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About this Issue

Statement of Purpose

The Rhodes Journal of Biological Science is a student-edited publication that recognizes the scientific achievements of Rhodes students. Volume XXXIII marks the twelfth year since Mark Stratton and Dr. David Kesler brought the journal back into regular publication in 2006. Founded as a scholarly forum for student research and scientific ideas, the journal aims to maintain and stimulate the tradition of independent study among Rhodes College students. We hope that in reading the journal, other students will be encouraged to pursue scientific investigations and research.

Acknowledgements

The editorial staff would like to thank Dr. Honsa of the Biology department for her support and guidance in preparing this publication.

Image Credits

Colleen Hulsey
This image was taken as part of the Plant Genetics and Diversity course.
Colleen is a Biomathematics and Environmental Science double major, and she has participated in research in coordination with the Overton Park Conservancy.

Editorial Staff

Erin Deery '18 (Senior Editor) is a Biomathematics major from Winter Park, FL. She has spent time researching both at Rhodes and off campus. Within the Rhodes Mathematics Department she worked to model the spread of 1878 yellow fever epidemic in Memphis with the help of the Biomathematics Summer Research Award. For these efforts, her and her research partner were published in *SPORA, A Journal of Biomathematics* and awarded the Outstanding Undergraduate Research in Biomathematics and Ecology Scholarship and Teaching Prize by the Intercollegiate Biomathematics Association. She also interned at St. Jude Children's Research Hospital in the Epidemiology and Cancer Control Department working under the St. Jude LIFE longitudinal research study. Erin was also a Mellon Fellow from 2014 to 2017 and worked in coordination with the Memphis Zoo and the Overton Park Conservancy. Additionally, Erin was a Rhodes Summer Service Fellow during the summer of 2015 where she worked with the Mid-South Chapter of the American Red Cross. She is a captain of the women's basketball team, a member of the track and field team, a member of Chi Omega fraternity, and Beta Beta Beta and Delta Epsilon Iota Honors Societies. She will continue her education next year at the University of Central Florida College of Medicine in Orlando, FL.

Allison Young '19 (Junior Editor) is from Florence, South Carolina, where she graduated from the South Carolina Governor's School for Science and Mathematics. She is a biology major and religious studies minor. She has spent time at St. Jude Children's Research Hospital as both an anesthesiology intern and a general volunteer, and has worked in Cafiero's lab on computation chemistry research. She is a Rhodes College diplomat, leader of the Rhodes Outdoor Organization, and a captain of the women's basketball team. She also is a leader of the Lynx Club, which works with Special Olympics of the Greater Memphis. She plans to spend a gap year working and volunteering abroad and then coming back to Memphis to pursue a Doctor of Nursing Practice degree at UTHSC.

Rachel Bassett '18 is a Biology major and a Chemistry minor from West Palm Beach, FL. On campus she is a Peer Academic Coach, a Rhodes College Diplomat, and the Biochemistry Tutor. Rachel is also an active member of Chi Omega and served as the Philanthropy Chair for her chapter in 2017. Rachel has conducted research in the Department of Chemical Biology and Therapeutics at St. Jude Children's Research Hospital since 2016. She studies how natural products, such as plants, can be used to treat resistant acute lymphoblastic leukemia. Rachel is a member of Beta Beta Beta, Gamma Sigma Epsilon, Mortar Board, Rho Lambda, Omicron Delta Kappa, and Phi Beta Kappa. Following Rhodes, she will attend Pharmacy School at University of Tennessee Health Science Center and hopes to become a clinical pharmacist.

Mark Massey '20 is a Biology major with a double minor in Spanish and Religious Studies. He was born and raised here in Memphis, and says the city has been integral in shaping him. At Rhodes, he was able to stay home and work in the community. In addition to serving as an editor for this journal, he serves as a Student Assistant Coach on the women's basketball team, an RSAP for the athletics department, and the activities coordinator for our own campus Special Olympics organization, Lynx Club. He hopes to continue his education and influence in Memphis, eventually serving in the community as a dentist.

Christopher Parrish '18 is from Largo, FL, where he graduated from Indian Rocks Christian School. He is a senior Biology major. His current areas of research include biological phase separation in the nucleolus at St. Jude Children's Research Hospital in the Kriwacki Lab in the Structural Biology department in conjunction with the Rhodes-St. Jude Summer Plus Program and fungal cell wall synthesis interactions in the Jackson-Hayes lab at Rhodes College. He also works for the Intramurals Sports department and is involved in Reformed University Fellowship. He will be attending UNC Eshelman School of Pharmacy in the fall to pursue a Doctor of Pharmacy degree after completing a summer in the Yang Lab in the Department of Pharmaceutical Sciences at St. Jude.

The Effect of Directly Observed Treatment in a Tuberculosis Outbreak

Sam Crowell and Peter Dorn

The purpose of this research was to analyze the effects of incorporating Directly Observed Treatment, Short Course (DOTS) into a Tuberculosis (TB) outbreak. TB is an infection that is spread through contact between individuals. In modeling the different types and progression rates of infection we were able to identify the level of treatment necessary to keep an epidemic at bay. The model also helps identify populations that need prolonged treatment.

Introduction

Tuberculosis (TB), an infection stemming from the bacteria *Myobacterium tuberculosis*, affects mainly the lungs in the human body. The bacteria has been around for thousands of years, but little is known about the prevalence of the disease prior to the industrial revolution. During the 1800's, almost 80 percent of individuals infected with TB in the US died (Blower et al., 1995). At the turn of the century, vaccinations became increasingly prevalent and as the 1900s progressed, TB infections and deaths greatly decreased. However, there has recently been a resurgence of resistant TB in rural Alabama (Blinder, 2016). The TB bacteria itself is spread through the air and infects the lungs as people breathe in the bacteria (Feng et al., 2000). Upon infection, the bacteria manifests itself in two main forms: latent and active infections (Blower et al., 1995). Latently infected individuals experience no symptoms of TB, but may later develop symptoms. Actively infected individuals immediately experience symptoms, such as coughing up blood, night sweats, and high fever. S.M. Blower, in his 1995 paper (see (Blower et al., 1995)), modeled a TB outbreak in a population, taking into account all stages of the TB infection. To this model, we have added Directly Observed Treatment, Short-course (DOTS) and non-observed treatment without the supervision of medical professionals. DOTS has been one of the most effective TB treatment regimens in developing countries in the last half century (Das et al., 2014). DOTS has five main components: accurate detection of the TB infection, government involvement in the process, medication, standardized reporting, and close observation to ensure patients take the medication as prescribed (Pasipanodya and Gumbo, 2013). We will use the percentage of infections treated with DOTS as a parameter in our model to determine the threshold of treatment necessary to prevent the large scale spread of the infection. We present the modified model in Section 2, the derivation of the basic reproduction number in Section 3, and the results of numerical analysis including parameter uncertainty and sensitivity analysis in Section 4. In Section 5, we draw some conclusions from the analysis of our model and make some policy recommendations based on our conclusions.

A Transmission Model of TB with Treatment

Our model assumes a population can be divided into seven subgroups, each represented as a model variable. Subgroup X is the susceptible population. These individuals can either die of natural causes not related to TB, become latently infected, or become actively infected. Actively infected individuals are either infectious or non-infectious meaning that both show symptoms of TB but non-infectious cases do not spread the disease. The subgroup L contains the individuals who are latently infected with the disease. Since latently infected individuals can become actively infected, two subgroups form: T_i and T_n. Those with infectious TB are grouped into T_i, and those with non-infectious TB are grouped into T_n. Individuals with infectious cases of TB can be treated. Those who are treated with the DOTS protocol are moved to the D class. Those treated with nonobserved treatment are moved to the N_0 class. Patients in either treatment classes can be cured and move to the recovered class or die. The recovered group, R, is composed of the individuals who have recovered from the infection. Recovered individuals, however, can become reinfected and move back into an infectious or noninfectious state. This model can be found in Figure 1.

Parameters

The rate of movement into and out of each subpopulation of our model depends on sixteen model parameters (see Table 1 and Figure 1). The recruitment rate, Π, is the rate at which new patients are added to the susceptible category through either birth or other expansions. The transmission coefficient, β, is the probability that an infectious individual will spread TB to a susceptible individual. The proportion of new infections that develop tuberculosis within a year is represented by p, and the force of infection is represented by λ equals βT_i. The rate of endogenous infection (i.e. a latent infection becoming an active infection) is q. From every

population sub-group, the natural death rate (deaths unrelated to TB) is μ. The death rate due to TB is μ_t. For those being treated with DOTS, the death rate is α, and for those being treated with non-observed treatment, the death rate is σ. The rate of relapsing infections is 2ω, and the natural cure rate, or the rate at which patients are cured without the use of medicine, is c. The probability of developing infectious TB from the susceptible subgroup is f, and the probability of developing infectious TB from the latent subgroup is q. The parameter $\varkappa$ is the rate at which infectious TB cases are treated, whether with DOTS or non-observed treatment. The parameter θ is the proportion of those cases that are treated with DOTS. With DOTS, the cure rate is represented by ψ, and for non-observed treatment, the cure rate is ξ.

Parameter	Interpretation	(Min, Max)	Units	References
μ	Average death rate	(0.0133, 0.04)	per year	
β	Transmission coefficient	(10^{-5}, 0.000104)	per year	Blower (1994)
Π	Recruitment rate	(4000, 5000)	ppl per year	
p	Proportion of new infections that develop in one year	(0.01, 0.30)		Blower (1994)
v	Progression rate to TB	(0.00256, 0.00527)	per year	Blower (1994)
f	Probability of developing fast TB	(0.5, 0.85)		Blower (1994)
q	Probability of developing slow TB	(0.5, 1.0)		Blower (1994)
μ_t	Mortality rate from TB	(0.058, 0.2)	per year	Blower (1994)
2ω	Relapse Rate to active TB	(0, 0.03)	per year	Blower (1994)
c	Natural cure rate	(0.021, 0.086)	per year	Blower (1994)
κ	Rate at which T_i receive treatment	(0.2, 0.6)	per year	Das (2014)
ψ	Cure rate of observed treated	(0.62, 0.8)	per year	Das (2014)
ξ	Cure rate of non-observed treated	(0.4, 0.6)	per year	Dye (1998)
σ	Death Rate of non-observed treated	(0.05, 0.35)		
α	Death Rate of observed treated	(0.036, 0.15)		Das (2014)
θ	Proportion treated with DOTS	(0.6, 0.9)		Das (2014)

Table 1: Model Parameters

Model Equations

$$\frac{dX}{dt} = \Pi - \lambda X - \mu X \tag{1a}$$
$$\frac{dL}{dt} = (1-p)\lambda X - (v+\mu)L \tag{1b}$$
$$\frac{dT_i}{dt} = pf\lambda X + qvL + \omega R - (\mu + \mu_t + c + \kappa)T_i \tag{1c}$$
$$\frac{dT_n}{dt} = p(1-f)\lambda X + (1-q)vL + \omega R - (\mu + \mu_t + c)T_n \tag{1d}$$
$$\frac{dD}{dt} = \kappa\theta T_i - (\psi + \mu + \alpha)D \tag{1e}$$
$$\frac{dN_o}{dt} = (1-\theta)\kappa T_i - (\xi + \mu + \sigma)N_o \tag{1f}$$
$$\frac{dR}{dt} = c(T_n + T_i) - (2\omega + \mu)R + \psi D + \xi N_o. \tag{1g}$$

Basic Reproductive Number: R_0

The variable R_0 is the basic reproduction number, and it corresponds to the number of secondary infections caused by one infectious individual in a perfectly susceptible population (Kajita et al., 2007). This number is important in modeling TB outbreaks, as it can predict whether the outbreak will either diminish or spread to epidemic levels. If $R_0 > 1$, TB will continue to spread through the population, but if $R_0 < 1$, TB incidences will decrease to a negligible level. R_0 assumes that the

entire population is susceptible, and our model reflects that assumption (Heffernan et al., 2005).

Calculating R_0

The next-generation method was used to calculate R_0, the threshold condition (see (Heffernan et al., 2005) and (van de Driessche and Watmough, 2001)). The variable R_0 determines the stability of the disease-free equilibrium in a TB outbreak. The disease free equilibrium of our model is as follows:

$$((X^*, L^*, T_i^*, T_n^*, D^*, N_0^*, R^*) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0, 0\right).$$

Under this assumption of the disease-free equilibrium, the rate of new infections coming into each of the infected classes are the F sub-equations:

$F_L = (1 - p)\beta XTi$,

$F_{Ti} = pf\beta XTi + \omega R$,

$FTn = p(1 - f)\beta XTi + \omega R$,

$F_D = 0$, *and* $F_{N0} = 0$.

The V sub-equations give the rate of movement into and out of the infected classes other than new infections:

$V_L = (v + \mu)L$, $VT_i = -qvL + (\mu + \mu t + c + \varkappa)T_i$,

$V_{Tn} = -(1 - q)vL + (\mu + \mu t + c)T_n$,

$V_D = -\varkappa\theta T_i + (\psi + \mu + \alpha)D$,

and $V_{N0} = -(1 - \theta)\varkappa Ti + (\xi + \mu + \sigma)$.

From these equations, we create the following matrices:

$$F = \begin{bmatrix} 0 & (1-p)\beta X & 0 & 0 & 0 \\ 0 & pf\beta X & 0 & 0 & 0 \\ 0 & p(1-f)\beta X & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} v+\mu & 0 & 0 & 0 & 0 \\ -qv & \mu+\mu_T+c+\kappa & 0 & 0 & 0 \\ -(1-q)v & 0 & \mu+\mu_T+c & 0 & 0 \\ 0 & -\kappa\theta & \theta & \psi+\mu+\alpha & 0 \\ 0 & -(1-\theta)\kappa & 0 & 0 & \xi+\mu+\sigma \end{bmatrix}$$

We then multiply F by V^{-1}, and R_0 is the largest eigenvalue that results from this FV^{-1} matrix. We found this eigenvalue to be:

$$R_0 = \frac{\beta(fp\nu + q\nu - pq\nu + fp\mu)\Pi}{\mu(\nu + \mu)(c + \kappa + \mu + \mu_t)} .$$

Numerical Analysis

With the equations for our modified TB model, we were able to use Mathematica to perform single simulations, multiple simulations with uncertainty, and calculate measurements related to sensitivity and uncertainty.

Single Simulation

Using the midpoint of parameter values given in Table 1, we ran two single simulations of the model over 100 years. One simulation included the possibility of treatment and the other did not. We then plotted the population of each state variable and new incidences of TB. Plots of each simulation are shown in Figure 2. divide each range into 500 equally distributed values and randomly sample each parameter without replacement 500 times. Thus, 500 sets of randomized parameters are created that can be used to measure output in the model. We calculated the value of R_0 using the 500 randomized parameter sets. Figure 3 presents the range of R_0 in a box and whisker plot. Approximately 25% of all simulations resulted in an R_0 below one.

Sensitivity and Uncertainty Analysis

After calculating R_0, we performed sensitivity and uncertainty analysis on the parameters of which it is composed. Latin Hypercube Sampling (LHS) is one tool used in sensitivity and uncertainty analysis. Given a range of values for each parameter (see Table 1 for parameter ranges), we divide each range into 500 equally distributed values and randomly sample each parameter without replacement 500 times. Thus, 500 sets of randomized parameters are created that can be used to measure output in the model. We calculated the value of R0 using the 500 randomized parameter sets. Figure 3 presents the range of R_0 in a box and whisker plot. Approximately 25% of all simulations resulted in an R_0 below one.

Uncertainty analysis was also performed on the incidence rate of TB and cumulative deaths from an outbreak. The box and whisker plot in Figure 4a shows that initially, fast TB incidences have the greatest amount of variation. The range of incidence of slow TB is greater than that of relapse TB, but slow and relapse TB incidences are still lower and less varied than fast TB. As time progresses, the incidence of fast TB decreases, the incidence of slow TB increases slightly, and the incidence relapse TB remains relatively constant. From these results, it can be predicted that in the long run, latent infections will

persist. From the simulations in Figure 4b, it is evident that as time progresses, the variation in cumulative deaths increases. Some simulated outbreaks have almost no deaths across all time periods, but the maximum simulations accumulate around 208,000 deaths over 100 years. The cumulative cured is shown in Figure 4c. It has great variation that spans the entirety of the simulated time period. The median year, though, occurs at year 13.

Following this, we calculated the partial rank correlation coefficients (PRCCs) of the parameters with respect to cumulative deaths, cumulative cured, and the basic reproductive number. The measure of cumulative cured accounts for all the cured individuals over the course of the simulation. The measure of cumulative deaths accounts for all deaths from TB over the course of the simulation. As stated earlier, R_0 corresponds to the number of secondary infections caused by one individual. PRCCs quantify the effect of a change in one parameter on the value of an output and range from negative one to positive one. A PRCC near positive one means that an increase in the parameter is highly correlated to an increase in the output. Similarly, a PRCC near negative one means that an increase in the parameter is highly correlated with a decrease in the output, and a PRCC value near zero means that there is almost no correlation between changes in the parameter and the output. The PRCCs for these outputs, given in Table 2, show which parameters should be manipulated in order to shorten the duration of a TB outbreak and lower the number of cumulative deaths. From these results of uncertainty and sensitivity analysis of R_0, several conclusions can be made regarding TB outbreaks and treatment with DOTS and non-observed treatments. The treatment parameter values tested lead to fewer epidemics than no treatment scenarios, as shown by the simulations in Figure 2. However, around 75 percent of the calculated R_0 are greater than one and would lead to epidemics, while the median value is approximately 2.77 (see Figure 3). From the simulations, it is evident that the risk of widespread infection occurs because of relapse infections. The next observation from the sensitivity analysis is that the most highly correlated parameters with cumulative deaths are β, the transmission coefficient, which is positively correlated with cumulative deaths, and p, proportion of new infections that develop in a year which is also positively correlated with cumulative deaths. Other significant correlations exist with μ, the natural death rate and c, the natural cure rate. Both μ and c are negatively correlated with cumulative deaths. Manipulation of these four parameters would thus have significant impact on the outcome of a TB outbreak.

Table 2: Partial Rank Correlation Coefficients with respect to cumulative deaths, cumulative cured, and the basic reproductive number, $\mathcal{R}_0$.

Parameter	Cumulative Deaths	Cumulative Cured	$\mathcal{R}_0$
μ	-0.492	-0.517	-0.893
β	0.838	0.711	0.923
Π	0.192	0.124	0.218
p	0.896	0.749	0.725
v	0.268	0.231	0.424
f	0.248	0.279	0.228
q	0.191	0.064	0.342
μ_t	0.199	-0.259	-0.324
ω	0.170	0.210	0.006
c	0.025	0.017	-0.203
κ	-0.398	-0.137	-0.588
ψ	0.069	-0.062	-0.043
ξ	0.065	-0.024	0.023
σ	0.015	-0.123	-0.029
α	-0.028	0.036	-0.119
θ	0.035	0.090	- 0.019

Discussion

Our model simulates the spread of TB with the inclusion of both directly observed treatment and non-observed treatment. This model is an adaptation of the Blower (Blower et al., 1995) model and is used to gage the effectiveness of differing treatment types (DOTS and non-observed) in handlingthe spread of TB across a time frame of 100 years. Prior studies have shown that upwards of 90% of individuals undergoing DOTS recover, and our findings further confirm the effectiveness of DOTS beyond treating individual patients (Pasipanodya and Gumbo, 2013). There have been many studies of DOTS that show its effectiveness in treating individual patients, but few studies have shown the treatment's role in preventing outbreaks' progressions to epidemics. For this, integrative research is important and quite necessary to understand the powerful effects of treating individuals in the prevention of widespread outbreaks. Our models show the stark difference that treatment can have in preventing a TB epidemic. In the simulations with treatment, the number of recovered cases are higher than in the simulation without treatment. The incidence is also much lower in the simulation with treatment. This occurs because the spread of TB is strongly correlated with the number of individuals currently infected. By lowering the number of infected individuals and treating them, further infections are prevented.

Treatments such as DOTS are powerful tools, however, latent infections still persist in simulations. DOTS and other treatment protocols only treat actively infectious cases, so individuals with latent infections do not receive treatment, and are therefore unaffected by the introduction of treatment. Beyond this, the model is idealistic in that it assumes all individuals that undergo DOTS complete the treatment, and it is assumed that all infected individuals have access to treatment. In reality, there are often financial and geographical constraints that hamper equal access to medical care. In rural communities, access to medical facilities and

trained clinicians may be limited. Going forward, future TB models could analyze the effect of individuals transferring between DOTS and non-observed treatment. This feature would more closely reflect real world scenarios, as it would allow a cost analysis between DOTS and other less costly treatments.

The modeling within this paper is still relevant, though, for within the past five years, a TB outbreak has ravaged parts of rural Alabama. With a distrust of medical professionals, many individuals may forgo treatment, and the outbreak could quickly reach epidemic levels (Blinder, 2016). Our model has shown the stark difference in outcomes of outbreaks in which different levels of treatment are administered.

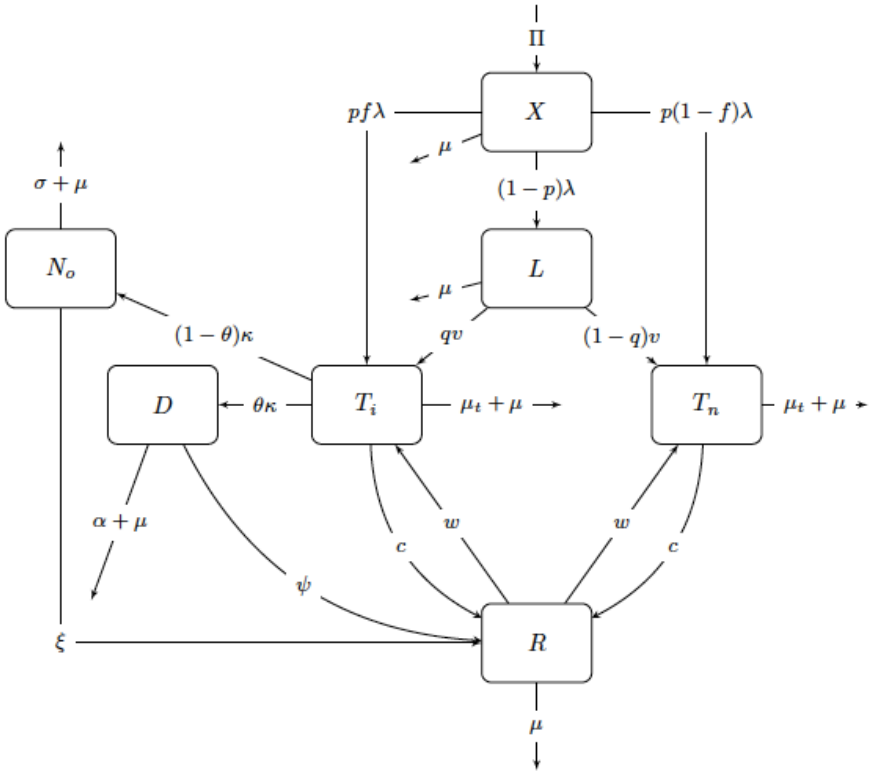
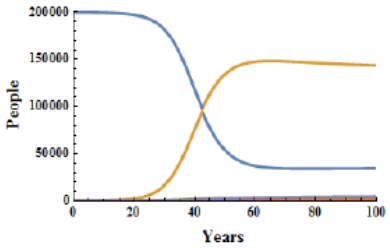
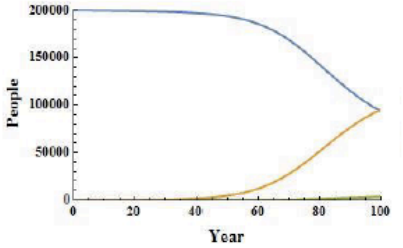


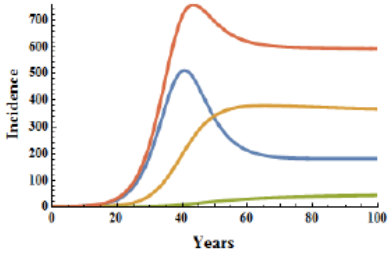
Figure 1: A flow diagram of our proposed model with the inclusion of DOTS and non-observed treatment. The boxes represent state variables, and parameters are depicted in the path lines.



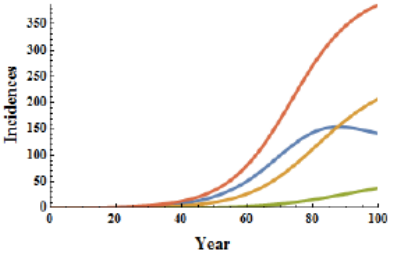
(a) Progression of TB virus without treatment. Blue is the susceptible state, yellow is the latent state, green is infectious TB, orange is non-infectious TB, and purple is the recovered state.



(b) Progression of TB virus with treatment. Blue is the susceptible state, yellow is the latent state, green is the recovered state, and the remaining states are too small to be visible in this graph.



(c) Incidences of new infections without treatment. Yellow is slow TB, blue is fast TB, green is relapse TB, and red is the sum of fast, slow and relapse TB.



(d) Incidences of new infections with treatment. Yellow is slow TB, blue is fast TB, green is relapse TB, and red is the sum of fast, slow and relapse TB. treatment.

Figure 2: Single simulations over 100 years, with and without treatment.

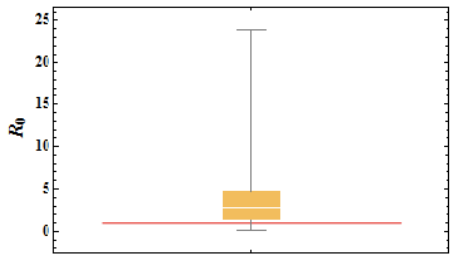
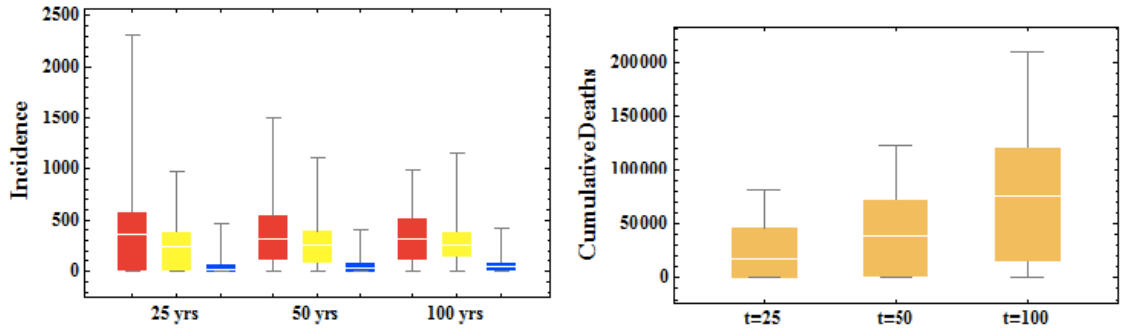
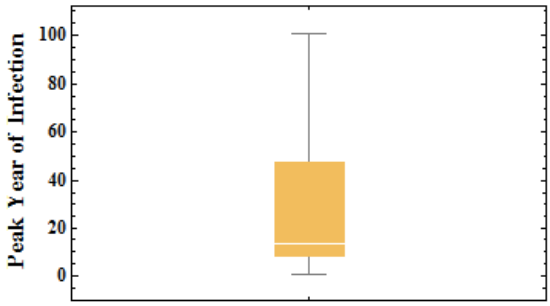


Figure 3: The box and whisker plot shows the ranges of the threshold parameter. The red line at $R_0 = 1$ marks the point below which an endemic will die out.



(a) Incidences of fast TB (red), slow TB (yellow), and relapse TB (blue) at $t = 25, t = 50$, and $t = 100$

(b) Cumulative Deaths from TB at $t = 25, t = 50$ and $t = 100$.



(c) Cumulative Cured from TB

Figure 4: Box and whisker plots for TB incidence, cumulative deaths, and cumulative cured. The boxes indicate the interquartile range, and the bars indicate the minimum and maximum values.

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The Mind of a Murderer

Sydney Watts

This paper is a collection of different psychological and neurological studies that show a connection between murderous behavior and specific types of brain damage. There are different factors. One factor is that a person usually goes through some type of childhood trauma, such as sexual, physical, or emotional abuse, bullying, a death in the family, etc. Once a person has experienced trauma, they are more likely to develop some type of personality disorder. The development of these personality disorders coincides directly with damage to certain areas of the brain. In summary, different parts of the brain control different aspects such as behavior, feelings, decision-making skills, emotions, and self-control. If one of these areas is damaged, so are its functions. These functional abnormalities along with some type of genetic predisposition or a childhood trauma may result in behaviors characteristic of murderers.

Introduction

The brain of a murderer is different than that of an average person. The majority of murderers, if not all murderers, share abnormalities in their brain anatomy and psychology that affect the overall function of their brains. There have been multiple studies conducted that connects murderers with brain damage and/or psychological disorders. According to Healthline Medical Team, Oler and Fox, and Raine, most of the structural abnormalities are related to reduced gray or white matter in the prefrontal cortex, amygdala abnormalities, and an asymmetrical hippocampus. These structural abnormalities coexist with personality disorders that are common in murderers.

A Review

Mental Disorders

The most common mental disorder among murderers is called anti-social personality disorder (APD). People with APD can not decipher between right and wrong and they can not care about other people's feelings (Pemment, 2013). APD can be an explanation for psychopathic behavior in murderers (Pemment, 2013). Another common psychological disorder recognized in murderers is borderline personality disorder (BPD). People with BPD experience severe emotional instability, anxiety, and psychotic-like behavior in which they could randomly become paranoid or suspicious of other people (Pemment, 2013). Doctors identify BPD patients as having "zero degrees of empathy," which means they are physically unable to understand other people's feelings (Pemment, 2013). Impulsive aggression is also a characteristic in BPD. Killers who have BPD tend to be more spontaneous when they kill due to their impulsiveness. This method of killing is a characteristic of a sociopath (Pemment, 2013).

Psychopathy vs. Sociopathy

The majority of murderers with mental disorders can be classified as a "sociopath" or a "psychopath." These are different, however, they share a few basic traits. They have a disregard for laws and common social knowledge, a disregard for the rights of others, a failure to feel remorse or guilt, and a tendency to display violent behavior (Bonn, 2014). Sociopaths are more nervous, are easily agitated, and appear to be constantly disturbed (Bonn, 2014). This causes them to have a higher risk of having an emotional outburst of rage. Sociopaths are typically uneducated and live at a lower class standard. They are unable to stay in one place for an extended time or hold a steady job (Bonn, 2014). They are able to form an emotional attachment to a group, but they do not have regard for society or its rules. The factor that sets apart a sociopath from a psychopath is that it is difficult, yet not impossible for a sociopath to form emotional connections with others (Bonn, 2014).

Alternatively, a psychopath is completely unable to form emotional attachments or feel empathy for others (Bonn, 2014). They learn to mimic certain emotions to deceive people. They are charming, narcissistic, and manipulative (Bonn, 2014). They easily gain people's trust or manipulate themselves into a relationship in order to appear "normal." Their families often do not suspect their psychopathic behavior. Their crimes are organized and planned with great detail, which makes it hard for police to identify any clues (Bonn, 2014). Sociopathy is the result of environmental factors, such as childhood trauma or abuse, while psychopathy is the result of physiological defects that cause the brain to be underdeveloped (Bonn, 2014). The FBI has stated

that the traits of a psychopath are linked to murderers (Pemment, 2013).

Brain Damage

These traits that cause murderous behavior are linked to physical brain damage. One type of brain damage that is commonly linked with violent behavior is a dysfunctional central nucleus region of the amygdala (Oler & Fox, 2010). The amygdala is responsible for emotions, survival instincts, and memory (Raine, 2000). If the amygdala is damaged, it becomes difficult to control emotions (Raine, 2000). Damage to this region causes people to have negative judgement and reactions and lower levels of empathy, which is one of the key characteristics of a sociopath (Raine, 2000). The hippocampus is another part of the brain that influences behavior because it is responsible for storing long-term memories (Healthline Medical Team, 2015). Damage to the hippocampus contributes to violent and abnormal behavior (Healthline Medical Team, 2015). The prefrontal cortex is the part of the brain that is the most influential if it becomes damaged (Raine, 2000). The prefrontal cortex is the control-center for behavior and personality development (Raine, 2000). If there is an abnormal function in the prefrontal cortex, personality disorders are developed, such as APD, BPD, psychopathy, and sociopathy, and an inability to control rage (Fallon, 2009). Damage to the prefrontal region of the brain is most often the cause of personality disorders (Fallon, 2009).

The other most common type of brain damage in murderers is reduced gray and white matter in the brain (Pemment, 2013). The gray matter is in the part of the brain that is responsible for muscle control, seeing, hearing, memory, emotions, speech, decision making, and self-control (Fallon, Jim). Poor decision making and poor self-control are key characteristics of murderers. All of these structural abnormalities are common in murderers because the damage can easily result in the development of psychopathic or sociopathic behavior. The majority of notorious serial killers—Charles Manson, John Wayne Gacy, Jeffery Dhamer, Aileen Wuornos, Ted Bundy, and many more—have had their brains analyzed and their psyches studied through interviews by multiple professionals with the mission of detecting the links they had with these abnormalities.

Psychological and Neurological Studies

There have been multiple case studies on serial killers' minds. In the late 19th century, French pathologist Alexandre Lacassagne had prisoners write notebooks with their stories so he could publish them as criminal autobiographies. Through this study, he learned that most of the violent offenders had endured some form of abuse as a child and had a history of tension, criminal records, and physical and mental disease. One of the most well-known criminal autobiographies, *Der Sadist,* was written by Dr. Karl Berg in 1945. This autobiography was about Peter Kürten, a German serial killer. He recounted his crimes in gory, dream-like details with no signs of remorse. Kürten told the judge at his trial, "Never have I felt any misgivings in my soul. Never did I think to myself that what I did was bad." This statement classified Peter Kürten as legally insane because he was not be able to comprehend that the crime he committed is against the law (Ramsland, 2013). Berg is responsible for inspiring the origins of the FBI's Behavioral Analysis Unit. This study was a new method when it came to criminology because he went beyond an isolated case analysis. He had a team of professionals to try and understand the psychological explanation behind serial sexual sadism and serial killing. These new methods of behavioral and psychological analysis had challenged the methods of regular law enforcement (Ramsland, 2013).

Additionally, Dr. Melvin Reinhardt, professor of criminology at the University of Nebraska, interviewed serial killer Charles Starkweather. Dr. Reinhardt had noted that Starkweather was bullied at a young age, and he had a severe head injury. This further supported that there is a relationship between murderers, childhood abuse, and brain damage. This study also helped to influence the BAU.

Similarly, in the 1960's, prison psychiatrist Marvin Ziporyn worked a study on a mass murderer named Richard Speck. Ziporyn came to the conclusion that Speck had a head injury and suffered substance abuse. He was also diagnosed with organic brain syndrome (Ramsland, 2013). After Speck died, Dr. Jan E. Leestma, a neuropathologist, scanned his brain and found blurred boundaries between the hippocampi and the amygdala. These two organs infringed upon each other (Ramsland, 2013).

Another doctor, Dr. Adrian Raine, was one of the first to perform a brain comparison experiment. He scanned 41 brains of violent offenders and 41 brains of normal, civil individuals. He saw a lot of damage in the brains of the violent offenders. Dr. Helen Morrison did a study similar to this, but she went beyond the brain damage and sought answers in genetics and DNA (Ramsland, 2013). Dr. Helen Morrison interviewed and analyzed brains of 135 serial killers and found that almost all of them shared a chromosome abnormality (Ramsland, Katherine). This specific chromosome is mostly abundant in males because it is a gene passed from mother to son (Fallon, 2009). If the chromosome is abnormal, it will not be activated until puberty (Fallon, 2009). This provides evidence for why serial killers start

displaying violent behavior in their early teens, after having some type of childhood trauma. Jim Fallon collected evidence from Morrison and other professionals and expanded it to conduct his own studies of how killing is linked to genetics.

Jim Fallon's approach was different, while still trying to accomplish the same goal as some of these other doctors. He scanned his own brain and compared it to the brain scan of a serial killer (Fallon, 2009). He noticed that he had the same reduced activity in the orbital cortex as the serial killer. Low activity in the orbital cortex meant low suppression of behaviors such as rage, violence, eating, sex, and drinking. This confused Fallon because he is not a serial killer, so he did not understand why his brain looked like one. The question was answered after studying his family history and scanning brains of family members. He learned from his mother that he has a line of serial killers at the roots of his family tree. He found out that he is related to Lizzie Borden—famous axe murderer. Further up the family tree is Thomas Cornell—the first person in history to commit matricide. When he studied the DNA of his family, he noticed the MAO-A gene—the violence gene. This gene regulates serotonin in the body and affects people's moods. The gene comes from the X-chromosome that boys get from their mothers. This could possibly explain why there are more male serial killers than female. The only way to activate this gene is by going through something traumatic or violent at a young age. If an already psycho-type person causes this gene to activate, the result could create a murderer because they already have the characteristic damage in their DNA (Fallon, 2009). This discovery opened up the possibility that psychopathic genes are hereditary, and can be passed on through generations of the family. Since Fallon shared the brain abnormalities but not the behavior, it is evident that serial killers are created from a combination of brain damage and a childhood trauma trigger.

Conclusion

There are physiological and psychological abnormalities in the minds of many murderers. Through brain scans, genetic testing, and interviews, most killers share similar types of brain damage and family history. Through behavioral profiling, it is understood that many killers are psychopathic. It is hard to pinpoint the exact cause of the brain damage that leads to their behavior. Regardless of whether or not their damage was caused from a head trauma, childhood abuse, drug abuse, or a mental illness, or in their DNA, many murderers share some type of damage to their brain.

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Cooperation and conflict: an agent-based model proposal of evolutionary tradeoffs exhibited in fetal microchimerism

Erin Deery

Fetal microchimerism, the bi-directional exchange of fetal and maternal cells during gestation, has broader impacts on the long-term health of the mother and child long after birth. In the mother, fetal cells can play roles in post-partum depression, breast, thyroid, and brain tumor development, and increased probability of miscarriage. For the child, the presence of these cells can affect the way that a mother interacts with her child after birth, thus directly affecting the care and ultimate survival of the child. The evolutionary tradeoffs between survival of the mother at the expense of her child and the survival of the child at the expense of their mother are useful in explaining and diagnosing adverse health outcomes that mothers experience post-partum. The interaction between fetal cells and maternal organs could be further explored through the use of an agent-based model that could potentially inform healthcare decisions made by mothers and their healthcare providers.

Introduction

Microchimerism, the presence of cells or DNA from two genetically distinct individuals in tissues or circulation of one body, can occur between mother and fetus during the gestational period (Gammill and Nelson, 2010). This phenomenon, exclusively observed in eutherian mammals, is a bidirectional flow of cells between the mother and fetus that can continue to impact both the mother and child postpartum (Binindia-Emonds et al., 2007). It is proposed that microchimerism occurred in the common ancestor of eutherian mammals approximately 93 million years ago, because an exchange of cells is observed in all placental types (Binindia-Emonds et al., 2007). It is most prevalent in hemochorical placental types, the most invasive placenta type, which allows for the greatest exchange of resources, and thus the exchange of cells, between the mother and fetus (Moffett and Loke, 2006). As depicted in Figure 1, fetal cells within the mother's body can also be transferred to that mother's future offspring, such that younger siblings may have both maternal cells and fetal cells from an older sibling transferred to them in the womb (Boddy, 2015). Additionally, microchimerism can endure generations when cells from new offspring's maternal grandmother are also present in the mother and then transferred to her offspring (Boddy, 2015).

Fetal microchimerism is specifically the presence and persistence of fetal cells in the mother. These fetal cells are often first detected at placental initiation, increase throughout the gestational period, and decrease in number after birth (Moffett and Loke, 2006). Postpartum, the mother's immune system often attacks fetal cells to induce apoptosis (Boddy, 2015). However, some fetal cells can persist within the mother's body for decades, potentially causing both benefit and harm to the mother (Haig, 2014). The presence of these cells both in the plasma of maternal blood and within maternal tissues can cause a variety of responses (Boddy, 2015).

Cooperation between fetal cells and maternal cells within the mother's body is important for the health of both individuals. Logically, if the mother is healthier, then the fetus will be healthier in the womb. Postpartum, the baby will receive greater care if the mother is able to provide adequate resources to her offspring (Gammill and Nelson, 2010). Fetal cells can act like stem cells within the mother's body. This trait can reduce the effects of aging by replenishing stem cell niches where maternal stem cells have depleted over time (Gammill and Nelson, 2010). The stem cell characteristics allow fetal cells to transform and be incorporated into healthy smooth muscle, bone marrow, and blood vessels (Boddy, 2015). Fetal

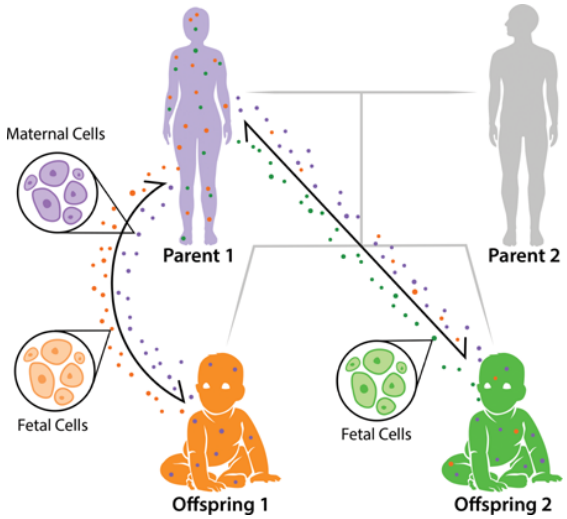

Figure 1: A diagram of microchimerism depicting the bidirectional flow of cells between a mother and her offspring (Boddy, 2015).

cells can also directly contribute to the mother's health through assistance in wound healing (Axiak-Bechtel et al., 2013). Fetal cells in mice were observed at sites of inflammation and are assumed to

aid in the repair and reproduction of blood vessels (Ngiyen Huu et al., 2007). In humans, fetal cells have been observed at cesarean section scars, suggesting that maternal wound healing aids both the survival of the mother and the survival of the offspring (Boddy, 2015). While these functions of fetal cells contribute directly to maternal health and indirectly to offspring health, these same functions can also provide direct fitness advantages to offspring by enhancing resource production and allocation to the offspring by manipulating maternal organs, exhibiting a push and pull of benefits and risk to the mother.

Due to the fitness advantages gained by the offspring, fetal cells are most often found in greater abundance in organs such as the breast, thyroid, and brain (Boddy, 2015). These organs are directly related to care for the offspring and reproduction of future offspring. In these organs, the cooperation between maternal and fetal cells to raise viable offspring, thus successfully passing on genetic information to the next generation, is met with limitations of the maternal organs. The evolutionary trade-offs exhibited by cells in these major organs demonstrate ways in which evolution has selected for microchimerism despite the negative health outcomes often observed in mothers with greater concentrations of fetal cells in their bloodstream and organs.

First, in breast tissue, the presence of cells can increase lactation by releasing factors that will stimulate mammary glands, or by using stem cell characteristics to differentiate to mammary cells (Gustbée et al., 2015). Milk is energetically costly for the mother to produce, but offspring receive vital nutrients, immunological defense, and calories from milk consumption. Because of these benefits, the offspring will often demand greater milk production than what is optimal for the mother (Boddy, 2015). Although breastfeeding, often induced by the presence of some fetal cells in healthy breast tissue, is associated with a reduced risk of breast cancer, excessive milk production has been associated with the genesis of breast tumors (Gustbée et al., 2015). Further evidence of conflict is observed in high-grade breast tumors that have greater concentrations of fetal cells than low-grade breast tumors (Boddy, 2015).

More cooperation and conflict is observed between fetal and maternal cells in the mother's thyroid. In the thyroid, resource allocation, particularly of heat, is again important because of the thyroid's roles in thermoregulation and metabolism (Winberg, 2005). Offspring will benefit from greater heat production and transfer from the mother, but this is energetically costly to the mother. While fetal cells have been observed in healthy thyroid tissue, elevated concentrations of fetal cells are known to cause diseases such as thyroiditis, Grave's disease, and cancer (Gammill and Nelson, 2010). These occur when when fetal cells proliferate or promote factors such as metabolic hormones T3 and T4 that can cause abnormal function or composition of the thyroid (Boddy, 2015).

Similar to the breast and thyroid, the mother's brain is also susceptible to manipulation by fetal cells. Although there are inconclusive results regarding the overall effect of fetal cells of the maternal brain, there are specific relationships between fetal cells and susceptibility to certain diseases such as Alzheimer and Parkinson's diseases (Boddy, 2015). In mothers with Alzheimer's disease, fewer fetal cells were detected in brain tissue (Chan et. al 2012). In mothers with Parkinson's disease, fetal cells were found in greater concentration in diseased tissue (Boddy, 2015). In addition to disease, fetal cells in the brain are also associated with elevated levels of oxytocin and prolactin (Carter, 2013). These elevated levels can increase lactation, attachment and interest in offspring, and resource transfer to offspring. Oxytocin in particular played a role in the development of the human nervous system and was selected for due to its role in human sociality, especially in coordination with child rearing (Carter, 2013). As previously mentioned, fetal cells are susceptible to maternal immune response, which may explain brain swelling that is associated with postpartum depression (Boddy, 2015). This response would be a conflict between the mother and offspring's interests as the mother's body attempts to optimize resource allocation to offspring at her offspring's cost. After birth, the evolutionary trade-off of child number versus child survival becomes apparent as mothers can either allocate resources to current offspring or invest in reproduction of future offspring. This period of time between offspring, known as the interbirth interval, becomes a conflict of interest for the mother and offspring (Haig, 2014). Fetal DNA favors a longer interbirth interval than maternal DNA because the fetus seeks to maximize the time and resources allocated to current offspring (Haig, 2014). Because of this, fetal cells in the brain can also act similarly to a contraceptive by influencing the interbirth interval necessary before the mother is able to have another child (Haig, 2014). Fetal cells have even been associated with SRM, secondary recurrent miscarriage, when found within a mother's brain and endometrial tissue, thus decreasing the mother's fitness but increasing the first born offspring's chance of survival (Haig, 2014).

These conflicting outcomes seen in the evolutionary trade-offs between maternal and fetal cells as observed in a mother's breast, thyroid, and brain could be further explored through agent-based mathematical modeling. An agent-based model is a

mathematical model that operates on an algorithm that codes for many possible outcomes in a system where entities, called agents, move and make decisions autonomously based on interactions with one another and their local environment (Bonabeau, 2002). At a time-step, an agent makes a decision according to both its surroundings at that moment and a set of behaviors that could potentially be executed, allowing for a flexible model that describes a system as it naturally exists (Bonabeau, 2002). Agent-based models have been used to study complex interactions, adaptive behaviors, and feedback loops in infectious and chronic diseases (Li, 2016). These models have emergent properties that arise from the analysis of individual agents' behaviors as it relates to the system as a whole (Bonabeau, 2002). Agent-based modeling works well in systems that can be described with thresholds that trigger changes in possible behaviors, when agent behaviors are based on probabilities but have an element of stochasticity, and when individual agent behavior is complex (Bonabeau, 2002). When behavior becomes more complex for agents, a differential equations model exponentially increases in complexity and still may not reflect important fluctuations in the model (Bonabeau, 2002). To parameterize, calibrate, and test simulations of an effective agent-based model requires large amounts of individual-level data, but studies on fetal microchimerism often include high volume tests of individual mothers, their numerical fetal cell concentrations, and quantitative health outcomes associated with the presence of these cells (Li, 2016). Based on the complexity and discontinuity of interactions between maternal and fetal cells, an agent-based model could harness these interactions and represent them spatially to provide explanations and structure to the data found in current literature on fetal microchimerism. Based on thresholds of fetal cell concentrations within the mother, such a model could predict maternal health outcomes that arise or are exacerbated by the presence of fetal cells. This research would increase our understanding of the correlations between fetal cell concentration and maternal health outcomes by analyzing the cellular interactions that have the greatest impact on maternal health and by quantifying the optimal fetal cell concentrations for maternal health based on the benefits and repercussions of fetal cell presence.

Hypotheses

If an agent-based model is built on the foundation of existing research that explores the cooperation and conflict of fetal cells within a mother's body, then the interactions between maternal and fetal cells will reveal emergent health outcomes for the mother that can be quantified and understood in an evolutionary context. The differential fitness interests of mother and offspring will be expressed even at the cellular level in the model, such that individual agents, fetal and maternal cells, will behave according to the interests of their original host. The concentration of fetal cells within a system of maternal breast, thyroid, and brain cells will effect each maternal organ in a different way based on the influence that fetal cells individually have on cells within these maternal organs. Interactions between cells of the maternal immune system and fetal cells will help determine the optimum concentration of fetal cells. Concentrations of fetal cells observable over many simulations of the model will reveal a threshold condition such that certain concentrations of fetal cells within maternal organ systems will raise the mother's negative risk factor to a level of alarm. These upper and lower thresholds of fetal cell concentrations can be quantified and generalized to mothers that have fetal cells detected in their bodies postpartum. Future samples of fetal cell concentrations within maternal organs could be compared to the model to raise awareness about health risks and influence future treatment plans for mothers with fetal cell concentrations outside the optimum threshold quantified in the model.

Research Plan

Using existing literature on fetal microchimerism and its effects on maternal fitness, an agent-based model will be constructed based on normalized data from current research like Lo and colleagues accomplished by quantifying the bidirectional transfer of cells (Lo et al., 2000), optimum fitness models for the mother and offspring like the model calculated by Haig (Haig, 2014), and probabilities that health benefits and risks will occur from interactions between fetal and maternal cells. Because this proposed model focuses on the health of breast, thyroid, and brain tissue and immune system response, extensive data from studies concerning how fetal cells interact with these types of cells will be the foundation of the model. Next, an algorithm, as seen in Figure 2, will be written that codes for the behaviors of the different agents; agents will be fetal cells and multiple types of maternal cells. This algorithm will be written based on possible cooperation and conflict between these cell types, probability that different health outcomes will occur, fetal cell concentrations at each time step, and fitness advantages of different behaviors.

This algorithm will be written and simulated on the software NetLogo, which is freely accessible online from Northwestern University. Then, the

model will be simulated many times with a range of initial fetal cell concentrations to determine the optimal concentration for maternal and offspring fitness. Finally, the results obtained from the model will be compared to literature values and revised to minimize error. Additional statistical analyses will be performed using Wolfram Mathematica software.

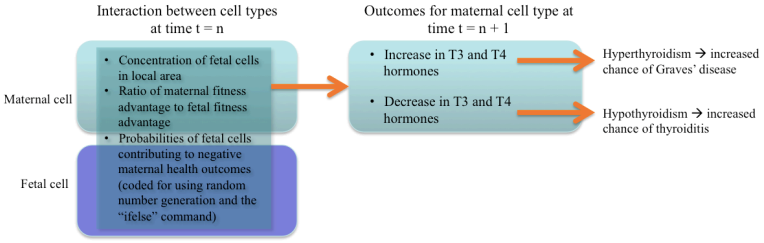

Figure 2: Example of behavioral algorithm for maternal thyroid cells

Anticipated Results

The result of this research would be an agent-based model of fetal microchimerism demonstrating how evolutionary trade-offs created by fetal and maternal cell interaction can affect maternal health. It is anticipated that the interactions of individual cells based on a their surroundings and probabilities of possible behaviors will have a significant impact on a mother's fitness as she allocates resources to her offspring. There is a projected quantitative optimum amount of resources – milk production from the breast, T3 and T4 production in the thyroid, and oxytocin and prolactin release in the brain – that allows a mother to best provide for her offspring without compromising her own health or her future potential to produce offspring. Through many simulations of the model, optimum concentrations of fetal cells within different tissues will be revealed. With these optimal concentrations and the consideration of statistical error, a threshold range surrounding the optimum will be calculated disclosing upper and lower bounds of concentrations of fetal cells.

Broader Impacts

Agent-based modeling has been used to model infectious disease, chronic disease, and tumor growth, but has not yet been applied to fetal microchimerism. Such a model would increase our understanding of how fetal cells negatively affect maternal tissue, and possibly inspire clinical methods of control to maintain fetal cell activity in an optimum range. A working model would help refine therapies aimed at quelling the negative effects of fetal cells before testing on animal models begins, thus saving time, resources, and animal life. The use of an agent-based model, a relatively recent innovation in mathematical modeling compared to differential equations models, would increase awareness in the scientific and mathematical communities about the biological applications of agent-based models. This model would demonstrate that agent-based models are proficient at analyzing positive and negative outcomes from complex cell-to-cell interactions. Results from this research will be publicly available with the hopes that mothers who fear adverse affects of fetal cells could be tested against the model to determine if they are at high risk for negative effects based on the concentration of fetal cells in their tissues.

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How Processed Foods Can Cause Heart Disease

Sydney Watts

Heart Disease is typically caused by a bad diet. The components of a bad diet that contribute the most to heart failure are sugar, fat, and sodium. Sugar causes high blood sugar, which leads to diabetes, which leads to heart disease. Sodium causes high blood pressure, which leads to heart failure. Fat causes high cholesterol, which leads to diabetes, which leads to heart failure. These components are mainly found in processed food. Since processed food is such a large part of everyone's diets, heart disease has become more common.

Introduction

According to the American Heart Association (AHA), Heart disease is the world's number one killer. Every year, 17.3 million people from all over the world die of heart disease (Heart, 2014). The Association has predicted that by the year 2030, heart disease will be the cause of almost 25 million deaths per year. In just America, one seventh of all deaths are caused by heart disease (Heart, 2014). There are many different conditions that can be classified as heart disease. The most common types are coronary artery disease, high blood pressure, cardiac arrest, congestive heart failure, arrhythmia, peripheral artery disease, stroke, and congenital heart disease (Heart, 2014). All of these conditions are preventable and they all share common causes. The American Heart Association has something called "Life's Simple 7" that consists of seven key health factors and behaviors that cause heart disease. These factors are smoking, limited physical activity, an unhealthy diet, overweight/obesity, high cholesterol, high blood pressure, and high blood sugar (Heart, 2014).

Diet

Processed Food

Five out of seven of those factors, excluding smoking and physical activity, are linked to diet. Even though diet is one of the seven factors, obesity, high cholesterol, high blood pressure, and high blood sugar are all linked to diet. Less than 1% of adults and practically 0% of children in America meet the AHA's definition of an ideal healthy diet. 69% of adults and 32% of children in America are overweight or obese; one third of Americans have high cholesterol; 33% of Americans have high blood pressure, also known as hypertension; 9% of the adult population has diabetes, which is a disease caused by high blood sugar. Studies have shown that from 1971-2004, eating habits have changed completely for the worst (Heart, 2014).

This recent change in eating habits is around the time that processed food became so popular in America (Ryssdale, 2013). Processed food makes up over 70% of the average American diet (Ryssdal, 2013). Processed food is defined as anything that has been changed in some way before consumption (Wolfram, 2016). This includes everything from pre-sliced apples, to canned tuna, to candy, to frozen pizza, to cereal. There are some processed foods that are harmless, like fruits, veggies and tuna. The problem lies within the chips, soda, pizza rolls, ice cream, candy, etc. These foods are dangerously high in sodium, sugar, fat, GMO's, chemical additives, and calories, all of which cause detrimental health problems. Since processed foods contain high levels of sodium, sugar, and fat, they are causing an increased rate of heart disease around the world. To combat this problem, the government could place a tax on sugar content in processed foods so the food companies will have a harder time accessing these harmful ingredients.

High Blood Pressure Caused by Sodium

According to the American Heart Association, one of the leading causes of heart disease is high blood pressure, which directly relates to sodium intake. The American Heart Association recommends a daily sodium intake of 1,500 mg, but the average American consumes around 3,400 mg. Over 75% of the sodium intake comes from salt that is added to processed food (Heart, 2014). People do not realize how much sodium there is hidden in food. Even if a label says "low sodium," the product may not truly contain a low level of sodium. Some foods like cereal and pastries that taste sweet, not salty, are some of the foods that contain the most sodium. Eating foods and not really knowing how much sodium it contains is one of the biggest reasons the sodium intake is dangerously high among Americans as well as other countries becoming more westernized (American Heart Association, 2017). Sodium plays a significant role in heart disease that the World Heart Federation has estimated that a universal decrease in sodium intake by just 1 gram would lead to a 50% decrease in the rate of heart disease, a 22% decrease in deaths caused by strokes, and a 16% decrease in the deaths

caused by coronary artery disease (World Heart Federation, 2017).

High Cholesterol Caused by Fat

Another ingredient found abundantly in processed food is fat, which causes high cholesterol, which causes heart disease. There are two types of fats commonly found in processed food: saturated fats, which are found in animal products, such as cheese and meat, and trans fats, which are found in bread products such as cakes, cookies, fast food, biscuits, etc (World Heart Federation, 2017). There are some types of fat that are beneficial to the body. Unsaturated, polyunsaturated, and monounsaturated fats are good for the heart and they are found in natural foods such as fish, nuts, seeds, and vegetables (World Heart Federation, 2017). However, if more than 37% of a person's total calorie intake consists of fat, whether it is good or bad fat, that person increases his or her risk of heart disease (World Heart Federation, 2017). If 70% of the average American's total calorie intake comes from processed food, it does not take much to exceed 37% fat in one day, because processed food is so full of saturated fat (World Heart Federation, 2017). Therefore, the AHA has concluded that eating so much processed food is increasing the risk of heart disease by causing abnormally high cholesterol.

Sugar

Sugar is widely considered the most unhealthy component of processed food. The effect that sugar has on the heart is one of the worst and most effective. Julie Corliss wrote in the Harvard Health Blog about a 15-year study that was conducted to prove that people who eat more added sugar than usual are twice as likely to die from heart disease (Corliss, 2016). In her blog, Corliss explains that the risk of heart disease that comes from eating sugar is real and inevitable, regardless of a person's age, sex, physical activity level, and weight. This means that a person does not have to be overweight or obese for sugar to hurt his or her heart. Even if a person exercises every single day, is in great shape, and eats mostly healthy food, heart disease is still a risk if he or she consumes more than the recommended amount of daily sugar (Corliss, 2016). The recommended amount of daily sugar for women is 6 teaspoons and for men 9 teaspoons (Corliss, 2016). To put that into perspective, a 6 oz can of soda contains 9 teaspoons. So, if one can of soda is already exceeding the recommended amount for women, it can be implied that it is very common for people to consume way more than the recommended daily amount. The most common sources of added sugar are drinks such as soda, energy drinks (gatorade, monster, red bull), and syrupy drinks (kool-aid, sweet tea, lemonade, juice) (O'Leary, 2017). Other common sources are pretty much any desert you can think of, including fruit. It is not able to be pinpointed exactly how sugar harms the heart. However, it has been proven that sugar raises blood pressure and blood sugar which stimulates the liver to dump more harmful fats into the bloodstream (Corliss, 2016).

There is a common misconception that the only thing sugar does to the body is cause weight gain due to it being "empty calories." People think that by eating excessive amounts of sugar, they will gain weight while simply gaining no nutrition. However, this is untrue. Sugar is not just "empty calories." It is a toxin, and instead of just emptily causing weight gain, it actually does a lot of harm (O'Leary, 2017). The reason that sugar is one of the most toxic substances to consume is because humans are biologically drawn to it, which makes it highly addictive, which makes it more likely to consume such high amounts (DiNicolantonio, 2015). According to Dr. James DiNicolantonio, sugar has the same effect on the brain as cocaine. Cocaine is a white crystal extracted from a coca leaf, while sugar is a white crystal extracted from sugar cane, but according to Dr. DiNicolantonio's study, sugar is even more addictive. Once a specific amount of sugar has been consumed, it alters the neurochemistry of the brain which causes it to become addictive. Sugar causes people to experience dopamine depletion and even withdrawals, which is why people crave sugar so much and eat it in excess amounts (DiNicolantonio, 2015). When people ingest sugar, there is a release of dopamine in the brain, and when people start eating too much sugar, over time, the dopamine receptors stop working as well, they do not regulate the chemical release, and they become less responsive (DiNicolantonio, 2015). So, since sugar has such a drastic effect on the brain, it is one of the most dangerous things to consume because it hurts the heart when eaten in excessive amounts.

Sugar causes insulin resistance, which causes diabetes; it causes hypertension; it causes obesity. All of these things are common causes of heart disease. According to Jared O'Leary's presentation at Vanderbilt, people who are diabetic have consumed so much sugar that their bodies have lost the ability to produce insulin. Insulin is an important hormone that breaks down sugar in the body and keeps blood sugar levels normal (O'Leary, 2017). Eating excess sugar alters insulin production which is how sugar causes diabetes. Excess sugar also causes hypertension, which leads to diabetes, obesity, and heart disease. Obesity is a specific problem that is a result of eating too much sugary, processed food (O'Leary, 2017).

Obesity

World hunger has always been a global issue, but in recent decades, the number of underfed people is equal to the amount of obese people, making obesity a global disease (Shah, 2010). Even though obesity is a bigger problem in America than in other countries, there are still a lot of other countries that struggle with the disease. Anup Shah has found that as of 1990, obesity is occupying more than 12% of the national health care budget. Even with all the money being wasted on obesity, it is being spent in non-productive ways. It is going towards surgical procedures, outrageous and unsafe diets, and food advertising (Shah, 2010). There are 400,000 liposuction operations every year, and as a result of obesity, $14 billion worth of expenses due to treating heart disease (Shah, 2010).

Obesity is becoming more and more of a problem every year and it is causing heart disease and death. The amount of processed food that America consumes is outrageous. Heart disease has always been one of the most natural causes of death because when people die of old age, their heart is usually failing. However, heart disease has increased around the world and it is now taking the lives of young people who have unhealthy diets. The amount of victims of all ages and all around the world is increasing because people hurt their hearts by eating too much sugar, sodium, and fat in processed foods.

Conclusion

Heart Disease is related to diet. The average American diet consists of so much processed food that it causes obesity, diabetes, and hypertension which all lead to heart failure. The sugar, fat, and sodium content found in processed food is the root of the problem. The best solution to the problem is to make it harder for food companies to access these ingredients.

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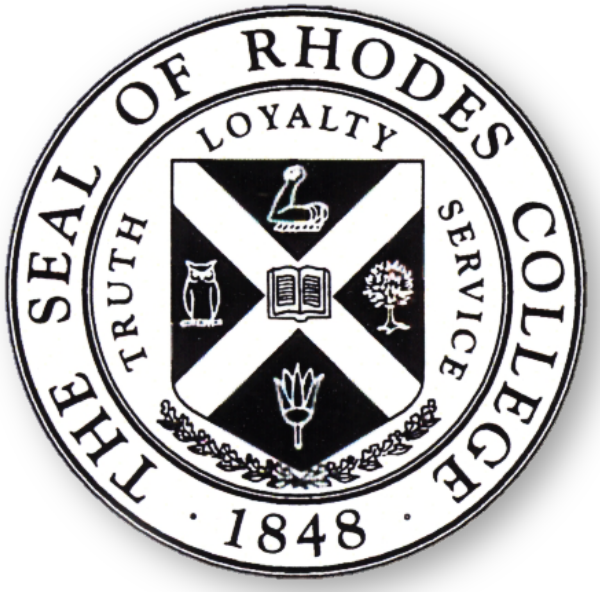

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