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A MODELING PERSPECTIVE OF DELIBERATE SELF-HARM

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ABSTRACT. Deliberate self-harm (DSH) is the act of deliberately harming your own body, such as cutting or burning yourself, without suicidal intent. It has especially become a problem among adolescents and college-age students in institutional settings such as boarding schools, Greek houses, detention centers and hospitals. We focus on contagion of DSH among adolescents and young adults by creating a deterministic epidemiological model. We study the impact of actual peer pressure, virtual peer pressure (the Internet) and treatment analytically in terms of a basic reproduction number through stability analysis of a system of ordinary differential equations. All parameters are approximated and results are also explored by simulations. The model shows that DSH is present in an endemic state in the population considered, and the control strategies are discussed.

1. INTRODUCTION

Deliberate self-harm (DSH), sometimes called self-injury(SI) or self-mutilation, typically refers to a variety of behaviors in which an individual purposefully inflicts harm to his or her body for purposes not socially recognized or sanctioned and without suicidal intent [1][7]. Self-injurious acts include cutting and carving of the skin and subdermal tissue, biting and skin burning among other behaviors [9][15]. We omit mental SI and minor self-injurious acts such as hitting a wall or biting nails in our study. DSH occurs as a maladaptive way of coping with intense feelings of anxiety and depression[2]. Experts say that individuals typically engage in self-injurious behaviors (SIB) in adolescence and early adulthood, with estimates of the first self-injurious episodes ranging from age 13 to 23 [12] and that the behavior can continue with increasing severity into one's late twenties and much longer without appropriate treatment. Self-injurious young adults who have difficulties expressing emotional states or with feeling neglected use DSH as a coping mechanism to relieve their emotional pain. The adolescents

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who want to fit in desperately with their peers may view DSH as a fad and use it to identify themselves. Yates[23] states that the lifetime prevalence of impulsive SIB in the general population is approximately 10-15%, with about 5-10% of individuals engaging in repeated episodes of DSH. However, the incidence of SIB rises to 40% to 61% in adolescent inpatient settings[3][4].

The epidemic-like patterns of SI in institutional settings such as hospitals and detention facilities were found by Matthews[13], Taiminen et. al.[17], and Rosen & Walsh [14] among others. In [17] some patients said that SI did not relieve their anger or anxiety at all, but they were involved in DSH mainly because they did not want to feel themselves outsiders. Rosen and Walsh[14] conclude that contagious self-mutilation may be viewed as a concrete display of affinity between two people and Walsh[19] reports a fad quality to the behavior in school settings. Matthews[13] discusses DSH in a unit for emotionally disturbed adolescents and reads, "One female member of the staff felt much attracted to the behavior and wanted to try it."

The Associated Press in Chicago[18] reported, "Nearly 1 in 5 students at two Ivy League schools say they have purposely injured themselves by cutting, burning or other methods." For adolescents and young adults, both Ross and Heath[16] and Favazza[8] obtained a similar overall prevalence rate of about 14%. The rapid spread and increase of SI in non-clinical populations has made many researchers wonder if there is a wider contagious effect through the Internet. Whitlock et. al.[20] identified more than 400 self-injury message boards, most of which were populated by people of ages between 12 and 20. They observed: online interactions provide essential support for otherwise isolated adolescents; they also expose vulnerable adolescents to a subculture in which SI is normalized and encouraged; lethal behaviors of DSH are shared online; easy access to a virtual subculture of like-minded others may reinforce the behavior for a much larger number.

SI is often associated with abuse, peer or family conflict, losses and social isolation among others. The majority of individuals who cut themselves replace their emotional pain temporarily by the physical pain. Therefore, the treatment for cutters is most often a combination of cognitive behavioral therapy, group or family therapy, and medications, which improve verbal competence and self-esteem, and reduce social isolation. The awareness of DSH acts among school counselors is crucial in preventing teens from being addicted to SI. Experts say that when persons injure themselves, endorphins are released in the body and function as natural pain killers and the SIB is associated with positive feelings, which is addictive. The progress in the reduction of cutting behavior is slow once addicted. Treatment varies and most often includes cognitive behavioral therapy although hospitalization is required in severe cases.

In this study, we focus on contagion of DSH in institutional settings of adolescents and young adults, and assume that the onset and progressing to addiction is through actual peer pressure and the virtual pressure (mainly the Internet) during the course of DSH. We take a mathematical modeling approach to the epidemiological patterns in DSH and construct an epidemiological model to examine the dynamics of DSH in institutional settings. We wish to discuss the impact of actual peer pressure and virtual peer pressure (the Internet) and treatment analytically through stability analysis of the system of ordinary differential equations.

We identify a threshold condition that describes different progresses of DSH by finding the basic reproductive number and using it to discuss the control strategies. All parameters are approximated to apply sensitivity analysis and our results are exploited through deterministic simulations.

2. MODEL

Our model consists of four classes: the susceptible class S, the addicted class A, the class in a treatment program P and the recovered class R. Individuals of S who try a self-injurious act move from S to A, the repeat self-injurious individuals stay in A, but individuals who do not want to harm themselves again move from A to R. When repeat self-injurers or one time self-injurers seek a treatment, they transit from A to P. Individuals in P may relapse back to A, but members of R do not fall into A. The total population is $N = S + A + P + R$. Note that this model has two types of infected classes, A and P. The model focuses on individuals of ages between 12 and 23 in an institutional setting such as boarding schools, residential colleges, Greek houses, juvenile detention centers, hospitals, or community treatment centers among others. Individuals move among the classes by a number of different processes. Since the self-injurers do not intend to suicide, we assume no death from DSH although some accidental visits to an emergency room occur. All rates are per capita rate. The parameter μ is the rate at which individuals enter and leave the population. α is the peer-driven rate of S into A by individuals in A and P, and β is the peer pressure rate of A to P by individuals in P and R. The parameters α and β may not be the same for A and P and for R and P, respectively, but using the same value is a good approximation in homogeneous mixing for our purposes. The linear term θ is the rate at which individuals in A seek treatment or the intervention rate. η denotes the rate at which individuals who tried SI only once or individuals who stopped without a treatment program transit to R from A. The positive influence of the Internet increases the values of θ and η, but the negative effect of the virtual pressure such as sharing the techniques or justifying self-injurious acts makes individuals in A stay longer in A and decreases θ and η. The recovery rate from the treatment program is ρ and the relapse rate is ω. The following figure summarizes the model in schematic form.

Under these assumptions the governing compartmental model of the model is the following system.

$$\begin{aligned}
\frac{dS}{dt} &= \mu N - \alpha\frac{S}{N}(A+P) - \mu S \\
\frac{dA}{dt} &= \alpha\frac{S}{N}(A+P) + \omega P - \beta\frac{A}{N}(P+R) - \theta A - \eta A - \mu A \\
\frac{dP}{dt} &= \beta\frac{A}{N}(P+R) + \theta A - \rho P - \omega P - \mu P \\
\frac{dR}{dt} &= \eta A + \rho P - \mu R \\
N &= S + A + P + R
\end{aligned} \tag{2.1}$$

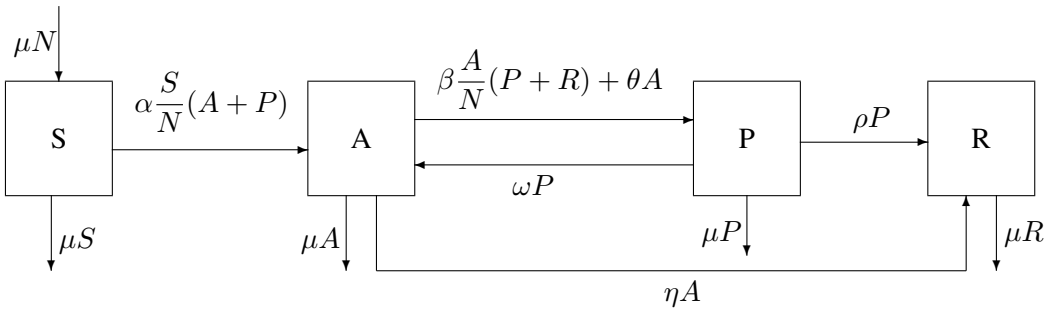

FIGURE 1. A schematic diagram of the model

3. ANALYSIS OF THE MODEL

Let $s = S/N,\ a = A/N,\ p = P/N$ and $r = R/N$ to define our variables as proportions of the entire population. The last equation of the system (2.1) provides $s = 1 - a - p - r$ and we obtain a rescaled system of ordinary differential equations.

$$a' = \alpha(1-a-p-r)(a+p) + \omega p - \beta a(p+r) - (\theta+\eta+\mu)a \tag{3.1}$$

$$p' = \beta a(p+r) + \theta a - (\rho+\omega+\mu)p \tag{3.2}$$

$$r' = \eta a + \rho p - \mu r \tag{3.3}$$

One possible end state is the DSH free equilibrium $(s, a, p, r) = (1, 0, 0, 0)$. We proceed to calculate R_0, the basic reproductive number of DSH, which is the average number of secondary cases produced by a single typical infection in the population near the disease free equilibrium in an epidemiological model. Since our model has two types of infected individuals, we use the next generation matrix method [5][6]. We solve for the equilibrium value r^* in Eq. (3.3), substitute it in Eq.s (3.1) and (3.2), and separate the new infections from the rest. Let f denote the new infection vector and w denote the vector of the negative of the remaining terms. Then the Jacobians of f and w evaluated at the DSH free equilibrium are

$$F = \begin{bmatrix} \alpha & \alpha+\omega \\ \theta & 0 \end{bmatrix} \text{ and } W = \begin{bmatrix} \theta+\eta+\mu & 0 \\ 0 & \rho+\omega+\mu \end{bmatrix}$$

and the next generation matrix G is

$$G = FW^{-1} = \begin{bmatrix} \dfrac{\alpha}{\theta+\eta+\mu} & \dfrac{\alpha+\omega}{\rho+\omega+\mu} \\ \dfrac{\theta}{\theta+\eta+\mu} & 0 \end{bmatrix} = \begin{bmatrix} g_{aa} & g_{ap} \\ g_{pa} & g_{pp} \end{bmatrix}.$$

Note that the ijth element of G, g_{ij}, is the expected number of secondary infections of type i caused by a single infected individual of type j. For example, g_{ap} is the expected number of

secondary infections of type A caused by a single individual in the P. The spectral radius of G is the basic reproductive number.

Result 1. *The basic reproductive number R_0 is*

$$R_0 = \frac{1}{2}\Big[\frac{\alpha}{\theta+\eta+\mu} + \sqrt{\Big(\frac{\alpha}{\theta+\eta+\mu}\Big)^2 + \frac{4(\alpha+\omega)\theta}{(\theta+\eta+\mu)(\rho+\omega+\mu)}}\Big].$$

Interpreting R_0 is complex since infected individuals have to go through class P before they recover. We apply the Minkowski inequality[10] to simplify R_0 algebraically. Then,

$$R_0 \leq \frac{\alpha}{\theta+\eta+\mu} + \sqrt{\frac{(\alpha+\omega)\theta}{(\theta+\eta+\mu)(\rho+\omega+\mu)}} \tag{3.4}$$

The first term of the inequality (3.4), $\dfrac{\alpha}{\theta+\eta+\mu}$, is the mean number of susceptibles infected by a typical individual of class A. $\dfrac{\omega}{\theta+\eta+\mu}$ is the average number of susceptibles that a relapsed (two staged) individual of A would infect. Hence, the sum of these two terms, $\dfrac{\alpha+\omega}{(\theta+\eta+\mu)}$, is the mean number of secondary cases infected by a single individual of A in the population close to the disease free equilibrium. The expression under the radical describes the mean number of susceptibles infected by an individual of A and progressed to class P. Note the multiplication by $\dfrac{\theta}{\rho+\omega+\mu}$, which is the proportion of addicted self injurers who enter treatment. The radical is part of R_0 because of two stages (generations) of the infected before recovery. That was why we used the generation matrix method to obtain R_0. Considering the overlapping individuals counted in both $\dfrac{\alpha}{\theta+\eta+\mu}$ and $\sqrt{\dfrac{(\alpha+\omega)\theta}{(\theta+\eta+\mu)(\rho+\omega+\mu)}}$, R_0 has to be less than or at most the sum of the two terms. Hence, if $R_0 < 1$, the disease dies out. We prove it analytically in the following.

We note that R_0 is independent of the peer-driven treatment rate, β. This is because R_0 measures the response to an initial individual of A in the population close to the disease free equilibrium, but β depends on peers in P and R. The role of β when P and R are populated is discussed below and summarized in Result 4.

In order to study the stability of the system at DEF we find the Jacobian of Eq.s (3.1)-(3.3) evaluated at the DFE,

$$J = \begin{bmatrix} \alpha-(\theta+\eta+\mu) & \alpha+\omega & 0 \\ \theta & -(\rho+\omega+\mu) & 0 \\ \eta & \rho & -\mu \end{bmatrix}.$$

Let M and N denote $\theta+\eta+\mu$ and $\rho+\omega+\mu$, respectively. One eigenvalue of the associated characteristic equation is $-\mu$ and the other two are

$$\Big(-(M-\alpha+N)\pm\sqrt{(M-\alpha+N)^2-4[(M-\alpha)N-(\alpha+\omega)\theta]}\Big)/2.$$

The expression under the radical is positive since it can be rewritten as $(M-\alpha-N)^2+4(\alpha+\omega)\theta$. Considering the model, $M-\alpha+N$ is positive. Note that

$$R_0<1 \Longleftrightarrow (M-\alpha)N-(\alpha+\omega)\theta>0. \tag{3.5}$$

Therefore, if $R_0<1$ then all eigenvalues are negative.

Result 2. *If $R_0<1$, the DSH free equilibrium is locally stable.*

For an endemic equilibrium, we solve Eq. (3.3) for the equilibrium value $r^*=\dfrac{\eta a^*+\rho p^*}{\mu}$, substitute it in Eq. (3.2), and solve for the equilibrium value p^* in terms of a^*,

$$p^*=\frac{a^*(\beta a^*\eta+\theta\mu)}{-\beta a^*\mu-\beta a^*\rho+N\mu}.$$

In order for $p^*>0$, $-\beta a^*\mu-\beta a^*\rho+N\mu>0$ is necessary, which provides

$$a^*<\frac{N\mu}{\beta(\mu+\rho)}. \tag{3.6}$$

Finally, substitute r^* and p^* in Eq. (3.1) to solve for the equilibrium value a^*. The cubic polynomial f in the resulting function F,

$$F(a^*)=\frac{-a^*}{(-\beta\mu a^*-\beta\rho a^*+N\mu)^2}f(a^*)$$

satisfies the following:

$$f(0)=\frac{1}{N}[(M-\alpha)N-(\alpha+\omega)\theta]$$

$$f\Big(\frac{N\mu}{\beta(\mu+\rho)}\Big)=\frac{(\theta\mu+\eta N+\rho\theta)^2\alpha N\mu^2}{\beta(\mu+\rho)^2}.$$

Note that it is enough to consider $f(a^*)=0$ for endemic equilibrium points since $a^*\neq 0$ and $a^*\neq\dfrac{N\mu}{\beta(\mu+\rho)}$. The equivalent statements (3.5) imply that $f(0)<0$ if $R_0>1$. $f\Big(\dfrac{N\mu}{\beta(\mu+\rho)}\Big)$ is clearly positive. Hence, depending on the value of R_0, we have the following.

Result 3. *If $R_0>1$, there exist one or three endemic equilibrium points. If $R_0<1$, there exist none or two endemic equilibrium points.*

Since Result 3 shows that a bifurcation occurs at $R_0=1$, we find $F'(0)$ in order to determine the type of bifurcation:

$$F'(0)=\alpha\Big(1+\frac{\theta}{N}\Big)+\frac{\omega\theta}{N}-M$$

By rewriting this, we obtain $NF'(0)=-[(M-\alpha)N-(\alpha+\omega)\theta]$. Hence, if $R_0<1$ then $F'(0)<0$, and if $R_0>1$ then $F'(0)>0$, i.e., a forward bifurcation occurs when $R_0=1$.

Since R_0 is independent of β, if any endemic equilibria ever exist when $R_0 < 1$, they should depend on β. If $\beta = 0$, $f(a^*)$ is a linear function and $f(0) = \mu^2 N[(M-\alpha)N - (\alpha+\omega)\theta]$. By (3.5), $f(0) > 0$ if $R_0 < 1$. Since it is linear, it can have at most one solution. From Result 3, we conclude that there is no endemic equilibrium point if $\beta = 0$ and $R_0 < 1$. Now, considering (3.6), we see that a^* decreases as β increases, and β is bounded by a real number $\dfrac{N\mu}{a^*(\mu+\rho)}$ since a^* is an endemic equilibrium point.

Result 4. *There is a forward bifurcation at $R_0 = 1$. A backward bifurcation may occur for a positive real number β when $R_0 < 1$.*

4. Parameter Estimation and Discussion

4.1. **Parameter Estimation.** To study the contagion of DSH, Taiminen et. al.[17] observed 51 adolescents who were treated in an adolescent psychiatric ward of Turku University Central Hospital in Finland for one year and Rosen and Walsh[14] did 35 patients of ages from 15 to 21 at the Community Treatment Center in Worcester, Mass. over 10 months. Both studies showed clear patterns of contagion by including sociogram of contagious SI. However, their DSH contagion was defined as two or more acts of self-mutilation that involved two or more individuals and occurred on the same day or consecutive days. Although we cannot apply their data directly to our model, we estimate the model parameters by reinterpreting their data in combination of other data published.

We assume that the average time an adolescent spends in an institution that our model applies to is about 10 months using Walsh[14]'s. The institution may be a community treatment center or a juvenile detention facility among others. So, we take $\dfrac{1}{\mu}$ = 10 months.

According to the study by [17], 12 subjects out of 51 were engaged in SIB and one third of self-injurers started SIB after they were hospitalized. Their mean length of hospitalization was about 3 months. This provides $\alpha \approx 0.17$ month^{-1} as a ratio of $\dfrac{4}{51}/3$ months and $\dfrac{8}{51}$.

Although the time it takes for severely addicted self-injurers to complete treatments varies, we estimate that it takes about a year, which gives $\rho = 0.08$ month^{-1}.

[16] reports that 18% of Canadian high school self-injurious students had self-mutilated on only one occasion. We estimate that about 10% of self-injurers try SI only once in a cohort environment since [17] and [14] show lower percentage. The parameter η should also consider the individuals who stop DSH without a treatment program. Whitlock et. al.[21] conducted an Internet-based survey to which 2,875 undergraduate and graduate students of ages at Cornell and Princeton responded. They reported that 40% of repeat self-injurers ceased SIB within one year and 79.8% within 5 years. We will take a conservative figure for this younger cohort institution and estimate that the proportion of self-injurers who move to the R class before leaving the institution is $\eta/(\eta+\mu) \approx 0.25$ and $\eta = 0.03$ month^{-1}.

According to [11], 75% were self-injury free two years post discharge from a treatment program, so we take for the proportion of self-injurers who relapse before leaving the population $\omega/(\omega+\mu) \approx 0.15$ and $\omega = 0.018$ month^{-1}.

For β and θ, we take for the proportion of self-injurers who seek treatment before leaving the institution $\dfrac{\beta(p+r)+\theta}{\mu+\beta(p+r)+\theta} \approx 0.05$ since [21] reports that 5.4% of repeat self-injurers disclosed SIB to a physician or allied medical health professional. In the absence of accurate data, we estimate that $4\beta(p+r) \approx \theta$. To estimate a, p, and r, we first average the prevalence rates of [16] and [17], 14% and 24%, which is about 19%. Then an estimate for p is 5% of 19%, $r \approx 0.19 \times 0.2$, and $a \approx 0.18 \times 0.75$. We now obtain $\beta = 0.024\ \text{month}^{-1}$ and $\theta = 0.0042\ \text{month}^{-1}$.

4.2. **Computer Simulation.** Using the parameter values we estimated in the previous section, we obtain $R_0 \approx 1.29 > 1$. This result is consistent with the observation of Taiminen et. al.[17] in the sense that DSH is present in the institution, but the endemic equilibrium value a^* is much less than 12/51 as Figure 2 indicates. This is due to the use of other data sets in addition to the data of [17], which was an institutional setting and did not have the rate for entering a treatment program or recovery rate among others. Since the stability of an endemic equilibrium point was too complex to show analytically, we have simulated the model multiple times by varying parameter values and initial values to see the stability. Both Figure 2 and Figure 3 show that DSH endemic equilibrium point is locally asymptotically stable.

Phase Portrait (a, r)

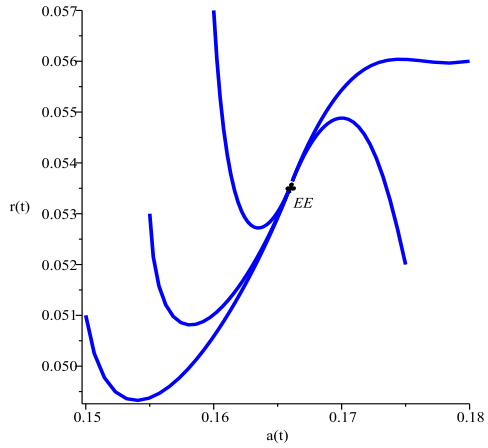

FIGURE 2. with $\alpha = 0.17$ and $\eta = 0.03$

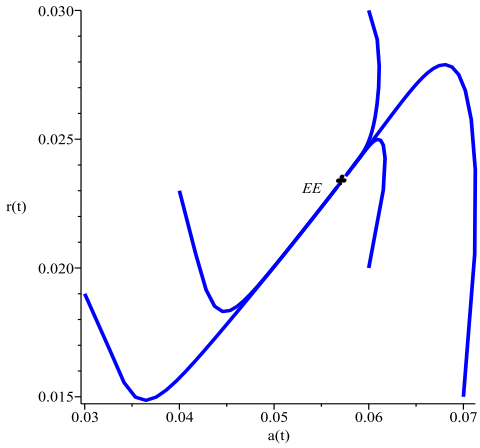

FIGURE 3. With $\alpha = 0.153$ and $\eta = 0.039$

4.3. **Sensitivity Analysis and Control Strategies.** The threshold condition R_0 in our model is often described as a tipping point. It is a point at which a stable system turns to an unstable one or vise versa. Hence, we find the sensitivity indices of R_0 for all parameters in order to determine parameters to which our system is most sensitive. To see how a small perturbation

made to a parameter q affects a threshold condition R, we define the sensitivity index of R for q as

$$S_q = \frac{\partial R}{\partial q}\frac{q}{R}.$$

We apply the same set of parameter values (obtained in Section 4.1) and find that the system is most sensitive to α over all, however, to η among the parameters that can be influenced by intervention strategies.

Although controlling α is hard, especially when DSH is motivated by desiring for group cohesiveness, some suggestions given by experts are as follows: target specific dyads or small groups rather than the whole milieu[14]; identify the primary status peer models and ask not to appear in school with visible wounds or scars (individuals, in some cases, may have to be dealt with in a disciplinary manner) [19]; clarify the possible contagious aspects of DSH directly because most adolescents avoid behavior that they themselves perceive as imitative[17].

Targeting η means minimizing the time that individuals of the A class stay in A. To transit individuals who have tried DSH to the R class before they become addicted to SI, experts suggest the following among others: teaching stress management techniques that may help the adolescents and young adults deal with feelings of tension and anxiety [16]; routine questions on DSH should be asked by school counselors [22]; encourage self-injurers to communicate with family and institution supporters[19]; educate residence assistants and counselors on DSH so that they can manage their personal reactions toward SIB [22] (any reaction with horror or helplessness isolates self-injurers since they already associate SIB with shame). Making medical information on the danger of addiction to DSH online widely as a public education campaign will be the positive effect of the Internet. Monitoring message boards actively and setting up a firewall to block the web sites that encourage and justify SIB will also increase the rate η.

Once the behavior starts, the endorphins released by SIB can become quite addictive. Like addiction to drugs, as tolerance builds up, self-injurers need increasing amount of DSH to achieve the same effect. It is well known that the rates of detection and treatment of SIB are remarkably low, so increasing θ is extremely hard. Mathematically, even if we increase θ to 0.03 (=ρ), $|S_\theta|$ is only one third of $|S_\eta|$. However, when we decrease α by 10% and increase η by 30%, the endemic equilibrium value a^* is reduced by about 65% (compare Figure 3 to Figure 2). In conclusion, the best strategies should be prevention and early intervention, which correspond to decreasing α and increasing η, respectively.

REFERENCES

[1] Alderman, T. (1997). The Scarred Soul: Understanding and Ending Self-inflicted Violence. Oakland, CA: New Harbinger.

[2] Carll, E. K. (2003). Self-injury behavior: Emerging trends. Bulletin of the Psychologists in Independent Practice, 23 (3).

[3] Darche, M. A. (1990). Psychological factors differntiating self-mutilating and non-self-mutilating adolescent inpatient females. Psychiatric Hospital, 21(1), 31-35

[4] DiClente, R. J., Ponton, L. E., & Hartley, D. (1991). Prevalence and correlates of cutting behavior: Risk for HIV transmission. Journal of the American Academy of Child and Adolescent Psychiatry, 30, 735-738

[5] Van den Driessche, P., Watmough, J. (2002). Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease tranmission. Mathematical Biosciences 180, 29-48
[6] Diekmann, O., Heesterbeek, J. A. P., & Metz, J. A. J. (1990). On the definition and the computation of the basic reproduction ration R_0 in models from infectious diseases in heterogeneous population. Journal of Mathematical Biology, 28, 365-382
[7] Favazza, A. R. (1996). Bodies Under Siege: Self Mutilation and Body Modification in Culture and Psychiatry (2 ed.). Baltimore, MD: Johns Hopkins University Press.
[8] Favazza, A. R. (1989). Why patients self-mutilate. Hosp. Comm. Pyschiatry, 40, 137-145.
[9] Gratz, K. L. (2001). Measurement of Deliberate Self-Harm: Preliminary Data on the Deliberate Self-Harlm Inventory. Journal of Psychopathology and Behavioral Assessment, 23(4), 253-263.
[10] Folland, Gerald B. Real Analysis: Modern Techniques and Their Applications. John Wiley & Sons, Inc.
[11] http://www.selfinjury.com/resources_faq.html S.A.F.E. Alternatives
[12] LLOYD-RICHARDSON, E., PERRINE, N., DIERKER, L. & KELLEY, M. (2007) Characteristics and functions of non-suicidal self-injury in a community sample of adolescents. Psychological Medicine, 37, 1183-1192
[13] Matthews, P.C. (1968). Epidemic self-injury in an adolescent unit. Int J Soc Psychiatry, 14, 125-133
[14] Rosen P. M. , Walsh B. W. (1989). Patterns of contagion in self mutilation epidemics. Am J Psychiatry, 146(5), 656-658
[15] Ross, S. and Heath, N. L. (2003). Two Models of Adolescent Self-Mutilation. Suicide & Life-Threatenig Behavior, 33(3), 277-287
[16] Ross, S. and Heath, R. (2002). A Study of the Frequency of Self-Mutilation in a Community Sample of Adolescents. Journal of Youth and Adolescence, 31(1), 67-77
[17] Taiminen TJ, Kallio-Soukainen K, Nokso-Kovisto H, Kaljonen A, & Helenius H. (1998). Contagion of deleberate self-harm among adolescent inpatients. J Am Acd Child Adolesc Psychiatry, 37(2), 211-217
[18] Tanner, Lindsey, AP Medical Writer, Associated Press in Chicago, June 5, 2006
[19] Walsh, B. W. (2005). Treating Self-Injury: A Practical Guide. New York, NY: Guilford Press
[20] Whitlock, J. L., Powers, J. L. & Eckenrode, J. (2006). The Virtual Cutting Edge: The Internet and Adolescent Self-Injury. Developmental Psychology, 42(3), 407-417
[21] Whitlock, J., Eckenrode, J., & Silverman D. (2006). Self-Injurious Behaviors in a College Population. Pediatrics, 117(6), 1939-1948
[22] White, V., Trepal-Wollenzier, H., & Nolan, J.M. (2002). College Students and Self-Injury: Intervention Strategies for Counselors. Journal of College Counseling, 5, 105-113
[23] Yates, T. M. (2004). The developmental psychopathology of self-injurious behavior: Compensatory regulation in posttraumatic adaptation. Clinical Psychology Review, 24, 35-74