PATENT WATCH

Supreme Court rules on Festo

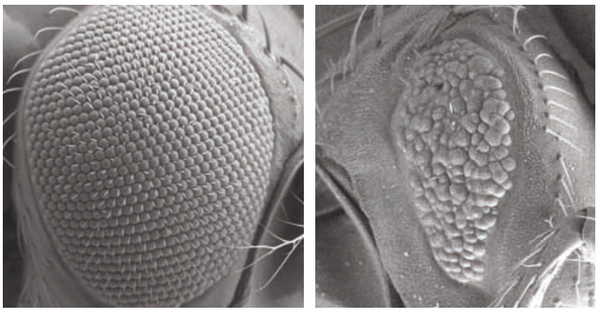
Patent holders worldwide have been nervously awaiting the result of a 13-year patent-infringement battle between Festo Corporation and Shoketsu Kinzoku Kogyo Kabushiki Co. (known as SMC), which has important consequences for patent-infringement protection. On 28 May 2002, the US Supreme Court unanimously decided to vacate the earlier ruling of the US Court of Appeals for the Federal Circuit, which had ruled that amendments that were made during the prosecution of a patent application effectively made the protection of the patent under the doctrine of equivalents unavailable.

The doctrine of equivalents is designed to prevent potential patent infringers from avoiding liability by simply making insubstantial changes to a patented invention. In the Festo case, Festo held a patent for a magnetic piston, which is used in a wide range of devices, that crucially contained an amendment stating that the device would contain a pair of one-way sealing rings and that its outer sleeve would be made of a magnetizable material. When SMC entered the market with a similar device, which instead used one two-way sealing ring and a non-magnetizable sleeve, Festo filed suit. In 2000, in the latest of a long line of cases and appeals, the Federal Court ruled that, in the process of amending the claims of their patent, Festo had relinquished the protection afforded by the doctrine of equivalents. This applied to all equivalents, not just to those equivalent products that related to the amended claim, and not just to those amendments that were meant to narrow the claim to avoid prior art. This decision reverberated through patent communities, because amendments to patent applications are commonplace, and the decision therefore threatened to render the doctrine of equivalents so narrow as to be useless for enforcing the 1.2 million patents in existence, as well as many others still under review.

Although the latest decision, written by Justice Anthony Kennedy, vacates the Federal Circuit's previous ruling, it does not offer patent applicants *carte blanche* to make amendments during the prosecution process. Kennedy wrote that, in a return to the Warner Jenkinson ruling of 1997, the Federal Courts can presume that patentees abandon the right to protection under the doctrine of equivalents with respect to any elements that are contained in an amendment. However, patentees can rebut this presumption and claim equivalent protection in such cases if they can prove that the equivalent could not reasonably have been foreseen at the time of drafting the amendment. In the case of *Festo Corp v. SMC*, the Supreme Court have left the decision of whether Festo can rebut the presumption to the Federal Circuit Court.

WEB SITES

US Department of Justice: http://www.usdoj.gov/
Festo Corp. v. Shoketsu Kinzoku Kogyo Kabushiki Co.
Supreme Court of the United States: http://www.supremecourtus.gov/
Supreme Court's decision in Festo v. SMC
Legal Information Institute Supreme Court Collection: http://supct.law.cornell.edu/supct/
Supreme Court's decision in Warner Jenkinson



NEURODEGENERATIVE DISEASES

TAU goes wild in *Drosophila*

A new model of TAU-related neurodegeneration that has been published in *Neuron* might serve to identify future therapeutic targets in progressive supranuclear palsy, Alzheimer's disease (AD) and frontotemporal dementia (FTD). Existing models of these tauopathies rely on mutant *TAU*. However, screening has failed to identify *TAU* mutations in AD or in most cases of FTD, which indicates that aberrant TAU hyperphosphorylation and the consequent neurofibrillary tangles (NFTs) might occur by unidentified regulatory mutations or other TAU-interacting proteins. George Jackson and colleagues have created a *Drosophila melanogaster* model of these tauopathies that does not rely on mutant *TAU*, but rather on modulation of human wild-type *TAU* expression.

Drosophila **lines were generated that overexpressed wild-type human *TAU* in the insects' eyes. Flies that expressed two copies of the transgene had severely reduced eye size and abnormal eye development; however, no NFTs were observed in the eye cells. The authors interpret this result as showing that there is a causal relationship between TAU modification and cell death.**

A major source of TAU phosphorylation in the human brain is glycogen synthase kinase-3β, (GSK-3β), which is the homologue of *Drosophila* Shaggy. Shaggy is a component of the Wingless (Wnt) signalling pathway, which phosphorylates several proteins. The WNT pathway comprises a group of genes that are responsible for mapping early brain development in both humans and flies, and it causes cellular degeneration. Components of this pathway have been shown to interact with other proteins that have been implicated in AD, such as the presenilins. By expressing different transgenes in this *TAU* model, the researchers were able to investigate the role of these members of the WNT signalling family in TAU-induced neurodegeneration.

Overexpression of *shaggy* induced TAU hyperphosphorylation and exacerbated TAU-induced cell degeneration. Importantly, for the first time, NFTs that resembled those found in the brains of patients with AD appeared in the flies' eyes. Conversely, reducing the amount of Shaggy reduced the amount of cellular degeneration. Further manipulation of other WNT signalling molecules indicated that Shaggy has a role that is distinct from the its classical role in the WNT pathway — it is likely that it exerts its effect on TAU dysregulation not through WNT, but through direct TAU phosphorylation.

Although no single model will reproduce all the pathological features of complex human neurodegenerative diseases, this work represents a powerful genetic system to identify modifiers of TAU-induced neurodegeneration and potential therapeutic targets in the tauopathies.

Melanie Brazil

References and links

ORIGINAL RESEARCH PAPER Jackson, G. R. Human wild-type Tau interacts with *wingless* pathway components and produces neurofibrillary pathology in *Drosophila*. *Neuron* **34**, 509–519 (2002)