Factoids

The first manuscript of *The Potbelly Syndrome* that I (Farris) sent to the publisher had an appendix called Factoids. It contained information that was interesting and relevant, but which did not fit very well into the main body of the book. When an editor removed the appendix, I put some of the factoids into the main body of the manuscript, but most of them were scrapped. Some of them have been resurrected and scattered around this website. I plan to add more as time permits.

Azithromycin and *C. pneumoniae*

Gupta et al. (1997) explored the relationship between CPN antibodies and future adverse cardiovascular events in 220 consecutive male survivors of myocardial infarctions. The subjects were stratified into groups according to their levels of antibodies, and those in the highest (seropositive) group were further subdivided into treatment (azithromycin), placebo, and not-recruited groups. Follow-ups were done for up to 22 months. The incidence of adverse cardiovascular events increased with increasing anti-Cp titer, but the risk for adverse events in patients receiving azithromycin was the same as for the group with no detectable CPN antibodies.[1] The results are shown in table A-1:

Table A-1. Effects of Azithromycin on Future Myocardial Events.

Group	N	Adverse events
No CPN antibodies detected	59	7%
Intermediate titers	74	15%
Seropositive subgroups:		
Placebo group (not treated)	20	25%
Not-recruited	20	30%
Treated with Azithromycin	40	7%

At the time Gupta et al. were conducting this study, it was assumed that CPN was like most bacteria, and that it could be eradicated by a single antibiotic. We know now that it takes multiple antibiotics to kill CPN. Still, that single antibiotic, azithromycin, produced pretty spectacular results in Dr. Gupta's study. However, if those patients had been followed for a longer period of time I think the beneficial effects of azithromycin would have been washed out by reinfections. That's why the Vanderbilt and Wheldon protocols advocate using a mix of antibiotics to kill CPN in all of its forms.

***Chlamydophila pneumoniae* (CPN)**

CPN is a small bacterium that attacks lungs, arteries, and nerves. It infects most adults, and it has been linked to more than forty diseases, including obesity, diabetes, and heart disease. It is discussed at considerable length in *The Potbelly Syndrome*.

CPN has three forms, and one of them is a spore-like form that is extremely difficult to eradicate. The best treatments for it appear to be the

Vanderbilt and Wheldon protocols, which use multiple antibiotics to kill all of CPN's forms. You can get more information on anti-CPN protocols from http://www.cpnhelp.org.

Cholesterol Ester and Cortisol

The cholesterol in our arteries has been modified by macrophages to form *cholesterol ester* (CE). Cortisol causes macrophages to produce huge quantities of CE.

Dexamethasone (DEX) is a synthetic glucocorticoid that resembles cortisol. Cheng et al. wanted to know what affect DEX would have on the accumulation of CE in human macrophages. To find out, they grew macrophages in their laboratory and supplied them with varying amounts of cholesterol. They then measured the amount of CE produced by the macrophages. They then repeated the experiment, but this time they gave the macrophages various amounts of DEX. These researchers found that adding DEX caused a fourfold increase in the amount of CE in the macrophages.[2] They concluded: "In general, the data suggest that high levels [of DEX] enhance lipid accumulation by macrophages and thus would have an atherogenic action that is independent of serum cholesterol."

This is extremely important--**the amount of CE created by the macrophages was determined by the available dexamethasone, not by the available cholesterol**.

A few years later Cheng, et al. did a similar study using different hormones.[3] Here is an extremely simplified summary of their results:

- Cortisol increased CE production five-fold.
- Prednisolone, another cortisol-like medicine, increased CE production.
- Progesterone prevented cortisol and prednisolone from increasing CE production.

(Progesterone is a hormone that is abundant in young women, and which is thought to protect them from heart disease.)

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C-reactive protein (CRP) and coronary artery disease (CAD)

CRP is an acute phase protein produced by the liver during infections. It is thought to be the best single indicator of the amount of inflammation in the body. Researchers from Nancy, France, found that the CRP levels of patients with coronary artery disease (CAD) were three times as high as those of healthy subjects (7.1 vs. 2.3 mg/L).[4]

Cushing's syndrome (CS)

CS is a metabolic disorder that is unequivocally caused by cortisol or cortisol-like medicines. It is similar to potbelly syndrome in many respects. You can find out more about CS at the following sites:

- http://www.csrf.net/ (A Cushing's syndrome support site)
- http://www.cushings-help.com/intro.htm (a Cushing's syndrome support site)
- http://www.endocrine.niddk.nih.gov/pubs/cushings/cushings.htm (NIH site)
- http://en.wikipedia.org/wiki/Cushing's_syndrome (Wikipedia)

Cytomegalovirus (CMV)
CMV infects half of all people under thirty, and nearly everyone over age 60. It usually does not present any symptoms, but it may cause blindness and a mononucleosis-like illness. CMV causes pneumonia in people with impaired immune systems, and it kills many people who receive organ transplants. CMV is a cortisol-loving pathogen; *in vitro*, cortisol multiplies CMV's ability to infect human cells eleven-fold.[5]

CMV raises IL-6 levels and it suppresses the immune system.[6]

Diabetes and Inflammation (part 1)
Acute phase proteins (APPs) are markers for inflammation. If type 2 diabetes were caused by infections, we would expect to find higher levels of APPs in diabetics, and we do.

Fibrinogen, PAI-1, and orosomucoid are important APPs, and orosomucoid is often used as a marker for acute phase responses (APRs). Researchers in the Czech Republic found that patients with type 2 diabetes had significantly higher levels of these APPs than healthy controls did.[7] These researchers concluded:

> According to these findings we suppose that an "inflammation" plays an important role in the evolution of atherosclerotic process [and] NIDDM together with the known influence of glucose and lipid metabolism pathology.

Diabetes and Inflammation (part 2)
If type 2 diabetes were caused by infections, we would expect to find higher levels of acute phase proteins (APPs) in diabetics, and we do. Pickup et al. from Guy's Hospital in London compared the APP levels of three groups of people:

- Type 2 diabetics with 4 or 5 features of the dysmetabolic syndrome
- Type 2 diabetics with 0 or 1 features of the dysmetabolic syndrome
- Healthy controls

APP levels were highest in the diabetics that had 4 or 5 features of DSX and lowest in the healthy controls.[8]

Dysmetabolic Syndromes
Insulin resistance, Syndrome X, metabolic syndrome, type 2 diabetes and potbelly syndrome are all "dysmetabolic" syndromes.

The history of dysmetabolic syndromes illustrates how long it can take the medical world to recognize an important idea. In 1939, H. P. Himsworth and R. B. Kerr wrote:

> On the whole the insulin-sensitive diabetics tend to be younger, thin, to have a normal blood pressure and healthy arteries; in them the disease is sudden and severe at onset; they easily develop ketosis and react to a slight excess of insulin with a hypoglycaemic attack. The insulin-insensitive diabetics on the other hand tend to be older, obese, to have hypertension and to exhibit arteriosclerosis; in them the onset of the disease is insidious; they rarely develop ketosis and can tolerate over-dosage of insulin without showing symptoms of hypoglycaemia.[9]

The paragraph quoted above reports two remarkable findings. First, Drs. Himsworth and Kerr were the first researchers to make a clear distinction between the two major types of diabetes, insulin dependent and non-insulin dependent (type 1 and type 2). Second, they were the first to describe an important cluster of risk factors for heart disease:

- Insulin insensitivity (now called *insulin resistance*)
- Hypertension (high blood pressure)
- Obesity
- Non-insulin dependent diabetes (NIDDM; type 2)

Working without any knowledge of Himsworth and Kerr's article, Gerald Reaven and his colleagues came to similar conclusions many years later when they defined Syndrome X.[10]

Dr. Reaven was careful to restrict the definition of Syndrome X to a small number of elements that were tightly linked to insulin resistance. There were good reasons, however, for wanting an expanded term that included more of the disorders that are linked to insulin resistance. The terms currently being used for expanded versions of Dr. Reaven's famous syndrome include:

- Dysmetabolic syndrome
- Dysmetabolic Syndrome X (DSX)
- Insulin resistance syndrome
- **Metabolic Syndrome** (this is becoming the standard term)
- Metabolic syndrome X
- Syndrome X+

Each of these terms is defined a little differently from the others, and different authors may define the same term differently. The situation was, as one researcher called it, "a mess."

Since 25% of the people in the U.S. exhibit symptoms of dysmetabolism and are therefore at increased risk of heart disease, it became important to pick a reasonable name and start using it.[11] In 2001, acting on a request from the American Association of Clinical Endocrinologists (AACE), the CDC's ICD-9-CM Coordination and Maintenance Committee chose the name *Dysmetabolic Syndrome X* (DSX). [12] The ICD-9-CM code number for DSX is 277.7. This will be the name used to report the syndrome to the CDC and the name used on insurance forms.

The CDC did not specify criteria for diagnosing DSX, but the AACE has proposed a definition that is broad enough to include most of the expanded versions of Reaven's original Syndrome X.[13] The final, formal definition may be years away.

Potbelly syndrome (PBS) is one of many versions of Dysmetabolic Syndrome X (DSX).

Exercise

In *The Exercise Myth*, Henry Solomon, M.D., explains that fitness and health are not the same thing. Fitness has to do with your ability to work or play; health has to do with how well you feel and how likely your are to continue living. Generally you need to be healthy to be fit, but you do not need to be fit to be healthy. Exercise contributes a lot to fitness, but very little to health.

Healthy people love to move, and they do a lot of it. People with insulin resistance can't get enough glucose into their muscle cells. They feel "dysphoric" when they move, so they move less than healthy people do.

Pleasant exercises boost our immune systems and make us well and fit. Unpleasant exercises, and most exercise is unpleasant to people with insulin resistance, have few benefits.

Fatty Acids and Blood Pressure

Fish oils are rich in polyunsaturated omega-3 fatty acids, and large amounts of fish oils may help reduce high blood pressure.[14] Omega-3 oils are also found in flax seeds, and there are flax-seed breakfast cereals available now with omega-3 oil in them. You can even buy eggs with omega-3 oil in them (the hens have been fed fish meal).

One of the problems with trying to lower your blood pressure with polyunsaturated oils is that you may have to consume as much as four ounces a day to get any noticeable effect. Four ounces of these oils would cause diarrhea, obesity, and immune suppression.[15]

Foam Cells

Cells (other than fat cells) that are stuffed with fat. The foam cells of greatest interest in *The Potbelly Syndrome* are diseased white blood cells (macrophages) that fill themselves up with cholesterol and then die in our arteries, thus causing atherosclerosis.

Food-Pyramid Follies

Some anti-fat crusaders have given up on getting us to cut down on the amount we eat, so they are focusing on getting us to eat more carbohydrates and less fat. These high-carbohydrate enthusiasts created the infamous nutrition pyramid, with its broad base of grains and a tiny dollop of fat at the top.

Should we eat lots of grains? Probably not. Judging from their skeletons, our hunter-gatherer ancestors had bodies like the top professional athletes of today.[16] As soon as they became grain farmers, they became shorter, weaker, and subject to more of the degenerative diseases of civilization.

Gerald Reaven, a world-renown authority on metabolic processes, reviewed the literature on high carbohydrate/low fat diets and concluded that they do not decrease weight or insulin resistance. He recommended that people with Syndrome X avoid these diets.[17] Virtually everyone who is overweight has Syndrome X, so Dr. Reaven is saying in effect that no one should use high carbohydrate/low fat diets.

After studying the effects of these diets on healthy, postmenopausal women, some of Dr. Reaven's colleagues concluded:[18]

> Because all of these changes would increase risk of ischemic heart disease in postmenopausal women, it seems reasonable to question the wisdom of recommending that postmenopausal women consume low-fat, high-carbohydrate diets.

Is it just a coincidence that obesity and type 2 diabetes are now being called epidemics among children raised under the influence of the food pyramid?

HIV/AIDS and Heart Disease.
Hypercortisolism and heart disease are both common in HIV patients. Researchers from the University of Turin studied 1042 patients with HIV. They found that 52% percent of the patients treated with NRTI, and 19% of the patients treated with HAART, experienced at least one of the following cardiovascular problems: arrhythmia, pericarditis, ischemia, endocarditis, pulmonary hypertension, myocarditis, or dilated cardiomyopathy.[19] Cardiomyopathy is common in HIV patients.[20]

From the late 80s until the mid-90s the most effective treatments for AIDS patients were nucleoside reverse transcriptase inhibitors (NRTI). About 1995 the highly active antiretroviral therapy (HAART) became available. Both treatments have greatly extended the life-expectancy of AIDS patients. The Cushingoid symptoms found in many HIV patients are sometimes thought to be a side effect of NRTI and HAART treatments, but Cushing's symptoms were common in HIV patients before these treatments were developed.[21]

Atherosclerosis is common in patients with HIV. Researchers from Bordeaux, France compared 30 HIV patients with 18 controls. They found atherosclerosis in 37% of the HIV patients, but only 11% of the controls. Cortisol lowers levels of the "good" HDL cholesterol. HIV patients with atherosclerosis had lower levels of HDL cholesterol than the controls.[22]

Coinfections—and nearly all HIV patients have coinfections—greatly increase the likelihood that a patient will have heart problems.[23] The same is true of those of us with less famous infections; the more infections we have, the more likely we are to have heart disease.[24]

Sensitivity to Cortisol. In addition to having high cortisol levels, HIV patients may be more sensitive to cortisol than other people; HIV produces a protein called "VPR" that makes human cells more sensitive to cortisol.[25]

Leptin and High Blood Pressure

Leptin is a hormone produced by fat cells, and it tells the appetite and fat-storage systems how fat we are.

The NIH says that being overweight makes us much more likely to develop high blood pressure.[26] This is a true, but it is misleading because it implies that fat is a root-cause of hypertension when fat is at most an intermediate cause. Cortisol makes people fat. The excess fat produces leptin, then cortisol and leptin together raise blood pressure.

ACE inhibitors. Since leptin raises blood pressure, we would expect that anything that reduced leptin production would also reduce blood pressure. This seems to be the case. K. Masuo et al., from the Osaka University Graduate School of Medicine in Japan, performed a complex series of experiments to find out why blood pressure dropped when weight dropped.[27] Their results were as complicated as their experiments, and we won't go into them except to quote the next-to-last sentence of their abstract:

> A novel finding from this study is that ACE inhibition had a striking effect to lower plasma leptin.

Leptin enhances the immune system, increases the rate at which we burn fat, and reduces our appetites. If ACE inhibitors reduce our leptin levels, we would expect them to make us gain weight and have more infections. No research has been done on either of these topics.

Lyme disease

A chronic disease caused by a bacterium called *Borrelia burgdorferi*. Patients often lose weight during the early acute phase, then gain much more weight during the chronic phase.

Doctors who understand Lyme disease thoroughly are often referred to as "Lyme-literate." You can learn more about Lyme disease at the following websites:

- http://www.cdc.gov/ncidod/dvbid/lyme/
- http://www.lymediseaseassociation.org/

Macrophages

Large white blood cells that begin life as monocytes, then change into macrophages. They are the body's garbage collectors and they normally help the body dispose of unused cholesterol. When macrophages are infected with CPN, or exposed to too much cortisol, they lose their ability to process cholesterol. They fill up with cholesterol and turn into foam cells. Foam cells die in large clumps that often block our arteries.

Palatability's Paradoxical Effect on Appetite

Attractive foods stimulate our appetite, but they also raise our metabolic rate.[28] One author has gone so far as to claim that the French do not have as many cases of obesity as Americans because French food is more attractive, tasty, and satisfying than American food, so smaller portions are eaten. Maybe.

I'm a "chocoholic" and I frequently go on binges and eat embarrassingly large quantities of anything with chocolate in it. A few years ago a candy shop opened near my house, and it sells Grand Marnier-flavored chocolate truffles that are mind-bogglingly wonderful. After eating my first one I was so in love with them that I had to have another and another. I ate

them secretly, in my car or in the movies, because I didn't want to be seen as a stereotypical fat guy stuffing himself with candy.

Over time, I noticed that the second truffle didn't taste nearly as good as the first one, the third was still less wonderful, and the fourth and fifth were kind of blah. Eventually I discovered that the best way to eat my Grand Marnier-flavored chocolate truffles is to buy one, then go sit at an outdoor table and slowly, while watching the pretty girls walk by, eat my truffle in front of the whole world. I do this about once a week, and I seldom touch lesser chocolates.

Is it possible that blah-tasting food, wolfed down while worrying about our waists, is stressful enough to raise our cortisol levels and make us eat more than we should? Maybe.

Potbelly Syndrome (PBS)
A metabolic disorder that includes insulin resistance, obesity, high blood pressure, and, in the worst cases, type 2 diabetes. The technical name for potbelly syndrome is chronic subtle hypercortisolism.

PubMed
Most of the information in *The Potbelly Syndrome* was winnowed from thousands of medical abstracts obtained from the PubMed database.

PubMed is a service provided by the U.S. National Library of Medicine (NLM). It lists about fifteen million medical papers that have been published since the Fifties. About half of the citations are accompanied by abstracts, and many of the citations have links to PDFs of the papers from which they were taken. You can get access to this treasure-trove by going to: http://www.pubmed.gov.

To search PubMed, enter the terms, or combinations of terms, that interest you. PubMed will list all of the papers related to those terms. Here are some pairs of terms that will bring interesting results:

- Infections inflammation
- Inflammation heart disease
- Inflammation cortisol
- Cortisol metabolic syndrome
- Metabolic syndrome obesity
- Metabolic syndrome diabetes

There are lots of other interesting pairs of terms you can look up on PubMed, but the ones listed above should keep you busy for a few weeks.

Salt (sodium chloride)

The public stance of the NIH is unambiguous about the dangers of sodium, but reports circulated among NIH-insiders are full of doubts. The studies discussed below, all of which are described in NIH documents, are presented here to illustrate how complicated the salt problem is, not to convince anyone that salt is harmless.

The Multiple Risk Factor Intervention Trial (MRFIT). After telling us for years about the dangers of salt, fat, and other cardiovascular risk factors, the experts at the U.S. National Heart Lung and Blood Institute decided to actually test their theories. The result was the $115,000,000.00 MRFIT study in which thousands of men were divided into "usual care" and

treatment groups. The men in the usual care group did pretty much what they had been doing, while the men in the treatment group reduced their caloric intake, their cholesterol, their blood pressure, their smoking, etc. At the end of the test it was found that more men had died in the treatment group.

The failure of MRFIT reflects, I think, the futility of treating the symptoms, and not the causes, of chronic diseases.

Years later there was a MRFIT Follow-up study to see whether sodium consumption had affected the mortality of the subjects. It had not.[29]

National Health and Nutrition Examination Survey (NHANES I). If salt causes hypertension, and if hypertension is a killer, we would expect that the people who ate the least salt would live the longest lives. Not so. NHANES I measured the nutritional status and health of a large sample of U.S. residents. Twenty years later a follow-up study showed that the subjects who consumed the *least* salt were the most likely to die from cardiovascular disease. Furthermore, they were the most likely to die from all causes.[29]

Scottish Heart Health Study. The amount of sodium excreted in our urine is an excellent measure of the amount of sodium consumed. The Scottish Heart Health Study found that the amount of sodium excreted in the subjects' urine was linked to future heart attacks in women, but not men.[29]

Worksite Cohort Study. Alderman et al. measured the amount of sodium excreted in the urine of 2,937 people with high blood pressure.[30] Then they waited a few years to see how many of their subjects would have heart attacks. When the data was analyzed, a strong correlation was found between the sodium excreted by men and their likelihood of having a heart attack, but the correlation was negative, i.e., the men who excreted the *least* sodium were about four times as likely to have a heart attack as the men who excreted the most.[29]

NIH recommendation. Despite the clear evidence that low-salt diets are bad for many people, the NIH still tells us:

> Since there's really no practical way to predict exactly who will be affected by sodium, it makes sense to limit intake of salt and sodium to help prevent high blood pressure.[31]

Is this good advice? Sodium's effect on blood pressure varies with age, race, and sex, and within these groups it varies widely between individuals. The foods you eat and the medicines you take can all affect your sensitivity to sodium. The length of time you are exposed to a certain level of sodium intake can affect your sensitivity. Your sensitivity to sodium can change over a short period of time for reasons no one understands.[29]

It seems like it would be a good idea for a person to find out whether he or she is salt sensitive before making large changes in salt consumption. Unfortunately, there is no easy way to find out whether you are salt-sensitive. Here is a great research opportunity for some bright young post-doc student.

Snacking

How we eat can affect our cortisol levels. Jenkins et al. put men on two metabolically identical diets for two weeks. In the regular diet, the subjects ate three meals a day. In the "nibbling" diet, the subjects ate seventeen snacks a day. At the end of the two weeks, the nibblers had 28% lower insulin levels, and 17% lower cortisol levels, than the men who ate the

three regular meals.[32] Low-density cholesterol—the "bad" cholesterol—was 14% lower in the nibblers.

Sucrose and Myocardial Hypertrophy
Sucrose (table sugar) is a simple sugar often used to make unhealthy foods more appealing. There is some evidence that sucrose raises cortisol levels. It reduces our immunity to infections, and is generally bad for us.

Researchers from Milano, Italy gave a group of rats regular rat chow, and gave a second group rat chow plus sucrose. After exercising the rats regularly for three weeks, the rats that got the sucrose developed myocardial hypertrophy.[33]

Vanderbilt Protocol
A protocol for treating *Chlamydophila pneumoniae* (CPN) infections. It uses multiple antibiotics to kill all stages of the CPN organism. You can find out more about it at http://www.cpnhelp.org.

Wheldon Protocol
An anti-*Chlamydophila* protocol similar to the Vanderbilt protocol, but designed for multiple sclerosis patients. It is described at http://www.cpnhelp.org.

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