IN BRIEF

NANOTECHNOLOGY

In vivo biodistribution and highly efficient tumour targeting of carbon nanotubes in mice.

Liu, Z. *et al. Nature Nanotech.* **2**, 47–52 (2007)

Determining the fate and biological effects of carbon nanotubes *in vivo* are crucial to their potential therapeutic applications, such as targeted drug delivery. Liu and colleagues have demonstrated efficient targeting of single-walled carbon nanotubes functionalized with phospholipids bearing polyethylene glycol to an integrin-positive tumour in mice. A high tumour accumulation was achieved, and the nanotubes exhibited relatively long circulation times and low uptake by the reticuloendothelial system.

ANALGESIA

An *SNC9A* channelopathy causes congenital inability to experience pain.

Cox, J. J. *et al. Nature* **444**, 894–498 (2006)

Cox and colleagues show that a rare autosomal recessive condition in which individuals have an inability to experience pain is caused by loss of function of the *SCN9A* gene, which encodes the α-subunit of the voltage-gated sodium channel $Na_v1.7$. The finding that disruption of this single gene leads to a complete loss of nociceptive function could stimulate research for analgesics that target this ion-channel subunit.

ANTICANCER DRUGS

Identification of NVP-TAE684, a potent, selective and efficacious inhibitor of NMP–ALK.

Galkin, A. V. *et al. Proc. Natl Acad. Sci.* USA **104**, 270–275 (2007)

Constitutive activation of the nucleophosmin–anaplastic lymphoma kinase (NPM–ALK) fusion protein drives anaplastic large-cell lymphoma (ALCL) proliferation. Galkin and colleagues have identified a selective ALK inhibitor — NVP-TAE684 — which induced apoptosis and cell-cycle arrest *in vitro*. In murine models of ALK-positive ALCL, NVP-TAE684 suppressed lymphomagenesis and induced regression of established lymphomas. The compound also downregulated CD30 expression, suggesting that CD30 could be used as a biomarker for NPM–ALK kinase inhibition.

NEURODEGENERATIVE DISEASE

Selective inhibitors of death in mutant huntingtin cells.

Varma, H. *et al. Nature Chem. Biol.* 31 Dec 2006 (doi:10.1038/nchembio852)

Huntington's disease (HD) is caused by mutations in the huntingtin protein, which leads to cellular dysfunction, but how this contributes to HD is unclear. Varma and colleagues developed a high-throughput neuronal cell-culture assay to screen more than 40,000 compounds, of which 29 were selective inhibitors of cell death in mutant-huntingtin-expressing cells and 4 were active in diverse HD models. These results suggest a role for cell death in HD, and identify mechanistic probes and potential drug leads for this condition.