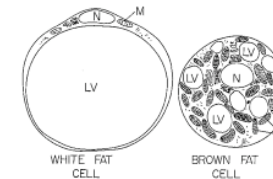


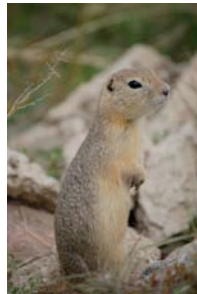
In the news...

Brown fat stores in adult humans

Remember that brown adipose tissue (brown fat) is used by small mammals and newborn humans for quick generation of heat:

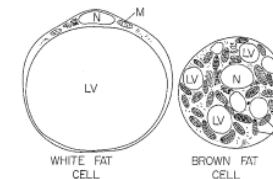


Rather than store fuel like white fat, brown fat contains LOTS of mitochondria that lack the enzyme ATP-synthase; instead they contain an enzyme that produces heat, and lots of it.



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Rather than store fuel like white fat, brown fat contains LOTS of mitochondria that lack the enzyme ATP-synthase; instead they contain an enzyme that produces heat, and lots of it.

Until recently, we did not think adult humans had brown fat stores...



- ~100 out of 1000 people had brown fat reserves in their necks
- ~50 of those people were less than 50 years old, and had BMI less than 23.5
- Tended to be more prevalent in women (less muscle mass?)
- Tends to decrease with age, similar to bone mass (may explain increased weight gain in older adults)
- Exact function is not clear

Migration of the bar-tailed godwit

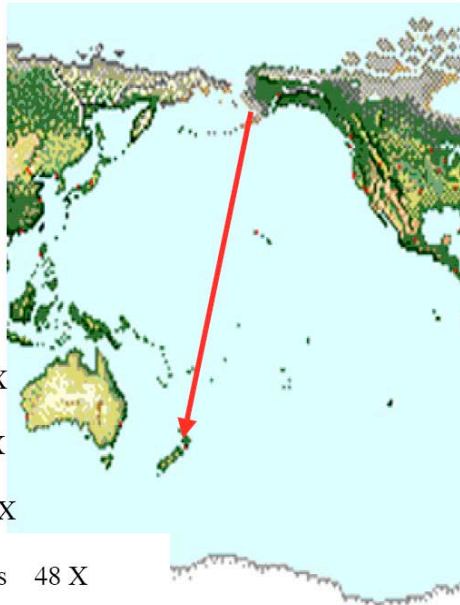


Breeds in Alaska...

Winters in New Zealand...

*Some fly this route
NON-STOP!!!
(don't eat: stored energy only)*

Migration of the bar-tailed godwit



Marathoner	godwit	ratio
42 km	11,000 km	262 X
3 hours	120 hours	40 X
13.5 km/h	90 km/h	6.7 X
21,000 strides	~1 million flaps	48 X

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They may not eat, but do they sleep?

Bird naps

Captive Swainson's Thrushes fall asleep almost immediately in captivity and nap in short (5-10 s) bursts during drowsiness



Sometimes, they kept one eye open and alert (watching for predators), while the other eye (and 1/2 of the brain) slept.

Other birds, and some aquatic mammals exhibit this "unihemispheric" sleep

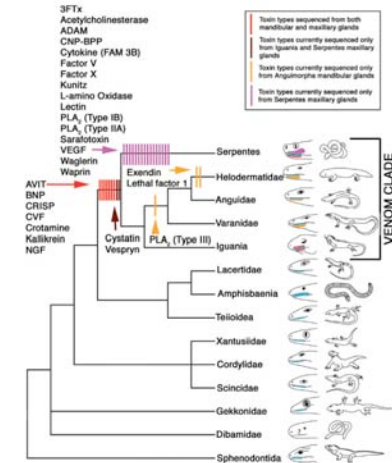
Its not clear if birds make up for all of their lost sleep in this fashion, but it can help us understand how to cope with human sleep loss.

The evolution of venom

You surely are familiar with the idea of venomous snakes, but if you look at what they are injecting into their prey, we see something cool:

A collection of proteins that falls into a nested hierarchy of descent!

Fry BG, Roelants K, Norman JA (2009) Tentacles of venom: toxic protein convergence in the Kingdom Animalia. J Mol Evol Mar 18. [Epub ahead of print].

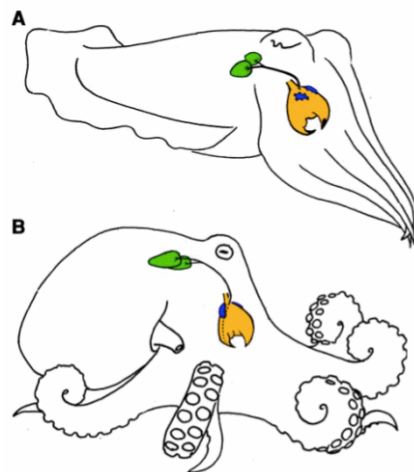


The evolution of venom

Let's look at a group of venomous protostomes: the cephalopods (after all, that's where the real action is!)

Brian Fry sequenced the proteins in cephalopod (as well as vertebrate) venoms and found a range of proteins present —some were unique and some present in reptiles.

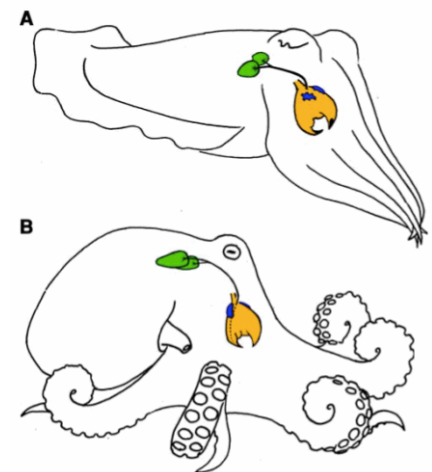
This teaches us some important lessons about evolution...



The evolution of venom

1. Evolution doesn't invent things; it uses what's available:

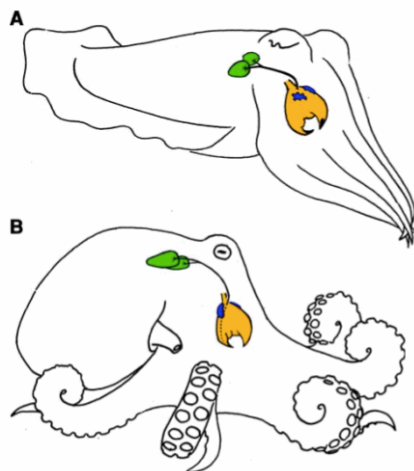
Phospholipase A₂, when produced in moderation is helpful a regulatory protein that controls inflammation, etc., but when venomous animals overdose us, it sends our control systems into a frenzy, often causing a tissue to inflame out of control



The evolution of venom

1. Evolution doesn't invent things; it uses what's available:

Peptidases are useful enzymes for digesting proteins in our stomachs, but when they are injected into our skin or muscle by a snake (for instance), all of a sudden our muscles are being digested.



The evolution of venom

2. There's an amazing amount of convergence in the proteins used for venom:

Table 1 Cephalopod toxic mutant proteins convergently recruited into other venomous lineages

	CAP	Chi	Hya	Kal	PLA ₂
Cephalopod	X	X	X	X	X
Cnidarian					X
Cone snail	X				
Fish			X		
Insect Bristle				X	X
Proboscis	X		X	X	X
Stinger	X	X	X	X	X
Hook worm	X				
Scorpion			X		X
Shrew				X	
Spider	X		X		
Reptile	X		X	X	X(3)
Tick	X			X	X

CAP CRISP, Antigen 5 (Ag5) and Pathogenesis-related (PR-1), *Chi* chitinase, *Hya* hyaluronidase, *Kal* kallikrein, *PLA₂* phospholipase A₂. X(3): independent recruitment of Group IB, IIA, and III PLA₂ into reptile venoms

The evolution of venom

3. Not just any proteins are being used; the same ones are being used over and over by various critters as venoms.

Table 1 Cephalopod toxic mutant proteins convergently recruited into other venomous lineages

	CAP	Chi	Hya	Kal	PLA ₂
Cephalopod	X	X	X	X	X
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Fish			X		
Insect Bristle				X	X
Proboscis	X		X	X	X
Stinger	X	X	X	X	X
Hook worm	X				
Scorpion			X		X
Shrew				X	
Spider	X		X		
Reptile	X		X	X	X(3)
Tick	X			X	X

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How neat is that?

...speaking of venom



Southern Pacific rattlesnake
Crotalus oreganus helleri

Most rattlesnake venoms are hemolytic (they digest tissue around the wound), but the southern Pacific's venom is one of a few that is also neurotoxic—it affects the nervous system as well as the muscular system.

In southern California, hospitals that treat snakebites formerly saw patients with severe neurological symptoms every 1-3 years...

...now they see several of these types of bites every year. *Why?*

...speaking of venom



Southern Pacific rattlesnake
Crotalus oreganus helleri

One hypothesis is that the southern Pacific tends to not rattle like other southern California species—this keeps it safe from human predators... and their shovels.

In other words, they are adapting to human habitats, and are increasingly coming into contact with humans (and more humans are being bitten).

...speaking of venom



Southern Pacific rattlesnake
Crotalus oreganus helleri

It could also reflect a change in the species' venom. The very deadly (and aggressive) Mojave green rattlesnake produces a neurotoxic venom as well. Could interbreeding have caused the SPR to 'turn on' genes that previously weren't?

The northern Pacific rattlesnake also has neurotoxin, but not nearly as much as SPRs.

...this would represent a case of very rapid evolution though.

...speaking of venom



Southern Pacific rattlesnake
Crotalus oreganus helleri

Probably it is a combination of humans encroaching on snake territory, snakes expanding their range, and possibly, humans becoming more susceptible to venom (reduced immune system efficacy because of pollution, etc.)

“Running man, revisited”

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Walking, running and the evolution of short toes in humans

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The phalangeal portion of the forefoot is extremely short relative to body mass in humans. This derived pedal proportion is thought to have evolved in the context of committed bipedalism, but the benefits of shorter toes for walking and/or running have not been tested previously. Here, we propose a biomechanical model of toe function in bipedal locomotion that suggests that shorter pedal phalanges improve locomotor performance by decreasing digital flexor force production and mechanical work, which might ultimately reduce the metabolic cost of flexor force production during bipedal locomotion. We tested this model using kinematic, force and plantar pressure data collected from a human sample representing normal variation in toe length ($N=25$). The effect of toe length on peak digital flexor forces, impulses and work outputs was evaluated during barefoot walking and running using partial correlations and multiple regression analysis, controlling for the effects of body mass, whole-foot and phalangeal contact times and toe-out angle. Our results suggest that there is no significant increase in digital flexor output associated with longer toes in walking. In running, however, multiple regression analyses based on the sample suggest that increasing average relative toe length by as little as 20% doubles peak digital flexor impulses and mechanical work, probably also increasing the metabolic cost of generating these forces. The increased mechanical cost associated with long toes in running suggests that modern human foot proportions might have been selected for in the context of the evolution of endurance running.

Key words: phalanges, gait, foot biomechanics, bipedalism, Australopithecus

