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Written and published by Ann Gerhardt, MD
Approximately 6 issues per year. Request a free email subscription or information about sponsorship at: algerhardt@sbcglobal.net. Read past articles on the website: www.drsgmedisense.com **I promise, an index is coming soon!**



Note from Dr G: DrG'sMediSense Newsletter is merging with WINS (We Insist on Natural Shapes) to form a 501(c)3 non-profit organization dedicated to providing health information to the public. That should finalize soon, and you'll be able to make tax-deductible donations of support to WINS, DrG'sMediSense newsletter and Dr G's health education efforts, and get tons of thanks and a few goodies in return!

My **teaching and volunteering** efforts have blossomed lately. From talking to students at the health professions high school, to caring for indigent patients in Honduras, to writing syllabi and lectures for UC Davis Medical Students (Future doctors are finally getting a good course, organized by Drs. Turgeon and Kulkarni-Date, about metabolism and nutrition!), I've been pretty busy. More info in the next newsletter.

SECOND HAND DEATH by Ann Gerhardt, MD
Subscribe at www.drsgmedisense.com 2/20/07

The city of Pueblo, Colorado passed a **smoke-free ordinance** in 2003. It disallowed smoking in all workplaces and buildings open to the public, including restaurants, bars, bowling alleys, and business establishments. Law enforcement officials strictly enforced the rule, imposing significant fines on violators.

Within 3 months, the heart attack rate suddenly fell. Since the ordinance, acute heart attack hospital admissions fell by seventy per 100,000 Pueblo residents per year. That represents a 27% reduction in heart attack rate, in spite of the fact that over 22% of Pueblo residents continued to pollute their own lungs with tobacco. The heart attack rate in surrounding areas fell only slightly.

A similar study in Helena, Montana mirrored the Pueblo results. Helena had also passed a don't-blow-smoke-on-my-dinner-or-work law and saw a 40% drop in heart attack

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admissions. Arguing that it was the passive smoking ban and not some other factor that reduced heart attacks was the fact that only city residents, those affected most by the rule, who had fewer heart attacks. Not recognizing a good thing when they had it, Helena **stopped enforcement and the heart attack rate rebounded to pre-ordinance rates.**

A smoking ban in Iceland led to drastic drops of air nicotine levels in bars. Blood cotinine (by-product of nicotine) levels in non-smoking hotel workers plummeted.

Being a smoker doubles the risk of having a heart attack. Within a year of quitting smoking, a person has a 50% lower risk of coronary heart disease. A never-smoker living with a smoker has a 30% greater risk of developing vascular disease than if he/she had lived with smoke-free air. Some scientists calculate that **passive smoking confers that same heart attack risk as does smoking ½ pack of cigarettes per day.**

Smoke attacks arteries on all battle fronts. Inflammation flared up by toxins in second-hand smoke damages blood

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SECOND HAND DEATH *continued from page 1*
vessel walls. The damage and inflammation make blood vessels more likely to close off and cause a heart attack. Second-hand smoke lowers *good cholesterol (HDL) and raises bad (LDL) cholesterol levels*. It changes sugar metabolism in a way that *promotes diabetes*, and stimulates blood platelets to *clot*.

The nicotine in second-hand smoke constricts blood vessels and *stiffens arterial walls*. It's far better to have pliable arteries that can rapidly change diameter in response to physiologic demand, than to have rigid ones whose only reflexive capabilities are to make the doctor reach for a prescription pad. Nicotine induces plaque to flip off, move downstream and completely block an artery. Nicotine and smoke hit just about all the pathologic mechanisms of heart attack.

As of July 1, 2006, there were 8 countries and 474 U.S. municipalities and 11 U.S. states with smoke-free ordinances. Do all the others have too much stock in RJR and Philips Morris to do the right thing and cut smoke time? Or maybe they just like to see people die young. •|

HOPING FOR HEALTH IN HONDURAS

by Ann Gerhardt, MD 2/20/07

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At least there was only one chicken on the bus. And it was in a box and didn't make much noise. Almost everyone, including chickens, use buses to get around in Honduras. Unless there is a cab to share with as many people as the cabby can pack into it. Or a pick-up truck to jump into, rap on the back window to get off and pay the driver a quickly negotiated fee.

The ancient school bus, with high-backed, rock-hard seats and empty soda containers rolling between our feet, did its little-engine-that-could trek up the winding mountain road to La Esperanza. La Esperanza, meaning 'the hope,' is where my friend Ann works for the Peace Corps and where she would connect me with World Vision. A World Vision supervisor would drive us into the hills, where the people are poorer than poor, carry all their water from dirty streams and live in empty stone and mud huts with dirt floors.

There I would see patients who had washed up just for the occasion of seeing the 'gringa' doctor from the U.S. They had dressed in the better of their two outfits of clothing. The children would withdraw into their mother's skirts, giggling and whispering. They would not question the wisdom of climbing on a cold hard desk (the 'exam' table) for me to poke on their belly and ask them to stick out their tongues.

I had been told that I would see children with 'gastritis,'



Azacualpa make-shift clinic

which is extremely common in Central America. I took medicines for gastritis, diarrhea, nausea and pain, knowing that there would not only be no facilities to do testing, but also no money for treatment.

The variety of disease I actually saw was astounding, far from simple gastritis. From three siblings with a very rare genetic disease of sugar metabolism, to a little boy with a loud heart murmur and weakness, each patient presented a new surprise. The 20 year old woman who should have been admitted to the hospital months ago shocked me: Her brothers had no money for treatment of her pernicious vomiting, nearly obtunded mental state, swollen belly and leg sores. World Vision would arrange and pay for her admission to the hospital, but would not happen for weeks.

There is hope in La Esperanza. These people hung on my every broken-Spanish word (my U.S. patients know better). Parents tried to hide crest-fallen faces when I suggested they feed their children protein foods far less affordable than tortillas, rice and potatoes. They didn't question my advice. Such is the faith in foreign medicine that two add-on, adult patients saw me just to confirm the wisdom of their Honduran doctors' advice (which, by the way, seemed to be sound).

World Vision in La Esperanza focuses on children's health, water sanitation and disease prevention. Ann had spent two years with them, teaching AIDS prevention, safe sex, and biology. Her roommate, Marianne, works with them to convince people to chlorinate their water. Yuki, a young man from Japan, tries to rid the area of a parasite that causes the severe Chagas heart disease.

I gave a stethoscope to their nurse and tried to teach her to examine an abdomen, attempting to leave behind something that might enable her to care for these children. They asked me to return. •|

PFIZER PHARMACEUTICALS – DON'T IGNORE MOTHER NATURE

by Ann Gerhardt, MD www.drugsmedisense.com 2/20/07

Pfizer Pharmaceutical Company announced in December 2006 that they were halting development of their much anticipated HDL-cholesterol raising drug.

The mega-drug maker's stock value plunged 11% overnight, losing billions of dollars. Pfizer is now slashing their work force by 2000 employees to compensate for the 800 million dollars they spent on the drug's development and loss of anticipated revenue.

The drug, torcetrapib, would have debuted as the first drug aimed at preventing heart disease by boosting HDL-cholesterol, known as the 'good' cholesterol (see **Cholesterol Basics** article). Pfizer pulled the plug on it after a massive, 15,000 patient trial of a combination torcetrapib/atorvastatin pill showed that patients who took the drug were 60% more likely to die than those who didn't. I would argue that problems with torcetrapib were predictable, given that some naturally occurring states of high HDL-cholesterol don't automatically guarantee long life and good health.

Don't get me wrong, I'm not saying that HDL-cholesterol has been masquerading under false pretenses of beneficence. Since the 1970's *population* studies have shown that higher HDL-cholesterol levels generally predict less risk of having a heart attack. Also, medications that both lower LDL-cholesterol and raise HDL-cholesterol confer greater benefit with greater HDL-cholesterol elevation.

In spite of man's efforts to compartmentalize people's heart disease risk according to their HDL-cholesterol levels, nature continues to defy the 'rules'. Population studies do not guarantee that every individual in the population fits the pattern. Desirable HDL-cholesterol levels have been set at >45 mg/dl for men and >55 mg/dl for women, but no level ensures a coronary-risk-free life. Some people with HDL-cholesterol levels higher than 95 mg/dl have heart attacks. Most do not.

On the other end of the spectrum, some people with HDL-cholesterol levels as low as 20 mg/dl have no heart disease. Certain human genetic mutations cause very low HDL-cholesterol levels. One might think that they would have early and severe vascular disease. Most do, but a few of these mutations induce premature heart disease only if LDL-cholesterol levels are high.

We can breed animals with a variety of mutations affecting HDL-cholesterol levels. Some follow the 'rules', but one such mutation induces very low levels of HDL-cholesterol without associated atherosclerosis. Another variant dramatically elevates HDL-cholesterol levels, but has *more* clogged blood vessels, not less. **These variant animals and humans are telling us that there is something we don't know about HDL-cholesterol that determines whether or not it is beneficial.**

Now let's consider Pfizer's drug. Torcetrapib works by blocking an enzyme (CETP) that moves cholesterol out of HDL particles into VLDL. From there, VLDL particles either get stuck in blood vessel walls or convert to LDL, both of which are bad. The promise of torcetrapib was that, by blocking CETP, cholesterol would stay in HDL and out of VLDL and LDL. HDL-cholesterol levels would rise, which has always been thought to be good.

Individuals with a genetic deficiency of CETP exist, giving us a natural example of what might torcetrapib might accomplish. These people all have very high levels of CETP. In Japan, one cluster of CETP-deficient individuals live long, heart disease-free lives, but most of these people have normal coronary risk. Some even have heart attacks at younger ages than usual.

Bottom line in the middle: Nature told us that CETP deficiency didn't necessarily protect against heart disease and, in might even increase risk. Science and medicine repeated prove that we don't know as much as we think we do about the body's complex mechanisms. Pfizer should heed these lessons. It takes arrogance and

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CHOLESTEROL BASICS by Ann Gerhardt, MD

Cholesterol travels through the bloodstream inside little balls of fat and protein called lipoproteins. I'll call them particles. We name the particles LDL, HDL and VLDL, and distinguish them from each other by the proteins, fats and cholesterol they contain.

Imagine these particles as party cheese balls, with a variety of cheeses crammed together inside a coating of chopped nuts. The cheeses are lipids – cholesterol, cholesterol esters and triglycerides (fat). The nuts are proteins and phospholipids, which do the 'work' of the particles: They

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Pfizer & Mother Nature *continued from page 3*
blind faith to think that replicating a defect (knocking out CETP) that in nature does not uniformly confer benefit would result in benefit when we do it with a drug.

In individuals with low HDL-C levels, high dose torcetrapib induces HDL-cholesterol elevation by up to 54%. It affects both the number and size of circulating HDL particles. It lowers LDL-cholesterol slightly, but only in people with normal triglyceride levels. Because of the inconsistent effect on LDL-cholesterol, Pfizer proposed that torcetrapib be administered with a statin, in order to assure LDL lowering. Offering torcetrapib as a combination pill with soon-to-be-generic Lipitor would also guarantee Pfizer an ongoing share of the Lipitor market. Torcetrapib also raises some people's blood pressure, and it's not clear how that happens. **What is clear is that we can't predict the fall-out of blocking CETP.**

CETP, one of HDL's proteins, transfers cholesterol out of HDL and into VLDL, in exchange for triglyceride moving the opposite direction. Pfizer and others decided that this is bad, but the body must have some reason for doing it this way. Blocking CETP also blocks the movement of triglycerides (fat) from VLDL particles into HDL particles. Once in HDL particles, any triglycerides that have been damaged get fixed and cleared out of the blood. Perhaps blocking this clean-up mechanism is counter-productive.

An HDL-cholesterol level is just that, a level. The level of *anything* in the blood represents a balance between how much is entering and how much is leaving, and at any given time tells us only what is present at that moment. It tells us very little about how it got to that level. Think of a bathtub you are trying to fill while the drain is open: The water level reflects the relative contributions of faucet flow and the size of the drain opening. A high level may result from either huge inflow or measly outflow. A low level might mean very little gain or excessive loss. A median level can represent the balance of large flux in both directions *or* not much happening in either. **A high HDL-cholesterol level could mean the cholesterol loading mechanisms are exceptionally active or suffering from bloated inertia.**

Many years ago probucol, also known as Lorelco, was pulled from the U.S. market by its maker, Hoechst-Marion-Roussel, under pressure from Food and Drug Administration. Probucool lowered total cholesterol levels and had an anti-oxidant effect, but also had the unfortunate side-effect of lowering HDL-cholesterol levels. The fear of any HDL reduction verged on phobia.

In spite of lower HDL-cholesterol levels, animals prone to clogged arteries that were given probucol cleaned up their arteries. Outcomes of patients on probucol improved, in spite of their lower HDL-cholesterol levels. There had been no deaths. The bias against low HDL-cholesterol levels was

so strong that probucol got the ax, in spite of evidence that it cleans out clogged arteries.

Probucool is still available in Canada, where doctors continue to study the drug's mode of action. They report that probucol cuts the need for repeat angioplasties in half. Probucool works by blocking a protein, ACAT, that transports cholesterol from cells to HDL. Not only does it reduce HDL particle cholesterol but it increases tissue cholesterol. The potential fall-out of letting cholesterol languish in tissue is not known and necessitates much more study before probucol returns to the U.S. market.

For now, people at risk of vascular disease will have to raise their HDL-cholesterol with exercise, niacin and nuts, and hope that the HDL particles they have are oxidizing, esterifying and transporting their lipids in the healthiest possible way. -|

Cholesterol Basics *continued from page 3*

enable the particles to attach to cells, transfer cholesterol and fat to and from various particles and reverse oxidation of lipids to make them less inflammatory.

The blood levels we measure are cholesterol and fat, not number or mass of particles. An ideal *total* cholesterol is <180 mg/dl. The total cholesterol is the sum of the cholesterol in all the particles. An ideal LDL-cholesterol is < 80, HDL-cholesterol is > 45 in men and 55 in women and VLDL-cholesterol is <20. An *ideal* triglyceride level is < 75, though "normal" in the U.S. is generally given as < 150 mg/dl. VLDL particles carry most of the triglycerides.

People talk about 'good' and 'bad' cholesterol, but cholesterol is just cholesterol. If it serves as a main ingredient to make hormones or as a component of cell membranes, it is good. If it becomes oxidized and trapped in blood vessel walls, it clogs arteries and is bad. The good or bad fate of cholesterol depends on the company it keeps - what type of particle carries it and which proteins modify it.

We call LDL particles (L for lousy) 'bad' because they tend to get lodged in blood vessel walls and promote inflammation and heart attacks. Put VLDL in the 'bad' category also. HDL (H for happy) is the 'good' particle because it pulls fat and cholesterol out of LDL and blood vessel walls, then neutralizes and converts them to forms that can be disposed of.

Since the particles and their proteins, not the cholesterol content, determine the fate of cholesterol and our health, it would be useful to know their levels. Currently we can't measure the number of HDL or LDL particles, or truly assess how well they are working. Specialized laboratories

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Cholesterol Basics *continued from page 4*

measure a few of the HDL and LDL proteins, but even these numbers don't give us the whole picture. Measuring just the cholesterol content must suffice. Few people even distinguish between particles and cholesterol: We just say, "My LDL is 135", even though we mean the LDL-cholesterol is 135.

The various particles interact with each other and with the body's cells. Investigators like to say that HDL particles clear cholesterol out of the body by dumping it into the liver, but we actually know very little about how that is accomplished. Try as we might, we have yet to find what they call 'putative receptors' that clear whole HDL particles from the blood. The liver does have scavenger receptors that extract cholesterol from HDL particles and release the HDL particle back into the circulation.

The liver also pulls LDL-cholesterol from blood via LDL receptors. Even though the LDL receptor was identified first, somehow HDL got the reputation as being responsible for cholesterol transport to the liver. I'm still scratching my head over that one.

LDL-cholesterol trapped in inflamed artery walls becomes oxidized. Oxidized cholesterol stimulates further inflammation and the artery becomes progressively more inflamed, scarred and narrowed until it closes off and causes a heart attack. One of HDL's proteins, LCAT, pulls cholesterol out of LDL and blood vessel walls, converts it to the healthier ester form and incorporates it into HDL.

Under usual circumstances, HDL's proteins un-do LDL-cholesterol oxidation, thereby interrupting the atherosclerotic process and contributing to the body's anti-oxidant and anti-inflammatory defense system. This is good, but HDL doesn't always act positively. HDL's lose their anti-inflammatory activity when the body is physically stressed, as in severe infection. Under such circumstances, HDL might even contribute to inflammation.

A study of heart attack victims with normal cholesterol levels showed that even very high HDL-cholesterol levels were *not* protective or anti-inflammatory. For some reason their HDL contained lipid hydroperoxides which are pro-inflammatory. This is a perfect example of the HDL-cholesterol level not revealing how well HDL works.

Normally the body's tissues extract VLDL's triglycerides to burn for energy. Another disposal mechanism for triglycerides is CETP. One of HDL's proteins, it moves cholesterol-ester from HDL to VLDL in exchange for triglyceride. Another protein, hepatic lipase, then removes triglyceride from HDL. People with high triglycerides tend to have low HDL-cholesterol levels and more heart disease. CETP lowers VLDL triglyceride, which is good. It enriches

HDL with triglyceride, which seems to be good, because 'fluffy' HDL's that contain more triglyceride than usual are associated with less heart disease. But CETP, if acting alone, leaves the HDL depleted of cholesterol, which dogma says is bad.

My theories: The key to good cholesterol metabolism is a proper balance between CETP's action, mechanisms that load HDL with cholesterol ester and how well the LDL receptor works. Perhaps one of HDL's major and unappreciated functions is triglyceride disposal. Maybe HDL's major effect on cholesterol isn't so much to dispose of cholesterol, but to clean it up and shove it back out into the blood in a healthier form. •|

A wonderful lady, Marlena Gutierrez,

died on Valentine's Day. Marlena was one of the major forces behind the success of We Insist On Natural Shapes (WINS). Though her prolonged severe illness side-lined her from our day-to-day activities, she remained one of our most enthusiastic supporters until the end. She could find something positive to say about anyone and anything. I'll always miss her "Oh, Dr Gerhardt, It's so good to hear from you!!" and her sincere love that she shared with all her many friends and family.

I've reprinted one of her articles published in the 1998 WINS newsletter, as it reflects so much of who she was and, perhaps, what would be good for others of us to become.

I AM 66, I'M A WOMAN & I'M BEAUTIFUL! *by Marlena Gutierrez*

A funny thing happened to me on the way to old age. I became beautiful.

All my life I got the message that I was not pretty, and (shame on me!), I believed it. And according to the very narrow and prejudiced definition of beauty in our society, it is true: I am not beautiful. Because of our "Stepford Wives" mentality, every woman, whatever her age, is expected to conform to one standard of beauty. "Be thin and look young" is the media's mantra. Hence the billion-dollar industry of anti-wrinkle creams, diets, exercise gimmicks and body-altering surgeries.

Nature has a lot to teach us. For everything there is a season: The beauty of spring is not the loveliness of winter, summer or autumn. Each season has its own beauty, its own purpose, its own joy. What is beautiful and natural for the spring of adolescence is not normal for the autumn or the winter of a long-lived life. Nothing is sadder than an adult woman still caught up in adolescent fantasies. Even

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GETTING THE MOST OUT OF YOUR DOCTOR- Be Sure To Mean It If You Cry Wolf

Ann Gerhardt, MD *Subscribe at www.drugsmedisense.com*

If you don't make every cold and cut an emergency, you (hopefully) get the doctor's attention more when you do have an illness. You deserve care when you are ill, but illnesses vary in severity. **If you don't receive attention and you truly do not cry wolf with every illness, you need to make that point to get the care you need.**

Presumably your doctor takes care of sick people as well as the worried well. Please recognize that and trust that, if you don't get an immediate response, it is because someone who is more ill than you took the doctor's time.

Don't exaggerate your symptoms to get attention or to justify complaining. If you do, your doctor won't take you seriously in the future.

I have some patients to whom a few spots of red are "a horrible rash all over." To them a joint that hurts with no discernible signs of swelling or inflammation is "the most excruciating pain I've ever had." For them, every pain rates a severity of 10 out of a possible 10. How can I get worried enough about those patients to squeeze in an appointment for them (squeezing out and inconveniencing others), when reality proves time and again that they have a minor ailment and major anxiety?

If you rarely complain or you have a chronic illness with a significant change in status, you deserve to make those points and expect to be given attention. I did a medico-legal review of a case in which a male patient complained of sudden onset of lightheadedness, fatigue and abdominal pain. The doctor saw him, but ignored the signs of acute ulcer, instead attributing his complaints to anxiety.

The patient called again, with increasing anxiety as his misery escalated. The doctor grew more convinced that anxiety was the major problem. Apparently four years previously the patient had had some headaches associated with anxiety for which he had received counseling. He called the doctor rather infrequently after that, until the current symptoms arose.

The doctor erred in assuming that every call from an anxious patient always represents anxiety. **The patient's only error was that he didn't (vehemently) remind the doctor that these were new symptoms and that he doesn't usually complain.**

On the flip side, be sure you take your own symptoms seriously enough that you ask for help when you need it. ❖



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I AM BEAUTIFUL *continued from page 5*
so, a society that seems to prefer infantile-adolescent beauty does not recognize the beauty of maturity.

What I have come to realize is that we are each unique and different, that beauty comes in many shapes, sizes and colors. We need to change our ideas of what beauty really is. As an older woman, my beauty is more complicated than that of a twenty-year-old. I've been around the block a few times. I've had more experiences. Because of these, I'm more capable, competent, savvy. My body is a map of where I've been. Every wrinkle, every sag has a story to tell – my story.

Today I choose to see myself as attractive. I have come to appreciate the light in my eyes, the warmth of my smile, but even more, my loving nature and compassionate heart. I've come to accept and love my humanness – my mistakes, foibles, idiosyncrasies. I love the parts of me that sag, the parts that have expanded. I love them because they are me. When I don't compare myself to you, I am happy. When I am content with my self, I see my inner beauty and sometimes others get a glimpse of it, too. Because I see beauty in myself, I can see it in you.

So when I see yet another wrinkle, I know it's not a curse but a coming of age – a rite of passage to empowerment, freedom, wisdom, if I choose to make it that. As an older woman, I don't take things at face value. I know that sometimes I have to dig deeper to find the pearl, to find the prize. So I choose to see beyond the superficial to a more genuine kind of beauty. I have come of age: another passage, another season, another adventure, another me.

Something funny happened to me on my way to old age. I found myself. I found how powerful and valuable and beautiful I really am.

(and during that process, she helped so many others to do the same. We will miss her.) ❖



Mangosteen grove, Honduras Botanical Garden

While writing the mangosteen article in the last DrG'sMediSense issue, I didn't know I would actually be seeing a whole grove of these trees so soon. It seemed a waste that such a rare fruit was abandoned and dried up on the ground under the trees.

HERB OF THE MONTH GINGER – Good Stuff

*by Ann Gerhardt, MD Subscribe at
www.drgsmedisense.com 2/20/07*

Bottom line at the top: Ginger works to alleviate nausea and vomiting associated with pregnancy and motion sickness. It may have other clinical benefits, but so far results in humans, using usual doses, either don't exist or are underwhelming. Ginger is usually but not always well tolerated and safe. Ginger root or tea or powdered ginger spice probably work best.

Ginger is the rhizome (underground stem) of *Zingiber officinale*. It has been used **in traditional Asian, Indian and Arabic medicine since ancient times to treat digestive symptoms, like nausea, diarrhea and belly pain.** Reportedly, traditional herbalists also used it for arthritis, toothache, headache, colds and various respiratory and heart ailments. Current websites extol ginger's benefit for atherosclerosis, migraines, bronchitis, rheumatoid arthritis, colic, painful menstrual periods, high cholesterol,

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burns, ulcers, depression, impotence, liver toxicity, Raynaud's disease, sciatica, ulcerative colitis, seborrhea, sore throat, swelling, chilblains, poor circulation, fever, tendinitis, and viral infections. Bleah!

Nothing is that good. I was OK with the helps-nausea part, and maybe the atherosclerosis, but the I-feel-a-snake-oil-coming-on feeling gets stronger as the list of cures grows longer. The longer list probably makes someone some money at the expense of emptying the pockets of gullible consumers who won't benefit.

Quite a few studies have examined ginger's anti-emetic (nausea and vomiting) effect. Most of the studies were small, but some were done quite well. The documented finding that people can tell they are taking ginger as opposed to placebo makes appropriate 'blinding' of ginger studies problematic.

Beware of ginger supplement pills and capsules: They may not contain much ginger. When ginger's known bio-active components were measured in various commercially available ginger capsules, the quantity of each compound varied from *none* to 1% (by weight). As usual, you and I, the consumers, have no way of telling which ginger capsules are real and which are not. Ginger sold as food spice is probably real ginger.

Meta-analyses of all clinical uses for ginger conclude that firm clinical evidence of superior benefit exists only for pregnancy-related nausea and vomiting. In all pregnancy trials, ginger consistently bettered placebo in relieving nausea. Two trials comparing ginger to vitamin B6 showed equivalent benefit. Ginger did not harm mothers or fetuses in these trials.

Studies of motion sickness have shown that ginger outperforms placebo, but does not work as well as standard medications (meclizine, dimenhydrinate, and scopolamine) to reduce nausea.

Results are mixed, some studies showing improved and
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others worse symptoms, when ginger is used for nausea and vomiting after surgery. Even if it were proven to work, ginger has limited utility in this setting, since it only can be taken by mouth. In the face of severe vomiting, getting ginger to stay down long enough to work is problematic. The same applies to chemotherapy-induced nausea and vomiting. Forty-one leukemia patients given ginger experienced reduced, but not cured, nausea after chemotherapy. In other situations ginger did not help at all.

Individual components of ginger demonstrate anti-inflammatory, anti-prostaglandin, anti-oxidant, immunomodulatory, anti-tumor and anti-microbial activity in test tube experiments. Claims that ginger has clinically relevant effects in humans for these purposes are entirely unjustified by current data. **Very small studies suggest that ginger works to relieve arthritis pain better than does placebo and as well as low dose ibuprofen.**

For colds, many people drink a water-based, chilled, carbonated and sweetened ginger extract, well known as Ginger Ale. There are no controlled trials of its use for colds. Except for tasting good and making me think my mother cared, I never noticed any significant benefit.

Many diabetics suffer from abnormal stomach and bowel contractions that lead to nausea, poor stomach emptying and constipation or diarrhea. A Michigan group of scientists found that a single gram dose of ginger partially and temporarily alleviated these problems. **In animals' colons, ginger exerts both contracting and relaxing effects.** The relative strength of these conflicting effects in humans remains to be determined.

Lately herbal companies state as proven fact that ginger lowers cholesterol, prevents heart disease and improves circulation, all blatant misrepresentations. **It's too early to tell whether ginger is beneficial for preventing heart disease in humans.** Ginger reduces the amount of artery-clogging plaque that develops in rabbits fed a high-cholesterol diet, without affecting cholesterol levels. A water-based ginger extract lowers blood pressure in rats by both lowering heart rate and dilating blood vessels.

One company claims that ginger works to stop bleeding *and* prevent clotting, actions that are usually diametrically opposed. One company claims that ginger's anti-coagulant properties make it an "ideal replacement for synthetic blood thinners." Such claims are dangerous. To take away a tried and true medicine that prevents clots and replace it with an herb with unknown potency incurs a huge risk. Someday ginger's effect may be proven and standardized to the point that one could take it with assurance that it will act at least as well as current medications, but we're not there yet.

Just for kicks I tried making pure ginger tea to treat my Honduras-acquired stomach upset. A quarter teaspoon of powdered ginger (about 500 mg) in hot water made a slurry with quite a kick as it went down. I burped and my belly churned, without any nausea abatement. Sigh.

Dose: The standard dose of 1 to 2 grams (divided into 2-4 doses) of powdered ginger is a lot of ginger. A teaspoon is about 2 grams. Alternative dosing might take the form of 2 to 4 grams of fresh ginger root, 1.5 ml (30 drops) ginger oil, a ginger tea (made by boiling grated ginger root -for how long, I'm not sure), or inhaled steaming water containing ginger oil or fresh root. One source recommends rubbing ginger oil into painful joints or placing fresh root in a warm poultice or compress and apply to painful areas. For motion sickness, start taking it 2 days prior to the anticipated voyage.

Side effects: Some people may feel heartburn, bloating, abdominal churning or burping after ginger ingestion, which in some may be very uncomfortable. Allergies to ginger may be severe. Since ginger may 'thin' the blood, it incurs a danger of excessive bleeding in people who are also taking blood thinners. There are no reports of ginger causing bleeding in people taking aspirin or warfarin, but the absence of reports doesn't mean it doesn't happen. We always just assume that a person taking one of those drugs is bleeding because of the drug and look no further for contributing culprits. Those who undergo surgery should either stop it prior to surgery or warn the surgeon and anesthesiologist about your ginger use.

Why it works: Ginger contains a variety of potentially bio-active compounds which may or may not account for its medicinal effects. The oil has volatile oils and pungent phenol compounds (such as gingerols and shogaols). Water extracts contain natural saponins, flavonoids, amines, alkaloids and terpenoids. Different cultivars vary with respect to their bio-active compound content.

Ginger's blood pressure lowering effect occurs via **muscarinic stimulation and calcium channel-blocking properties**, the former slowing heart rate and the latter dilating blood vessels. Ginger may work to reduce inflammation **by blocking prostaglandin production.** It's **cholinergic (spasmogenic) and prostaglandin reducing (spasmolytic) activities** are responsible for the effects on gastrointestinal motility. Ginger exerts anti-clotting effects, possibly via an anti-platelet action. •|

Two impulses struggle with each other within man: the demand for repetition of pleasant stimuli, and the opposing desire for variety, for change, for a new stimulus.
Arthur Schoenberg

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