

PROFESSOR TRIM'S WAISTLINE

THE PROFESSOR'S FEATURES

NO. 21; SPRING 2007

Don't ditch your fat friends yet

Despite the media's interpretation of some fascinating research, fat is not contagious.

Put a cat in charge of the pigeons and you'll wind up with pigeon soup.

That's what happened recently when the popular media reported on a fascinating, but complex study of social networks in obesity carried out in the US and reported in the prestigious *New England Journal of Medicine*.

The study, carried out by two researchers at Boston Medical School, followed people involved in one of the longest running health studies in the US, based in Framingham, Connecticut. Using complex mathematical models, they looked at participant's obesity levels and their proximity to friends over 6 periods from 1975 to 2000.

Lo and behold, they found that people who stayed in close social contact with overweight friends, tended to have a greater risk of becoming overweight themselves. Geographical closeness had no effect, but reported social closeness did, suggesting that there might be more to weight gain than what we eat and drink.

Indeed, the popular media went wild, reporting on the 'social contagion' of obesity, as if an obesity 'germ' can hop through the telephone or other forms of contact between individuals who remain close irrespective of physical distance.

This notion of course received credence from fast food manufacturers and marketers of leisure saving devices, all keen to allay their own complicity in the biggest epidemic ever to strike humanity.

If close friends get fat, it would seem an easy step to imply that fat is contagious –

socially acceptable for us to do the same thing. It's the phenomenon that explains 'canned' laughter on TV sitcoms, copycat suicides, and copycat massacres like the epidemic of high school shootings in the United States.

Social proof has been tested in experiments where a 'victim' purports to be in trouble on the street. If no-one stops to help, then – no-one stops to help. If one collaborator helps, this increases the chances by 25% of another bystander coming to help. If two or more stop to help, the chances of others stopping increases to 100%.

Similarly, if my friends happen to be getting fatter and fatter in a society where this is rife and where it is easier to follow suit than to try to buck the trend, it's more likely I'll

get fatter (well, not me, because I know better. But you know what I mean).

So, before you consider ditching those friends who have a bigger waist than Jumbo the elephant, use this knowledge to turn the tables. Show them that being lean and mean can also be socially contagious.

For reference:

Chistakis NA and Fowler JH. The spread of obesity in a large social network over thirty-two years. *New England Journal of Medicine* 2007;357(4):370-379.



even socially contagious could be enough to take the pressure off the fat floggers, who, after all have had a bit of a hard time of it lately.

'Social Proof' and Obesity

However, all this ignores what social researchers and marketing experts have known for years and have described as 'social proof'. Social proof suggests that if other people, particularly those close to us, have done or are doing something, it is

THE PROFESSOR'S POINT

We seem to find it hard to accept the fact that economic prosperity can have a down side – ie, it can make us fat and unhealthy. Hence we look for all the other options: Social contagion is the latest one sought by the popular media, as referred to in the *New England Journal of Medicine*. But this is not what the research has shown. In fact, it suggests the opposite: that people who fatten up readily in an environment in which this is easy, will sub-consciously suggest to others that this is OK. We are social creatures. The Professor for one however, suggests we don't let the economists and politicians off the hook this easily. Obesity – and the health problems that stem from this comes from an 'obesogenic' environment. We need to make that environment less fattening if we want to enjoy the fruits of the economic system that has created this environment.

also in this issue

- Fat or inflamed?
- Depression and inflammation
- Genetics, big bellies and bums
- Syndrome ZZZZZZ

(click titles for article links)

Are you fat — or just ‘inflamed’?

New research suggests that inflammation, rather than fat, may be the problem stemming from obesity.

Partly fuelling the ‘contagion’ view of fat discussed in this issue’s lead story, are new findings that the main diseases associated with being overweight, like diabetes, heart disease and some cancers, may come from inflamed cells and arteries, rather than just the fat itself. This has created a flurry of activity amongst scientists, which may change our views on whether obesity is really a health issue, or whether it is our total modern lifestyle that needs to be corrected.

How does this happen?

For some time now, heart disease researchers have suspected that the blockages in coronary arteries that can lead to a heart attack, aren’t just caused by the ‘mechanical’ build up of cholesterol and other fats in the bloodstream and arteries (the process called atherogenesis), but by an inflammatory process in the arteries, similar to that which occurs in a microbiological infection.

The immune system it seems, can react to some non-infectious, lifestyle-based event, just like to a new kid (bacteria or virus) on the block. White blood cells surround a certain area and pump out inflammatory chemicals, designed to neutralise the invader. Then, when this is done, anti-inflammatory processes occur which dampen down the potentially damaging long-term effects of these pro-inflammatory chemicals.

The whole process is akin to attacking a house invader with a flame-thrower. If your aim is good, the intruder will be brought to his knees with just a bit of yelling and screaming. However if it’s not so good, or the intruder is part of a gang, which just keeps on coming, it might result in the whole house catching fire – room by room.

For many years it’s been thought that obesity was the intruder, and if we got rid of this we’d save the house. Now, a new line of theory proposes that obesity might just be the open window through which the burglar climbs. In other words it may be a marker, as much as a cause of disease. So what of the real causes?

These include the direct causes of obesity ie. a high energy-dense diet and inactivity. But long standing knowledge



Inflamed bodies and an inflamed environment: all part of the same syndrome.

and recent research has shown that these factors can cause inflammation in cells lining the arteries (endothelial tissue) on their own, even in the absence of obesity. This explains why some fat people (perhaps 1 in 6), never show signs of lifestyle-related diseases, and some lean, fit people (also about 1 in 6), do.

On the other hand, other lifestyle factors, like stress, depression, lack of sleep, smoking, and possibly heavy alcohol use, have been found to cause the low level type of inflammation in the cells and arteries thought to be only associated with obesity, suggesting that it may be our whole modern lifestyle that is causing the fire, with obesity just a bystander.

So what does this mean? And does it matter in terms of treatment any way?

The implications are that our whole modern lifestyle may be at fault; that our immune systems, designed to fight

foreign bacterial invaders, might also attack a lifestyle to which it has never been accustomed. From an evolutionary perspective, humans have never, over millions of years, lived the kind of exuberant lifestyle they have for the last half-century. It is just possible then that our bodies are mistaking aspects of this as the biological invaders they were designed to repel. Some saturated fats in animal foods for example have been around forever, and the body can manage these quite efficiently (like a small backyard fire). But once they become overwhelming, the fire starts to spread to the house and is hard to contain.

Research shows clearly increased production of inflammatory proteins from increased saturated fats in the diet – with or without weight gain. High levels of activity throughout history have also managed to squelch the flames of inflammation. While our inactive

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How the Professor aims to help the world lose weight — and save the environment (before moving on to cure all known illnesses)

They laughed at the Professor when he said he wanted to become a comedian. They’re not laughing now!

Similarly, his scheme for solving all of the world’s known problems by the introduction of a personal carbon-trading scheme (first referred to in these pages; issue No. 20, Winter 2007), has been met with more than underwhelming enthusiasm. Yet believing that a prophet is never acknowledged in his own time, the idea marches on – albeit with greater clarity as expressed herein, and now legitimised in the August 6th issue of the *Medical Journal of Australia*. Now read on...

Depression and inflammation

Are we seeing a trend here?

Depression is defined as:

“... an abnormal state characterised by exaggerated feelings of sadness, melancholy, dejection, worthlessness, emptiness and hopelessness that are inappropriate and out of proportion to reality.”

Some psychiatrists suggest that this has an evolutionary function: that depression helps maintain the order in animal populations, such that when an alpha male is challenged and beaten for his position, he retires away quietly and doesn't upset the flock by going berserk.

But what of females? Why is it that they admit to suffering depression much more than males? Could it be that the one in four females who admit to suffering depression are being more honest than the 1 in 6 men? We don't know. But we do know that the incidence of genuine depression (as measured by the same tests) has risen ten-fold since the late 1940s.

One suggestion is that this is due to the big increase that has occurred in physical activity in the community, largely as a result of time saving (cars, washing machines, vacuum cleaners) and time using (TV, video games, the Internet) technology. And although such a physical past-time as activity would seem to have little to do with such a mental process as depression, the link is not so far-fetched. A common theme

also seems to be the low grade level of inflammation in the inside of the arteries, referred to in our 'inflamed' article in this issue.

Running and depression

Research with mice has shown that in comparison with those allowed to run daily in an activity wheel (a thing that mice do quite readily and in fact get 'edgy' if they can't do), mice confined to inactivity tend to lose the neural connections in the part of the brain (hippocampus) associated with feeling good. They also show mousey signs of depression and even Alzheimer's disease and dementia.

Of course it's difficult to do the same experiment with humans (carving up the brain and examining this under a microscope is the bit it's hard to get participants for). However, one clever group of researchers got around this by studying blood flow to that part of the brain under study using a computerised imaging technique which leaves the subject happy and able to go home to wife and kids. They found a big increase in blood flow in the hippocampus in regularly exercising humans, which implies a blood supply to those nerve cells which tend to die in inactive animals, but which can be regenerated if treated kindly.

Other tests, comparing exercise with the new generation of anti-depressants (SSRIs) has shown that exercise stacks up just as well in relieving depression.

Also of interest is the fact that a form of inflammation appears to be more common in the small blood vessels of depressed individuals, suggesting a possible reduced blood flow to the vital parts of the brain associated with neurogenesis (re-growth of nerve cells). It's not clear yet whether this is a cause or effect, but ongoing stress, as well as inactivity and obesity seem to be an issue in the inflammatory reaction of the body.

All this may lead to as a change in our thinking about psychological issues such as depression. Losing weight and becoming active might even become front-line treatment in the future to take over from drugs.

For reference:

1. Brenes GA and others. Treatment of minor depression in older adults: A pilot study comparing sertraline and exercise. *Ageing and Mental Health*, 2007;11(1):61-68.

2. Cotman CW and others. Exercise builds brain health: key roles of growth factor cascades and inflammation. *Trends in Neurosciences* 2007 (published ahead of print).

So what is inflammation anyway?

The Macquarie dictionary defines inflammation as: ...a reaction to injurious agents... characterised by disturbed function. In biological terms, inflammation occurs as a result of a cascade of immune reactions in response to injury or infection. It can happen like this:

In response to a foreign invader (antigen) such as a bacteria or virus, white blood cells get to work to protect the body. These send out a number of signals, which result in the foreign body being surrounded and attacked. In the process however an inflammatory 'wall' is set up, consisting of protein signalling agents (pro-inflammatory agents) to contain the problem. In time, these are counter-acted by a deconstruction squad (anti-inflammatories), which help pull down the wall and allow life to get back to normal.

Recent findings suggest that not only invading micro-organisms, like bacteria, viruses (and even dust) set off the immune cascade and cause inflammation, but even certain foods. Humans have not evolved eating large amounts of fat and sugars for example, and while small amounts of these every now and then can be overcome by the immune system, ongoing, continual intake, may result in pro-inflammatory chemicals overwhelming the anti-inflammatories, resulting in ongoing inflammation.

It is this ongoing inflammation (particularly in the lining of the blood vessels), that is now known to be associated with heart disease, diabetes and a range of other problems. Unlike a few years ago, when we thought that heart attacks were caused by simple mechanical blockages of the arteries, the process now seems to be much more active. The inflammatory process might be caused by, or at least accompanied by, an increase in body fat. In any case, excess fat, particularly around the waist, is not a good look and is better gone — thus also reducing your inflammatory potential.

How do you know if you're depressed?

Many depressed people often aren't aware they are. They may just feel they're not coping with their situation, but this is likely to pass.

Mild depression however can become more severe if not well managed, so a proper analysis can be vital. There are several questionnaires for doing this, but a positive answer to just two questions correlates highly with all other measures. The questions are:

1. During the last month have you often been bothered by feeling down, depressed or hopeless?

2. During the past month have you often been bothered by little interest or pleasure in doing things?

If the answers to both these are positive, now may be the time to seek help.

Big belly or big bum – it's in your genes



So you've put on weight – that happens when humans spend too much time in the good paddock. But where has it gone: to your belly (as with most men) to your hips (as with most women) or to other places unknown. Research with mice shows that this depends largely on the genes you've inherited from

your parents. It also shows that this might not be inconsequential as fat from some parts of the body are more dangerous than that from others.

Knocking out mice

Recent scientific developments have raised the bar in genetic research by allowing scientists to 'knock out' genes in animals so they no longer work, and test how this affects other bodily functions as the animal grows. By knocking out genes involved in fat cell functioning for example they can check on the importance of that function in the cell.

Work of this type has shown that 'apple' or 'pear-shaped' people – those who store fat mainly on the belly or the hips, have parents that are generally shaped the same, and that particular genes control for this. It means that no matter how you try, if you're a natural pear, and you lose a good amount of weight, you'll probably just become a smaller pear – never an apple.

US diabetes expert Professor Ron Kahn, speaking at a recent International diabetes conference in Christchurch, New Zealand has also found something interesting about the types of fat from different parts of the body. Internal, or visceral fat, exists around the organs of the trunk, and unlike fat in other parts of the body, can't be seen. Yet it's much more dangerous than the visible fat around the waist or hips. Animals (and people) with high visceral fat have a much greater disease risk.

But when even a small amount of fat from the hips in mice is transplanted into the internal fat stores, that fat becomes *less* dangerous. And when visceral fat is transplanted into sub-cutaneous stores it becomes *more* dangerous.

Implications

What does this mean for the average punter? It suggests that not all fat is bad. In fact some is absolutely necessary for survival. It also suggests that the most dangerous type can't readily be seen. And although someone with a big belly might be more likely to have more internal fat than someone with big hips, or a small belly, this isn't always the case. Even lean people can have high internal fat and hence a greater health risk than someone with a big belly but low internal fat.

In any case, there's little that can be done. You can get rid of your belly – and you can reduce internal fat in some cases by doing so. But this is not always the case. It's just the luck of the draw – and the pattern of your genes.

For reference:

Khan R. The adipocyte as an endocrine organ. Paper presented to the Australian and NZ Society of Endocrinology, Christchurch, New Zealand, September 2007.

Syndrome ZZZZ

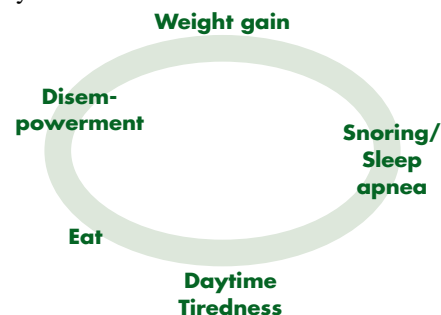
You may have heard of syndrome X, or the 'metabolic syndrome' which is a constellation of health risks like abdominal obesity, high blood pressure, high cholesterol and high blood sugars, that often seem to go together.

Now meet syndrome ZZZZ... It may not yet be a fully accepted medical condition, but it does explain why being overweight can cause snoring and snoring can cause being overweight. It happens like this:

Weight gain is not just in the form of fat on the body. A recent study of autopsies has shown that men with fat bellies, can also have fat tongues (see 'The Professor's Rear End'). These then lie across the wind-pipe, with extra pressure from fat jowls when lying on the back at night, and tend to block off the air supply, causing that steady ZZZZZ of snoring. The process makes breathing so difficult that the respiratory system often just shuts down and breathing stops.

Of course this can only happen for so long (but can be up to a minute in some cases), after which the body takes over to gasp for air like a formula one car taking off from a standing start. Several of these stoppages during the night (called apneas), are associated with several low-level awakenings, after which the sleeper wakes feeling tired and un-rested and of course sleepy all day.

Feeling tired during the day means being inactive (and probably eating more), which then leads to feeling disempowered and out of control, and gaining more weight, which makes the problem worse, as shown in the cycle below.



Having a big neck can also make this worse – even without excess body fat. Men with a neck circumference of >40cm and women >38cm are particularly at risk.

For reference:

Carey D., Type 2 Diabetes. Symposium presented at 15th Australian Society for the Study of Obesity Annual Conference, Canberra, Sept 2007.

Depression and body weight



While everyone thinks they know what it's like to be depressed, the different levels of this disorder make it a little more complicated than it seems. Also, as one psychiatrist has pointed out: "Depression (is) nearly incomprehensible to those who have not experienced it in its extreme mode." Although it is common enough to be called 'the common cold of psycho-pathology,' there are different levels of extremity.

1. The 'minor' depressive disorders; while not seeming 'minor' to sufferers, usually don't require pharmaceutical treatment and are generally thought to be psychologically based;
2. The 'major' depressive disorders on the other hand, have existed for much longer and are broken into melancholic depression and the psychotic disorders. At these extremes, all forms of medical treatment are required.

One line of theory suggests that while organic depression runs in families and is thus largely inherited, minor depression can develop an organic, or physiological base, if left to vegetate for long enough.

Everyone suffers periods of disappointment and sadness at some stage in life. However these are generally only short-term and are usually overcome returning us to some level of happiness. However if this sadness persists, leading to feelings of loss of control and worthlessness, and is accompanied by a genetic pre-disposition to depression, it can become biological for which medication then becomes vital. However, medication alone is usually not the long-term solution. Psychological techniques involving thinking (cognitive change) also need to be used to get the body back into equilibrium.

Understanding this process can help halt the problem in the early stages. If this is not done, the chances of a second episode after a first bout of depression are around 50% and up to 100% after 3 depressive episodes.

For reference:

Parker G.
Understanding Depression.

continued from page 2

modern lifestyles have been shown to do the opposite. More work is currently being done on other lifestyle factors and their direct effect on inflammation.

A solution then would need to be looked at in terms of managing modern lifestyles – hopefully without giving up the advantages of modern technology. Treating obesity by prescribing a diet, as we have often done, is akin to fighting the house fire by wearing a gas mask. Environmental change, like the personal carbon trading scheme proposed by the British (and elaborated by the Professor two issues ago)*, might also help the environmental and population crises which are rapidly engulfing humanity. At least it's worth a look.

* also see; Egger G. Personal carbon trading: a potential 'stealth intervention' for obesity? *Medical Journal of Australia*, 2007;187(3):185-187.

Depression and anxiety can cause diabetes

While it's known that being unfit, overweight and eating the wrong foods are all risk factors for Type 2 diabetes, there has been a feeling that psychological factors may also play a part. Now Norwegian researchers have confirmed this by following over 3,000 people diagnosed as anxious or depressed or with neither problem over ten years (*Journal of Psychosomatic Research*, 2007; 62(10):45-49). The researchers found that anxiety and depression are risk factors for the disease, independent of other, known risk factors. Surprisingly, there was no increased risk for depression as a result of having diabetes, which would seem to make more sense given that depression and diabetes are now known to be inflammatory states.

FIT BITS

Alcohol and the J-shaped curve

As you've no doubt heard, alcohol can be good for you. Its benefits come through its effects on insulin sensitivity and the good form of cholesterol (HDL). This is perhaps ironic because at one stage people with Type 2 diabetes (and hence insulin resistance) were advised not to drink. BUT, before you run off to the drinks cabinet, listen up: Alcohol also has a down side (see *Journal of the American College of Cardiology*, 2007;50(11):1009-14).

More than about one drink daily for women and two for men, can also cause heart failure, diabetes, stroke, congestive heart failure, liver problems dementia and early death. The clue is in how much, and how often. One or two drinks regularly is better than twenty-four on a Friday night, and moderation is better than excess. Given this, it seems to be the alcohol, rather than the type of drink itself that helps most – but not mixing drinks with sugary soft drinks or fruit juice is probably a good idea.

Female sexual desire and the metabolic syndrome

She might say she's got a headache, but there could be some other, more serious reasons for lack of female sexual desire. Austrian researchers have shown that, as with men, sexual dysfunction is higher in women who suffer from 'the metabolic syndrome', which includes abdominal obesity, high blood sugars, high blood pressure and high cholesterol (*International Journal of Impotence Research* 2007; Sept 20 – ahead of print). It's been known for years that this mix interferes with the downstairs region of overweight men, but the mechanical process, not required of women, was thought to make this irrelevant for women. According to the Austrian researchers however, decreased desire, as measured by a standard measure of female sexual dysfunction, was 3.3 times greater in women with the metabolic syndrome. Thinning her down might be a better option than paying more for headache pills.

THE PROFESSOR'S PRACTICE



Measuring your waist

It could be called the 'belly', but 'waist' is probably a better term – because that's largely what it is (or, more correctly, 'waste'). Waist (or belly) size, is now known to be predictive of health problems, and hence recommendations are to keep this down to about the following levels:

	Men	Women
Caucasian (white)	<100cm	<90cm
Asian/Indian	<90cm	<80cm
Pacific Islander	<110cm	<100cm

But where to measure? If you're as big as some of the men dealt with by the Professor, you could be in danger of collapsing in on yourself and causing a 'black hole'. Finding the part of the gut to measure is then often a task for an explorer, not a doctor. But here are a few alternatives:

1. Take a constant distance (eg. 28cm) from the top of the xiphi-sternum (just below the neck, where the two collarbones meet. This is regarded as a measure of the upper waist.
2. For the lower waist, measure a further 10cm down (38cm from top of the sternum)
3. Measure at the base of the lowest rib – where the belly starts and the ribs finish.
4. Use umbilicus (belly button) as a guide – but this may mean lifting the gut if this hangs low.

Irrespective of where you measure, the main aim is consistency. Take a spot that you know you can find again. Then aim for the goal of around 1% waist reduction per week (for at least 4-6 weeks).

"None of one, less of two, more of three" The Japanese come up with a handy way to remember health habits

Because we're told so many different things about what we should (and shouldn't) do for good health, it's often difficult to remember. Should I be eating less of this, or doing more of that to lose weight and feel better?

Now a group of Japanese clinicians have come up with a motto for how to do this – ironic because the Japanese have traditionally had less of a problem with obesity than most cultures. Their suggestion is "none of one, less of two, more of three" where:

The 'one' behaviour is smoking;

The 'two' things are intake of total food and alcohol, and

The 'three' are: exercise, rest and enjoyable activities.

In testing these 6 habits with over 18,000 people who attended the Health Science Centre at Jikei University in Tokyo, the doctors found that those who practiced more of these habits had less symptoms of the metabolic syndrome (high blood pressure, blood sugars and blood fats and abdominal obesity). Only 7% of those practicing all 6 habits had the metabolic syndrome, whereas this was almost 1 in 4 of those who did not practice any of the habits.

For reference:

Wada T and others. Effective prevention of metabolic syndrome: A motto for healthy habits – "none of one, less of two, more of three". *Obesity Research and Clinical Practice*, 2007;1:1330138.

FIT BITS

Fatties get it together

They say 'birds of a feather flock together' and it's apparently no different for overweight humans. Researchers at the University of Aberdeen in Scotland (*American Journal of Clinical Nutrition*, 2007; 86(2):316-323) have compared the body fat levels of a group of 42 Scottish husbands and wives and found that those who are disproportionately overweight seem to get together more frequently. They call this assortative mating, which, apart from anything else would suggest that the obesity epidemic is likely to continue for a while longer at least.

Exercise builds brain health

In a recent review that would take slightly more space to do it justice than the paragraph devoted to it here, University of California brain researchers (*Trends in Neurosciences*, 2007; ahead of print) have concluded that exercise is not just good for the body, but can also be good for the brain. It does this by allowing the production of growth factors which allow for neurogenesis (growth of new nerves) in the part of the brain (hippocampus) associated with learning and feelings of well-being. Inflammatory proteins caused by inactivity tend to block the growth factors and can increase the risk of Alzheimer's and Parkinson's disease as well as depression.

Spicy foods reduce inflammation

Given that we have talked a lot about cellular inflammation in this issue, it only seems right to suggest some ways of reducing this. The obvious ones are a change in lifestyle including a good diet, plenty of exercise, not smoking and moderate drinking. However, working with mice, scientists in North Korea (*FEBS Letters*, 2007; Aug 14 ahead of print) have shown that a good dose of capsaicin, the main ingredient from spicy foods such as chillies and curries, also tends to do the trick. Capsaicin has previously been found to increase metabolic rate and assist in weight loss, so the move to a good spicy meal (sans coconut and other fats) may be advisable.

TRIM TIP: Learn the Language of 'Energy density'

By learning to read the energy content of food labels (ie. energy per 100g of a food), it is possible to avoid foods (and drinks) that are likely to be most fattening. Avoid foods with an energy-density of >3kcal/100g (13kJ/100g), and drinks that are more than 0.4kcal/100ml (>1.5kJ/100ml).



PROFESSOR TRIM'S TABLE TALK

SPICY SPINACH AND ASPARAGUS SOUP

(serves 6 – virtually no fat)

1 bunch spinach (any kind)
4 stalks celery cut in small lots
1 medium carrot
6-8 asparagus heads
1 cup cauliflower
1 large onion cut finely
2 cloves of crushed garlic
1 tablespoon curry powder
1 fresh or dried chilli – cut finely
1 teaspoon fresh ginger
Sprinkle of pepper seeds
1 tablespoon of olive oil
3 cubes vegetable stock, mixed
with 1-2 litres of hot water
1 can low fat coconut milk
(or low fat evaporated milk plus
coconut essence)

1. Heat oil, onion, garlic, ginger, curry and pepper seeds in large pot till onion browns
2. Add cauliflower, carrot, celery, spinach and asparagus
3. Add vegetable stock to cover vegetables; bring to boil and then reduce to simmer for 45 minutes
4. Switch off heat, add coconut and whisk to the required consistency with a blender

THE PROFESSOR'S POETRY

LIFE ON A SOAP BOX

I'm just an average person
With an average share of strife;
But I'm terrified of dying
'Cause I'm really liking life;
So I try to do the right thing
By my body so that I
Get a few more years on Terra
And a few less in the sky.

Well I gave up eating butter
And I gave up munching cake;
And although I love me beer and prawns
I cut down my intake;
And I cut that lovely fat off
All that meat I just adore;
Then some doctor says cholesterol
Don't matter any more.

Now some other 'expert' tells me
That the biggest problem's stress;
And although I try to play it cool
I'm not that cool I guess;
'Cause you must admit it's kind of hard
When working in a job,
Where the boss is always at you
And you've got to earn a bob.

So I thought I'd quit and stay in bed
And then I'd find perfection;
'Till I checked on where my vote went
At the very last election;
Then at last the flaming coin dropped
And I realised the tricks,
If you want to live much longer
Have a go at Politics.

Think of Churchill, Menzies, Eisenhower,
And you'll see it's not quite cricket;
When for most of us it's six and out,
While they brickwall at the wicket;
Then there's Bolte, Breznev, Khrushchev,
Schmidt, Selassie, Baldwin, Mao,
And there's Truman, Gladstone, Bismark.
Playford, Johnson, Adenauer.

And if you think that their success
Just comes from being old;
What about the many others
Who've spent years out in the cold;
Like there's Lang and Gair and Little,
McEwin, Fullbright, Billy Jack,
Ho Chi Minh, Khomeni, Rhudolf Hess,
Petain, Rejaji, Colbach.

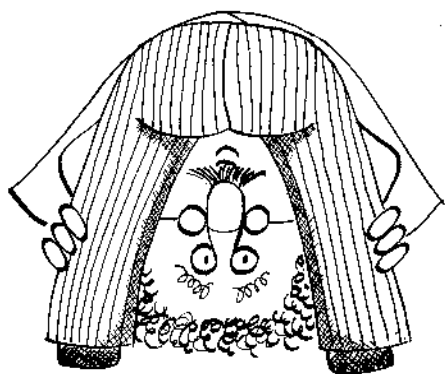
And although the good book tells us
We each have three score and ten,
That's the average, but they make it hard
For all we mortal men;
With the likes of Salisbury, Hoover, Brandt,
McMillan, Chou-en-lai,
Golda Meir and Hughes and Spear,
Mykoyan and Desai.

Then there's Chang Kai Chek and Chang Tso Ling,
Kan His, Chuteh, Chien Lung,
And there's Franko, Akbar and Lloyd George,
Who had their late bell rung;
There's the Singh's, the Khan's and Tito,
Mitchell, Ghandi, Ataturk,
Causescu, Dimitrov, Senghor,
Go to show it's all a lurk.

So now I've found the answer
I no longer am concerned;
I'm lapping up the beer and prawns
And my running shoes I've burned;
For I know my change of life
Will help me stay around so long,
Once I get a seat in Canberra
As the member for Grong Grong.



PROFESSOR TRIM'S REAR END



Resting heart rate and heart disease

Athletes have always known that resting heart rates tend to go down with an increase in physical fitness, so much so that elite athletes like Bjorn Borg were reputed to have a resting pulse of around 28 beats per minute (the average for men is 72bpm and for women 80 bpm). However medical scientists have largely ignored resting heart rate as a measure of good health, whilst knowing that a resting pulse over 100 bpm could indicate a form of racing heart known as 'tachycardia'.

Now a multi-national group of heart specialists have changed their tune on heart rate, suggesting from an analysis of heart disease cases that a high resting heart rate could be predictive of later heart disease (*Journal of the American College of Cardiology*, 2007, 50:823-8300). They suggest that the reason some drugs like beta-blockers for high blood pressure may help prevent further heart problems may be because they slow down resting heart rate. They also show that high resting rates are more likely to be associated with heart problems.

Another possibility however is not that a low resting pulse reduces heart attack, but that a low resting pulse is indicative of a high level of physical fitness. As it has been shown consistently that physical fitness lowers the risk of heart disease (even without weight loss), it may just be the fitness that is causing both the reduction in resting pulse and the reduction in disease risk. In any case, it might be an idea to keep an eye on that resting pulse – and definitely aim to keep it below 100bpm.

Eating less to live longer

For some time now it's been known that animals that are put on a calorie restricted diet tend to live longer than those given free access to food. This has also been offered as an explanation for

why some populations (ie. the Hunzas in Tibet and Okinawans in Japan) live to a ripe old age. It's not just (as the breakfast cereal marketers try to tell you) because they eat more cereal or oats, but because they actually eat 30-60% less in total – and what they eat is also low in energy density and fat. Extrapolations from animal studies suggest that a 48 year old man who eats 30% less till his normal life expectancy of 78, might live an extra 2.8 years (begging the question: 'is it worth it?')

Of course the hypothesis is hard to prove in a laboratory experiment. And it's also hard to cut people up to try to understand why this happens physiologically. But other ways are possible. In a cunning series of experiments, researchers at the Pennington Diabetes Research Centre in Texas (*PLOS Medicine*, 2007; 4(3): 1-9) have looked at the cells of people who are put on a restricted diet to see if they are different to those not restricted. They found that the mitochondria, or powerhouses of the cell, were increased in number and more effective, in the food restricted group. This suggested that these cells were more efficient at disposing of 'free radicals', probably because there were less of these in the lower calorie intake anyway.

Not happy to settle for this, other researchers have tried to develop what they call 'calorie restriction mimmetics', or a pill that will have the same effect as reducing food intake without having to reduce food intake. Why they would do so seems a little convoluted – particularly in an environment where less food production would help with environmental problems. But then, that's often the way of science: nobody understands some things – and somebody understands no things.

Fat body, fat tongue?

The idea that sleep apnea and snoring is at least partly caused by an increase in the size of the tongue in obese people has been around for some time. This makes perfect sense because an increase in tongue size would mean a blockage in the air passages and hence a reduced airway flow, thus resulting in the vibrating sound of snoring and reduced oxygen intake.

Pathologists carrying out autopsies however have often said they fail to find increases in tongue size in fat corpses. Does this mean the theory is wrong? And if so, how else do we explain the big increases in snoring and sleep apnea in the overweight. There are a couple of

lines of evidence here: In the first place a recent study published in the journal *Laryngoscope* (Nashi and others, 2007; June 22) reporting the autopsies on 122 medical examiner cases, not only found a high correlation between tongue weight and Body Mass Index (BMI), but a close link between fat content of the tongue and BMI. This was particularly so in men, perhaps explaining why snoring is generally more of a problem amongst the male gender.

But how do we explain the lean snorers and the obese individuals who don't snore (although there's not many of the latter)? The answer seems to lie in the concept of inflammation again (see story this issue). Inflammatory markers appear to occur in the airways passages of people whose lifestyle may pre-dispose them to obesity, but who, for some reason may not become obese. Some obese individuals on the other hand may be obese for genetic reasons, not lifestyle, and may not have developed the inflammatory processes of those with the poor lifestyle. Inflammation, like tongue enlargement, can amplify upper airway narrowing and hence result in the same type of airways restriction as a fat tongue itself.

In any case, the main cure for snoring – and sleep apnea – remains a healthy diet and lots of exercise, even if body weight loss is only minimal. In the few cases where this still exists alongside a healthy lifestyle, anatomical structures in the airways may need to be checked.

TRIM'S TUMMY TICKLERS

So you think you've had a bad day?

A guy goes into a hospital for a simple ingrown toenail operation. During the anaesthetic, the doctors find a major gangrene problem and decide to amputate the leg. However in the excitement, they initially take off the wrong leg. They then have to amputate the other one to get to the real problem.

When the patient awakes he is met by the operating doctor who says: "I have some bad news and some good news. The bad news is that we had to take off one of your legs, but we initially took off the wrong one so then we had to take off the other one as well."

Feeling down to his stumps, the patient says: "My God, after that, what can be good news?"

Says the doctor: "the guy in the bed next to you reckons he can use your slippers."