PHARMACEUTICAL MYTHOLOGY: Do Not Take Aspirin as a Blood Thinner

A definitive study on the uselessness of aspirin taken for cardiovascular disease is published in no less than the Journal of the American Medical Association (JAMA). The study finds no benefit at all in giving aspirin to people at risk for heart disease. Not only does aspirin not prevent heart attacks, but patients who take daily aspirin (or, who take ibuprofen regularly) actually double their risk of a fatal heart attack.

Furthermore:

- Thousands of people each year die of side effects of aspirin, including upper GI bleeding.
- Aspirin also increases the incidence of hemorrhagic strokes (which constitute about 20% of all strokes), and taking daily aspirin can actually cause the formation of a blood clot, rather than prevent it.
- Another side effect of regular aspirin use is hearing loss.
- Yet another alarming side effect for those who use aspirin regularly, particularly people who take aspirin daily in the mistaken notion that it reduces risk of heart attacks and strokes, is wet macular degeneration and ultimately blindness.

Fowkes, et al. Aspirin for prevention of cardiovascular events. JAMA, March 2010.

This study (also from JAMA) looks at the effectiveness of aspirin in preventing cardiovascular events in people with atherosclerosis, indicating an increased risk of cardiovascular and cerebrovascular events, but who have never experienced such an event. The results are:

- Aspirin gives zero protection against initial fatal or non-fatal coronary event.
- Aspirin gives zero protection against initial stroke.
- Aspirin gives zero protection against the development of angina.
- Aspirin gives zero protection against the development of intermittent claudication.
- Aspirin gives zero protection against transient ischemic attack (mini strokes).
- Aspirin gives zero protection against all-cause mortality.
- However --- the aspirin group is 70% more likely to be hospitalized for an initial event of major hemorrhage.

Ogawah, et al. Low-dose aspirin for primary prevention of atherosclerotic events in patients with Type II diabetes. JAMA, November 2008.

This study looks specifically at low-dose aspirin for prevention of cardiovascular events in those with Type II diabetes, and thus at high risk for heart attack or stroke.

The study concludes that in patients with Type II diabetes, low-dose aspirin does not reduce the risk of cardiovascular events, either heart attack or stroke.

Ridkerpm, et al. A randomized trial of low-dose aspirin in the primary prevention of cardiovascular disease in women. N Engl J Med. March, 2005.

This study looks specifically at the use of aspirin to prevent heart attacks and strokes in women. The study finds absolutely no benefit in preventing heart attacks. It does show a slight decrease in the incidence of ischemic stroke in women who take aspirin, but, an increase in hemorrhagic stroke.

Curhan, et al. Analgesic use and the risk of hearing loss in men. Am J Med. March, 2010.

Chronic use of painkillers such aspirin, Tylenol (acetaminophen), and ibuprofen causes hearing loss. Those who use Tylenol only twice weekly have double the risk of hearing loss; ibuprofen increases hearing loss by two-thirds, and aspirin by one-third.

Studies published in both the Journal of the American Medical Association and in the British Journal of Ophthalmology show that low-dose aspirin taken over a period of years increases the incidence of wet macular degeneration (the leading cause of blindness). The increased incidence of macular degeneration in aspirin users is only minimally significant when the study is adjusted for confounding factors such as smoking. However, the incidence of macular degeneration in aspirin users with cardiovascular disease (the very people who are snookered into taking aspirin as a blood thinner) is statistically significant to a high degree.

Research done at the University of California and reported in the British Medical Journal shows that elderly men and women who take aspirin every day almost double their chances of developing ischemic heart disease. This study also shows that there is an increased incidence of both kidney and colon cancer in those taking aspirin on a daily basis. The study also shows that

there is an increased death rate in the aspirin users from hemorrhagic stroke, along with morbidity from bleeding ulcers and other intestinal bleeding.

The truth regarding aspirin's failure to prevent heart attacks and strokes has been known for over 25 years.

A study done at the University of Sidney in Australia and reported in the Medical Tribune on June 25, 1992 shows that patients with some degree of blockage of arteries to the brain are three times more likely to have a stroke if they are taking aspirin, and that increased incidence of stroke is from as little as a half tablet daily.

The original study from long ago purporting to show that aspirin decreases the risk of a second heart attack did not use pure aspirin but aspirin combined with magnesium in the form of the product Bufferin. It turns out that the slight benefit that was achieved in decreasing heart attacks was entirely due to the magnesium supplementation and had nothing whatsoever to do with the aspirin. This study was done by the AMA and was called the Physicians Health Study. What the study also showed was that even though the incidence of heart attacks was reduced in these people taking the aspirin accompanied by magnesium, the incidence of strokes increased in an amount almost exactly equal to the decrease in heart attacks. --- So, the overall mortality rate of the group taking the buffered aspirin was unchanged from the control group. --- In summary, the incidence of heart attacks was reduced slightly but was entirely due to the magnesium, and the incidence of strokes increased.

Shortly after the Physicians Health Study a British study was done to confirm it and was completely unable to do so. The British study used only aspirin (with no magnesium) and proved conclusively that aspirin had absolutely nothing to do with lowering the incidence of heart attacks. Furthermore, the people in the British study suffered so severely from stomach and intestinal bleeding that many subjects had to drop out of the study.

The case against using aspirin as a blood thinner to protect against heart attacks and strokes when, as we have made clear above, it does no such thing, has recently grown even stronger. Newer research shows that those who are prescribed aspirin to protect against heart attack are far more likely to have a heart attack if they try to quit the aspirin. As soon as a person stops taking aspirin, whether it was prescribed to prevent a heart attack or stroke, the risk of a serious heart problem immediately increases by 35%. Yes, quitting aspirin causes heart attacks. Since the risk is immediate upon stopping the drug, it is clear that these heart attacks are not caused by the purported "protection" of

the drug being denied the patient --- no, the cause is the aspirin itself --- by a REBOUND EFFECT.

In those taking aspirin to prevent a first heart attack, the odds of getting a heart attack (from the rebound effect) increase by 28%. For those who have already had a heart attack and have been put on aspirin to prevent a second one, the risk of suffering a heart attack increases by 46% from the rebound effect.

What makes this dangerous drug all the more bizarre, is that countless people who are put on aspirin have no choice but to quit because it causes extreme bruising, or ulceration in the gut. More than 15% of people who are put on aspirin need to stop taking it because of serious side effects. --- So now what a predicament --- quitting aspirin to avoid bleeding to death can cause a deadly heart attack.

Final summary: Why would you take a drug that increases your chances of hemorrhagic stroke, increases your chances of forming certain types of blood clots, causes upper GI bleeding, causes hearing loss, and may increase your chance of wet macular degeneration and colon or kidney cancer, when all you get in return is blood that is a bit thinner? It is all the more absurd to consider taking a dangerous drug (--- yes --- from what you have read above, you must understand that aspirin is very definitely in the category of dangerous drug), when the blood thinning effects can be achieved quite naturally with a healthy diet of meat, fish, poultry, eggs, and cheese, plus lots of vegetables --- not to mention the NUTRI-SPEC supplements that are cardio protective, vascular protective, and which thin the blood …

There are nutrients abundantly supplied in NUTRI-SPEC supplements that thin the blood. These supplements include:

- Oxy-Max (Diphasic P.M.)
- Oxygenic D
- Oxygenic D-plus
- Potassium Citrate
- Phos Drops

Many other NUTRI-SPEC supplements are specifically protective against various forms of cardiovascular disease. These include:

- Adapto-Max (Diphasic A.M.)
- Formula ES
- Complex S
- Oxygenic A
- Oxy Tonic
- Taurine

Aspirin (as well as ibuprofen and naproxen, but not Tylenol/acetaminophen) is fine for occasional short-term use for relief of inflammatory pain. But to protect and strengthen your cardiovascular system, rely on NUTRI-SPEC rather than being snookered into taking drugs that are certain to hurt you.

Additional Notes for Advanced NUTRI-SPEC Practitioners:

Note: Classical NSAIDs are non-selective Cyclooxygenase (COX) inhibitors, inhibiting both COX-1 and COX-2. The result is inhibition of Prostaglandin (PG) and Thromboxane (TXA2) synthesis and thus reduced inflammation, as well as anti-pyretic, anti-thrombotic, and analgesic effects. But the most frequent adverse effect of NSAIDs is irritation of the gastric mucosa since PGE2 normally has a protective role in the GI tract. --- Specific COX-2 inhibitors achieve most of the benefits, without gastric irritation. However, the other side effects of NSAIDs are still a concern --- notably renal failure, and an increased risk of heart attack, strokes, and thrombosis because NSAIDs leave the inflammation-associated with TXA2 unbalanced by the anti-inflammatory PGI2 (which is reduced by COX-2 inhibition).

The (unsubstantiated) theory is that low-dose aspirin protects against heart attacks and strokes by preventing COX-1 from forming TXA2, but this benefit is more than overwhelmed by COX-2 inhibiting drugs with their suppression of PGI2. So, drugs like Celebrex increase the risk of cardiovascular events due to clotting.

Note: Aspirin should never be taken with either pharmaceutical or nutritional COX-2 inhibitors because of the increased potential damage to the gastric mucosa. Specifically, COX-2 is up-regulated when COX-1 is suppressed with aspirin. One molecule of aspirin destroys one COX-1 enzyme.

Aspirin, ibuprofen, and naproxen decrease PGs, but have no effect on Leukotrienes (LTs). When the formation of PGs is inhibited (e.g. aspirin), the Arichidonic Acid (AA) Pathway is diverted to LT production = increased allergies and asthma.

Aspirin = increases abortion, increases internal bleeding, increases hemorrhagic stroke, decreases bicarbonate secretion in the gut, depresses the immune system, increases histamine, causes hearing loss.

Several metabolites of the Eicosanoid Pathway, including AA, PGA1, PGA2, PGE1, PGE2, and PGI2 = all stimulate renin production → angiotensin --- which should theoretically increase blood pressure --- but --- the PG effect of decreasing blood pressure is stronger (vasodilation). (So, aspirin increases blood pressure by blocking PG production).

Both the number of ventricular ectopic beats and the incidence of ventricular fibrillation, are reduced by aspirin. Yet, PGE2, PGI2, and PGF2-α all also show anti-arrhythmic activity. It is concluded that the genesis of arrhythmias is associated with TXA2 release, and not the other Prostaglandins.

NSAIDs inhibit PG synthesis and melatonin synthesis. In human subjects, it is found that NSAIDs (aspirin and ibuprofen) do not affect daytime body temperature but do attenuate the normal nocturnal body temperature decrease (? Decreased PGD2 more than PGE2 at night?). NSAIDs also attenuate the normal melatonin release at night. The lower melatonin is associated with a relative flattening of body temperature. Aspirin and ibuprofen both disrupt sleep --- ? both by suppressing PGD2 and suppressing melatonin?

When the PGD2-sensitive sleep-promoting zone is infused by TNF-α, the TNF-α increases the amount of slow-wave sleep, but decreases the amount of paradoxical sleep, while also causing fever and anorexia. The slow-wave sleep enhancement, the fever, and the anorexia are all blocked by COX inhibitors indicating that the effects of TNF-α result from the Cyclooxygenase Pathway hyperproduction of Prostaglandins, but the effects of TNF-α in decreasing paradoxical sleep are associated with an entirely different mechanism. --- TNF-α activates NF-kappa B, which in turn promotes COX-2 in the brain, and thus increases production of PGD2. The importance of the COX-2 AA cascade and subsequent production of PGD2 is why COX inhibitors such as aspirin and other NSAIDs disturb sleep.

PGE2 opposes PGD2 in many ways. PGE2 causes insomnia (not so much an inability to fall asleep, but a shortening of the sleep time) and increased body temperature. PGE2 also has an antihistamine effect. That is why aspirin (which suppresses PGE2) can cause stomach ulcers --- it suppresses the protective action against excess histamine in the stomach by PGE2.