A komodo dragon fossil with the replica of a modern day komodo skull. Photo: Queensland Museum

Komodo Dragon Is Dinky-Di

Australia's disproportionate share of terrifying beasts was once even higher with the discovery that the world's largest lizard, the komodo dragon, dwelled and probably originated here.

"The fossil record shows that over the last four million years Australia has been home to the world's largest lizards, including a 5-metre giant called *Megalania* (*Varanus prisca*)," says Queensland Museum Curator Dr Scott Hocknull. "Now we can say Australia was also the birthplace of the 3-metre komodo dragon (*Varanus komodoensis*)." says Hocknull.

"Over the past 3 years we've unearthed numerous fossils from eastern Australia dated from 300,000 years ago to approximately four million years ago that we now know to be the komodo dragon. When we compared these fossils to the bones of present-day komodo dragons, they were identical."

The discovery refutes the theory that komodos evolved on the Indonesian island of Flores from smaller ancestors through the process of island gigantism, where species that have become geographically isolated often grow to extraordinary size.

Further confirmation of its local origin comes from genetic evidence that the dragon's closest living relative is the Australian lace monitor (*Varanus varius*).

In a third dramatic addition to our knowledge of giant lizards, the Queensland Museum announced the discovery of an extinct Timorese relative that is larger than the komodo but smaller than *Megalania*.

Hocknull says it is likely that further species filling the size gap between komodo and *Megalania* will be found. "Previously *Megalania* was an outlier, so to find an intermediate in Timor suggests dispersal westward from Australia," he says. "All Indonesian islands could hold remnants of a number of different giant goannas, but these have been very poorly explored for fossils."

Komodo dragons weighing 70 kg, the same as their modern descendents, roamed Queensland at least as recently as 300,000 years ago, and Hocknull imagines the komodo actually survived even more recently. "It's most likely we've found nothing younger because we haven't found the sites," he says. "We suspect it lived on into the late Pleistocene simply because it's a generalist feeder, and it would be expected to have eked out a living even as the climate changed."

Australian komodos were probably another victim of direct or indirect human behaviour. Hocknull says that the species survived on Flores for at least 900,000 years despite changes in fauna, volcanism and even meteorite impact. However, it recently disappeared from central Flores, apparently due to habitat destruction by humans.

Antioxidant Link to Diabetes

Health fanatics who dose themselves with antioxidants may be increasing their chance of getting Type 2 diabetes, if mice studies are to be believed.

Prof Tony Tiganis of Monash University's Department of Biochemistry and Molecular Biology found that reactive oxygen species (ROS) molecules are protective against the early stages of Type 2 diabetes in mice.

Mice with high ROS levels were able to overcome high-fat diets and avoid developing insulin resistance. However, antioxidants in the mouse diet mopped up the ROS, leading the mice to become diabetic.

"ROS molecules, such as hydrogen peroxide, are important for normal cell function," Tiganis says. "We have shown that ROS present in muscle enhance insulin action and help lower blood sugar levels."

However, it's not a matter of simply reversing the "ROS bad, antioxidants good" formula. "Our studies do not negate the role of ROS in late-stage disease," Tiganis says. "There's a 'yin and yang' relationship that takes place, wherein ROS are beneficial in the early stages of Type 2 diabetes and shift to being harmful at later stages of the disease. We are now trying to find out when ROS make the switch from being 'good' to 'bad'," Tiganis says.

While that research is going on Tiganis recommends against taking antioxidants, suggesting instead that people should "eat healthy and exercise".

Insurance Dampens the Demand for Genetic Screening

People are risking their lives rather than placing their insurance in peril, a study of genetic testing has found. The research has bolstered calls for reform on insurance premiums.

Researchers from the University of Melbourne and the University of NSW offered genetic screening to106 individuals from families with genetic predispositions to bowel cancer. Initially 20% refused. However, when the potential insurance consequences were explained to them the refusal rate increased to 50%.

"In Australia, while genetic information has no implications for health insurance, it can affect life, trauma, disability and sickness, and accident insurance policies," says Dr Christine Van Vliet of UNSW's School of Medical Sciences.

Bowel cancer was chosen because the benefits of genetic screening are substantial. One in 3000 Australians carries a mutation that greatly increases the chance of contracting the disease.

"For those at high genetic risk, screening for and removal of polyps reduces the risk of bowel cancer by more than 50%," says A/Prof Mark Jenkins of the University of Melbourne's School of Population Health. "Screening people at high genetic risk of bowel cancer is a highly cost-effective way to reduce deaths due to bowel cancer."

Van Vliet says that we can learn from what other nations have done. The United States has recently passed legislation so that those who have genetic tests as part of research programs do not have to declare the results to insurance companies. Van Vliet considers this a minimum step required to protect our capacity to learn more about genetic diseases.

However, the Dutch provide an even better model in Van Vliet's opinion. "Up to a certain-sized premium it is not necessary to declare the results of testing, but when applying for larger premiums the results must be declared," Van Vliet says. This makes basic insurance available to all, but prevents insurance companies from being bankrupted by those who take out huge premiums after discovering their risk.

"It's going to be a growing problem as we test for more genes," Van Vliet notes. However, she adds that insurers sometimes place too much significance on single genes whose operation may be influenced by the environment or other hereditary factors.

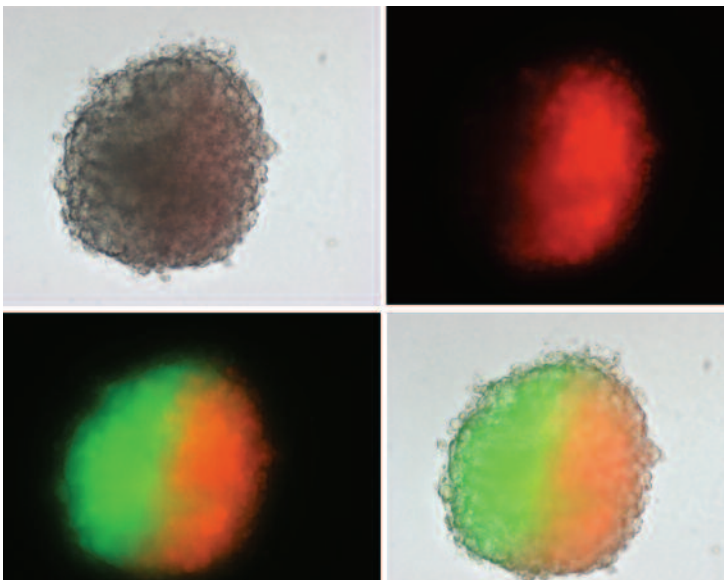
Although insurance companies provided the information used to inform participants of the consequences of being tested, they have so far not commented on the findings.

Blood that Glows

Stem cells have been altered so that they glow when they become red blood cells. The technology will enable researchers to track the development of stem cells.

Human embryonic stem cells (hESCs) have the potential to turn into any type of cell in the body. However, tapping that potential to make them consistently turn into a specific cell type remains a challenge. The ability to measure success will make such transformations more reliable.

"Generally it is easier to turn ESCs into cells that appear earlier in embryo development," explains Prof Andrew Elefanty. "Blood cells go through stages, and we have produced a cell line such that each of those stages can be recognised."



A new line of embryonic stem cells turns from green to fluorescent red as they transition through several stages to become red blood cells.
Photo: Tanya Hatzistavrou and ELizabeth Ng, Monash Immunology and Stem Cell Laboratories

Previously researchers had to rely on a hard-to-detect red tinge to cells once they had matured, or on studying the cell's RNA – a process that Elefanty says "terminated the experiment" by destroying the cells.

Elefanty's method produces a green background for cells that have not transitioned, followed by cells that fluoresce bright red under the right wavelengths of light.

"We hope that in living small animals we will be able to identify cells using the fluorescent colour. We'd like to be able to unequivocally identify the donor cells after a transplant," Elefanty says. The Monash Embryonic Stem Cell Differentiation Group that Elefanty leads has already produced other hESCs that indicate their transition to particular tissue cells, and are about to announce several more.

"By being able to identify when ESCs have become precursors to heart or pancreas or nerve cells we have a technological skill set that is not widely available," Elefanty says.