suggestive of an arrangement along perpendicular lines, rather than in rings (Fig. 1a; it is possible to determine these analytically); we also found many other irregular configurations (such as those shown in Fig. 1b,c). We stress that in each case the configuration is of the same type for several different values of N , although only one value of N has been shown here.

We have also revealed many new vortex patterns of the symmetrical, nested-ring type. All of these have so far turned out to be unstable, but their number and geometric richness has surprised us.

Finally, and most importantly, we have identified equilibria that have no rotational or reflectional symmetry at all. Examples for N values of 8, 9, 10 and 12 are shown in Fig. 1d–h, but we believe that such equilibria exist for all $N \geq 8$ (and we have found them for N values of 8–14). Note that the reflection of any of these patterns yields a new equilibrium.

These asymmetric equilibria appear to belong to several families, and we have established relations among some pairs of equilibria for different N by the method of growing and reducing equilibria that we describe here.

In a non-dissipative system, such as point vortices, unstable equilibria play an important role in determining system evolution: as such states are approached, evolution slows down. The existence of unstable, asymmetric equilibria suggests that such configurations will often be observed when a vortex system is started from general initial conditions.

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Scrapie infectivity found in resistant species

Experimental and epidemiological evidence indicates that bovine spongiform encephalopathy (BSE) has been transmitted to humans^{1–4}, although the mechanism of this transmission is unknown. Hamsters and chickens are clinically resistant to the transmission of BSE, but we report results that raise concern over the possible long-term

Table 1 Persistence of hamster scrapie agent in normal mice depends on expression of mouse PrP

Mice inoculated with hamster scrapie			Recipient hamsters		Recipient mice
Mouse	Mouse PrP genotype	Days after inoculation	Brain	Spleen	Brain
			No. positive/ total	Incubation period	No. positive/ total
1	+/+	204	4/4	313 ± 112	NT
2	+/+	310	3/3	178 ± 14	NT
3	+/+	574	7/7	140 ± 4	6/6
4	+/+	574	8/8	162 ± 38	6/6
5	+/+	693	8/8	156 ± 20	6/6
6	+/+	693	8/8	168 ± 42	6/6
7	+/+	782	8/8	128 ± 13	NT
8	0/0	112	0/12	–	NT
9	0/0	112	2/12	294–334	NT
10	0/0	217	0/12	–	1/8
11	0/0	217	4/12	296–352	0/8
12	0/0	330	0/7	–	0/8
13	0/0	330	0/8	–	0/8

PrP-null ($PrP^{0/0}$) mice⁷ (provided by J. Manson, Neuropathogenesis Unit, Edinburgh) were backcrossed four times to C57BL/10. The original donor mice were inoculated intracerebrally with 50 µl of a 1% suspension of hamster scrapie brain containing 1×10^7 ID₅₀ units. Mice were killed after the indicated number of days after inoculation with hamster scrapie. To test for persistent scrapie infectivity, 50 µl of a 1% suspension of brain or spleen was inoculated intracerebrally into recipient hamsters or mice. NT, not tested.

persistence of infectivity in such clinically persistent species and which may have implications for the control of BSE.

‘Natural’ transmission of BSE seems to result mainly, if not solely, from feeding animals with meat and bonemeal derived from BSE-infected cattle⁵. BSE is transmissible by inoculation or ingestion to mice, sheep, goats, marmosets, mink, pigs, certain members of the cat family and various exotic ungulates.

Interestingly, though, it is not transmissible to hamsters or chickens⁵. This resistance to BSE is borne out by an absence of clinical brain disease and a lack of the hallmark protease-resistant prion protein.

We injected 10^7 ID₅₀ (where ID₅₀ is the half-maximal infectious dose) units of hamster scrapie agent (strain 263K) into the brains of groups of C57BL/10 mice that either did or did not express the mouse gene that encodes the normal version of the prion protein (PrP). Mice are highly resistant to this hamster scrapie strain⁶ and, as expected, none of our mice developed any clinical symptoms of scrapie.

We found, however, that brain and spleen tissue from the PrP-positive mice obtained between 204 and 782 days after inoculation contained scrapie agent that was capable of infecting hamsters but not mice (Table 1). All of the recipient hamsters were positive, although the long incubation periods indicated that the amount of infectivity present was about 10^2 ID₅₀ per gram of tissue, far lower than is typically found in clinical scrapie in hamsters or mice.

By contrast, tissues of mice without a functional PrP gene ($PrP^{0/0}$ mice) were only very rarely able to transmit disease to recipient hamsters. Thus, expression of a normal mouse PrP gene seemed to enable hamster

scrapie agent to persist in the brains and spleens of normal mice, suggesting that perhaps mouse PrP could be functioning as a receptor for the infectious agent⁴. However, there was no evidence for any replication of the hamster scrapie agent in these persistently infected mice. The infectivity recovered remained specific for hamsters rather than mice (Table 1).

Although we have not tested whether similar results would be obtained after oral ingestion, this unexpected and prolonged survival of a foreign scrapie agent raises the possibility that BSE infectivity might persist in various ‘resistant’ species exposed to BSE-contaminated feeds. Of particular concern would be domestic animals such as poultry which are raised for human consumption.

So far, there is no evidence for the secondary transmission of BSE from such resistant species to more susceptible species. However, the results presented here would strongly favour a decision to stop feeding ruminant-derived products to all animal species. Additional experiments should be carried out to detect possible BSE infectivity in clinically normal BSE-exposed animal species. Titration in cattle may be necessary to achieve the sensitivity needed to detect the levels of BSE anticipated in such situations.

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