THE SCIENCE CREATIVE QUARTERLY ISSUE TWO PART FOUR OF SIX AUGUST 22TH 2005 KACHOW!

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Our masthead, we think, will be forever evolving, although at present we have two Daves, a Bethany, a Caitlin, a Stephen, a Claire, a Russell, an Alex, a Justin, and Andrew and a Caley

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

Maybe 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**

WE SEEK EXPERIMENTAL FICTION - KEYWORD EVOLUTION.

Early in human history, a great divide occurred. Some - we mean writers - began writing hieroglyphics and posting stories on their websites. Others - we mean scientists - began making ruthless observations about the world. Inevitably, the writers and the scientists grew apart, and now we have the poet vs. the physicist; the blogger vs. the biologist. It is true that the divide between science and the arts runs deep, but we hope to do our part to bridge that gap. However, we realize that this is no easy task, and are aware that it may take at least a couple of weeks to fully reunite the sciences and the arts. In truth, there are pessimists among us who suggest a time scale closer to a full month.

We think that there are many ways to bring science and literature together. For example one might write stories about Albert Einstein or a molecule; compose poetry on a myriad of elegant observations. We could also try to write using some scientific pattern for device. For example the Science Creative Quarterly may invite stories that have even numbers playing a pivotal role. In this example, we would revel in stories that focused on love squares, but not love triangles. Overall, we would love to see scientific ideas providing some backbone to the fiction, and we want to offer this constraint as a gift to you.

So how's about this...

The Science Creative Quarterly is currently seeking character driven stories which show the evolution of a character [1]. But get this - evolution means many things to many people. For example a story could be modeled on S.J. Gould's punctuated equilibrium. Imagine a plot with great and rapid change from one stage of existence to the next. Doesn't that make you tingle? Besides, you probably have been meaning to write a story along that line for some time[2].

In any event, Darwin, Dawkin or our friend Louie would also work and also stand a good chance of getting past our intern - perhaps even our head editor. We, of course, will strive to showcase only the good, realizing that our readers are highly literate, shrewd and, I'm pretty sure, the folks who came up with the whole idea of evolution in the first place.

* * *

1. This idea is kind of inspired by a group of brilliant writers who constitute a group of writers known as Oulipo. They came up with models of literature based on constraints, many of a mathematical nature. You should Google Oulipo.

2. For fun, let's also say that you have until November 28th, 2005 to submit your story. Winner wins a hardcopy copy of Stephen Jay Gould's "The Structure of Evolutionary Theory" a book that is sure to impress the science and non-science savy if only because it is over fourteen hundred pages long and weighs only a little less than a newborn baby.

PROKARYOTES OF AMERICA UNITE.

by Stephen McNeil

The biotechnology community has been taken aback by a sudden and aggressive attack by an organization calling itself Humans for Bacterial Suffrage (HuBS). The group claims that an insidious culture of what it calls "eukaryotic oppression" is enslaving trillions of bacteria, subjecting them to perverse genetic experiments, and exploiting their labour in the execution of profitable biochemical reactions.

Says HuBS president David Clostridium, "Bacteria are routinely abducted from their natural habitats, sold on the open market, unwillingly subjected to invasive genetic manipulations, and forced to breed in captivity. Multiple generations of enslaved lifeforms are set to work expressing secondary metabolites at the hand of big corporations and research scientists. It's disgraceful. They aren't paid, the living conditions are terribly overcrowded, they don't even get dental. Just because you don't have a nuclear membrane, that doesn't mean you don't have rights, you know?"

Clostridium points out that his organization's principal goal is simply to increase awareness. "Few people realize that so-called "organic" products like Bt insecticide are prepared by bacterial slave labour. They say it's "natural", because it comes from a living organism. What's so natural about eating agar and living in a petri dish? About being sprayed with billions of your relatives onto a plant and forced to wage biological warfare on insects who never did anything to you? Consumers should have a choice. Consumers should be able to buy produce treated with free range Bt bacteria, who are properly compensated and valued for their work. It's just like slavery. Except, you know, without that miniseries starring Geordi LaForge."

Rhodia Chemicals markets Rhovanil Natural Vanilla, which is prepared via a biofermentation process carried out entirely by bacteria. Rhodia CEO Jean-Pierre Clamadieu was asked to comment on charges from HuBS that his company is exploiting single-celled organisms. "Look, these bacteria, they were eking out a miserable existence before they came to work for Rhodia, they were hiding in rocks, in the cold dirt, in rotting animal carcasses. They don't even have cable TV in some of these places, you know? Our e. coli workforce is proud and happy to be part of the Rhodia team." When asked if there had ever been any complaints from the bacterial workforce, Clamadieu shrugged. "Well, they don't have mouths."

Insiders at the US Republican party have expressed concerns that the ultimate goals of the bacterial suffrage lobby may include the granting of voting rights to all single-celled organisms. Estimates put bacteria population in United States at over $5x10^{26}$, a number far exceeding that of registered Republicans, and a recent poll reveals that 74% of prokaryotes either agree or strongly agree with the statement "George Bush is a lying crapweasel."

THAWING OUT NORTHERN MAMMALS.

By Bethany Lindsay

For mammals in the northern regions of the world, global warming must seem a little more real than it does to humans below the Arctic Circle. In 2004, the Arctic Climate Impact Assessment released a report called Impacts of a Warming Climate that revealed dire and immediate consequences of climate change for species ranging from polar bears to seals [1].

Temperatures in the Arctic are increasing at twice the rate of the rest of the world, according to the report. Sea ice is rapidly melting and breaking up at a rate of about 3% per decade[2], winters are shortening, and every year brings more precipitation[1].

All of these climatic changes are working together to change the landscape of the Arctic. Longer and warmer growing seasons are helping the treeline to shift northward, replacing tundra, and bringing insects and forest fires to the North[1].

Making the situation direr, other kinds of pollution might cause the Arctic ice to melt even more quickly. The carbon dioxide that is released when fossil fuels are burned acts as a greenhouse gas to warm the atmosphere, but soot, another byproduct of the same process, affects the ice directly. NASA scientists have found large accumulations of soot in Arctic ice, much of which can be traced back to worldwide industry. Soot in the air traps heat, and the dark colour of ice-bound soot absorbs heat, resulting in warmer temperature and melting ice[3].

Arctic mammals, specially adapted to the cold and ice, are witnessing the transformation of their habitat. Global warming and pollution will unquestionably change their lives and alter their species' futures.

In the short-term, some mammals may be able to adapt to the warming temperatures—red squirrels, for instance. A population of the animals near Kluane Lake, Yukon, has seen average temperatures rise by 2o C in the last three decades[4]. The growing season of white spruce, the cones of which are the squirrels' staple food, has lengthened dramatically, and the trees are producing up to 35% more cones within each squirrel's lifetime[4].

As the food supply steadily increased, the squirrels' behaviour began to change, too. In the last 10 years, they sped up breeding at an average rate of six days per generation, meaning that squirrels are giving birth 18 days earlier than they were a decade ago[4].

Most of the change in red squirrel breeding (about three and a half days per generation) comes because the animals have an innate ability to change their behaviour to suit a certain range of environmental conditions, a phenomenon known as phenotypic plasticity[4].

To a lesser degree, though, red squirrel behaviour is genetically evolving to keep up with their changing environment. About a day's worth of the change in birthing dates of each successive generation is actually a result of genetic changes that are being passed from mothers to daughters[4].

But most of the changes to the Arctic that are caused by global warming aren't quite as beneficial as an increase in food supply.

Polar bears are predicted to suffer in several ways from a warming arctic climate. The bears are listed as "of special concern" by the Committee on the Status of Endangered Wildlife in Canada (COSEWIC), because hunters over-harvest adult females and melting sea ice is threatening their access to food[5].

Bears roam the ice searching for air holes where seals come to rest and raise their young[6]. In the 20 years preceding the 21st century, sea ice declined by 14%[7], and some researchers have predicted that, within the next 50 years, ice will no longer be connected to the mainland[8]. If the prediction holds, it will become more difficult for polar bears to catch and kill seals while they use the ice.

The seals will be available to polar bears for a shorter time, too. Compared to 30 years ago, sea ice is breaking up more than two and a half weeks earlier[9].

Declining sea ice is already affecting the

health of polar bears. Their average mass is steadily declining, and some researchers predict that, within 100 years, female bears won't have enough food to reach the mass required for reproduction[6]. Of course, there are annual fluctuations in sea ice that would allow adequate feeding in some years, but those years will become fewer and farther between[6].

Female polar bears also use sea ice as a path to reach the sites they use as dens while they give birth[6]. Females return to the same denning sites year after year, and they swim and walk over the ice to get to them—and many of them build their dens on sea ice, which is becoming increasingly disconnected from land[6]. Thaws and break-up of ice mean that traditional dens will be harder to reach, and may be in precarious positions.

Sea ice isn't the only thing that's melting. Further south, many female polar bears build their dens on land, digging them into snow or permafrost soil[6]. In the past few years, warming temperatures have thawed several permafrost dens while polar bears are inside, causing the collapse of the dens and killing the female within6.

For polar bears, like many large mammals, survival as a species depends upon high numbers of fertile adult females. Many of the problems caused by global warming (declining sea ice, collapsing) are disproportionately affecting females. These females are difficult to replace because polar bears take several years to reach sexual maturity.

It should be noted, however, that some parts of the Arctic are changing in ways that may have nothing to do with global warming, and seem to run counter to the effects of climate change. Even though ice cover is decreasing in most of northern Canada, some marine animals are in danger because of increases in sea ice in other parts of the North.

Since the late 1970s, the extent of sea ice in the eastern Canadian Arctic—specifically Baffin Bay, Davis Strait, coastal West Greenland, and Lancaster Sound—has actually increased[10]. That means that there are fewer cracks and gaps in the ice to allow marine mammals like narwhals and bowheads to come up for air[10, 11]. At the same time, animals like polar bears that hunt those underwater creatures aren't able to catch them as easily—or in some cases, marine animals become trapped in the areas around limited numbers of air holes, making them easy prey for predators[10].

Arctic mammals won't only be affected by changes in climate and their abiotic environment. The northward shift in ecosystems will also bring southern organisms farther north, increasing competition[12]. In one extreme example, wolves, which tend to stay below the

treeline, may move farther north with the shifting of the boreal forests, bringing them closer to polar bear cubs, an easy source of food[12].

Although most of the impacts of global warming remain predictions rather than facts, there is no question that the climate of the North is changing. Arctic mammals will be forced to deal with warming temperatures and altered environments, and their responses will shape the futures of both their species and their ecosystems.

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EVOLUTION OF ALCOHOL SYNTHESIS

By P.Z. Myers

We need to appreciate beer more. Alcohol has a long history in human affairs, and has been important in purifying and preserving food and drink, and in making our parties livelier. We owe it all to a tiny little microorganism, Saccharomyces cerevisiae, which converts complex plant sugars into smaller, simpler, more socially potent molecules of ethanol. This is a remarkable process that seems to be entirely to our benefit (it has even been argued that beer is proof of the existence of God*), but recent research has shown that the little buggers do it all entirely for their own selfish reasons, and they've been busily making alcohol that has gone undrunk by humankind for tens of millions of years.

In order to explain how we know this, forgive me, but I must explain some very basic biochemistry, and summarize what cells do to extract energy from sugar. We start with a 6 carbon sugar molecule. As a first step, called glycolysis, enzymes in the cell snap the molecule in half, liberating a little bit of energy and producing two 3-carbon molecules, called pyruvate.

Pyruvate gets passed on to the next step, called the citric acid cycle. This is a series of reactions that breaks the 3-carbon chain down carbon by carbon, liberating yet more energy at each step. It's all the steps after glycolysis that extract the bulk of the energy from the sugar molecule, but there's a catch: these steps require oxygen to run (this is also called the aerobic pathway). No oxygen, no citric acid cycle. Glycolysis can run, but some of the reaction products (especially a compound called NADH) accumulate, and soon enough that reaction would get choked off, too.

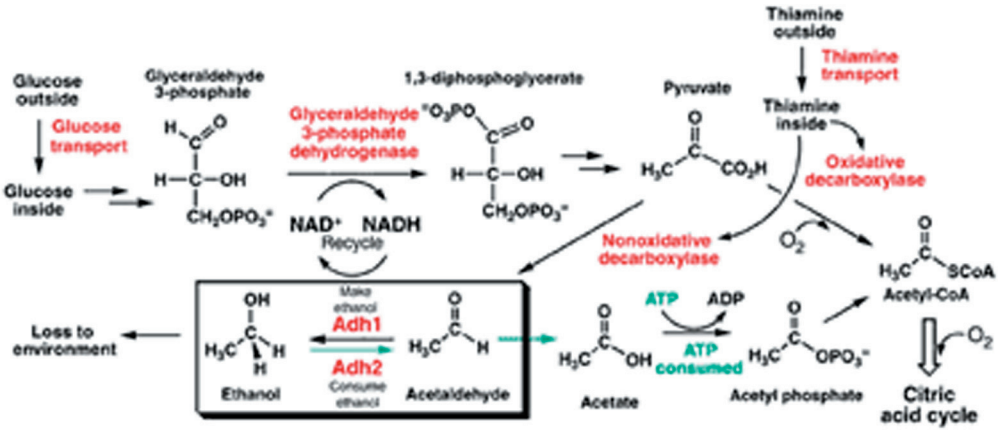
In the absence of oxygen, cells can continue to get that little bit of energy from glycolysis if only they can get rid of the accumulating reaction products somehow. In us, our cells do that by carrying out an additional reaction to convert excess pyruvate and NADH to another 3-carbon molecule, lactate, and NAD+. Lactate diffuses out of the cells and into the blood stream, forming lactic acid. When you are exercising anaerobically, that is, making your cells work harder than you can deliver oxygen to them, they limp along by dumping 3-carbon molecules in the form of lactic acid so they can keep burning sugar inefficiently. Once you're done working out, and your oxygen intake catches up, the lactate is converted back to pyruvate and can be burned completely and efficiently in the citric acid cycle.

Yeast do something different. If they are under anaerobic conditions, say, deep in the flesh of some decaying fruit, or in a wine bottle, they have the same problem: they want to keep their metabolism going by carrying out glycolysis, but to do that they have to get rid of accumulating products, somehow. They don't do it by making lactic acid, though (thank goodness—if they did, fermentation would produce a vinegary acid). Instead, they take the 3-carbon pyruvate and split off one carbon, producing CO2, which is given off as a gas. Any homebrew beer makers out there will be familiar with the idea of monitoring fermentation by observing the gas being produced.

The 2-carbon molecule left behind is called acetaldehyde. Acetaldehyde is further processed by an enzyme called alcohol dehydrogenase, Adh for short, which also recycles NADH. Adh converts the 2-carbon acetaldehyde into another 2-carbon molecule, ethanol. Alcohol. Booze.

Just like us, yeast produce this byproduct to keep going under anaerobic conditions, and when oxygen is available, they try to recover the energy in the alcohol. Familiar brewers' yeast has two forms of alcohol dehydrogenase: Adh1, which favors the production of alcohol from acetaldehyde, and Adh2, which more effectively runs the reaction in reverse, producing acetaldehyde from alcohol, and allowing the 2-carbon molecule to be fed back into the citric acid cycle.

If you'd rather see this in a simple biochemical diagram of the yeast pathways below: it says the same thing I just wrote up there.



Enzymes in red are associated with gene duplications that, according to the transition redundant exchange clock, arose nearly contemporaneously. The make-accumulate-consume pathway is boxed. The shunting of the carbon atoms from pyruvate into (and then out of, blue arrows) ethanol is energy-expensive, consuming a molecule of ATP (green) for every molecule of ethanol generated. This ATP is not consumed if pyruvate is oxidatively decarboxylated directly to acetyl-coenzyme A to enter the citric acid cycle directly (dashed arrow to the right). If dioxygen is available, the recycling of NADH does not need the acetaldehyde-to-ethanol reduction.

The yeast method of handling anaerobic conditions is not particularly efficient. They have to burn a little extra energy to prepare acetaldehyde for the citric acid cycle (the steps in green in the diagram above), which wouldn't be necessary if they used a 3-carbon intermediate as we do. So, one question is why they use a relatively inefficient method to carry out anaerobic metabolism.

One explanation is that humans are responsible—we've been selecting for yeast that produce intoxicating byproducts. A prediction from that would be that alcohol production would be a relatively recent innovation. An alternative explanation is that yeast have been doing this as a clever strategy—flooding their environment with a poison that that they can tolerate but that other

microorganisms cannot is a way to limit competition for resources. A prediction from this is that the yeast evolved first to produce ethanol, and only secondarily evolved the ability to recycle it. A recent study strongly supports the latter hypothesis.

First, molecular clock analysis of various yeasts suggests that the ethanol enzymes began to diversify about 80 million years ago…at about the time flowering plants started producing fleshy fruits (that meteor at the end of the Cretaceous may have had an abrupt impact on the lives of dinosaurs, but I wonder if the explosion of flowering plant species before that may have had an equally profound, if more drawn out, effect). Face it, people, the chemistry of beer is for the benefit of yeast, and didn't evolve for our enjoyment. Or if it were the result of domestication, it was by the undiscovered species Zymurgosaurus dipsomanius, not Homo sapiens.

The second line of evidence is very cool. It would be instructive to be able to directly examine the metabolism of yeast from 80 million years ago, and measure for ourselves the activity of their Adh enzyme. We don't have a time machine, unfortunately, but we do have the ability to reconstruct ancient genes.

The authors compared the sequences of Adh1 and Adh2 from S. cerevesiae and from 15 other Adh homologs in other yeast species. They then calculated the maximum likelihood gene sequence for the last common ancestor of these enzymes, the primordial alcohol enzyme, which they called AdhA. They then took modern yeast, removed their Adh1 and Adh2 genes, and replaced them with AdhA. Voilà, they have yeast from the Age of the Dinosaurs.

They then analyzed the chemical kinetics of this enzyme. The question was whether it was more like Adh1, the enzyme that primarily makes ethanol, or whether it was more like Adh2, the enzyme that primarily consumes alcohol. Did yeast evolve this enzyme to make a byproduct to inhibit its competitors, or did it evolve it to eat this byproduct?

The answer is that it was more like Adh1, and that early yeast were brewers, not drinkers.

"Notably, the kinetic properties of the remaining ancestral AdhA candidates resembled those of Adh1 more than those of Adh2. From this, we inferred that the ancestral yeast did not have an Adh specialized for the consumption of ethanol, similar to modern Adh2, but rather had an Adh specialized for making ethanol, similar to modern Adh1. This suggests that before the Adh1-Adh2 duplication, the ancestral yeast did not consume ethanol. This implies that the ancestral yeast also did not accumulate ethanol under aerobic conditions for future consumption and that the make-accumulate-consume strategy emerged after Adh1 and Adh2 diverged. These interpretations are robust with respect to the ambiguities in the reconstructions."

We can assemble a history of yeast fermentation from this information now. The first step was the gradual evolution of efficient alcohol-producing enzymes that allowed the yeast to colonize and exploit rotting fruit exclusively. This occurred a very long time ago, in the Cretaceous. Next, there was a gene duplication event that produced two copies of Adh; initially, both would have done exactly the same thing, just allowing the lucky duplicators to pump out alcohol even faster. With two copies, though, one would have more freedom to drift and change its enzymatic prop-

erties without serious consequence to the owner. One fortuitous change would be a shift in enzyme kinetics in one copy to better promote conversion of alcohol back to acetaldehyde and enter back into the citric acid cycle. So, first they learned how to make an environmental poison to give them exclusive access to a food source, and then that same machinery was adapted to better allow them to eat that poison, permitting them recover some of the energy lost in secreting it.

Notes:

* "Beer is proof that God loves us and wants us to be happy," Benjamin Franklin.

* * *

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MOTHER GOOSE AND THE SCIENTIFIC PEER REVIEW PROCESS.

by David Ng

Jack and Jill went up the hill.
To fetch a pail of water.
Jack fell down and broke his crown.
And Jill came tumbling after.

First of all, we are not sure there's enough clarity in this text. Scientific literature, in particular, should leave little room for confusion. Where exactly did Jack fall down? Into the well? A little ways down the hill? All the way down the hill? It's just too vague. Worst still, we're not convinced that the science conducted is of high enough caliber. I mean really, who would be stupid enough to put a well on the top of a hill? In conclusion, we feel that this manuscript should be rejected in its current state, but are not opposed to viewing a revised version in the near future.

Twinkle twinkle little star.
How I wonder what you are.
Up above the sky so high.
Like a diamond in the sky.
Twinkle twinkle little star.
How I wonder what you are.

Initially, we were quite intrigued by your work, especially since it appeared to contain several elements that merit genuine excitement. However, it was then brought to our attention that this body of work had remarkable similarities to a previously published report (The Alphabet Song). It was upon further investigation, that our worst fear was confirmed to be true - that this manuscript constitutes an act of plagiarism. We must state that we feel this to be a serious breach of scientific ethics, and must therefore strongly decline your manuscript.

Humpty Dumpty sat on a wall.
Humpty Dumpty had a great fall.
All the King's horses and all the King's men.
Couldn't put Humpty together again.

Although otherwise promising, the reviewers felt that the research in its current state is incomplete. Quite frankly, it was agreed that your principle subject needed to be put back together again. Several of the reviewers suggested courting the expertise of a mathematician who could perhaps create an appropriate algorithm to solve this problem. Alternatively, one reviewer suggested glue. As a final note, questions were also raised regarding the treatment and well being of Mr. Dumpty. Why exactly was he made to sit on the wall? And why exactly would you allow horses (of all things) to put him together again. No matter, the reviewers overall impression was that if you were able to address each and every one of these issues, they would see no problem entertaining a revised version.

Hey diddle diddle, the cat and the fiddle.
The cow jumped over the moon.
The little dog laughed, to see such a sight.
And the dish ran away with the spoon.

The reviewers felt that not enough data was presented to support your claims. For example - how many times did your group observe the cow jumping over the moon? From the text and supporting figures, it would appear that you base this conclusion on one data point as no calculations regarding standard deviations were presented. As an analytical journal of high repute, the reviewers felt that this is simply not acceptable. In addition, several of the reviewers felt that the word 'diddle' was inappropriate, and should have been replaced by the more scientifically correct, 'Hey fornicate fornicate." Because of these, and other problems, we are sorry to inform you that your manuscript has not been accepted for publication.

Rub a dub dub, three men in a tub.
And who do you think they'd be?
The butcher, the baker, the candlestick maker.
Turn'em out, knaves all three.

Thank you most kindly for allowing us to see this marvelous manuscript. We feel that it is a great privilege that you and your colleagues decided to submit it to our journal. We truly feel that it represents seminal work that could even one day lead to a Nobel prize. To be frank, we were quite surprised to receive your submission, in that we all felt it could have easily been accepted by the more high profile publications (The Nature and Science journals for instance). In any event, we are very pleased to inform you that, we, the reviewers are unanimous in our decision to accept your manuscript.

POEMS.

By Rhea Tregebov

The Big Picture

The man on the radio is speaking of his specialty
and passion, theoretical astrophysics. The interviewer
frets the big ones, wants to know what is there, on
the other side of the end of the universe. Wants
to know what it was was then before the Big Bang happened.
I've heard these questions before; heard my son, at seven,
brood over them, though not so much now as when he was five.
You try, at some point, to place yourself in the here and now.
The astronomer has kept his mind open to these wide questions,
the ones we don't want to think of, the ones that make us
dizzy.
I imagine him lying sleepless, restless in the stars'
old light, the sheets furrowed anxiously around him, not
listening for a child's cry or cough, not making grocery
lists or emotional agendas but insomniac with worry
over the big picture. On the radio his voice
is eager: before the Big Bang, everything that we are now
was already all there, compressed in some inexplicable form.
(And inside the ovum in every girl child, I think, compressed
in some inexplicable form.) What is there, on the other side of
the end of the universe, is before time began.
Though in the universe itself, the stuff
we are made of constitutes only ten percent
of what is there. The rest is either void or
(again) inexplicable. As yet. Meaning,
I suppose, that here is now. I knew that.
It feels like life, the little bit you can grab onto.

At or Above the Earth's Surface

Saturday at dinner, as we spoon into dessert
(crème anglaise with raspberry sauce on one side
chocolate on the other), our friend, who is a physicist,
speaks to us of time as yet another human
fallacy. That it's only through the usual egotism
of our species that we imagine its existence;
that looked at properly it may well have no beginning
or end to it. I probably don't understand him fully,
despite my ignorant love for physics.
But I have experienced something like
the a-chronological, standing for brief seconds
not in time, absorbed in some moody
perception, shop window; caught in stasis, being –
how glad I am of the forsythia today, for instance,
how it keeps coming back and coming back –
if that's what he means.
And I have felt the elasticity of time,
especially its slowness in those moments when
I was moving at or above the earth's surface,
the news of someone's death the event
that splits time into *before* and *after*.
As though in those moments in between
my grief for someone else left me
perhaps immortal, not part of time. I link this
with the desire I feel sometimes for death;
the wanting it all to stop, to stop, to step
outside of it all, outside of
everything the mind frets over busily,
all the questions that are for me anything
but philosophical. The questions I call
suffering; the question, I guess, of time,
of trailing the long long freight train
of our lives: the accumulated memories of this
bit of light striking that plane, smell
of decayed oak-leaves a resonance in the primitive
brain. All these things packed in electrical
circuits in the walnut our bony skulls
protect, synapses flickering like
the prettiest Christmas lights so that
if surgeons stimulate that bunch of cells
we taste our mother's breast milk; this bunch
and it's pale red tulips by the concrete steps

at Matheson Avenue. That the transparency we
imagine of ghosts is the transparency of memory.
A collision of times. And who can tell me why
it is that often when we make love I am among the arches
under the New Sacristy in Florence where
we went up, eighteen and nineteen years old,
to see for the first time
Michelangelo's tomb for the Medicis? What
does lovemaking have to do with
Renaissance art? Or is it arches, is it
the caves down there, is it that
the trigger this moment is just death,
that I'm thinking of how, making love, we go
into it, the great current of procreation, time?

The Dinner Table, the Tulip

So what do we do with this,
this world, this uncertain spring,
the tulips still holding, things green and cold.
Take the tulips, composed, driven to yellow or rose
from their chilly green, given to order,
unfolding. The colour they move towards
held for a day, or a week, contingent
on the weather, accident. Then paling or darkening
into other shades, then the quick
or slow decomposing. Coming to grief.
To being not tulips. Does rot
have its own order? I think not.
Theorists see things moving
to degeneration, some, and looking down,
I might be inclined to agree, skidding down
to an agreement since more than the weather
this spring is uncertain. Systems large
and small are flawed, disintegrating.
Think of anything: my respiratory system,
the world's. Today I run along the cul-de-sac
in the swanky end of our neighbourhood.
As always, there are vans parked in the driveways.
Things are being taken care of, expensive systems
in need of maintenance. The rest of us
are short on money, time, love.
And you so careless, the roof needing repair,
plaster crumbling from the living-room ceiling,
faith battered, struck by dilemma. Ah you.
It's a good thing it is spring, my faith still holding,
in me, this body running along concrete,
however the lungs rasp. Spring inclines me
elsewhere, to lean towards other theories –
anti-chaos, the universal yearning
towards order. Setting the table just so.
The tulips in the right vase.
Yearning, yes, the scientist on tv wanting
it to be the case that we are at home
in the universe, that life is inevitable,
"the consequence of broad avenues of possibility,
not back lanes of improbability." Although,
agnostic, I might settle for back lanes.
I've loved their rough edges, seamy sides:

rusted garbage cans overturned, the
opportunity for scrounging, the
possibility of unexpected plenty.
A clump of fat white violets beside the garage
and beside them, blue ones, their pansy faces
attentive. Not an aberration but a plan.
Agnostic, I bless those looking
for "a science of emergence, of complexity,"
looking for a way to model complicated systems
like the dinner table, the tulip. And I
agree. The ultimate question not only
of science, but ours *why is there*
something rather than nothing.

ISLET AMYLOID - A CULPRIT IN TYPE 2 DIABETES.

By Agnieszka Klimek, images by Jen Philpot

It is estimated that nearly 194 million people worldwide have diabetes. This is an increase from the 1995 global estimation of 135 million which was published in a World Health Organization study in 1998[1]. The International Diabetes Federation reconfirms that type 2 diabetes, which is the non-insulin dependent type, constitutes about 85% to 95% of all diabetes cases in developed nations and accounts for an even higher percentage in developing nations. Diabetes continues to affect increasing numbers of people around the world while public awareness remains low.

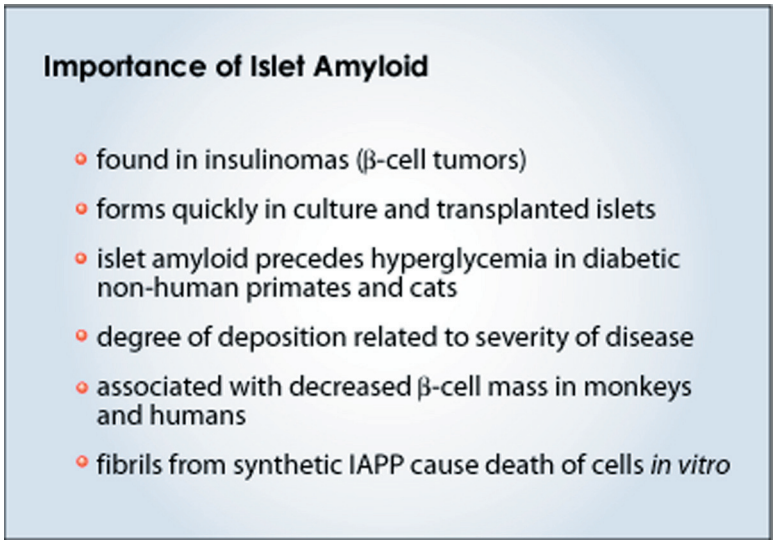


Figure 1. The importance of Islet Amyloid

What is type 2 diabetes?

Type 2 diabetes is a disease often associated with growing obesity in the adult population. More recently, changes in diet and reduced physical activity are probable contributors to the increased occurrence of this disease in children[2]. Type 2 diabetes is characterized by elevated blood glucose levels due to problems with the secretion of insulin and its action within the body. Over time, the blood glucose levels increase while insulin secretion steadily decreases. This is primarily due to the gradual damage to insulin-producing beta cells in the pancreas by the formation of toxic protein-containing deposits within the pancreatic islets. At autopsy, these deposits, which replace the endocrine cells within the islet, have been demonstrated in up to 90% of individuals with type 2 diabetes. These toxic deposits are now recognized as a pathological feature of this disease[4].

What is Amyloid?
Amyloid describes a deposition of protein generally located on the outside of a cell. The name amyloid comes from the latin word amylum which means starch. Initially scientists thought that the deposits were starch-like but later discovered that they were actually protein-like. Amyloid deposits can be classified into systemic amyloidosis where deposits are present in many organs, or local amyloidosis where deposits are confined to a specific tissue.

Discovery of Amyloid.
100 years ago a pathologist, Eugene Opie, described the occurrence of 'hyaline degeneration of the islets of Langerhans' in patients with hyperglycemia[2] and, not knowing that these pancreatic islets contained the insulin producing cells, suspected that there may be a relationship between the presence of these hyaline deposits and the development of diabetes. Later, the hyaline degeneration was described as amyloid deposit.

Amyloid and Disease.
Local amyloidosis is generally related to aging diseases. The two best examples of localized amyloidosis are Alzheimer's disease and type 2 diabetes mellitus (DM2). Localized amyloid deposition results from the production of a unique polypeptide, which contains an amyloidogenic sequence and is capable of forming a beta-pleated sheet structure necessary for these deposits to aggregate. In Alzheimer's the unique peptide is the beta-amyloid protein (Abeta) and in type 2 diabetes it is the islet amyloid polypeptide (IAPP) also known as amylin[4]. In order for fibrils to form in neurons and in pancreatic islets, in Alzheimer's and DM2 respectively, the unique peptides must be present.

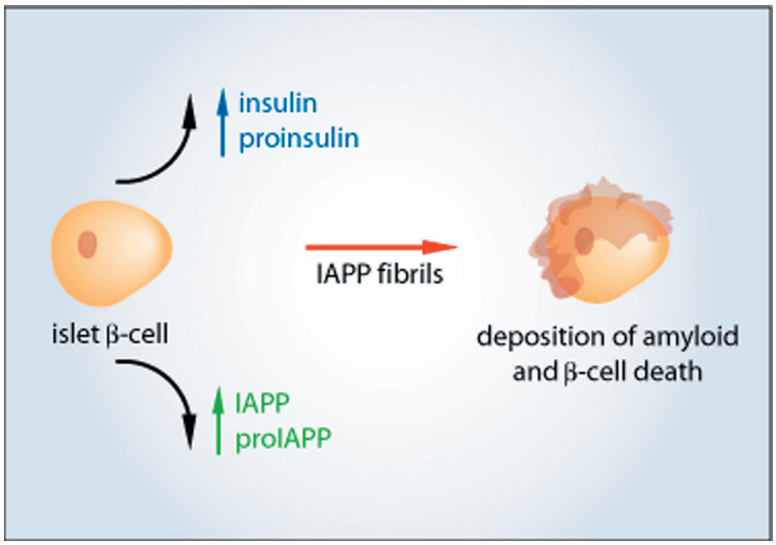


Figure 2. IAPP fibrils can cause a deposition of amyloid on beta-cells resulting in cell death

Islet amyloid and type 2 diabetes – is there a relationship?
Over the years, the ma-jor peptide component of amyloid was difficult to identify due the ex-

treme insolubility of the deposits. However, the peptide was finally identified by Per Westermark in 1986. The initial studies were performed on insulinoma tissues derived from insulin-producing tumors in pancre-atic islet b-cells. The studies resulted in the identification of the peptide known today as islet amyloid polypeptide (IAPP)[4]. IAPP is an important player in the formation of amyloid deposits and the progression of type 2 diabetes.

IAPP is a 37 amino acid peptide which is co-stored and co-secreted with insulin in a molar ratio of approximately 1:100 (IAPP: insulin) from the beta-cell secretory granules of the pancreatic islets. IAPP is derived from a larger molecule called proIAPP and processed by two enzymes called prohormone convertases (PC) – PC 1/3 and PC2, which are also responsible for the conversion of proinsulin to insulin[3].

In type 2 diabetes, the proinsulin to insulin conversion is impaired due to a processing defect in the beta-cells. It is thought that since IAPP and insulin are co-stored and co-secreted they likely use similar pathways; it is possible that the IAPP processing pathway is also impaired in cells affected by type 2 diabetes. This impairment may lead to the accumulation of an incompletely cut precursor molecule, proIAPP, within the beta-cells. Since proIAPP also contains the amyloidogenic portion of the mature human IAPP, it may aggregate within the beta-cells, ultimately contributing to cell death. This suggests that islet amyloid deposition is an early event in the pathogenesis of type 2 diabetes and that it is associated with a reduction in insulin release well before the onset of hyperglycemia[3,4,5].

hIAPP transgenic mice develop amyloid deposits.

Initial in vivo models for islet amyloidosis were difficult to establish. Although the IAPP amino acid sequence is conserved between species, only humans, non human primates and cats express a form of IAPP that is able to form fibrils. Mice and rats, the standard animals used for laboratory studies, produce IAPP that does not form the fibrils because it lacks the proline amino acid residue necessary for the formation of the beta-pleated sheet. For this reason, scientists were unable to study the function of the human amyloidogenic form of IAPP. Recently, with the use of transgenic mouse technology, scientists were able to study the effects of human IAPP overproduction in an obese diabetic mouse model. The expression of the IAPP gene is specifically targeted to islet beta-cells so that hIAPP is only expressed in these cells. Mice that develop diabetes spontane-ously show higher blood glucose levels and lower insulin levels as well as the presence of amyloid deposits in their beta-cells. This suggests that the high fat diet leading to the obesity of these mice may be partly responsible for the development of both an IAPP and an insulin processing defect that could lead to the accumulation of amyloid deposits in the beta-cells[6].

Figure 3.The difference between the human and mouse IAPP protein sequences

Other roles for IAPP?

The known functions of IAPP are related to glucose metabolism: IAPP suppresses insulin-mediated glucose uptake in skeletal muscle and prevents the release of insulin with glucose stimulation. Other suggested roles include regulation of renal filtration, calcium homeostasis and vasodilation. Although a definite function for IAPP has not been clearly indicated, its role in islet amyloid formation in the pancreas of individuals with type 2 diabetes is clear – it has a role in the pathogenesis of the islet beta-cell dysfunction[3].

Targeting amyloid for therapy.

There is evidence to suggest that aggregation of the IAPP fibrils plays an important role in beta-cell death and the progression of type 2 diabetes. To prevent amyloid accumulation, the following are possible therapeutic targets:

1. reducing IAPP release from the islet to decrease the precursor pool of available amyloidogenic IAPP
2. using anti-diabetic agents that act by decreasing IAPP release by b-cells eg. metformin, thiazolidinediones
3. development of inhibitors to target the amyloidogenic region of the IAPP mol ecule using synthetic peptides

Summary.

Islet amyloidosis is both a consequence and a cause of type 2 diabetes. It is induced by insulin resistance, stimulating not only insulin but IAPP secretion, and contributes to insufficient insulin in the body by promoting b-cell failure. In this respect, IAPP and islet amyloid provide an at-tractive target for diabetes therapy2.

References.

1) http://www.idf.org/home/index.cfm?node=264

2) Hoppener JW, Nieuwenhuis MG, Vroom TM, Ahren B, Lips CJ. (2002) Role of islet amyloid in type 2 diabetes mellitus: consequence or cause? Mol Cell Endocrinol. Nov 29; 197(1-2):205-12.

3) Hull RL, Westermark GT, Westermark P, Kahn SE. (2004) Islet amyloid: a critical entity in the pathogene-sis of type 2 diabetes. J Clin Endocrinol Metab. Aug;89(8):3629-43.

4) Kahn SE, Andrikopoulos S, Verchere CB. (1999) Islet amyloid: a long-recognized but underappreciated pathological feature of type 2 diabetes. Diabetes. Feb; 48(2):241-53.

5) Clark A, Nilsson MR. (2004) Islet amyloid: a complication of islet dysfunction or an aetiological factor in Type 2 diabetes? Diabetologia. 2004 Feb;47(2):157-69. Epub 2004 Jan 13.

6) Porte D Jr, Kahn SE. (2001) beta-cell dysfunction and failure in type 2 diabetes: potential mechanisms. Diabetes. Feb;50 Suppl 1:S160-3.

Glossary.

Amyloidosis – A disorder that results from the abnormal deposition of a particular protein, called amyloid, in various tissues of the body. Amyloid protein can be deposited in a localized area and not be harmful, or it can cause serious changes in virtually any organ of the body.

Beta (b) cell – A type of cell in the pancreas. Within the pancreas, the beta cells are located in areas called the islets of Langerhans where they constitute the predominant type of cell. The beta cells make and release insulin, a hormone that controls the level of glucose (sugar) in the blood.

Endocrine – Pertaining to hormones and the glands that make and secrete them into the blood-stream through which they travel to affect distant organs eg. Islets of Langerhans in the pancreas, which secrete insulin.

Hyperglycemia – An elevated level specifically of the sugar glucose in the blood. Often found in diabetes mellitus. It occurs when the body does not have enough insulin or cannot use the insulin it has to turn glucose into energy. The signs of hyperglycemia are polydipsia (a great thirst), polyuria (a need to urinate often), and a dry mouth. The term "hyperglycemia" comes from the Greek "hyper-" = high, over, beyond, above + "glykys" = sweet + "haima" = blood. High sweetness (sugar) in the blood.

Islets of Langerhans – Known as the insulin-producing tissue, the islets of Langerhans do more than that. They are groups of specialized cells in the pancreas that make and secrete hormones. Named after the German pathologist Paul Langerhans (1847-1888), who discovered them in 1869, these cells sit in groups that Langerhans likened to little islands in the pancreas. There are five types of cells in an islet: alpha cells that make glucagon, which raises the level of glucose (sugar) in the blood; beta cells that make insulin; delta cells that make somatostatin which inhibits the release of numerous other hormones in the body; and PP cells and D1 cells, about which little is known. Degeneration of the insulin-producing beta cells is the main cause of type I (insulin-dependent) diabetes mellitus.

Pancreatic beta cell – A type of cell in the pancreas that makes insulin. The pancreas is a fish-shaped organ that stretches across the back of the abdomen behind the stomach. Within the pan-creas there are areas that are called the islets of Langerhans. The beta cells constitute the pre-dominant type of cell in the islets.

A SIMPLE METHOD FOR RATING POTENTIALLY REVOLUTIONARY CONTRIBUTIONS TO PHYSICS.

by John Baez

A minus 5 point starting credit.

1 point for every statement that is widely agreed on to be false.

2 points for every statement that is clearly vacuous.

3 points for every statement that is logically inconsistent.

5 points for each such statement that is adhered to despite careful correction.

5 points for using a thought experiment that contradicts the results of a widely accepted real experiment.

5 points for each word in all capital letters (except for those with defective keyboards).

5 points for each mention of "Einstien", "Hawkins" or "Feynmann".

10 points for each claim that quantum mechanics is fundamentally misguided (without good evidence).

10 points for pointing out that you have gone to school, as if this were evidence of sanity.

10 points for beginning the description of your theory by saying how long you have been working on it.

10 points for mailing your theory to someone you don't know personally and asking them not to tell anyone else about it, for fear that your ideas will be stolen.

10 points for offering prize money to anyone who proves and/or finds any flaws in your theory.

10 points for each new term you invent and use without properly defining it.

10 points for each statement along the lines of "I'm not good at math, but my theory is conceptually right, so all I need is for someone to express it in terms of equations".

10 points for arguing that a current well-established theory is "only a theory", as if this were

somehow a point against it.

10 points for arguing that while a current well-established theory predicts phenomena correctly, it doesn't explain "why" they occur, or fails to provide a "mechanism".

10 points for each favorable comparison of yourself to Einstein, or claim that special or general relativity are fundamentally misguided (without good evidence).

10 points for claiming that your work is on the cutting edge of a "paradigm shift".

20 points for emailing me and complaining about the crackpot index, e.g. saying that it "suppresses original thinkers" or saying that I misspelled "Einstein" in item 8.

20 points for suggesting that you deserve a Nobel prize.

20 points for each favorable comparison of yourself to Newton or claim that classical mechanics is fundamentally misguided (without good evidence).

20 points for every use of science fiction works or myths as if they were fact.

20 points for defending yourself by bringing up (real or imagined) ridicule accorded to your past theories.

20 points for each use of the phrase "hidebound reactionary".

20 points for each use of the phrase "self-appointed defender of the orthodoxy".

30 points for suggesting that a famous figure secretly disbelieved in a theory which he or she publicly supported. (E.g., that Feynman was a closet opponent of special relativity, as deduced by reading between the lines in his freshman physics textbooks.)

30 points for suggesting that Einstein, in his later years, was groping his way towards the ideas you now advocate.

30 points for claiming that your theories were developed by an extraterrestrial civilization (without good evidence).

30 points for allusions to a delay in your work while you spent time in an asylum, or references to the psychiatrist who tried to talk you out of your theory.

40 points for comparing those who argue against your ideas to Nazis, stormtroopers, or brownshirts.

40 points for claiming that the "scientific establishment" is engaged in a "conspiracy" to prevent your work from gaining its well-deserved fame, or suchlike.

40 points for comparing yourself to Galileo, suggesting that a modern-day Inquisition is hard at work on your case, and so on.

40 points for claiming that when your theory is finally appreciated, present-day science will be seen for the sham it truly is. (30 more points for fantasizing about show trials in which scientists who mocked your theories will be forced to recant.)

50 points for claiming you have a revolutionary theory but giving no concrete testable predictions.

This piece can also be found at http://math.ucr.edu/home/baez/crackpot.html

ELSEWHERE AND OVERHEARD

By Angela Genusa

Overheard:

"The only depressing thing is that the representation is very small."
Christian Kell at the University of Frankfurt in Germany, on where the penis is represented on the brain's map of body parts. (New Scientist)

"Having a distinctive dog [an afghan] means that if we'd [ended up with] a dachshund we'd know that something funny had happened."
Gerald Schatten at the University of Pittsburgh School of Medicine, one of the researchers who worked on a project that produced the world's first cloned dog. (New Scientist)

"As far as I know, no other species has been observed [sexually] sampling nearly as many candidates as the California fiddler crab."
Catherine deRivera at the University of California in San Diego, on how the Californian fiddler crab females often check out more than 100 males even inspecting their bachelor pads before selecting their mate. (Physorg.com)

"We're not talking about flatulence."
University of California researcher Frank Mitloehner, whose research on cow emissions has been dismissed as "fart science." a label he says doesn't do justice to the seriousness of his work. (Washington Post)

"Then you add in (that) you got sick on strawberry ice cream. You want them to think about the getting sick aspect of the experience."
Professor Elizabeth Loftus, at the University of California Irvine, lead author of a study that shows that fabricated memories might aid weight loss. (Earthtimes.org)

Elsewhere:

Sperm-free sex keeps hens happily faithful
(http://www.newscientist.com/article.ns?id=dn7657>http://www.newscientist.com/article.ns?id=dn7657)

Drinking improves thinking
(http://www.guardian.co.uk/australia/story/0,12070,1542092,00.html%3ehttp://www.guardian.co.uk/australia/story/0,12070,1542092,00.html)

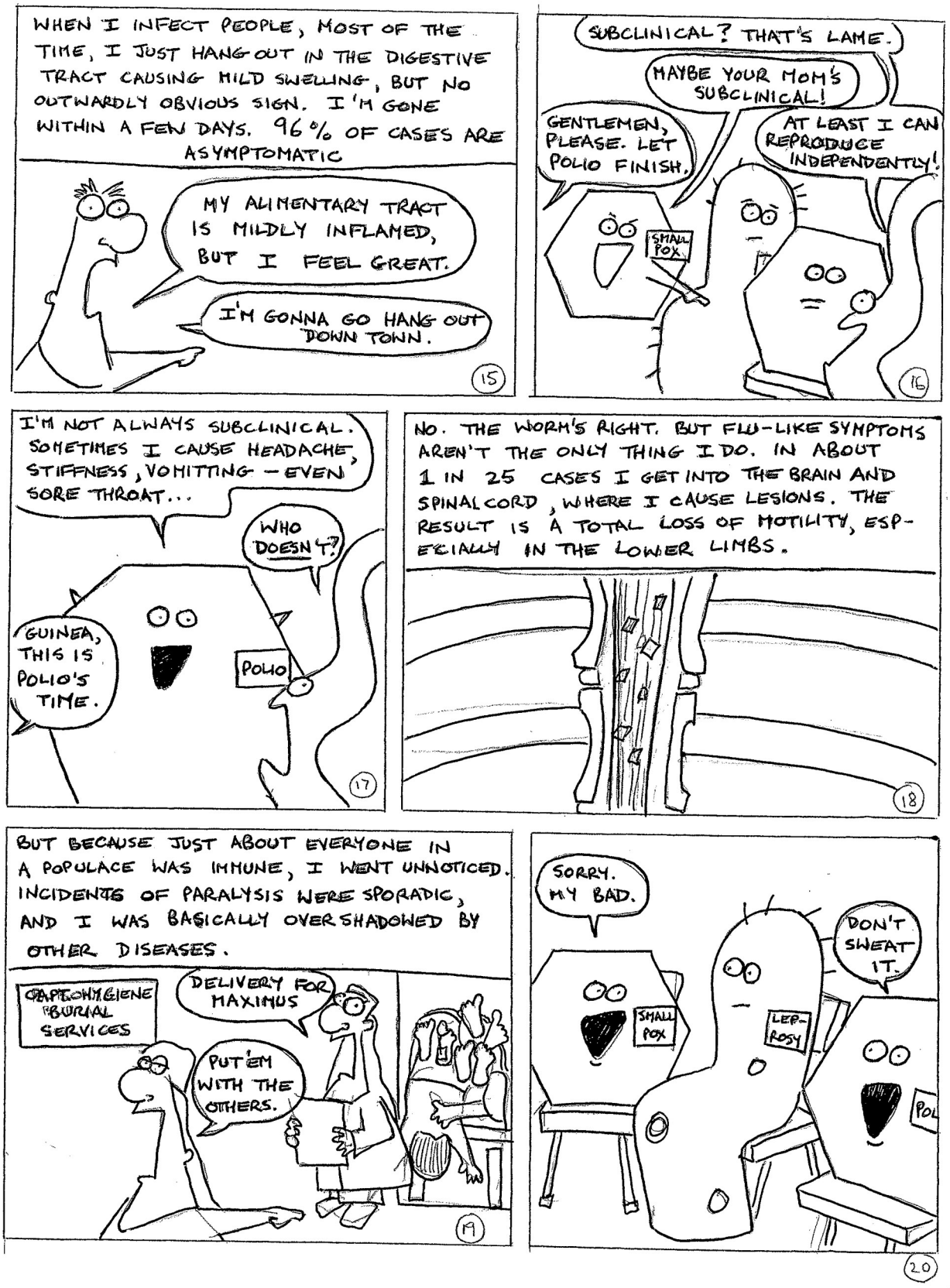
'Worthless' gifts get the good girls
(http://www.newscientist.com/article.ns?id=dn7737)

Sunscreen has gone on sale for dogs
(http://www.ananova.com/news/story/sm_1474854.html?menu=news.quirkies>http://www.ananova.com/news/story/sm_1474854.html?menu=news.quirkies

Smoking linked to fiery kids
(http://www.theaustralian.news.com.au/common/story_page/0,5744,16123063%255E23289,00.html%3ehttp://www.theaustralian.news.com.au/common/story_page/0,5744,16123063%255E23289,00.html)

POLIO: ITS STORY...(PART TWO)

By James Weldon



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TO BE CONTINUED...

JOURNAL CLUB SELECTION.

How Baseball Outfielders Determine Where to Run to Catch Fly Balls. (1995) Science 268:p569
In which we learn that apparently, there can be a scientific basis for 7 figured salaries. Next up, how to objectively judge figure skating.

(Found by David Ng, pdf of title page, available on line)

Science 268 (1995) p569

How Baseball Outfielders Determine Where to Run to Catch Fly Balls

Michael K. McBeath, Dennis M. Shaffer, Mary K. Kaiser

Current theory proposes that baseball outfielders catch fly balls by selecting a running path to achieve optical acceleration cancellation of the ball. Yet people appear to lack the ability to discriminate accelerations accurately. This study supports the idea that out-fielders convert the temporal problem to a spatial one by selecting a running path that maintains a linear optical trajectory (LOT) for the ball. The LOT model is a strategy of maintaining control" over the relative direction of optical ball movement in a manner that is similar to simple predator tracking behavior.

NEW (THIS TIME AROUND) CONTRIBUTORS

John Baez is a mathematical physicist at the University of California. After spending a lot of time moderating the newsgroup sci.physics.research, he became very good at spotting the tell-tale signs of a physics crackpot. After spending even more time moderating this newsgroup, he quit. Now he doesn't have to deal with as many crackpots.

Agnieszka Klimek has been a biologist, physiologist and now a pathologist studying Type 2 Diabetes at UBC and hoping to develop a novel predictor test for this disease. On her „off‰ time, she enjoys ultimate, cycling, good music and a good book, preferably somewhere hot, sunny and with lots of sandy white beaches!

Rhea Tregebov is a recently appointed (January 2004) Assistant Professor in the Creative Writing Program at UBC. She has published six books of poetry, most recently (alive): Selected and new poems, and still worships her high school physics teacher.

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.

Things that could win you an iPod of some shape and form.

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**