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## LETTER TO THE EDITOR

# Field theoretic formulation of an epidemic process with immunisation

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**Abstract.** We describe a model of the spread of an epidemic on a lattice, in which sites may be infected by their neighbours, after which they recover and are subsequently immune. It corresponds to a modification of the directed percolation problem, and also to a growth model of clusters. The model is analysed using a continuum field theory, both within a self-consistent approximation and using an  $\epsilon$  expansion below six transverse dimensions.

The problem of directed percolation, which has applications in many different fields, has been discussed extensively in recent years (Kinzel 1982). The directed site percolation problem may be realised on a lattice of sites  $(\mathbf{r}, t)$ , where  $t$  is an integer-valued 'time' coordinate. The site  $(\mathbf{r}, t)$  is connected to a set of ancestor sites  $(\mathbf{r}', t-1)$  by bonds. A set of source sites at  $t=0$  is prescribed. Then  $(\mathbf{r}, t)$  is connected to the source with probability  $p$  if at least one of its ancestors  $(\mathbf{r}', t-1)$  is connected to the source. Otherwise it is not connected. This defines a Markov process. There is a critical value  $p_c$ , such that, for  $p < p_c$ , the number of sites connected to the source decays exponentially as  $t \rightarrow \infty$ , while for  $p > p_c$ , this number grows like  $t^d$ ,  $d$  being the number of transverse dimensions. Close to  $p = p_c$ , the large-scale behaviour is characterised by universal critical exponents.

Directed percolation may be interpreted as an epidemic problem (Mollison 1977, Bailey 1975). Infected sites are those connected to the source. An infected site  $(\mathbf{r}, t)$  itself infects its neighbours at time  $t+1$  with probability  $p$ . However, in this model, it then immediately recovers, and is as equally likely to be re-infected as before.

This problem may now be modified by supposing that a site which has been infected at times  $t' < t$  has a probability  $p - q$  of being re-infected at time  $t$ , given that one of its neighbours is infected. The case  $q > 0$  corresponds to increased immunity, while  $q < 0$  represents increased susceptibility to re-infection. The extreme case  $p = q$  of total immunisation corresponds to a growth model of a colony of organisms on a food substrate. Infected sites correspond to live tissue, and immune sites to those where the food has been used up.

The model is now non-Markovian in that the development of the system at time  $t$  depends not only on the configuration of infected sites at time  $t-1$ , but also on their whole previous history. It shares this property with other kinetic growth models. It is important to realise that the model described above is different from that described by Alexandrowicz (1980), which generates percolation clusters. In that model, an infected site may infect its neighbours with probability  $p$ , but if it fails to infect a particular site, that site may not be infected subsequently. By contrast, in the epidemic

model described above, we may attempt to infect a given site an arbitrary number of times, until we succeed, when it becomes immune. It will turn out that this model is in a different universality class from percolation, although its upper critical dimensionality is the same,  $d_c = 6$ . Thus, it seems different from a similar epidemic model discussed by Grassberger (1982).

In this letter we describe the field theoretic formulation of this problem, and present the results of an  $\varepsilon$  expansion about the upper critical dimensionality, near the critical point  $p = p_c$ . We also formulate a self-consistent approximation which neglects correlations, but which should be valid away from the critical region.

Although it is possible to give a systematic derivation of the field theory as an approximation to the microscopic theory at large distances and times, we shall give only an heuristic derivation here. Recall that the effective field theory for directed percolation is given by an action (Cardy and Sugar 1980)

$$S_1 = \int dt d^d x (\bar{\varphi} \partial_t \varphi + D_0 \nabla \bar{\varphi} \cdot \nabla \varphi + \Delta_0 \bar{\varphi} \varphi + \frac{1}{2} \mu_0 \bar{\varphi} \varphi^2 - \frac{1}{2} \sigma_0 \bar{\varphi}^2 \varphi) \quad (1)$$

where  $\Delta_0 \propto (p_{c0} - p)$  and  $p_{c0}$  is the mean-field value of  $p_c$ . In the site problem, the probability that  $(\mathbf{r}, t)$  is connected to the source  $(\mathbf{0}, 0)$  is<sup>†</sup>

$$G(\mathbf{r}, t) = p \langle (1 - e^{-\varphi(\mathbf{r}, t)}) (e^{\bar{\varphi}(\mathbf{0}, 0)} - 1) \rangle. \quad (2)$$

Now consider the case when  $p$  depends locally on  $(\mathbf{r}, t)$ . Neglecting irrelevant terms, this may be accounted for by including this variation in  $\Delta_0(\mathbf{r}, t)$ . In the case in question,  $p(\mathbf{r}, t)$  is determined by whether the site  $\mathbf{r}$  has been infected in the past. The probability that this has *not* happened is  $\prod_{t' < t} \langle e^{-\varphi(\mathbf{r}, t')} (e^{\bar{\varphi}(\mathbf{0}, 0)} - 1) \rangle$ . Thus

$$p(\mathbf{r}, t) = p - q \left[ 1 - \left\langle \exp \left( - \sum_{t' < t} \varphi(\mathbf{r}, t') \right) (e^{\bar{\varphi}(\mathbf{0}, 0)} - 1) \right\rangle \right] \quad (3)$$

which leads to a similar dependence of  $\Delta_0(\mathbf{r}, t)$ . Because the averaging in (3) is carried out at times  $t' < t$ , it may be shown that, in computing correlating functions of quantities at time  $t$  with  $(e^{\bar{\varphi}(\mathbf{0}, 0)} - 1)$ , it is permissible to replace (3) by the operator expression

$$p(\mathbf{r}, t) = p - q \left[ 1 - \exp \left( - \sum_{t' < t} \varphi(\mathbf{r}, t') \right) \right]. \quad (4)$$

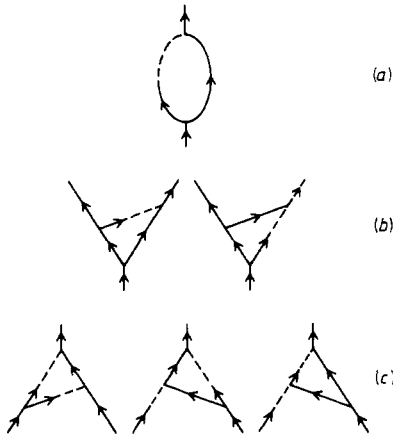
A similar argument has been used in constructing the field theory for the true self-avoiding walk (Amit *et al* 1983, Obukhov and Peliti 1983). Expanding the exponential in (4), we see that (1) has to be modified by the substitution

$$\Delta_0 \rightarrow \Delta_0 + \lambda_0 \int_0^t \varphi(\mathbf{r}, t') dt' + \dots \quad (5)$$

where  $\lambda_0 \propto q$ . The higher-order terms may be shown to be irrelevant near six dimensions. The new interaction term which has to be added to  $S_1$  is then  $\lambda_0 \int dt d^d x \int dt' \theta(t - t') \bar{\varphi}(\mathbf{x}, t) \varphi(\mathbf{x}, t) \varphi(\mathbf{x}, t')$ . The non-locality in time is a consequence of the non-Markovian property.

The one-loop diagrams contributing to the renormalisation of the propagator and  $\lambda_0, \sigma_0$  are shown in figure 1. The most relevant diagrams near  $d = 6$  transverse dimensions do not involve  $\mu_0$ , and depend on  $\lambda_0, \sigma_0$  only through the combination

<sup>†</sup> The fields  $\varphi, \bar{\varphi}$  are related to those of Cardy and Sugar (1980) by  $\varphi = i\psi, \bar{\varphi} = -\bar{\psi}$ .



**Figure 1.** One-loop diagrams contributing to the renormalisation of (a) the propagator, (b)  $\sigma_0$ , (c)  $\lambda_0$ . The broken line represents  $\theta(t-t')$ .

$g_0 = \lambda_0 \sigma_0$ . A study of the diagrams to all orders shows that no more relevant non-local couplings, for example  $\iiint dt dt' dt'' \bar{\varphi}(t) \varphi(t') \varphi(t'')$ , are generated. In  $6 - \epsilon$  transverse dimensions, the theory has a stable fixed point at  $g_0 = 320\epsilon/1229 + O(\epsilon^2)$ , whose basin of attraction is  $g_0 > 0$ , corresponding to  $q > 0$ . This fixed point controls the critical behaviour of the epidemic process with immunisation. The scaling law for  $G(\mathbf{r}, t)$  (the probability that a given site is infected) is summarised by

$$G(\mathbf{r}, t) \sim |p - p_c|^{-\gamma + \nu_{\parallel} + d\nu_{\perp}} \Phi(t|p - p_c|^{\nu_{\parallel}}, |\mathbf{r}| |p - p_c|^{\nu_{\perp}}) \quad (6)$$

which becomes, at  $p = p_c$ ,

$$G(\mathbf{r}, t) \sim t^{-(\nu_{\parallel} + d\nu_{\perp} - \gamma)/\nu_{\parallel}} \Psi(|\mathbf{r}| t^{-1/z}) \quad (7)$$

where  $z = \nu_{\parallel}/\nu_{\perp}$ . To first order in  $\epsilon$ ,

$$\gamma = 1 + 160\epsilon/1229, \quad (8)$$

$$\nu_{\parallel} = 1 + 40\epsilon/1229, \quad (9)$$

$$\nu_{\perp} = \frac{1}{2} + 303\epsilon/4916. \quad (10)$$

The number of infected sites is  $\sum_{\mathbf{r}} G(\mathbf{r}, t)$ , which scales at  $p = p_c$  as  $t^{(\gamma-1)/\nu_{\parallel}}$ . On the other hand the number of immune sites scales as  $\sum_{t' < t} \sum_{\mathbf{r}} G(\mathbf{r}, t')$ , and therefore behaves like  $t^{(\gamma-1)/\nu_{\parallel}+1}$ . The fractal dimensions of the sets of infected and immune sites, defined in terms of their volumes and radii of gyration, are  $(\gamma-1)/\nu_{\perp}$  and  $(\gamma-1)/\nu_{\perp} + z$ , respectively.

For  $q < 0$ , the case of increased susceptibility, the fixed point is not accessible, and trajectories flow out of the region of validity of perturbation theory. We are therefore able to make no definite statement about this case. One possibility is that such trajectories end on  $p - q = 1$ . In this case we may expect the cluster to grow compactly (with fractal dimension  $d$ ), even at  $p_c$ .

The qualitative behaviour of the model away from criticality may be analysed in a self-consistent approximation, which is equivalent to neglecting interactions in the field theory. The probability that  $(\mathbf{r}, t)$  is infected is written  $G(\mathbf{r}, t) = p(\mathbf{r}, t)G_0(\mathbf{r}, t)$ ,

where  $G_0$  has the same behaviour as in directed percolation (Cardy and Sugar 1980), namely

$$G_0 \sim \begin{cases} (4\pi Dt)^{-d/2} \exp(-r^2/4Dt - \Delta t) & (p \leq p_c), \\ \theta(Vt - |r|) & (p > p_c). \end{cases}$$

Neglecting correlations, the probability that  $(r, t)$  is immune is

$$\sum_{t' < t} G(r, t') - \sum_{t'' < t' < t} G(r, t'') G(r, t') + \dots = 1 - \exp\left(-\sum_{t' < t} G(r, t')\right). \quad (11)$$

Thus we may write, using a continuum approximation,

$$G(r, t) = G_0(r, t) \left\{ p - q \left[ 1 - \exp\left(-\int_0^t G(r, t') dt'\right) \right] \right\}. \quad (12)$$

This integral equation may be solved readily by converting it to a differential equation. For  $p < p_c$ ,  $G$  has the same qualitative behaviour as  $G_0$ . For  $p > p_c$  we find

$$G(r, t) = p(p - q)\theta(Vt - |r|) / \{p - q \exp[-(p - q)(Vt - |r|)]\}. \quad (13)$$

The infected sites form an expanding sphere. The density of infected sites is  $p$  near the edge of the sphere, but is reduced (for  $q > 0$ ) to  $p - q$  deep inside. The density of immunised sites, from (11), is

$$p\{1 - \exp[-\sigma(p - q)(Vt - |r|)]\} / \{p - q \exp[-\sigma(p - q)(Vt - |r|)]\} \quad (14)$$

which vanishes on the surface of the sphere, but tends to unity deep inside. Interesting special cases are  $p = q$  (total immunity), when the density of infected sites inside the sphere tends to zero like  $t^{-1}$ , and  $p - q = 1$  (total susceptibility), for which all sites within the sphere become infected.

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