idler (i) photons emerge from both crystals. The signal photons s_1,s_2 are mixed by beam splitter BS_A and the mixed beam falls on detector D_A. Similarly, idler photons i_1,i_2 are mixed by beam splitter BS_B and detected by D_B. In quantum mechanics interference is the manifestation of the addition of probability amplitudes for several different, indistinguishable, possible paths. As the alternative paths from NL1 to BS_A, BS_B and from NL2 to BS_A, BS_B are completely indistinguishable, the corresponding probability amplitudes add. Interference should therefore show up in the joint detection probability, or the rate of coincidence detection, by D_A and D_B as a function of path difference. This was indeed observed in the experiment[3].

The interference can, however, be destroyed by a 'delicate' change in the experiment, for example the removal of beam splitter BS_B, which would at first sight appear to have no effect on the signal photons. But because signal and idler photons are produced simultaneously, once BS_B is removed it becomes possible to tell from the output of D_B whether the corresponding signal photon detected by D_A comes from NL1 or NL2. Restoring BS_B and mixing the idlers restores the interference, but only in the coincidence counting rate. In the language of Scully *et al.*[1], the insertion of BS_B, mixing of the idlers and subsequent detection by D_B 'erases' the path information, restoring the interference.

It is possible to change the configuration of Fig. 1 in such a way that mixing the idlers restores the interference without the need for coincidence detection. An example of such an experiment is shown in Fig. 2 (refs 4,5). Here, the two nonlinear crystals NL1 and NL2 are arranged so that two idlers i_1 and i_2 are aligned in direction and idler i_1 passes from NL1 to NL2. It is now impossible in principle to tell whether the photon detected by D_S comes from NL1 or NL2, so long as the i_1 trajectory from NL1 to NL2 is not interrupted. The alignment of i_1,i_2 therefore serves as the 'quantum eraser' in the experiment. However, as soon as the NL1 to NL2 coupling is broken, say by deflection of the i_1 beam, or by insertion of a stop or even a sufficient time delay, it becomes possible to tell from a measurement made with an auxiliary (very efficient) detector D_i whether the photon registered by D_S comes from NL1 or NL2. Evidently, it comes from NL2 if D_S and D_i register counts simultaneously, and it comes from NL1 otherwise. The interference effect should therefore disappear when the NL1 to NL2 path is blocked. This too has been observed[4,5].

In this case i_1 'induces coherence' between the signals without inducing emission, even though opening or closing the path NL1 to NL2 does not physically perturb the s_1,s_2 beams. Thus the state vector reflects not only what is known about the photon, but also what is knowable in principle. The 'auxiliary' measurements to identify the source or the path of the detected photon need not be carried out; it is sufficient for them to be possible, in principle, for the interference to be destroyed.

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Actin in ribbons

SIR — We have proposed that actin filaments can be transformed by a twist and a stretch into 'ribbons', which we believe might be important for understanding the mechanism of force generation[1]. Our conjecture has been dismissed[2] on the grounds that it is inconsistent with the 'known' atomic structure of the thin filament[3]. This criticism is largely based on our use of the terms 'small' and 'large' to designate the axial and distal domains of the actin monomer as it packs into the ribbon. Notwithstanding the obvious confusion caused by the near identical size of the two domains, our analysis rested on the consistency of the radial position of the penultimate cysteine residue with fluorescence transfer data. Recent gold-labelling experiments[4] establish the similarity of actin-monomer orientation in the ribbon[1] and helix[2] states.

In agreement with DeRosier[5], we take issue with the claim that the structure of F-actin has been established at high resolution as the 'atomic' model is restrained by diffraction data limited to at best 8.4 Å in the meridional direction and even less radially. Furthermore, the axial contact (the 'finger') is produced by *ad hoc* intervention without any supporting high-resolution data. We do not understand Holmes and Kabsch's insubstantiated contention[2] that a 30° rotation of the actin monomer would give a model consistent with F-actin but with contacts different from those in the profilin:actin crystals, as we have not presented a high-resolution analysis. The crucial issue remains whether the genetic helix contacts are strong, as we think the balance of experimental data supports, or rather tenuous, as seen in the Holmes and Kabsch model.

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Where next for canine virus?

SIR — Canine distemper has been recognized as a lethal infectious disease of the domestic dog for more than 200 years. Later observations have extended the known host range to include the coyote, wolf, fox, ferret, mink, skunk, raccoon, badger, panda and macaca, to name but a few. In the past 3 years, studies of mass mortalities in seals, porpoises and dolphins have revealed canine distemper or canine distemper-like infection, hitherto unknown in such marine mammals. In seals two agents are involved: canine distemper virus (CDV) and 'phocid' distemper virus, the latter perhaps a mutant of the classical dog agent.

We have investigated an episode of a fatal central nervous system disease in javelinas (collared peccaries) which live as feral animals in Arizona[1]. Javelinas resemble small pigs but are not directly related to pigs. We discovered that the deaths are caused by CDV encephalitis; other, healthy animals showed serological evidence of subclinical infection. By monoclonal antibody studies we found that the isolated virus is classical CDV, and not the mutant form. The javelina is also susceptible to bovine Rinderpest virus[2], a morbillivirus closely related to CDV and human measles.

Clearly, CDV is a pervasive pathogen that can cause infection and disease in an unusually wide spectrum of domestic, wild and marine animals. It has recently been incriminated in some cases of Paget's disease of bone in humans[3]. Where will it turn up next?

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