Epidemic models and percolation

LETTER TO THE EDITOR

Epidemic models and percolation

John L Cardy† and Peter Grassberger‡

† Department of Physics, University of California, Santa Barbara, CA 93106, USA
‡ Physics Department, University of Wuppertal, Gauss-Strasse 20, D 5600 Wuppertal 1, West Germany

Abstract. We argue that local epidemic models with immunisation are in the same universality class as percolation cluster growth models, and show that the static exponents are equal to all orders in the ε expansion. We calculate the dynamic exponent $\nu_t = 1 + \frac{1}{28}\varepsilon + O(\varepsilon^2)$ and relate this to other exponents involving the chemical distance.

In the past few years, a number of papers have appeared which describe the growth of random clusters on a lattice (Alexandrowicz 1980, Grassberger 1983, Cardy 1983, MacKay and Jan 1984; for reviews of previous work, see Mollison 1977, Bailey 1975).

These models have in common the inhibition of growth at previously occupied sites which prevents them from being branching processes, and the existence of a growth parameter p. There is a critical value p_c, such that for $p < p_c$ the clusters grow only to a finite size, while at $p = p_c$ power-law behaviour is obtained.

In this letter we describe the field-theoretic formulation of these problems and show that they fall in the same universality class which also describes the problem of chemical distance in percolation clusters.

For definiteness consider the following microscopic kinetic models of cluster growth.

(A) At a given time t a site is either vacant, occupied or immune. An immune site is one which has been occupied in the past. At time $t+1$, a vacant site becomes occupied with probability p if at least one of its neighbours is occupied at time t. Otherwise it remains vacant. An occupied site at time t becomes immune at time $t+1$. However, immunisation is not perfect, and an immune site may become re-occupied with probability $p-q$ if, once again, one of its neighbours is occupied. This model has been considered by Cardy (1983) who gave a field-theoretic version and calculated§ the critical exponents to first order in an $\varepsilon \equiv (6-d)$ expansion.

(B) More generally, one might assume that the probability that a site becomes occupied depends on the number n of occupied neighbours. In model (B) we assume that the chances of 'infection' across each bond are independent. Then this probability is $P = 1 - (1-p)^n$ for a vacant site, and $P' = 1 - (1+q-p)^n$ for an immune.

In the case $p = q$ any bond can be tried only once, since at a second try one of the neighbouring sites is completely immune and no infection can pass. Thus, the clusters of immunes are, for $t \to \infty$, clusters of bond percolation. This model has been studied by Grassberger (1983) as a model of epidemics. A similar model was treated by MacKay and Jan (1984).

§ An error was made in this calculation which is corrected in this letter.

(C) In addition to models leading to bond percolation, one can discuss processes leading to site percolation (Alexandrowicz 1980, Pike and Stanley 1981, Grassberger 1983).

(D) This is a reaction-diffusion model in which random walkers W diffuse across the lattice, giving birth to offspring and dropping 'poison' as they go. The poison neither diffuses nor disappears, and walkers are killed if they hit a poisoned site. The microscopic processes are

(i) $W \xrightarrow{\sigma} W + W$

(ii) $W \xrightarrow{\alpha} W + P$

(iii) $W + P \xrightarrow{\beta} P.$
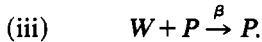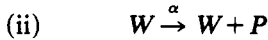
The connection with models A and B is that the positions of the poison define the immune sites and those of the walkers the infected sites; (i) corresponds to the process of infection, and (ii) to immunisation.

While models A, B, C and D are microscopically different, the essential processes involved are identical and we may expect them to be in the same universality class. In what follows we construct a field theoretic formulation of model D and show that the most singular diagrams are those considered by Cardy for model A. We then show that the static limit of these diagrams gives those of the percolation problem in $6 - \varepsilon$ dimensions, to all orders. Thus all these models fall into the same universality class. The static exponents (to be defined below) are those of ordinary percolation, and we are able to calculate the dynamic exponent in the ε expansion.

The field theory for the reaction-diffusion model D is readily derived using the methods of Grassberger and Scheunert (1980) (see also Doi 1976a, b). Introducing fields W^+, W and P^+, P which create and annihilate walkers and poison respectively, the Liouvillean operator is

$$\mathcal{L} = D\nabla W^+ \cdot \nabla W - \sigma(1 + W^+)W^+ W - \alpha(1 + W^+)P^+ W + \beta(1 + P^+)W^+ WP. \tag{1}$$

This corresponds to an action

$$A = \int dt\, d^d x [W^+ \dot{W} + P^+ \dot{P} + \mathcal{L}]. \tag{2}$$

In the Feynman diagrams, there are two causal propagators shown in figure 1, corresponding to walkers and poison respectively. The vertices are shown in figure 2. The most infrared singular diagrams have a maximum number of insertions of the vertex (c) $(P^+ W)$. This means that they will involve only the vertices (a), (c) and (d) and not the vertices (b) or (e). These diagrams have precisely the same form as those of the field theory version of model A (see figure 1 of Cardy (1983)). Note, however,

Figure 1. Bare propagators. Here $\Delta = \sigma_c - \sigma$ and $\varepsilon \to 0^+$.
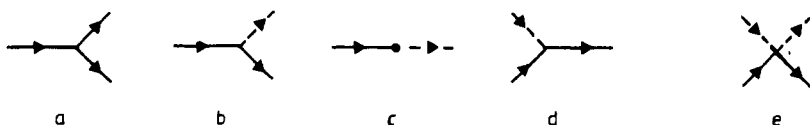
Figure 2. Vertices of the field theory. Only (a), (c) and (d) are relevant.

that this model did not include the irrelevant vertices (b) and (e). Thus we may assert that these models are in the same universality class (to all orders in the ε expansion) but they are not identical.

The renormalisation group analysis of this model follows closely that of directed percolation (reggeon field theory, Abarbanel and Bronzan 1974, Cardy and Sugar 1980), and we shall not give the details. The Fourier transform of the correlation function $\langle W(t, x) W^+(0, 0) \rangle$, which gives the probability of finding walker at (t, x), given that there was just one at $(0, 0)$, satisfies a scaling law

$$G(\omega, k) = k^{-2+\eta} \Phi(\omega \Delta^{\nu_t}, k\Delta^{\nu}) \tag{3}$$

where $\Delta = \sigma_c - \sigma \propto (p_c - p)$. To first order in the $6 - \varepsilon$ expansion we find

$$\eta = -\tfrac{1}{21}\varepsilon, \qquad \nu = \tfrac{1}{2} - \tfrac{5}{84}\varepsilon, \qquad \nu_t = 1 + \tfrac{1}{28}\varepsilon. \tag{4}$$

We see that the static exponents η and ν agree with those of percolation to $O(\varepsilon)$ (Harris *et al* 1975, Amit 1976). We now show that this result is valid to all orders. The argument proceeds by induction. Consider the nth-order contribution to the $(N \to M)$ one-particle irreducible vertex function, $\Gamma^{(N,M)}$. It satisfies an equation shown diagrammatically in figure 3, where the vertex function $\Gamma^{(N,M+1)}$ will be evaluated to
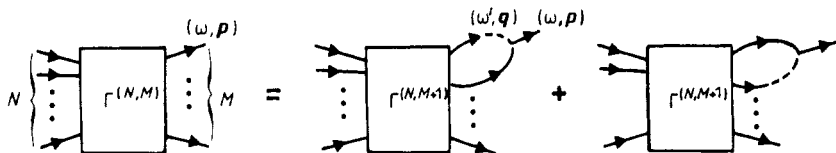

Figure 3. Integral equations for the vertex functions.

order $(n-1)$. Suppressing all non-essential dependences, this equation is

$$\Gamma^{(N,M)}(\omega, p) = \beta \int \frac{\mathrm{d}\omega' \, \mathrm{d}^d q}{(2\pi)^{d+1}} \frac{\Gamma^{(N,M+1)}(\omega', q; \omega - \omega', p - q)}{(-\mathrm{i}\omega' + Dq^2 + \Delta)[-\mathrm{i}(\omega - \omega') + D(p - q)^2 + \Delta]}$$

$$\times \left(\frac{1}{-\mathrm{i}\omega' + \varepsilon} + \frac{1}{-\mathrm{i}(\omega - \omega') + \varepsilon} \right). \tag{5}$$

In the static limit $\omega = 0$ the factor in braces becomes $2\pi\delta(\omega')$, which therefore sets $\omega' = 0$ in $\Gamma^{(N,M+1)}$. Completing the induction, we see that the static vertex functions satisfy the equations illustrated in figure 4, where now the propagator means $(Dq^2 + \Delta)^{-1}$, and the loop integrations involve $\int \mathrm{d}^d q / (2\pi)^d$. These then are the vertex functions
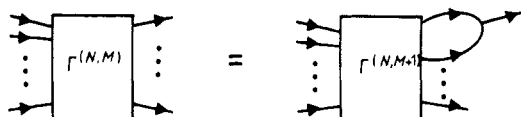

Figure 4. Static limit of figure 3.

of a theory with the vertices of figure 5, but the diagrams will have the same topological structure as those of the original dynamic theory, that is they will be causal. No diagrams with closed loops of arrows will appear. This is the field theory formulation of ordinary percolation as given by Essam (1980) and Benzoni and Cardy (1984), which gives a simpler structure than the more conventional $q \rightarrow 1$ Potts model.
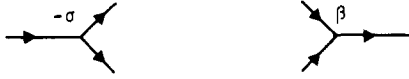
Figure 5. Vertices of the field theory corresponding to figure 4.

We conclude that to all orders in the ε expansion the static exponents of models A, B, C and D should agree with those of percolation. In particular the fractal dimension of the clusters at p_c will be

$$d_F = d - \beta/\nu = \tfrac{1}{2}(d + 2 - \eta) = 4 = \tfrac{10}{21}\varepsilon + O(\varepsilon^2).$$ (6)

On the other hand, the exponent ν_t is unrelated to the static exponents and gives the average radius of a cluster at p_c at time t as $R \sim t^{\nu/\nu_t}$. The shortest path, or chemical distance (Pike and Stanley 1981, Hong and Stanley 1983a, b, Herrmann *et al* 1984, Rammal *et al* 1984, Vannimenus *et al* 1984, Grassberger 1984) between two points on the infinite cluster a distance r apart scales like r^δ, where $\delta = \nu_t/\nu = 2 - \tfrac{1}{6}\varepsilon + O(\varepsilon^2)$. In a recent paper, Havlin and Nossal (1984) conjectured that

$$d_F = \nu^{-1} + \delta.$$ (7)

We see that this is incorrect to $O(\varepsilon)$.

In conclusion we have argued that, contrary to a previous claim by one of us, epidemic models with immunisation are in the same universality class as percolation. Our field theoretic formulation gives the first ε-expansion result for the dynamic exponent ν_t which is related to the chemical distance. It would not be difficult to extend this result to higher orders in ε if desired.

Note added. After submission of this letter, we learned that results identical to those reported above have been obtained by H K Janssen (University of Düsseldorf preprint).

References

Abarbanel H D I and Bronzan J B 1974 *Phys. Rev.* D **9** 2397
Alexandrowicz Z 1980 *Phys. Lett.* **80A** 284
Amit D J 1976 *J. Phys. A: Math. Gen.* **9** 1441
Bailey N T J 1975 *The mathematical theory of infectious diseases* (London: Griffin)
Benzoni J and Cardy J L 1984 *J. Phys. A: Math. Gen.* **17** 179
Cardy J L 1983 *J. Phys. A: Math. Gen.* **16** L709
Cardy J L and Sugar R L 1980 *J. Phys. A: Math. Gen.* **13** L423
Doi M 1976a *J. Phys. A: Math. Gen.* **9** 1465
—— 1976b *J. Phys. A: Math. Gen.* **9** 1479
Essam J W 1980 *Rep. Prog. Phys.* **43** 833
Grassberger P 1983 *Math. Biosci.* **63** 157
—— 1984 *Preprint*
Grassberger P and Scheunert M 1980 *Fortschr. Phys.* **28** 547

Harris A B, Lubensky T C, Holcomb W K and Dasgupta C 1975 *Phys. Rev. Lett.* **35** 327
Havlin S and Nossal D C 1984 *J. Phys. A: Math. Gen.* **17** L427
Herrmann H J, Hong D C and Stanley H E 1984 *J. Phys. A: Math. Gen.* **17** L261
Hong D and Stanley H E 1983a *J. Phys. A: Math. Gen.* **16** L475
—— 1983b *J. Phys. A: Math. Gen.* **16** L525
MacKay G and Jan N 1984 *J. Phys. A: Math. Gen.* **17** L757
Mollison D 1977 *J. R. Stat. Soc.* B **39** 283
Pike R and Stanley H E 1981 *J. Phys. A: Math. Gen.* **10** L69
Rammal R, Angles d'Auriac J C and Benoit A 1984 *J. Phys. A: Math. Gen.* **17** L491
Vannimenus J, Nadal J P and Martin H 1984 *Preprint*