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Nonlinear Dynamics and Probabilistic Behavior in Medicine: A Case Study

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1. Introduction

Nonlinearity is ubiquitous in medicine and life sciences, from the molecular and cellular to the organismic and population levels, owing to the presence of a variety of interactions, feedbacks and other kinds of regulatory processes that ensure the harmonious coexistence of the multitude of simultaneously ongoing activities (Mosekilde, 1996).

Nonlinearities arising from the cooperative interactions between the subunits constituting a system in conjunction with appropriate environmental stimuli, give often rise to collective behaviors transcending the individual subunits. A striking example of such collective behavior is contagion, be it in the form of propagation of a disease, of a rumor or on a more microscopic scale of a mutation, whereby a previously unaffected unit becomes affected in its turn following an encounter with the information-carrying unit. In this chapter we will be concerned with a particularly dramatic instance of contagion arising in the context of adolescent psychiatry, namely, adolescent suicidal outbreaks.

Suicidal trends rank among the most serious disorders of adolescence. In most countries, mortality from suicide is the second or the third leading cause (depending on the surveys) of teenage deaths. The incidence of suicide attempts peaks during mid adolescence (Becker, Schmidt, 2004). It is estimated that 20% of adolescents have suicidal thoughts and among them as much as 5 to 8% have attempted to commit the act (Pommereau, 2001). Each of these suicidal acts leaves behind surviving family members, friends and acquaintances who must cope with the loss (Bridge et al, 2003).

A number of risk factors for adolescent suicidality have been identified. Among these the most important are depression and exposure to suicide, suicide attempts or suicidal thoughts by family and friends, suggesting that the adolescent can be considered at potential risk of contagion with suicidality stimulations. Here, suicide contagion refers to the link between adolescent's exposure to a suicide stimulus and subsequent rise in the frequency of suicide attempts or suicide rate and is considered most likely to occur in already suicidal adolescent and to be a time-limited risk. In this respect, it appears reasonable to view a suicidal trend as a behavioral attribute. If so, suicide contagion could be regarded as a particular manifestation of behavioral contagion whereby, much like in an infectious disease, an attitude or a mood passes from a person to the next. Jones and Jones (1994) provided statistical support of behavioral contagion in a number of situations, and

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the perspectives opened in their analysis constitute one of the principal motivations of the present work.

Generally speaking, if a behavior is contagious, its prevalence increases with the number of susceptible adolescents rather than the total number of individuals present. Wheeler (1970) identifies behavioral contagion by 4 criteria:

1. An observer is motivated to behave in a certain way;
2. The observer knows how to perform the behavior in question but is not performing it;
3. The observer sees a model perform the behavior;
4. The observer after observing the model performs the behavior.

The theory of contagion rests on three central concepts apart from contagion itself: susceptibility, mode of transmission and exposure. Susceptibility is necessary for contagious transmission.

One aspect of youth suicide of particular concern is the repeated reports of suicide outbreaks among young people. These outbreaks have been reported from as long ago as ancient Greece and from around the world. They have been called suicide clusters, a term that describes three or more suicides occurring within a defined space and time. The incidence of cluster suicides is highest among teenagers and young adults (Gould, 2001) and a growing concern has been that adolescents exposed to a peer's suicide may be at increased risk to engage in suicidal behavior (Brent et al, 1993 a, b). Many studies have also addressed the question of whether indirect exposure to suicide through media or Internet accounts contributes to subsequent suicide (Baume et al, 1997; Davidson et al, 1989).

The most common explanation for the above noted phenomena is that of imitation. This mechanism is consistent with reported epidemics of suicide involving unusual methods such as immolation etc...Imitation is also consistent with the short latency between publicity and the increased rate of suicide within 1 to 2 weeks. According to McKenzie et al (2005) there is indirect evidence that imitative suicide occurs among people with mental illnesses and may account for about 10% of suicides by current and recent patients.

One could argue that individuals are influenced in their suicidal thoughts mainly through their direct exposure to an actual suicidal attempt. If so, suicidal trend would be a spontaneous process occurring at a rate equal to the size of the population of concerned individuals multiplied by proportionality constant whose value depends on the exposure in question. In this context, Joiner (1999) wonders if the pernicious agent of the hypothetical contagion in suicide exists. He insists on the important role of exposure, external influence rather than contagion and suggests that the concept of imitation may be not needed. He emphasizes that the vulnerable people may become socially contagious via assortative relating and thus simultaneously susceptible to the effects of life stress. Other studies report that the predominant psychiatric sequelae observed in adolescents exposed to violent deaths are anxiety, depression and post traumatic stress disorder. It has been suggested that the degree to which the second person identifies with or feels similar to the deceased person may influence the degree to which he is affected by this exposure.

While these mechanisms are undoubtedly operating in a number of circumstances of interest, our main thesis here is that they cannot account properly for suicidal outbreaks, as they lack the necessary ingredient of feedback. The alternative we thus propose is that of cooperativity, when a population of susceptible individuals is mixed with a population of suicidal ones. The nature of the suicidal attempt is in this perspective completely different, as it now depends on the size of two subpopulations in close interaction. This double

dependence calls for a nonlinear approach to the problem and opens the way to self-acceleration, abrupt transitions and other analogous behaviors concomitant to the well-established syndrome of outbreaks.

In this chapter, the propagation of suicidal trends is viewed as the result of encounters in the course of which a susceptible individual can change its mental state with some probability following interaction with a suicidal one. The encounters can be short ranged like e.g. a physical encounter in a hospital unit and a school class, or long ranged like e.g. communication through the Internet. Different contagion scenarios are explored and the main trends to be expected are identified. The results are confronted to the data available and different strategies for improving current prevention practices are suggested. Two types of methodologies are employed. In a first approach, the variability arising from the individual decision making is ignored and a mean view is adopted. This maps the problem to a problem of population growth in a medium of limited resources (here the total number of susceptible and suicidal individuals). Various growth patterns are highlighted depending on the contagion probability and the initial percentage of suicidal individuals. In a second approach, variability is incorporated by means of the technique of Monte Carlo simulation well suited to treat populations of limited size where randomness is expected to play an important role. This approach has been used with success in several problems arising in chemical kinetics, biochemistry and social insect behavior (Gillespie, 1992). A number of different evolution scenarios are explored and some unexpected effects are brought out. The novelty here is to give access to situations limited in space and time like e. g. those arising in a given hospital unit over the usual hospitalization time, as opposed for those accounted for in surveys where local and short scale trends are smeared out.

2. General setting

Let X_1 , X_2 be the populations of suicidal and of susceptible individuals respectively. In order to bring out the role of nonlinearity and cooperativity in a clearcut manner, it is stipulated that during the phenomenon of interest there is no major reshuffling of the organization, entailing that the total population remains essentially constant:

$$X_1 + X_2 = N = \text{constant} \quad (1)$$

In addition to the above two types of individuals, a third type may also be present. In what follows its role is viewed as that of a buffer, in the sense that while it does not participate directly in the dynamics, it may play a role in determining the values of some of the parameters present.

A first instance (hereafter referred as case I) explored in the sequel is that of contagion arising through direct, physical encounters of type 1 and type 2 individuals, hereafter denoted as S_1 and S_2 which are schematized as follows:

The first two steps correspond to the obvious idea that encounters of individuals of the same kind do not give rise to a mental transition. On the contrary, the last two steps account for

imitation and thus cooperativity: upon encountering a susceptible individual, a suicidal one can either switch to the susceptible state with a certain probability p_2 or induce a suicidal trend to the susceptible partner with another probability p_1 . The corresponding probabilities p_1 and p_2 are expected to fulfill the inequality $p_1 > p_2$. Although there seems to be no direct statistical evidence in support of this, we argue that in the absence of medical treatment such a property reflects the well-established tendency of susceptible and suicidal individuals to evolve “uphill” in the search of increasingly dramatic experiences rather than to evacuate stress and evolve to the opposite way toward normality. We refer to Pommereau (2001) for the definition of susceptible individuals. In fact adolescents with mental dysfunction express affective immaturity, sensibility to frustrations, massive dependence to genitors, depressivity of the mood without depressive episode and tendency to acting out. These susceptible adolescents refer to the most deviant repairs including suicidality. We emphasize that p_1 , p_2 are intrinsic parameters associated to individual 1-2 encounters, independent of the respective sizes of the populations X_1 and X_2 . Depending on the latter, the overall process of contagion will of course become accentuated, as seen below. It should also be noticed that in writing scheme (2), we tacitly assumed that individuals of the type 1 and 2 can only exist in a single state. In a more refined analysis one could account for further differentiation within a single subpopulation, like e.g. different degrees of susceptibility in individuals of type 2. Other refinements would be to account for memory effects and for changes in the parameters N , p_1 , p_2 arising for instance from medical care, environmental stimuli or population renewal. Such extensions are likely to be important on a long time scale. They are not carried out here, as our main purpose is to identify the role of nonlinearity and cooperativity in the outbreak of suicidal attempts, a phenomenon expected to be initiated in the short to intermediate time regime.

A second instance of interest (hereafter referred as case II) pertains to contagion through long range interactions. To account for this possibility, we imagine that individuals constitute the nodes of a network and the interactions between any two individuals give rise to a connection between the corresponding nodes. In the previously presented case I, only nearest neighbor nodes are connected (e.g. 1-2, 2-3 etc.). In the other extreme each node is connected to any other node (e.g. 1-2, 1-3, 1-4..., 2-3, 2-4,..., etc...). This corresponds to the longest possible range that interactions can achieve. Intermediate cases may also be envisaged. We emphasize that the model as defined above is in many respects generic. It should thus apply suitably adapted to other types of behavioral contagion beyond the suicidal one that constitutes the main focus of the present work.

We are now in the position to formulate the evolution of the subpopulations X_1 and X_2 in a quantitative manner. Two complementary points of view are adopted for this purpose, as specified below. The results to be reported depend crucially on the values of the contagion probabilities p_1 and p_2 . These quantities or, more to the point, their difference $p_1 - p_2$ determine the time scale over which the suicidal trend will spread. In view of the scarcity of relevant data, different values will be considered and the sensitivity of the results on the choices will be assessed. Another important parameter, responsible for the sharpness of contagion and for the importance of stochastic effects, is the total number N of the individuals in the group and the initial numbers $X_1(0)$ of suicidal ones. In the following a sensitivity analysis with respect to these parameters will be carried out and some robust trends will be identified. The following possibilities will be considered.

1. All individuals $N-X_1(0)$ other than the suicidal ones are likely to be affected by the contagion. This can be the case in a hospital unit or in an institution where non-suicidal patients are already subjected to psychiatric disorders.
2. Among the $X_2=N-X_1(0)$ individuals only a fraction $\gamma X_2(0)$ (γ much smaller than 1) are likely to be affected, the remaining ones being immune to any psychiatric disorders. This can correspond to a school class or to hospital unit in which the adolescent patients are treated for a completely different kind of disease.

3. Population dynamic approach: An averaged view

In this view, encompassing case I as well as case II above, it is assumed that individuals 1 and 2 are well mixed and interact at random. The strength of the interactions is proportional to the corresponding fractions $\Theta_1=X_1/N$, $\Theta_2=X_2/N$, and only encounters between 1 and 2 lead to changes in the populations of either 1 or 2. This leads us to a rate law of the form

Rate of change of 1 over a time interval

$$=p_1 \times (\text{frequency of 1-2 encounters}) - p_2 \times (\text{frequency of 1-2 encounters})$$

Taking the limit of the shortest time interval over which interactions become effective one obtains the quantitative expression

$$d\Theta_1/dt = (p_1 - p_2) \Theta_1 \Theta_2$$

or, with eq. (1)

$$d\Theta_1/dt = p \Theta_1 (1 - \Theta_1) \quad (3)$$

where we set

$$p = p_1 - p_2 \quad (4)$$

This equation is formally identical to the logistic equation (Pielou, 1969). It can be integrated exactly, yielding

$$\Theta_1(t) = \frac{\Theta_1(0)}{[1 - \Theta_1(0)] e^{-pt} + \Theta_1(0)} \quad (5)$$

which is seen to depend solely on p and on the initial fraction $\Theta_1(0)$.

The two quantitatively different evolutions predicted by this equation are depicted in Fig. 1 and 2 corresponding respectively to $\Theta_1(0)$ being greater or smaller than $1/2$. As can be seen, in the first case one witnesses a smooth evolution toward a contagion of the entire population, bound to occur on the time scale of

$$T_{\text{cont}} \sim 1/p \quad (6)$$

In the second case one observes on the contrary a first period of quiescence during which individuals 1 seem to have no contagion effect, followed by an explosive growth and eventual saturation. The explosion time, corresponding to the inflexion point of the Θ_1 versus t the curve of Fig. 2, can be evaluate explicitly and is given by

$$t^* = \frac{1}{p} \ln \left[\frac{1 - \Theta_1(0)}{\Theta_1(0)} \right] \quad (7)$$

For $\Theta_1(0)$ much smaller than unity it is therefore much longer than the contagion time associated to the case of Fig. 1. In practice, saturation and explosion may never be achieved if the corresponding times are longer than the hospitalization period. Nevertheless, the above results may provide valuable indications on the trends that may be in elaboration within the populations in interaction. They will also serve as reference for the Monte Carlo approach presented below.

Fig. 1. Time evolution of the fraction of individuals of type 1 as deduced from eq. (5) under the condition $\Theta_1(0)>1/2$. Parameter values $p=0.15$, $\Theta_1(0)=0.55$.

Fig. 2. As in Fig. 1 but with $\Theta_1(0)=0.01$.

4. Monte Carlo simulation

When dealing with complex realities one is often led to recognize that a modeling approach may be limited by the lack of detailed knowledge of the laws governing the system at hand and of the values of the parameters involved in the description. A central point of the present work is that to cope with this limitation it is important to set up a complementary approach aiming at a direct simulation of the underlying process, rather than at the solution of the evolution laws suggested by a certain model. The Monte Carlo simulation approach described below provides an efficient way to achieve this goal. It also allows one to incorporate in a natural way the role of individual variability expected to be of the utmost importance, since the quantities featured are now fluctuating in both space and time rather than being fully deterministic. Two types of studies have been conducted. In both cases, the population sizes have deliberately been taken to be small to emulate real world situations as they arise in a single hospital unit or in a school class. As it will turn out stochastic effects will then play a very important role. Still, the averaged description serves as a useful reference for apprehending the specific role of stochasticity in the overall process.

Case I

The physical space (school class, recreation area, hospital unit, space of common patient activities, ...) is modeled as a regular square planar lattice. Each individual performs a random walk between an initial position and its first neighbors. When two individuals are led to occupy through this process the same lattice site processes (2) are locally switched on. The various steps are weighted by the corresponding probabilities and the particular transition to be performed at a given time is decided by a random number generator (amounting essentially to throwing dice) compatible with these probabilities. After this particular step is performed the populations X_1 , X_2 are updated and the process is restarted. The simulation, which records the numbers of X_1 and X_2 at different parts of space, is stopped at a number of steps beyond which the process becomes stationary in the sense of reducing to fluctuations around a constant (time-independent) plateau. In addition to a single realization of the simulation (referred as "stochastic trajectory") averages over realization giving access to mean values, variances etc are also performed.

The following instances are considered.

- i. An institution or a big hospital unit with $N=30$, $X_1(0)=6$ suicidal individuals and $X_2(0)=24$ individuals presenting other kinds of psychiatric disorders. The contagion probabilities are set $p_1=0.25$, $p_2=0.1$ and the individuals are initially taken to be distributed randomly.
- ii. As before, but with $N=20$, $X_1(0)=4$ in order to test the role of population size.
- iii. A school class or a mixed hospital unit with $N=30$, $X_1(0)=2$ suicidal individuals. It is supposed that of the $N-X_1(0)=28$ individuals 4 are susceptible of being affected and the remaining 24 ones constitute the environment within which the process will take place. Accordingly, the contagion probabilities are set to lower values $p_1=0.1$, $p_2=0.05$ since the encounters are expected to be more scarce.
- iv. $N=8$ individuals of which $X_1(0)=4$ are suicidal and $N-X_1(0)=4$ subject to other types of disorder, functioning as a "clan" independent of its environment. This is accounted for by resetting p_1 , p_2 to the values of 0.25 and 0.1 respectively.
- v. As in iv. but now the two subpopulations are initially segregated (say in different hospital rooms) and meet only in common activities.

Figures 3a,b depict the time dependence of the population density X_1/N of X_1 averaged over many realizations of the process and of the associated variance $\langle \delta X_1^2 \rangle = \langle X_1^2 \rangle - \langle X_1 \rangle^2$. Figure 4 provides a reformulation of the results of Fig. 3 when all cases (i) to (v) are normalized to the same mean population. Figs 5 and 6a,b provide a more refined view of the role of inherent variability by showing respectively a single stochastic trajectory under the conditions of case (iii) and the probability histograms associated with (i) and (iii).

Case II

The physical space (e.g. Internet, a newsletter etc...) is here lumped into a single cell within which each individual may interact with any number of other ones with probabilities determined as before. Again, stochastic trajectories recording the individual transitions as well as averaged quantities over all trajectories are deduced. The context is now that of a small number of heavily affected individuals communicating via Internet, newsletter or any other kind of multimedia means with a small number of susceptible partners not attained so far by the disease. Fig. 7 summarizes the results for $N=6$, $X_1(0)=3$ using the same values for parameters p_1 and p_2 as before.

5. Discussion

Building on evidence supporting the existence of suicidal contagion, we proposed and developed a predictive model of how suicidal trends propagate in an adolescent population. The principal feature underlying the model is the cooperative character of the contagion process (last two steps in (2)). The model predictions depend entirely on two kinetic parameters, the contagion probabilities p_1 and p_2 for susceptible and for suicidal individuals to switch to the suicidal and susceptible state respectively; and on two population like parameters, the total number N of individuals that may undergo a transition in their mental state and the number $X_1(0)$ of suicidal individuals initially present.

A first result of interest has been that contagion is not always a smooth process but may rather take an explosive form, depending on the values of $X_1(0)/N$ and $p=p_1-p_2$. In this latter case there exists a well-defined time t^* of switching toward a collective suicidal state (Figs 2, 3a and 4a). This provides a quantitative basis for the phenomenon of outbreak referred in the Introduction as well as a strong support of the idea of contagion as a generic mechanism of adolescent suicidal trends. Subsequently, the population attains a mean saturation level on which is superimposed a random signal reflecting individual variability. This level may actually never be attained since on a long time scale the refinements to the original model discussed in section 2 will begin to play an increasingly crucial role.

A second series of results pertains to the role of stochasticity. The following comments are in order on inspecting the key Figure 3.

- In all cases the mean value $\langle X_1 \rangle$ is increasing in time, in qualitative agreement with Figs 1 and 2.
- The evolution is initially slower for segregated sub-populations (case (v)). What is happening here is that few among 1 and 2 types first meet in a limited space which constitutes a front of some sort, from which the trend can subsequently propagate.
- In cases (i), (ii), (iv) and (v), a saturation level in which the entire population of susceptible individuals switches to the suicidal state is eventually reached. The time scale for this to happen may be long with respect to the hospitalization or school period times. Still, the explosive growth for short times should be emphasized, confirming the prediction made in eq. (7) and Fig 2.

- The saturation level reached in case (iii) is significantly less than 100% in the same time scale as (i), (ii), (iv) and (v). This at first sight unexpected emergence of a state of undecidability is robust with respect to changes in the values of p_1 and p_2 . It arises primarily from individual variability, here exacerbated by the smallness of the size of the overall population compared to $X_1(0)$. There are long periods of hesitation and in some realizations of the process the trend is inverted and the entire population reaches the more favorable state.

Fig. 3. (a): Time dependence of the mean density of individuals of type 1 as deduced from the Monte Carlo simulation; the full, dashed, heavy dotted, dashed-dotted and light dotted lines refer to cases (i) to (v), respectively. (b) : Time dependence of the variance under the conditions of Fig 3a. The physical space considered is a square planar lattice of size 10X10 space units, the number of statistical realizations is 10,000 and the initial positions of the populations are random in space.

These trends are further illustrated in Fig. 3b where the variance $\langle \delta X_1^2 \rangle = \langle X_1^2 \rangle - \langle X_1 \rangle^2$ is represented. In all case but (iii) $\langle \delta X_1^2 \rangle$ is seen to reach a low final value, but prior to this it goes through a well - marked maximum grossly at a time corresponding to the inflexion point of the curves in Fig. 3a. As for case (iii), $\langle \delta X_1^2 \rangle$ steadily increases and reaches a final value orders of magnitude larger than for (i), (ii), (iv) and (v) which is comparable to the mean value itself. This is in agreement with and provides an explanation of the statement in Jones and Jones on the behavior of variance.

Fig. 4. (a): As in Fig. 3 but under conditions of identical overall population densities. Full, dashed and dotted lines refer to cases (i), (ii) and (iii), respectively. Initial positions and number of realizations as in Fig. 3.

Interestingly, when all cases above are normalized to the same mean population density, cases (i), (ii), (iv), and (v) are essentially reduced to a “universal” behavior both for the mean and the variance while case (iii) still constitutes a different class (Fig. 4a, 4b). This suggests that the model of eq. (3) is rather adequate for intermediate to long times as long as N is sufficiently large (which in practice could be reached already for the rather modest value of $N=8$), but even in these cases it may prove inadequate for short times and especially for times around the maximum of the variance.

Fig. 5. (a): Quasi-deterministic behavior modulated by small scale variability under the conditions of case (i). (b): Situation of undecidability induced by the individual variability in a small size population (case (iii)).

At the level of a single stochastic realization of the process (the analog of the type of evolution observed in practice) variability and undecidability are reflected by the fact that while in case (i) the switching of the population to state 1 occurs quite early in time (Fig.5a), it needs a much longer induction time under the conditions of case (iii) (Fig. 5b). We next comment on Figs 6a,b which depict the probability histograms associated with (i) and (iii) respectively. In 6a, drawn after 80 time units (the time at which the variance reaches its maximum in Fig. 3b) the histogram is clearly unimodal. It is peaked at a value corresponding

Fig. 6. Probability histograms associated with cases (i), Fig. 6a and (iii), Fig. 6b with an initial population density 0.3. Initial positions as in Fig. 3 and number of realizations is 20,000.

to the instantaneous X_1/N as deduced from Fig. 3a. For longer times the maximum slides to the right and eventually tends to 1. The structure is radically different for Fig. 6b drawn after 300 time units (the time for the value of the variance to exceed that of cases (i), (ii), (iv) and (v)) which displays a bimodal structure. As can be seen, the two peaks are located at low (close to 0) and high (close to 1) density of X_1 , reflecting the possibility of switching from individuals of type 1 to type 2 with a non-negligible probability. Clearly, this type of structure is quite different from the binomial distribution usually featured when interpreting results of surveys (Jones & Jones, 1994). This reflects the cooperative character of the contagion dynamics, an idea that has been central throughout this chapter.

Fig. 7. Time dependence of the mean density of individuals of type 1 and 2 (7a) and of the variance of individuals of type 1 (7b) in the presence of long range interactions. Number of realizations as in Fig. 3.

The results discussed so far pertain to Case I. Regarding now the new features concerning Case II, summarized in Fig. 7, their most striking difference with Figs 3 and 4 is that the process is now accelerated dramatically, such that saturation level is reached within an observable time scale. Owing the small numbers involved this level is less than 100% in a way analogous to case (iii) above. The variance remains substantial at saturation (Fig. 7b) and goes through a maximum.

6. An augmented model

The results in the preceding sections depend crucially on the validity of the conservation condition of the total population of suicidal and susceptible individuals (eq. (1)). Although this may be a reasonable assumption over short to intermediate time scales it is bound to fail in the long run, as the system becomes open to different kinds of interactions with its environment. In this section we develop an augmented version of the model of eqs (2) accounting for key processes expected to be present in real-world situations. Specifically, we allow for the following additional steps.

- The influx of susceptible individuals S_2 from an external population A of size much larger than S_2 :

- The possibility that suicidal individuals may be removed from the population S_1 (recovery or on the contrary isolation):

- The possibility that susceptible individuals may likewise be removed from the space of coexistence with S_1 , spontaneously or deliberately:

The rate equations associated to this augmented model read

$$\begin{aligned} \frac{d\Theta_1}{dt} &= p\Theta_1\Theta_2 - k_1\Theta_1 \\ \frac{d\Theta_2}{dt} &= a - p\Theta_1\Theta_2 - k_2\Theta_2 \end{aligned} \quad (9)$$

Choosing as before $p > 0$, we notice that in the limit $a = 0$, $k_1 = k_2 = 0$ the total population $\Theta_1 + \Theta_2$ is conserved and one recovers for Θ_1 the logistic equation (3). Here we are interested in the new effects arising (a), from the opening of the susceptible population towards the influx a of freshly arriving individuals; and (b), from the process by which both suicidal and susceptible individuals tend to leave the system through the above mentioned mechanisms of medical treatment, recovery or spatial constraints.

Contrary to eq. (3), eqs (9) do not admit an explicit analytic solution. We therefore proceed by identifying first the stationary states in which the variables Θ_1 and Θ_2 no longer evolve in time. Setting $d\Theta_1/dt = d\Theta_2/dt = 0$ in eqs. (9), one finds:

- A semi trivial solution

$$\Theta_1 = 0, \quad \Theta_2 = \frac{a}{k_2} \quad (10a)$$

- A fully non-trivial solution

$$\Theta_1 = \frac{1}{k_1} \left(a - \frac{k_1 k_2}{p} \right), \quad \Theta_2 = \frac{k_1}{p} \quad (10b)$$

To determine the conditions under which the system will eventually settle in (10a) or (10b) we perturb slightly each of these states and seek for conditions on the parameters under which the perturbations are amplified or on the contrary damped. In the first case the state - which will be qualified as unstable- will not be sustainable under real-world conditions, where perturbations of different origins are inevitable. In the second case the state -which will be qualified as stable- will represent the asymptotic regime towards which the system will evolve after a transient period whose duration depends on the values of the parameters. A standard linear stability analysis (Nicolis, 1995)) leads to the conclusion that there is a well-defined transition separating these two situations, occurring at a value of the influx parameter a given by

$$a_c = \frac{k_1 k_2}{p} \quad (11)$$

For $a < a_c$ state (10a) is the unique, stable steady state solution of eqs. (9) since state (10b) is physically unacceptable ($\Theta_1 < 0$). For $a > a_c$ state (10a) still exists but is unstable, and the system evolves spontaneously towards state (10b) which becomes physically admissible as Θ_1 is now positive. Notice that in the limit $a=0$, $k_1=k_2=0$, $p>0$ the semi-trivial state is always unstable and the non-trivial one is always stable. This corresponds, in fact, to the situation depicted in Figs 1 and 2 pertaining to the model of eq. (3).

Figures 8a,b summarize the time evolution of the fractions of Θ_1 and Θ_2 prior to the steady state, under the condition $a > a_c$ (state (10b) is stable). We start with a sizable pool of susceptible individuals in which a small fraction of suicidal ones has been introduced. The evolution of Θ_1 follows first a course quite similar to that of Fig 2, but once near the plateau the situation changes radically: owing to the increasing effect of suicidal contagion, the pool of susceptibles tends to be depleted and this in turn induces a sharp decrease of suicidal incidents. The result is the appearance of a marked overshoot in the population of Θ_1 and a concomitant undershoot in Θ_2 . Subsequently both Θ_1 and Θ_2 experience a slight undershoot and overshoot respectively, before settling to their long terms values. We have here a second manifestation of suicidal outbreak beyond the one identified for the model of eq. (3), where outbreak was associated with the occurrence of an inflexion point of the function $\Theta_1(t)$ prior to the attainment of the plateau (eq. (7)).

Fig. 8. Transient evolutions of the fractions of Θ_1 (a) and Θ_2 (b) obtained by solving numerically eqs. (9). Parameter values $a=1$, $k_1=0.12$, $k_2=0.01$, $k=0.02$ and initial conditions equal to 0.001 and 0.999, respectively.

Following the logic of the Monte Carlo analysis previously carried out for the scheme of eqs (2), we now inquire on the effect of variability in the results derived so far in this section. Rather than perform a full scale Monte Carlo study, we resort to a more phenomenological approach in which variability is accounted for by adding to the right hand sides of both eqs (9) uncorrelated random noises sampled from a Gaussian distribution. Fig 9 depicts the

response of Θ_1 to a variability source of this kind. Keeping parameters values as in Fig. 8 we see that variability tends to depress the extent of suicidal outbreak, presumably by desynchronizing the action of the suicidal individuals that would otherwise have manifested itself in a concerted fashion.

Fig. 9. Effect of variability in the form of uncorrelated Gaussian noise sources of variance equal to 10^{-2} added to eqs (9), on the evolution of the fraction of Θ_1 . Parameter values and initial conditions as in Fig. 8.

7. Conclusions and perspectives

We believe that the ideas put forward in this work have a methodological interest that may be further enhanced by e.g. refining the model to account for several internal states or for memory effects. In addition to this fundamental aspect we suggest that our results as they stand can be the starting point for two kinds of applications. Firstly, the reassessment of some of the results available from surveys. In particular the bimodal character of the probability in Fig.6b, reflecting the cooperativity and the smallness of the population size, suggests that the process does not always follow the trend of a purely random event as reflected by a binomial probability distribution. Secondly, the elaboration of prevention strategies. In particular, one may use the switching time t^* (eq. (7)) and inflexion point in Figs 2 and 8a) as alert level beyond which the process may get out of control. It may happen as mentioned in Sec. 3 that under the conditions actually prevailing in a given environment this time is much too long compared to the time scale imposed by the local conditions. If so one should switch to a second indicator of an imminent catastrophic evolution, which in our view is provided by the standard deviation $(\langle \delta X_1^2 \rangle)^{1/2}$ or more significantly the ratio

$(\langle \delta X_1^2 \rangle)^{1/2} / \langle X_1 \rangle$. As seen in Sec. 5 this quantity, easily monitored, tends to be enhanced in the vicinity of a collective transition encompassing the populations of interest.

For all the situations analyzed in Sec. 4 with the exception of (iii), the propagation of suicide is explosive and inevitable. The evolution of the propagation of suicide in case (v) is slower because of the limited cooperativity between individuals who have few contacts between them. It would be worthwhile to analyze in the future from this perspective contagion trends in other behavioral disorders typical of adolescence such as running away and addictions.

Another potential application pertains to prevention of situation (iii) in connection with the nature of the class group. There is much discussion about the possibility to create classes with mixed difficult adolescents, that is teenagers with conduct and affective disorders inhibiting the faculty to learn and to succeed in school. In fact the adolescents suffering of conduct disorder have often difficulties in mentalization of their essential depressive symptoms. Even if they do not have the problem of suicidal symptoms in first place, they commit repeatedly a lot of accidents, such as motor vehicle fatalities or even delinquent acts, equivalent to suicidal act. Regrouping this kind of adolescents may be, in our view and according to our results, an error as it will tend to induce further accidents. We see actually that the mixing of susceptible individuals in a "healthy" class group limits the risk of suicidal contagion.

Finally, there is according to our results an interactive "Werther" effect in the form of cyber suicide. In 1774 Johann Wolfgang von Goethe published his by now famous novel "Die Leiden des jungen Werther", in which his hero a young artist, takes his own life after a series of failed attempts to gain the love of beautiful Lotte. The novel had an immediate and an immense impact: men of society used to dress like Werther and as many as 2000 readers seem to have imitated the way he acted and died. As a result of this catastrophic situation, Goethe's novel was banned for a long time in many European countries. More than 200 years later, it appears that the availability of easy communication channels through the mass media and in particular through the advent of the Internet, an increasingly important mode of information and communication among adolescents and young adults is at the origin of a comeback of an interactive "Werther effect". Many studies have addressed the question of an observer copying suicidal behavior that he has seen modeled in the media. Case reports about cyber-suicide have been published, whereby indirect exposure to suicide through media or Internet accounts contributes to subsequent suicide.

Suicide information is easily accessible over the web, as are special chat rooms for discussions with like-minded people. Chat rooms are typical of adolescents and young adults, a group at the highest risk for imitative suicidal behavior (Davidson et al, 1989). In fact mass clusters are media related phenomena. They are regrouped more in time than in space, and are purportedly in response of actual or fictional suicide.

Our results (case II, cf. Fig. 7) provide insights on the mechanisms underlying this collective behavior. They also suggest certain ways of control of the phenomenon and of its follow ups. Health group sites and qualified treatment for suicidal youths should be better promoted. Psychiatrists, parents and teachers should take more interest in their patient's/children's Internet consumption and discuss with them. Question on media and Internet should be part of the anamnesis. The legal options to prevent cyber suicide should be discussed from a national and international perspective because of the dramatic

contagion and the criminal abuse of the Internet communities (Becker, Schmidt, 2004). This is crucial especially in view of our results on the dramatically fast pace of the process.

In summary, the major clinical insights afforded by our models are: the elaboration of guidelines for slowing down the propagation of suicide; the identification of possible “alert” indicators; and controlling Internet consumption. The main limitations of the models in their present form are that memory effects are not incorporated and that an individual is taken to be in either of only two mental states.

All in all we believe that in addition to and as complement of the all-important insights afforded by the statistical analysis of surveys, a “first principles” approach of the kind suggested in this chapter may contribute to the unveiling of some of the multiple facets of the dramatic episodes surrounding adolescent suicidal trends.

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