Neurobiology

Agents against cocaine addiction

J. Neurosci. **25,** 1889–1893 (2005)

The search for treatments for cocaine addiction has been frustrating. Most notably, many of the compounds that bind to the drug's apparent biological targets, dopamine transporters, induce cocaine-like behavioural effects.

However, analogues of a drug called benztropine are showing promise as potential medications. Rajeev I. Desai and colleagues now show that one such analogue, JHW007, has a high affinity for dopamine transporters but not the same stimulatory effects as cocaine. When cocaine itself binds to these transporters, it prevents the reuptake of dopamine; this neurotransmitter therefore accumulates and begins to activate other cells, creating an intense 'high'. The researchers think that the ten times slower binding of JHW007 to dopamine transporters is responsible for its lack of cocaine-like effects.

Moreover, JHW007 has the virtue of crossing the blood–brain barrier, which means that it reaches the brain comparatively readily and, once there, it antagonizes the stimulant effects of cocaine. As well as pointing to the attributes of this analogue as a potential treatment for cocaine addiction, Desai *et al.* mention the scale of the cocaine problem: according to some surveys, there are more than two million cocaine users in the United States alone. **Roxanne Khamsi**

Developmental biology

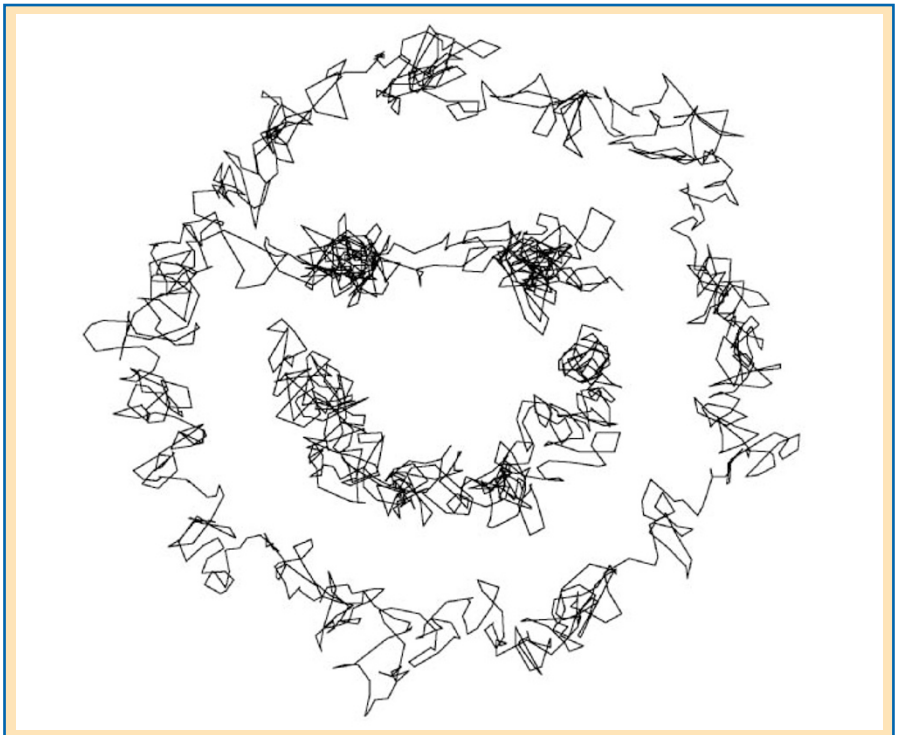
DiGeorge mice

Genes Dev. **19,** 530–535 (2005)

People with a rare congenital condition called DiGeorge syndrome have an array of facial, heart, endocrine and immune abnormalities. Heiko Wurdak *et al.* show how problems with signalling by a molecule called transforming growth factor-β (TGF-β) may contribute to this condition.

The researchers examined the role of TGF-β in neural-crest cells, which migrate from the neural tube in the growing embryo to various locations that are affected by DiGeorge syndrome, including the face, heart and thymus. They found that mice genetically engineered to lack TGF-β in their neural-crest cells mimic many of the defects seen in DiGeorge syndrome patients. Their observations suggest that the cells are unable to adopt the fates of smooth muscle or other tissues.

Wurdak *et al.* propose that TGF-β partly controls the activity of another protein, called CrkL, which lies in a segment of chromosome 22 that is missing in DiGeorge patients. Thus, they speculate that at least part of the syndrome is caused when loss of this gene disrupts TGF-β signalling. **Helen Pearson**



Nanotechnology

Stop for brownian motion

Appl. Phys. Lett. **86,** 093109 (2005)

Making nanoparticles is one thing; controlling them in solution is another. Laser tweezers, alternating electric currents and magnetic fields can all be used to trap and manipulate micrometre-sized objects, but fail for much smaller particles.

Adam E. Cohen and W. E. Moerner have an answer that can raise a smile. They have used a direct electrical current hooked up to a digital feedback system to cancel out the brownian motion of a nanoparticle in aqueous solution, allowing them to trap fluorescent polystyrene nanospheres as small as 20 nm across.

As the particle moves around, a fluorescence microscope monitors its progress and alters the voltage across the solution to correct that drift. This keeps the particle locked into place, while careful variation of the voltage allows the particle to be moved at will — hence the smiley face shown here, which was drawn with a 200-nm-diameter particle and is about 10 μm across.

The trap should work for any object that can be imaged optically and acquires a charge in water, the authors say. With the present set-up the particle can be confined to within 1 μm. But an improved apparatus can offer increased precision and power, as they will report in a forthcoming paper in *Physical Review Letters*. **Mark Peplow**

Metrology

Requiem for a mass?

Metrologia **42,** 71–80 (2005)

One would think that objects kept in a vault would not gather much dust. Not so. The platinum–iridium prototype of the standard kilogram must be thoroughly cleaned before each use and, despite this, its mass may have varied by as much as 100 μg (about five grains of sugar) over the past century.

Redefining the kilogram using a fundamental constant such as Planck's constant h or Avogadro's constant N_A could eliminate this source of uncertainty. Ian M. Mills and co-workers propose to redefine the kilogram by fixing the value of either h or N_A, rather like fixing the speed of light to define the metre.

Currently, h and N_A are themselves known to within only about 10^{-7} (one part in ten million), which is similar to the uncertainty in the mass of the prototype kilogram. Previous proposals for a new standard kilogram have always suggested waiting until the accuracy of h and N_A has been improved by a factor of at least ten.

But the authors observe that three advantages will follow from fixing h or N_A now: the kilogram will be defined in terms of an 'invariant of nature', rather than an unstable artefact; the uncertainties of many other fundamental constants (such as the elementary charge e and electron and proton masses m_e and m_p) would be reduced by more than a factor of ten; and future changes in the best estimates of the fundamental constants from one review to the next would be markedly reduced. **May Chiao**