39, issue 1, pp. 77–94, Apr. 2018, doi: 10.1146/annurev-publhealth-040617-014317.
- [7] M. Tracy, E. Gordis, K. Strully, B. D. L. Marshall, and M. Cerdá, "Applications of agent-based modeling in trauma research.," *Psychological Trauma: Theory, Research, Practice, and Policy*, vol. 15, issue 6, pp. 939–950, Sep. 2023, doi: 10.1037/tra0001375.
- [8] A. S. Zannas, N. Provençal, and E. B. Binder, "Epigenetics of Posttraumatic Stress Disorder: Current Evidence, Challenges, and Future Directions," *Biological Psychiatry*, vol. 78, issue 5, pp. 327–335, Sep. 2015, doi: 10.1016/j.biopsych.2015.04.003.
- [9] T. N. Brockie, M. Heinzelmann, and J. Gill, "A Framework to Examine the Role of Epigenetics in Health Disparities among Native Americans," *Nursing Research and Practice*, vol. 2013, pp. 1–9, 2013, doi: 10.1155/2013/410395.
- [10] D. Somasundaram, "Addressing collective trauma: conceptualisations and interventions," *Intervention*, vol. 12, pp. 43–60, Dec. 2014, doi: 10.1097/WTF.000000000000068.
- [11] R. M. MacNair, "Causing trauma as a form of trauma.," *Peace and Conflict: Journal of Peace Psychology*, vol. 21, issue 3, pp. 313–321, Aug. 2015, doi: 10.1037/pac0000116.
- [12] M. Bracher, "Healing Trauma, Preventing Violence: A Radical Agenda for Literary Study," *JAC*, vol. 24, issue 3, pp. 515–561, 2004.
- [13] P. Dashorst, T. M. Mooren, R. J. Kleber, P. J. De Jong, and R. J. C. Huntjens, "Intergenerational consequences of the Holocaust on offspring mental health: a systematic review of associated factors and mechanisms," *European Journal of Psychotraumatology*, vol. 10, issue 1, p. 1654065, Dec. 2019, doi: 10.1080/20008198.2019.1654065.
- [14] I. G. Barron and G. Abdallah, "Intergenerational Trauma in the Occupied Palestinian Territories: Effect on Children and Promotion of Healing," *Journ Child Adol Trauma*, vol. 8, issue 2, pp. 103–110, Jun. 2015, doi: 10.1007/s40653-015-0046-z.
- [15] P. González-Rodríguez, J. Füllgrabe, and B. Joseph, "The hunger strikes back: an epigenetic memory for autophagy," *Cell Death Differ*, vol. 30, issue 6, pp. 1404–1415, Jun. 2023, doi: 10.1038/s41418-023-01159-4.
- [16] J. A. B. Sousa and E. Costa, "Designing an Epigenetic Approach in Artificial Life: The EpiAL Model," in *Agents and Artificial Intelligence: Second International Conference*, J. Filipe, A. Fred, and B. Sharp, Eds., in Communications in Computer and Information Science. Valencia, Spain: Springer Berlin Heidelberg, 2011, pp. 78–90. doi: 10.1007/978-3-642-19890-8_6.
- [17] F. Mukhlish, J. Page, and M. Bain, "Reward-based epigenetic learning algorithm for a decentralised multi-agent system," *IJIUS*, vol. 8, issue 3, pp. 201–224, Apr. 2020, doi: 10.1108/IJIUS-12-2018-0036.
- [18] R. Boero and F. Squazzoni, "Does Empirical Embeddedness Matter? Methodological Issues on Agent-Based Models for Analytical Social Science," *Journal of Artificial Societies and Social Simulation*, vol. 8, issue 4, Oct. 2005, [Online]. Available: https://www.jasss.org/8/4/6.html
- [19] I. C. Weiss, T. B. Franklin, S. Vizi, and I. M. Mansuy, "Inheritable Effect of Unpredictable Maternal Separation on Behavioral Responses in Mice," *Front. Behav. Neurosci.*, vol. 5, 2011, doi: 10.3389/fnbeh.2011.00003.

- [20] L. Song, M. I. Singer, and T. M. Anglin, "Violence Exposure and Emotional Trauma as Contributors to Adolescents' Violent Behaviors," *Arch Pediatr Adolesc Med*, vol. 152, issue 6, Jun. 1998, doi: 10.1001/archpedi.152.6.531.
- [21] A. Jawaid, M. Roszkowski, and I. M. Mansuy, "Transgenerational Epigenetics of Traumatic Stress," in *Progress in Molecular Biology and Translational Science*, vol. 158, Elsevier, 2018, pp. 273–298. doi: 10.1016/bs.pmbts.2018.03.003.
- [22] K. M. Magruder, K. A. McLaughlin, and D. L. Elmore Borbon, "Trauma is a public health issue," *European Journal of Psychotraumatology*, vol. 8, issue 1, p. 1375338, Jan. 2017, doi: 10.1080/20008198.2017.1375338.
- [23] B. E. Carlson, "The Most Important Things Learned About Violence and Trauma in the Past 20 Years," *J Interpers Violence*, vol. 20, issue 1, pp. 119–126, Jan. 2005, doi: 10.1177/0886260504268603.
- [24] C. for S. A. Treatment (US), "Understanding the Impact of Trauma," in *Trauma-Informed Care in Behavioral Health Services*, Substance Abuse and Mental Health Services Administration (US), 2014. Accessed: Dec. 28, 2023. [Online]. Available: https://www.ncbi.nlm.nih.gov/books/NBK207191/
- [25] R. J. Sampson and S. W. Raudenbush, "Seeing Disorder: Neighborhood Stigma and the Social Construction of 'Broken Windows," *Soc Psychol Q*, vol. 67, issue 4, pp. 319–342, Dec. 2004, doi: 10.1177/019027250406700401.
- [26] M. Masiero, K. Mazzocco, C. Harnois, M. Cropley, and G. Pravettoni, "From Individual To Social Trauma: Sources Of Everyday Trauma In Italy, The US And UK During The Covid-19 Pandemic," *Journal of Trauma & Dissociation*, vol. 21, issue 5, pp. 513–519, Oct. 2020, doi: 10.1080/15299732.2020.1787296.
- [27] "Epigenetics," https://www.cdc.gov/genomics/disease/epigenetics.htm. Accessed: Oct. 24, 2023. [Online]. Available: https://www.apa.org/topics/trauma
- [28] A. T. Natarajan, "Induced Transgenerational Genetic Effects in Rodents and Humans," *JRR*, vol. 47, issue SupplementB, pp. B39–B43, 2006, doi: 10.1269/jrr.47.B39.
- [29] B. G. Dias and K. J. Ressler, "Parental olfactory experience influences behavior and neural structure in subsequent generations," *Nat Neurosci*, vol. 17, issue 1, pp. 89–96, Jan. 2014, doi: 10.1038/nn.3594.
- [30] R. Painter, C. Osmond, P. Gluckman, M. Hanson, D. Phillips, and T. Roseboom, "Transgenerational effects of prenatal exposure to the Dutch famine on neonatal adiposity and health in later life," *BJOG*, vol. 115, issue 10, pp. 1243–1249, Sep. 2008, doi: 10.1111/j.1471-0528.2008.01822.x.
- [31] M. Veenendaal, R. Painter, S. De Rooij, P. Bossuyt, J. van der Post, P. Gluckman, M. Hanson, T. Roseboom, "Transgenerational effects of prenatal exposure to the 1944-45 Dutch famine," *BJOG*, vol. 120, issue 5, pp. 548–554, Apr. 2013, doi: 10.1111/1471-0528.12136.
- [32] W. Jiang, T. Han, W. Duan, Q. Dong, W. Hou, H. Wu, Y. Wang, Z. Jiang, X. Pei, Y. Chen, Y. Li, C. Sun, "Prenatal famine exposure and estimated glomerular filtration rate across consecutive generations: association and epigenetic mediation in a population-based cohort study in Suihua China," *Aging*, vol. 12, issue 12, pp. 12206–12221, Jun. 2020, doi: 10.18632/aging.103397.
- [33] J. Li, S. Liu, S. Li, R. Feng, L. Na, X. Chu, X. Wu, Y. Niu, Z. Sun, T. Han, H. Deng, X. Meng, H. Xu, Z. Zhang, Q. Qu, Q. Zhang, Y. Li, C. Sun, "Prenatal exposure to famine and the development of hyperglycemia and type 2 diabetes in adulthood across consecutive generations: a population-

- based cohort study of families in Suihua, China," *The American Journal of Clinical Nutrition*, vol. 105, issue 1, pp. 221–227, Jan. 2017, doi: 10.3945/ajcn.116.138792.
- J. W. Smoller, "The Genetics of Stress-Related Disorders: PTSD, Depression, and Anxiety Disorders," *Neuropsychopharmacol*, vol. 41, issue 1, pp. 297–319, Jan. 2016, doi: 10.1038/npp.2015.266.
- [35] D. Barrett, Supernormal Stimuli. New York, NY: W. W. Norton & Company, 2010.
- [36] "mesa-examples." Project Mesa, Dec. 13, 2023. Accessed: Dec. 27, 2023. [Online]. Available: https://github.com/projectmesa/mesa-examples
- [37] R. Dirks, G. Armelagos, C. Bishop, I. Brady, T. Brun, J. Copans, V. Doherty, S. Fraňková, L. Greene, D. Jelliffe, E. Jelliffe, D. Kayongo-Male, C. Paque, E. Schusky, R. Thomas, D. Turton, "Social Responses During Severe Food Shortages and Famine [and Comments and Reply]," *Current Anthropology*, vol. 21, issue 1, pp. 21–44, Feb. 1980, doi: 10.1086/202399.
- [38] S. Finnerty, "An Analysis of the Association between Food Insecurity and Violent Crime in Georgia in 2020," Master's Thesis, Dept. Public Health, Georgia State University, Atlanta, GA, 2023.
- [39] H. F. Lee, "Cannibalism in northern China between 1470 and 1911," *Reg Environ Change*, vol. 19, issue 8, pp. 2573–2581, Dec. 2019, doi: 10.1007/s10113-019-01572-x.
- [40] N. Zemon Davis, "Cannibalism and Knowledge," *Historein*, vol. 2, p. 13, May 2012, doi: 10.12681/historein.109.
- [41] M. Frank, L. Daniel, C. Hays, M. Shanahan, R. Naumann, H. Reyes, A. Austin, "Association of Food Insecurity With Multiple Forms of Interpersonal and Self-Directed Violence: A Systematic Review," *Trauma, Violence, & Abuse*, p. 152483802311656, Apr. 2023, doi: 10.1177/15248380231165689.
- [42] P. S. Mahajan and R. G. Ingalls, "Evaluation of Methods Used to Detect Warm-Up Period in Steady State Simulation," in *Proceedings of the 2004 Winter Simulation Conference*, 2004., Washington, D.C.: IEEE, 2004, pp. 651–659. doi: 10.1109/WSC.2004.1371374.
- [43] C. Di Francescomarino, A. Burattin, C. Janiesch, and S. Sadiq, Eds., *Business Process Management Forum: BPM 2023 Forum, Utrecht, The Netherlands, September 11–15, 2023, Proceedings*, vol. 490. in Lecture Notes in Business Information Processing, vol. 490. Cham: Springer Nature Switzerland, 2023. doi: 10.1007/978-3-031-41623-1.
- [44] R. Tedeschi, C. Park, and L. Calhoun, *Posttraumatic Growth: Positive Changes in the Aftermath of Crisis*, 1st ed. New York, NY: Routledge, 1998. [Online]. Available: https://doi.org/10.4324/9781410603401

AUTHOR BIOGRAPHIES

NICHOLAS BISHOP is a student pursuing a Master's of Science degree in Computational Science at George Mason University. He is a simulation engineer by profession and finds interest in research topics involving data science, agent-based models, physics simulations, and time series analysis. His email is nbishop3@gmu.edu.

HAMDI KAVAK is an Assistant Professor in the Computational and Data Sciences Department and codirector of the Center for Social Complexity at George Mason University. His research interests lie at the intersection of data science and modeling & simulation. His email address is hkavak@gmu.edu and website address is hkavak@gmu.edu and website address is hkavak@gmu.edu and website