THE SCIENCE CREATIVE QUARTERLY ISSUE ONE PART FIVE OF SIX JUNE 6TH 2005 UMMFFFF!

Our masthead, we think, will be forever evolving, although at present we have two Daves, a Bethany, a Caitlin, a Stephen, a Claire, and a Russell.

Tom, Moebius and Richard continue to be happy to help.

Maybe Justin and Willow?

We did follow up on Chris and his friends, and for now, we know that Chris is on board.

Isn't Jen really good at drawing pictures?

Our exotic sounding Azar is sort of still with us, but involved with a different project that will likely have an affiliation with the SCQ later this summer.

Email us at **tscq@interchange.ubc.ca**

EUREKA!

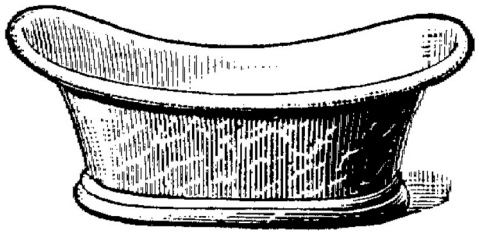
By The Science Creative Quarterly

Dear Reader,

In a nonchronological theory of literary history, T.S. Eliot proposes a simultaneous existence and order of all written art. This manifests itself in the evolution of a continuous string of exposition so that earlier work is always being altered through the introduction of later work. Perhaps this is personified in a Palimpsest. As writing materials were not readily accessible in ancient Greece, it was commonplace to wash or scrape a piece of parchment or vellum, and reuse it. Infra-read and digital enhancement techniques employed by modern historians can recover the erased text to reveal older works. In this way, Palimpsests subvert the concept of author as sole originary, much in the way Eliot redefines the order of new work into the pantheon of "existing monuments" by deferring significance to an infinite sea of previous sources.

This is how we came to know Archimedes. Hidden in layers of liturgical writing, a Method was revealed in a hybrid of mathematics and physical phenomena. Codes in a codex of religious rites to fill in the lacunae of imagination and curiosity.

We at The Science Creative Quarterly are looking for modern and metaphysical Palimpsest writers, something to make us jump out of the bath and run around naked and screaming.



ABOUT SUBMISSIONS:

Anything will do, but if you like more direction, we are happy to look at:

Things with some link (however weak) to science.

Things in English.
Things in other languages that are more or less readable when translated with Google tools.

Things with many words.
Things with few words.
Things with pictures.

Things that are news worthy.
Things that are not terribly so.

Things that educate.
Things that entertain.
Things that both educate and entertain.

Things that are important to ones well being, or perhaps to the global community at large.
Things that (at the end of the day) are really only there for the sake of being there.

Things from famous people who think that this is a pretty neat thing going on here.
Things from infamous people - they're interesting too.
Things from everyone else.

And things whose copyright ultimately remain with the author, although it would be nice to be acknowledged as being involved in presenting it to others.

Submissions are preferred as attached word documents, or text pasted directly into the body of the email. Please send us your good work to **tscq@interchange.ubc.ca**

AND COMMENTS ALWAYS WELCOMED...

DEATH: AN INTERDISCIPLINARY ATTEMPT AT A DEFINITION

By Justin Kahn

(1: Holding the Tail)

The mouse is shaking his head and tail as if he is saying no, no, no. Honest, he is.

Yesterday, I couldn't do the mouse in, but that was yesterday. I've learned to do other things that I didn't enjoy. Annihilating a mouse is a yet another one of those unpleasantaries, that I must own up to. Killing mice is a part of life and accepting life is a part of growing up, so it follows that making this mouse give up his ghost is a part of my maturing process. Besides, death is happening everywhere, I think.

Death is already in this very mouse. Inside my clutch, inside the body, cells are dying. We don't hear them dying, because we don't want to hear them dying. Cells in their little black turtleneck's reading molecular size Camus. Dying cells, dead cells.

No one tears up over dandruff, a very visible manifestation of dead cells

I believe, the mouse is bearing his teeth.

(2: The Body)

Those pleading eyes of his.

Why can't you buy death in a package? Is it simply because no one wants packaged death? Or does some kind of technical problem prevent bottled death? Why couldn't you package something found only in bodies, which are kinds of containers? Death is not ubiquitous like some kind of ether. (Although, I don't know that for sure. I'm not real current on my physics.)

Maybe I should have gone into physics. They don't kill mice, not qua physicists anyway. Neither do these guys embody the primitive élan vital. In fact, some shoot for the very opposite. One went so far as to say, "I am become death". He touched the thing with a needle. Not that all of them personify death.

Sometimes they just talk about death—as out there, as not yet. Heat Death. The most morbid week of my undergraduate course in cosmology was reading Paulson's article, "Don't Sweat the Small Stuff and It is All Small Stuff." The mood in the room suggested that death is most certainly an inescapable ether. An ether, one would very much doubt light's ability to pass through.

If such a thing is possible, then this mouse is moping.

(3: Snap)

Why did I think that I could do this? I couldn't sleep for a week after seeing Ingmar Bergman's Seventh Seal. A week without sleep, knowing that I could play the its-only-a-film card against my imagination.

My impression is that artist's tend to figure, 'better dead than never', but there is no room for that kind of sloppy thinking in the lab, where either there is a heartbeat or there is not. Unless you go by a brain stem definition, but that would considerably complicate things.

In the end, there is just me, my mouse and this metal rod.

We can summarize our research on death so far by recalling the words of a (deceased) philosopher. Not precisely his words, but to paraphrase Pascal, between the death of the universe and the death of a cell is dandruff. Sort of puts things in perspective.

Was J. Robert Oppenheimer's comment, I am become death, really about his need for Head and Shoulders? Might dandruff be an evolutionary adaptation to keep us aware of the Unbecoming? Or was Oppenheimer, in a sense making a much more serious comment about the status of abstract thinking in toto, in relationship to that which we call life?

Finally, both the mouse and I reach the fifth phase of Kübler-Ross: acceptance.

From where I stand, for a moment, death is a warm fuzzy feeling.

Then, I draw a vial of blood and I'm not sure exactly what death is or is not.

ASPARAGUS, STINKY PEE, AND SCIENTIFIC CURIOSITY

By Willow King

I was recently enjoying a nice dinner with a few close friends of mine. Our conversations are often less than delicate, so it was no surprise that talk soon became a little tactless. The subject turned to the unpleasant yet inevitable consequences of our meal that would visit each of us within a few hours. No, we weren't talking about beans (the so-called "musical fruit"), but about asparagus, a seemingly benign green delicacy that goes especially well with hollandaise sauce. Strangely, one member of our group had no idea what we were talking about, despite describing asparagus as her favourite (and also oft-consumed) veggie. Eventually, I had to spell out for her what I assumed anyone who had ever tried asparagus must know – it tastes great, but later gives its consumer horribly, wretchedly, and obviously stinky pee. Of the six people at dinner, she was the only one who had never noticed such a phenomenon. She promised to pay special attention on her next bathroom visit, and eventually reported back to the group: no stink.

Being a geeky scientist type who also happened to be taking biochemistry, I supposed that my friend differed from the rest of us in how her body broke down the asparagus – in other words, that she metabolised some component differently. The food we eat is digested, both mechanically and chemically, into numerous minute components; the fate of these components can include storage for later use, immediate recycling, or excretion. Complex proteins called enzymes catalyze these processes, and as is the case with all our body's proteins, they derive from a blueprint in our genetic code, or DNA. I had learned of genetic errors of metabolism in biochemistry class, including the very literally named maple syrup urine disease, a condition that imparts a sweet odour to the urine (along with a number of other far more severe symptoms). Remembering this disease prompted my suspicion that a genetic variation likely accounted for whatever my friend didn't smell.

Surveys can be helpful tools in science, but to draw meaningful conclusions from the results, scientists must use a large enough sample size to ensure their numbers are significant (i.e. they're not biased, a fluke, etc.) Clearly, my dinner-table survey was not statistically relevant, so I set to work. I began asking everyone I know if they noticed anything unusual in the hours after eating asparagus. I learned a number of things:

1) Most people do not eat asparagus often enough to even remember when they last had it;

2) Many people had no clue what I was talking about, but when prodded to have some asparagus for dinner and report back, the majority said something along the lines of "Oh, that's where that smell comes from!"

3) Some people prefer not to discuss the scent of their pee, even in the name of science;

4) I don't have enough friends to gather sufficient data for any kind of survey.

Nevertheless, I will report that of the 22 asparagus-eaters willing to discuss the matter with me, three swore up and down they had no stink to report. That's about 14%, but given the margin of error inherent to my small sample size, it doesn't mean much. (Interestingly, one of the non-stinkers was my own mother – presumably, I inherited the stinky trait from my father.)

My survey being a bit of a bust, a turned to another tool in the scientist's arsenal: research. (Truth be told, I really should have gone here first.) It turns out that many before me have been riddled by the mystery that is asparagus-induced stinky pee (or malodorous urine, as those fancier types call it). Incredibly, some scientists have been curious enough to actually study the phenomenon. Indeed, some 50 years ago, a study found about half of the British population to be "excretors" – people whose bodies make the foul-smelling compound (1). It was later found that, as with any genetic trait, frequency varies with population; a study of French citizens found all subjects produced the smelly compound (2).

So what exactly is this stinky chemical? The answer is a matter of dispute, with a surprising number of scientists weighing in. There are a number of fancy machines and complex techniques that are used to identify the chemical makeup of a substance, but none of them will tell you which of the numerous chemicals in a urine sample is causing a certain smell! A veritable laundry list of chemicals has been proposed as the offender, with some suggesting it may be a combination of many. All agree it is likely some sulphur-containing compound, one likely contender being asparagusic acid (3).

Reading this paper, you may have noticed a rather serious error in my early logic. My stinky pee hypothesis was based on what I was familiar with; that is, metabolic processes and variations. However, had I been studying sensory processes (particularly, our sense of smell) instead of metabolic biochemistry, might I have considered a different theory? Isn't it possible that we differ not in our ability to make a certain compound, but in our ability to smell it? The answer to this question, I discovered, is yes. Perhaps operating on the same bias as I was, early studies of the stinky pee phenomenon had subjects eat asparagus, do their business, and report any smell (or lack thereof) to the researchers. Those reporting no smell were then designated non-excretors, and the study results based on these reports. However, it never occurred to the researchers to have someone else smell the urine samples, ideally, someone known to be able to identify the smell. Some of the supposed non-excretors may have indeed been producing the offensive odour, but not been able to smell it. This error underscores the importance of considering all possible solutions when faced with a scientific puzzle.

A 1980 study examined the possibility that anosmia (the inability to smell the compound) might actually account for those though previously to be non-excretors. Interestingly, the results showed that, indeed, some people are simply unable to smell the foul odour, no matter how strong it may be (4). Thus was identified a new category of people, the "nonpercievers", with those of us able to smell the stench called "perceivers".

So is the mystery now solved? Are we all stinkers, but only some of us smellers? Not exactly. The 1980 study found that, as earlier reported, nonexcretors do exist. But these people were not exclusively nonpercievers – some could sense the smell in other's urine. Conversely, some excretors were unable to detect the compound in urine others had identified as outright putrid. Apparently, this riddle, like much of genetics, has more than one answer. We have tens of thousands of genes, each wielding influence over one or several traits. From an evolutionary perspective, it's hard to imagine why some of these genes exist; really, what survival advantage is gained with the ability to smell asparagus pee stink? And why should some of us make the compound while others don't? I have no answers to these questions, but do know that genetics can make for fascinating dinner conversation, particularly when the meal includes asparagus.

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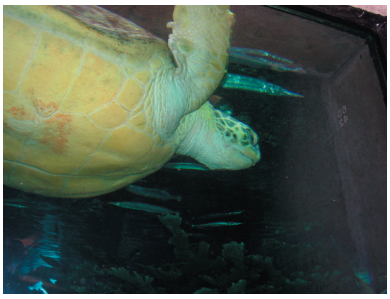
TRASH TALKIN' AT THE AQUARIUM

By Christopher Monks



Wut up, tortoise? You think you're all that 'cause you can swim really well and stuff? Well, sorry to disappoint you, son, but I can swim really well, too. Sucka. I need to wear water wings, on account I'm scared of deep water, but that's still swimming. So bite me, fool.

Ooo, shark, what big sharp teeth you have! Too bad four out of five dentists think you're a doophis. Boo-ya!

"Arf! Arf! Look at me: I'm a big fat sea lion! I can wave hello with my big fat flipper and spin a beach ball on my big fat nose. Arf! Arf!" Shoot, you ain't nothin'—nothing but a seal that needs to lose mad weight. That's right: you need to go on a diet, boy. I'm on Weight Watchers, chump. I got, like, eleven points left for today, too. Gonna get me a yogurt pop. Jealous? Ha-ha. Loser.

Hold up, octopus: you did not just call me "your bitch" because no way in hell I'm your bitch. I ain't no invertebrate's bitch. You're my bitch, octobitch. That's right, wut you gonna do about it? Huh? Wut? Wut? I didn't think so. Pussy.

Hey, catfish, Sylvia Plath called: she wants her depression back. Snap out of it, sad sack. I didn't pay seventeen bucks to watch some half fish/half cat have a nervous breakdown. Been there, done that, fish.

Yo, hottie cleaning the seal tank: me likes what me see. What you say after you finish scrubbing seal feces off that rock we kick this joint and go dutch on some daiquiris? No? Okay, whatever then. I was just jokin. Shoot, no way I'd go out with you. Think I might be gay, anyway. So later for you!

Penguins suck!

Well, what do we have here? Looks like a lazy-ass starfish. Is it hard work sitting on your lazy ass eating crud off that rock all day long? Shoot, that I ain't hard. I used to do that all the time back when I was living with my mom. Got my own place now, though. Still get lonely at times, but all in all I'm a lot happier. [Sigh].

DATING OILY ROCK

By David Secko

The giant oil sands of Alberta finally have a date. And it's a lot older than anyone expected.

David Selby and Robert Creaser, from the University of Alberta, recently put an age of 112 ± 5.3 million years ago for the migration and accumulation of oil in the Alberta oil sands – a date over 60 million years earlier than previously thought.

Although, the date isn't the first to be done on the sands, it's the first in the world to be done with such accuracy.

"This has only ever been done on a relative scale before with something like fossil dating," says Selby, a postdoctoral fellow and lead author of the study. "We are quite excited by it," he says.

Dating the formation of oil is no easy task. This is because oil and gas forms from a variety of organic material and also tends to migrate through rock. In fact, as the oil migrates it either winds up escaping to the surface or is trapped and forms petroleum fields like in the western Canadian sedimentary basin in Alberta. This process tends to remove any memory of where and when these fields first formed.

As a result, "there was an immense debate on when the oil sands formed," says Selby. This has also left explorers looking for oil with little information on what type of rocks might contribute to petroleum fields.

To tackle this problem, Selby and Creaser decided to use a naturally occurring chronometer in the form of isotopes from two elements found in the Earth's crust – rhenium and osmium. This chronometer is based on an unstable rhenium isotope, rhenium-187, which decays into osmium-187. This rate of decay is constant allowing researchers to use it as a geological clock.

The first question for Selby and Creaser therefore became whether they could detect rhenium and osmium in oil. For an answer, they turned to a type of rock termed black shale, which is the source of oil as it migrates and matures into a petroleum fields, says Selby.

Upon examination of the shale, Selby and Creaser found 3 to 50 parts per billion rhenium and 25 to 290 parts per thousand osmium. Since oil picks up rhenium and osmium as it migrates through the shale, the researchers were then able to examine rhenium/osmium ratios in oil to accurately date it.

What Selby and Creaser found places the creation of the Alberta oil sands at a much more ancient date then previously suggested (researchers had thought the oil sands formed during the Late Cretaceous Laramide Orogeny, ~ 60 million years ago.) The findings also suggest the oil formed from a single source.

"This helps answers some huge questions," says Selby, "about when the oil sand formed and how they were trapped." The results of the study are published in the May 27, 2005, issue of Science.

In an accompanying comment piece in the same issue, Bruce Schaefer, from Monash University, Australia, says these findings are important for petroleum exploration.

"For petroleum explorers, knowing the ori-

gin of hydrocarbons in a sedimentary basin places constraints on where they might be able to accumulate, or whether they are able to accumulate at all. With oil exploration drillholes costing multiple millions of dollars, every piece of data informing site location is of immense worth," writes Schaefer.

Selby isn't quite so sure of the immediate impact to petroleum exploration, but says "we are beginning to look at other oils from all over the world."

So, the technique may help some day. But right now, "it's very very new," says Selby.

2ND LAW OF THERMODYNAMICS

By Claire Salvador

eyed with atlantic possibilities
far beyond our sensibilities
a depth so indigo
starfalls time in a recycled surge and flow
to obfuscate be wilder wisdom
from such limited dimension
(an inconceivable expanse of sea)
by our gracefoolish linearity

(onward ever we cycle
persisting because a
system can be ordered at
the expense of disordering
its surroundings)

so much randomness rearranged
we can never reach from this narrow frame
equilibrium (enlightenment)
through mere scientific disestablishment
destined to be continously reborn
into joy reversed, forlorn

THE GIRAFFE: A FAVOURITE TEXTBOOK ILLUSTRATION OF EVOLUTIONARY THEORIES

By Richard Peachey

High-school biology texts regularly present Darwin's theory of evolution in contrast with Lamarck's earlier explanation, and the organism most often used to illustrate the difference between the two views is the giraffe (e.g., Creager et al., pp. 233-240). Lamarck, it is said, told a story of giraffe necks becoming longer as the animals tried to stretch their necks to reach food (Law of Use and Disuse). The longer necks acquired in this way would then be passed on to their offspring (Law of Inheritance of Acquired Characteristics). Continued stretching over the generations led to today's long-necked giraffes. Darwin, on the other hand (it is said), proposed that early giraffes had necks of different lengths, some longer and some shorter (Variation). Limited food supplies meant that not all giraffes could obtain enough food to survive (Competition). Giraffes with longer necks could survive better and reproduce, passing their long-necked trait to their offspring, while those with shorter necks more often died off before being able to reproduce (Natural Selection). Over the generations the average giraffe neck became longer due to this process. But a number of things are wrong with this story:

1. Historically, there is no evidence that either Lamarck or Darwin used the giraffe as a significant part of their presentation of evolution. In his sixth edition of Origin of Species (though not in the first five editions), Darwin (201-203) did discuss the giraffe's neck, as part of a new section attempting to refute St. George Mivart's objections to the theory of natural selection (Gould 54). But then, Darwin explicitly accommodated Lamarckian thinking in his explanation; he accounted for the giraffe using natural selection "combined no doubt in a most important manner with the inherited effects of the increased use of parts" (202, cf. 133-139). Harvard evolutionist Stephen Jay Gould states: "When we look to presumed sources of origin for competing evolutionary explanations of the giraffe's long neck, we find either nothing at all or only the shortest of speculative conjectures. . . . The giraffe's neck just wasn't a big issue for the founders of evolutionary theory—not as a case study for arguing about alternative mechanisms, not for anything much at all. No data from giraffes then existed to support one theory of causes over another, and none exist now [emphasis added]" (21).

2. Female giraffe necks, on average, are two feet shorter than male necks! "If a longer neck were needed to reach above the existing forage line, then the females would have soon starved to death and the giraffe would have become extinct" (Davis and Kenyon 71).

3. Many researchers now suggest that the primary function of giraffe neck length is not for reaching leaves on tall trees, but for male combat ("necking"), or for spotting predators, or for shedding heat through increased skin surface area. All of these functions "have been viewed by prominent scientists as a chief reason for the evolution

of the long necks" (Gould 56f.). Darwin himself (202) alludes to some of these as alternate possibilities.

4. There is no fossil record showing a gradual increase in giraffe neck length. "All giraffes belong to a single species, quite separate from any other ruminant mammal, and [allegedly] closely related only to the okapi (a rare, short-necked, forest-dwelling species of central Africa). Giraffes have a sparse fossil record in Europe and Asia, but [alleged] ancestral species are relatively short necked, and the spotty evidence gives no insight into how the long-necked modern species arose [emphasis added]" (Gould 56).

5. The giraffe neck is not simply a longer version of an okapi neck; it is a well-designed "adaptational package" — a combination of unique features that work together to help the giraffe survive in its environment:

"To drive blood eight feet up to the head, the heart is exceptionally large and thick-muscled, and the blood pressure—twice or three times that of a man—is probably the highest in any animal" (Foster 409). "When a giraffe is standing in its normal erect posture, the blood pressure in the neck arteries will be highest at the base of the neck and lowest in the head. The blood pressure generated by the heart must be extremely high to pump blood to the head. But when the giraffe bends its head to the ground it encounters a potentially dangerous situation. It must lower its head between its front legs, putting a great strain on the blood vessels of the neck and head. The blood pressure plus the weight of the blood in the neck could produce so much pressure in the head that the blood vessels would burst. Mercifully, however, the giraffe is equipped with an adaptational package, including a coordinated system of blood pressure control. . . . Pressure sensors along the neck's arteries monitor the blood pressure, and can signal activation of other mechanisms to counter any increase in pressure as the giraffe drinks or grazes. Contraction of the artery walls [which have increased muscle fibre toward the head], a shunting of part of the arterial blood flow to bypass the brain, and a web of small blood vessels (the rete mirabile, or 'marvelous net') between the arteries and the brain all serve to control the blood pressure in the giraffe's head. Notice that adaptations require other adaptations so that a specialized organism such as the giraffe can function optimally" (Davis and Kenyon 71). The giraffe also has special "control valves in the jugular veins" (Foster 409); these "heavily valved veins control return of blood to the heart" (Davis and Kenyon 70).

"The lungs are oversize to compensate for the volume of dead air in the long trachea. Without this extra air-pumping capacity a giraffe would breathe the same used air over and over" (Foster 409). "The giraffe's lungs are eight times the size of those of humans, and its respiratory rate is about one-third that of humans. Breathing more slowly is necessary in order to exchange the required large volume of air without causing windburn to the giraffe's rippled 3.6 metres (12 feet) of trachea. When the animal takes in a fresh breath, the oxygen-depleted previous breath cannot be totally expelled. For the giraffe this problem is compounded by the long trachea that will retain more dead air than man can inhale in one breath. There must be enough lung volume to make this 'bad air' a small percentage of the total" (Hofland 12).

"Equally marvellous is the fact the blood does not pool in the legs, and a giraffe does

not bleed profusely if cut on the leg. The secret lies in an extremely tough skin and an inner fascia [fibrous connective tissue] that prevents blood pooling. This skin combination has been studied extensively by NASA scientists in their development of gravity-suits for astronauts. Equally helpful to prevent profuse bleeding is that all arteries and veins in the giraffe's legs are very internal. The capillaries that reach the surface are extremely small, and the red blood cells are about one-third the size of their human counterparts, making capillary passage possible. It quickly becomes apparent that these unique facets of the giraffe are all interactive and interdependent with its long neck. But there's more. The smaller red blood cells allow for more surface area and a higher and faster absorption of oxygen into the blood. This helps to retain adequate oxygen to all extremities, including the head" (Hofland 12).

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RESURRECTING DAMAGED NEURONS: ARE WE FIGHTING A HOPELESS BATTLE?

By Melvin Kwok, images by Jen Philpot

Spinal cord injury (SCI) has been documented throughout history. Early physicians described patients with this affliction to be conscious and aware yet unable to innervate their limbs. Up to the mid 1960s the proper course of treatment for SCI was to stabilize the spinal cord to avoid further injury. Repair, however, was deemed impossible. It wasn't until recent developments in molecular biology and biotechnology over the past decade that an understanding of the true molecular mechanisms behind SCI has been unraveled to provide a small glimpse of hope. To this day, there is not a cure for SCI but studies in biochemistry and genetics are starting to paint a clearer picture of the events that occur during SCI. With these insights comes the hope that there is a cure buried among the depths of this information. However, there exists some doubt as to whether a cure is really possible. Are scientists and physicians fighting a hopeless battle?

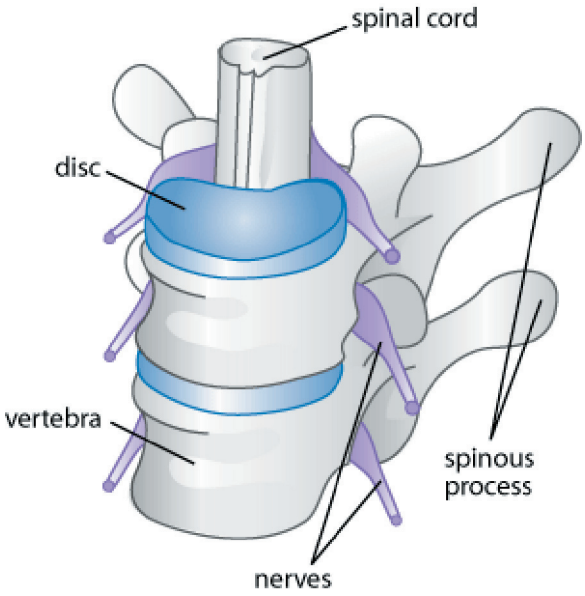
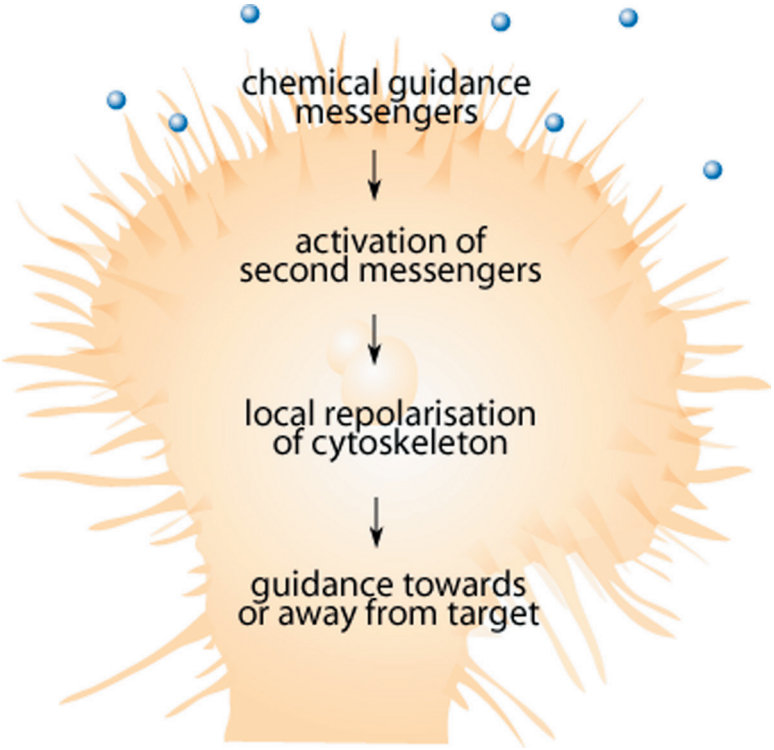


Figure 1: a diagram of a healthy spinal cord

Since the mid 1990s when the first neuronal chemical attractant was identified by Tessier-Lavigne and his group[1-3], numerous studies have identified chemicals that attract and repel growing neurons during development [4-6]. These chemical signals are important because they guide the developing neuron toward its target so that it can innervate specific neurons or muscle fibers. Among the chemicals and receptors being identified, there were some identified as responsible for destroying the path-finding portions of growing neurons known as the growth cone [4-8]. Growth cones are the sensing unit of the growing nerve cell that is designed to sense the surrounding environment in order to help make decisions about which direction to grow [9]. Immediately following SCI, injured nerves have demonstrated the ability to form new growth cones that explore the surrounding environment and try to make connections that have been lost due

to the injury [10,11]. Figure 2 shows how a growth cone should function either during development or in the event of nerve damage. Why, then, are previous connections not reestablished?



Severed neurons have difficulty growing and finding their original targets because the environment surrounding these new growth cones is a hostile one [12,13]. The nerve and the surrounding area has sustained significant trauma. As a result, the insulating myelin sheath has usually been torn and there is inflammation of the surrounding area in response to the injury [14,15].

Secondary Damage

Inflammation under normal circumstances is a welcomed primary immune response to injury because the series of events: heat, swelling, redness and pain are all useful to the body [16]. It promotes the recruitment of macrophages and other immune cells [16]. Macrophages are responsible for the clearance of cellular debris, production of toxins to kill any foreign bacteria or viruses as well as the generation of chemicals that promote the healing process [16]. The spinal cord is usually an immune privileged site, which means that immune cells normally do not have access to this area. However, when SCI is sustained, the barrier that separates the nervous system from the rest of the body is damaged and macrophages gain access.

Under these circumstances, inflammation can do more damage than good because the toxins that macrophages create are deadly not only to bacteria and viruses, but to the cells of the nervous system as well. These toxins include reactive oxygen and nitrogen species that are capable of destroying proteins and damaging cellular membranes [12-15]. Also, macrophages will produce and secrete chemicals known as interleukins. Interleukins have the ability to trigger the activation of many intercellular signals by activating nuclear factor kappa B (NFkB)[16-18]. Active NFkB will lead to the transcription and activation of genes that cause cellular decay and programmed

cell death, known as apoptosis, to occur. As a result, the normally positive actions of the immune cells turn out to have disastrous effects on the newly injured spinal cord, causing an effect known as "secondary damage" [12-15]. Through the actions of secondary damage, the initial mechanical damage done to the spinal cord and surrounding tissue is made worse by chemical damage due to inflammatory events [14]. Secondary damage will spread outwards from the point of injury killing damaged cells and, eventually, even healthy cells that are adjacent to the injured areas [14].

Growth Cone Collapse

Although the inflammatory response can make things worse, given enough time inflammation does dissipate, the reactive oxygen and nitrogen species are no longer created and the area is allowed to recover to a normal healthy state. Why, then, will the damaged neurons not send out new growth cones across the damaged area to reestablish previously severed connections?

When the nerve is damaged, the surrounding glia cells tend to up-regulate the production of chondroitin sulfate proteoglycans (CSPG)[19]. These proteins are normally responsible for facilitating interactions with cell adhesion molecules and growth factors necessary to maintain a healthy nervous system [19]. However, the over-production of CSPG causes the formation of a blockade that surrounds the broken nerve endings. This is known as the glial scar and has inhibitory effects towards axon regeneration [20]. Embedded in the glial scar are molecules that have been demonstrated in several lines of experiments to have repulsive effects on the growth cone [19,20].

Nogo protein was the first to be identified as having inhibitory effects towards newly developing growth cones [21]. It is a small transmembrane protein that is found on the inner-most layer of the myelin sheath [22]. This protein was identified by isolating protein fractions from rat brain myelin that displayed inhibitory effects on growing growth cones [22]. Through subsequent characterization, an extracellularly exposed region of 66 amino acids known as Nogo-66 was found to be responsible for the inhibitory effects of Nogo [23-25]. Myelin-associated glycoprotein (MAG), a protein initially implicated in the formation and maintenance of myelin sheaths was also identified to have inhibitory effects on neurite (cultured neuron) outgrowth [26,27]. MAG turns out to have dual roles of developmental significance: it can promote the outgrowth of immature neurons and inhibit the growth of mature neurons [28]. Finally, oligodendrocyte myelin glycoprotein (OMgp), a protein involved with the onset of myelination during the development of growing neurons was also identified to have neurite outgrowth inhibitory effects [29,30]. Nogo, MAG and OMgp together are responsible for the majority of the inhibitory effects observed at the site of injury [31].

Contrary to the conventional single ligand to single receptor model, all three of these proteins have strong affinity towards the same receptor known as the Nogo-66 receptor (NgR) [24,30,32]. The binding of just one of these inhibitory proteins to NgR on the newly developed growth cone is enough to cause the initiation of downstream events. Activated NgR has the ability to signal a downstream cascade. This cascade starts with the activation of Rho, one of the 3 main proteins responsible for actin remodeling within the cell [33-35]. Rho has the ability to

down-regulate the effects of Rac and Cdc42, causing destabilization of actin filaments and leading ultimately to growth cone collapse [33,34,36]. Once the growth cone has collapsed, the growing nerve axon can no longer grow in that direction until a new growth cone is formed.

Fighting A Hopeless Battle?

Certainly, with the evidence presented it would seem that regenerating a severed spinal cord is an impossible dream. There are layers upon layers of complications that make it difficult for neurons to grow even if they wanted to. But why are there so many obstacles in place? If the regeneration of peripheral nerves from a cut at your finger tip is possible, why is it so difficult to regenerate damage sustained by the central nervous system? Although the answers still elude scientists, progress in molecular medicine has provided important insights. For example, pyrrolidine dithiocarbamate, an anti-inflammatory drug, when administered to rat models of SCI demonstrate that an attenuated inflammatory response following injury will limit the degree of secondary damage sustained [37]. In mice, stopping NgR from binding to its partners demonstrated partial functional recovery following experimental SCI [38]. Ongoing experiments are underway to identify the genetic determinants that are responsible for inhibiting the growth of recovering neurons. Researchers are looking into treatments such as gene therapy, synthetic nerve fibers, surgical grafting approaches and even the use of specialized electromagnetic fields to stimulate the regeneration of damaged nerves [39-42]. With more work and an even greater understanding of the mechanisms mentioned in this article, potential cures are just around the corner. The once impossible battle against spinal cord injury may not be so hopeless after all.

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AFRICAN LION FAMILY OBJECTS TO THEIR PORTRAYAL IN RECENT DISCOVERY CHANNEL DOCUMENTARY

By Steve Caldes

Papa Lion (Carl)

First off, that tree we were lying around in the shade by, that wasn't even our tree. The producers literally brought in that tree and told us it was going to be our new tree. They said our tree—the tree we've lay under for years—"didn't have a river view" and was "a little smaller than what we were looking for." This was all said to me in front of my kids, I might add. Real class act that Discovery Channel!

Just like any family we like to have our place look clean, so we moved all the twigs and tried to flatten out some nice spots. We worked hard for over three hours, but did any of that stuff make the final cut? No way, José. They just showed me lying by the trunk of the tree resting my back and cleaning myself. We were cleaning and my damn sciatica starting acting up. I can't be on my feet as much as I used to. Betty knows that. The kids know that. But I come off as some lazy bum who just lies there and yawns all the time. You know, they were telling me to yawn! Do you really think lions yawn that much? Think again, Bub.

Goddamn Hollywood. I didn't even want to do this from the get go. I knew it would end up like this. Filmmakers and their agendas. I'd rather leave the kids with a poacher than a documentary filmmaker.

Mama Lion (Betty)

Carl is really all bent up about this. He was on us for weeks prior, showing us how to behave and whatnot. I must say, the kids were really good about it—I think they were excited, you know, being on TV and all. Halfway through the shooting Carl knew something was up. The director kept having the kids "play fight," and that got the kids going and pretty soon Carl was yelling at us all. "We're a family!" he kept saying. "Now goddamn it, let's act like one!"

I will say that when they had Carl mount me, like I was his play thing or something, that really got me upset—and I don't anger easily. That director just kept on saying that they needed some good mating shots and the stuff they were getting was great stuff. That's all the kept saying, "Oh yeah, great stuff. Really great stuff." Carl is usually much more of a romancer. And it's never in front of the kids like that.

Brother Lion (Tommy)

So like, I tell everyone by the grassy plains and everyone by the river that we're going to be on TV and boy, was that ever a mistake. We all looked like chumps. It's all in the editing. Sure, I guess I must have chased a couple zebras and gazelles before I caught one, but they just played my missed attempts all in a row—like two weeks worth of chasing—and talked about how sometimes we are unsuccessful. What the eff! Everybody by the river has been on me about that, calling me Wendy and telling me I hunt like girl. And when I did get one, they ran out of film and told me I had to do it again. So that last zebra that you see, they brought that guy in. He was old and slow and it was like, embarrassing chasing him down. I actually didn't even want to do it. Mama said she actually knew that zebra, they took a ceramics class together or something—but they made me do it. I like it better when it's just those old Land Rovers piled with wealthy people with hats and scarves on their heads taking pictures and clapping.

Sister Lion (Tiffany)

They took shots of me pooping! Can you believe that? Get a life! I was all the way over by the far shrubs, doing my business—my business!—and I turn and notice everybody right there behind me. One guy who was holding this huge mirror thing, reflecting light on my behind, was giggling. Getting his kicks. Becky and Vanessa say that that is an invasion of privacy and I can get all those guys fired. Becky said that her cousin's best friend knows this crocodile and she got this guy fired for touching her where she didn't give him permission to touch.

Those shots of me and Tommy cleaning each other! Fakes! We're family, we don't do that stuff. Gross! That was me and Todd Sherman. We were dating, but now Mama won't let me see him and when I finally snuck off and found him he was all, "I don't clean girls who poop on camera" and stuff. And those shots of me with like, my whole head in the zebra, they told me they stopped filming and I could dig in. I hadn't eaten in days. The buffet they had set up was horrible—little sandwiches and salads—I was starving.

ELSEWHERE AND OVERHEARD

By Caitlin Dowling

Overheard

"This is the first human-poultry interaction system ever developed."
Professor Adrian David Cheok who has been developing a system to enable people to stroke chickens over the Internet. I'm serious. (ananova.com)

"I've smelt one and tracked several since then and you cannot come much closer than that"
The fact that the Tasmanian "tiger" was officially declared extinct nearly 70 years ago does not deter Col and his fellow tiger hunters one bit. (BBC News)

"Wildlife and baby boys will be the losers."
Gwynne Lyons, toxic adviser to the WWF, regarding gender-bending chemicals in plastics that leak into foods affecting male development. (New Scientist)

Elsewhere

Llamas to offer a new way of tackling disease…
(http://news.bbc.co.uk/2/hi/health/4584409.stm)

Shocking new way to heal major wounds (Guardian)
(http://www.guardian.co.uk/life/news/story/0,12976,1495065,00.html)

Scientists crack the code for sarcasm? Yeah, right! (ananova.com)
(http://www.ananova.com/news/story/sm_1406656.html?menu=)

The US is officially searching for ET (Washington Post)
(http://www.washingtonpost.com/wp-dyn/content/article/2005/05/29/AR2005052900966.html)

THE GALLON CLUB

By Jonathan Cohen

Today, when I gave blood, the petite, Hispanic technician informed me that I was now a member of the Gallon Club. She explained that I had made eight visits to the blood center over the years, and, having given eight pints, was now in the ranks. Opening a white file cabinet, she removed a gaudy gold plastic pin in the shape of a drop of blood. It was emblazoned with a red cross on a white field, and in nearly invisible writing below the insignia, it said GALLON (1) DONOR. I graciously accepted the honor. Now, however, I was curious about "the rights and privileges appertaining thereto."

In a better world, there would be an actual Gallon Club, like the Yale Club of New York. Wearing the pin, I would proceed under the umbrella awning and be saluted by the doormen. I would pass up a grand, semi-circular marble staircase, and be invited into the Gallon Room, an expansive taproom where people drank two-quart steins of beer and ate French-dip sandwiches with plenty of jus. Light would come in through round leaded portholes set with brown glass panes. Later, in the wood-paneled great common room, complete with twinkling Christmas tree, there would be wine, hors d'oeuvres, and a speaker decrying the metric system—in particular, the SI units for volume. The audience would nod knowingly, as this was the traditional invective hurled yearly against the Gallon Club's malevolent counterpart, La Fédération des Litres. There, blood was not taken in good honest pints, but in units of 450 millilitres. How was anyone supposed to get a gallon out of those? How could they have a club for litres in the first place, as the closest they ever got to a litre was 900 millilitres, a paltry amount to begin with? An outrage! As a member of the Gallon Club, I would be bound to act. We would block the stairs to the building of those Litre chaps with fifty-gallon drums filled with miserable plonk. That would certainly show them a thing or two. Perhaps a couple of infernal mechanisms of death lifted from Jules Verne would assist us.

Somehow, I made it out of the donation center without incident.

NEW (THIS TIME AROUND) CONTRIBUTORS

Steve Caldes is a recent graduate of the MFA program at New Mexcio State University. He's still living in Las Cruces, NM, but that will soon change. Steve's girlfriend lives nine hours away by car. Steve is currently in bewteen cars. Steve's the two time winner of the Joe Somoza/Keith Wilson Prize for Poetry and the 2004 winner of the Frank Waters Fiction Award. His work has appeared here and there, most recently at McSweeneys.net. He's tall and will be a year older on July the 8th. His piece originally appeared in the most excellent but sadly retired haypenny.com

Justin Kahn writes to ward off death, bring order to chaos, and generally lighten the mood. His email is Justin_Kahn@hotmail.com. He welcomes spam, since most of his correspondence is blocked by the filter..

Willow King recently completed her BSc in Cell Biology & Genetics at UBC. She can be found celebrating this achievement at the beach.

We know Melvin Kwok is a graduate student at the Department of Pathology at UBC. Beyond that, we assume he is, perhaps, on holiday.

Richard Peachey is a public school science teacher with a background in biology and chemistry. He finds himself in agreement with T. H. Huxley and E. O. Wilson, both of whom dismissed the logical possibility of believing evolution and the Bible at the same time. Formerly a friend of the prevailing evolutionary worldview, he now takes his stand with the Bible, and with Jesus Christ, who taught concerning humankind, "At the beginning the Creator made them male and female."