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REPORT

S-Adenosylmethionine (S-AdoMet)

Part 2: Fast-Acting Natural Antidepressant

S-Adenosylmethionine (S-AdoMet) Part 2: Fast-Acting Natural Antidepressant Last month, we introduced you to an exciting new anti-aging therapy (S-AdoMet) that is currently unavailable anywhere else in the U.S. S-AdoMet is a substance synthesized in the body from the amino acid, methionine. An enzyme called methionine S-adenosyltransferase (MAT) catalyzes a reaction between methionine and ATP to form S-AdoMet. S-AdoMet has numerous actions within the body: its importance has been demonstrated in numerous published studies.



In the first installment of this series, we told you about a study in Hepatology, where S-AdoMet's ability to protect rat liver cells against alcohol was demonstrated. That study exemplifies S-AdoMet's three important actions: Methylation-S-AdoMet is a "methyl donor" for the synthesis of neurotransmitters, DNA, RNA, protein, and phospholipids; Transsulfuration-S-AdoMet is the precursor for cysteine, glutathione and taurine; Polyamines-S-AdoMet and Arginine catalyze the synthesis of spermine, spermidine and putrescine, which are essential for cell growth and differentiation.

A METHYL DONOR

S-AdoMet "donates" methyl groups to other molecules in order to stimulate biochemical reactions that transform these molecules into bioactive substances. For example, when methyl groups are transferred from S-AdoMet to certain phospholipids, phosphatidylcholine is produced. This important lipid is found in all cell membranes. Its presence or absence affects how cells react to stimuli from the outside environment because it controls accessibility of the cell membrane to signals from the outside.

Here's how it works:

Phosphatidylcholine makes cell membranes pliant. The other lipid in cell membrane-cholesterol-makes them stiff. Stiff membranes do not transmit signals as well as pliant membranes because more receptors are exposed in pliant membranes. There is also evidence that there may be distortion in the receptors of overly viscous cells. Receptors and molecules that occupy them are like parts of a jigsaw puzzle. With a malformation, and the piece may fit into its "receptor", but the fit will be imperfect. And any signal between the two pieces will be impaired. Cells with an overabundance of cholesterol simply "don't get the message."

Aging causes "hardening" of cell membranes. With age, the ratio of phosphatidylcholine- to-cholesterol decreases, and cholesterol becomes predominant. Decreased methylation which occurs with age plays a part in this lipid alteration. This is one area where the increased methylation that S-AdoMet causes, protects and enhances cell integrity.

Methylation of DNA is another area where S-AdoMet goes into action. The methylation of DNA causes the activation or inactivation of genes. Activated genes transcribe proteins. Without the proper transcription of proteins, cells cannot grow or function optimally. Activating or inactivating genes can stop tumor growth.

Other important processes that involve methylation are the suppression of viruses, the activation of heat shock proteins, and the synthesis and signaling of cytokines.

TRANSSULPHURATION

S-AdoMet is the precursor for the sulfur amino acids cysteine and taurine, as well as the tripeptide glutathione. S-AdoMet is first transformed into S-adenosylhomocysteine, which is then converted into cysteine and taurine. Sulfur compounds are so important that it has been written that "under conditions of absolute deficiency of sulfur, there is no living material." Every cell in the body contains sulfur compounds.

The end products of the transsulphuration pathway-free radical scavengers-are important. Glutathione is the most important substance in the liver. The liver's principle function is to break down damaging substances the body encounters. These may be drugs, or the body's own products. Liver malfunction- whether caused by alcohol, viral infection or other disorder-is invariably accompanied by glutathione depletion. When glutathione is depleted, the liver simply can't do its job. Glutathione is also found in other organs. It inhibits the deleterious effects of inflammation throughout the body. And it is an extremely potent free radical scavenger in the eye, where it protects against cataracts caused by UV sunlight. By providing the building blocks of glutathione, SAME contributes to maintenance of this important natural antioxidant.

POLYAMINES

These biochemical bombshells bind DNA and regulate gene expression. They make cell membranes act younger (more fluid), and repair DNA. We will be telling you much more about the importance of polyamines in future issues.

NATURAL ANTIDEPRESSANT

The most compelling clinical evidence for SAME is the hundred-plus published studies regarding its benefit in depression. SAME is the most well-documented non-drug antidepressant available today.

According to the latest data from the Center for Disease Control's (CDC) National Center for Health Statistics (NCHS), suicide is the 9th leading cause of death in the U.S., after AIDS, which ranks 8th. In people aged 25-44, it is the 5th leading cause of death; and in those aged 15-24, it is the 3rd leading cause of death, following accidents and homicides. In 1995, the number of suicides exceeded the number of homicides in the U.S. Clearly suicide is a major health problem.

It was estimated that successful suicides and suicide attempts cost over \$16 billion in 1994-in lost earnings, hospitalizations, and the like. This year, thousands of Americans will suffer a serious bout of depression, which is the Number One cause of suicide! It has been reported that every American will suffer at least one bout of depression during their lifetime. People with serious physical illness are often depressed, and it is occurring with greater frequency in the elderly and in young adults. What can be done?

Antidepressant drugs are part of a billion dollar psychopharmacology industry that, according to some physicians, churns out dangerous, addictive products. While antidepressants work in most patients, there are drawbacks. According to statistics from the Substance Abuse and Mental Health Services Administration (SAMSHA), 53% of drug-related admissions to emergency rooms are due to overdose. People frequently overdose on tricyclic antidepressants during the lag time between the time the drug is prescribed, and when it starts working. In 1994, 90% of emergency room visits related to tricyclic antidepressants were for overdose (intentional and unintentional).

EUROPE'S BEST-KEPT SECRET

In the 1970s, while testing SAME as a treatment for schizophrenia, Italian researchers discovered that their patients were becoming less depressed. This set off a wave of studies that continues to the present. In study-after-published-study, SAME is equal, or superior, to tricyclic antidepressants. Not only is it usually more effective, it works faster, and without significant side effects.

SAME has been proven effective in every type of depression, and seems particularly good for the endogenous form, where people are depressed without any apparent external cause. Even people with depression so severe they were contemplating electroshock therapy (ECT), have been "saved" by SAME. Bipolar depression (manic depressive) may be an exception to SAME's otherwise good record. SAME can cause some people with this type of depression to switch from depression to mania. This effect does not always occur.

SAME's anti-depressant effect begins anywhere from immediately to 5 weeks. Most patients benefit within 4 days, which is faster than most antidepressant drugs.

CLINICAL STUDIES SUPPORT EFFICACY, SAFETY AND QUICK ACTION

In 1987, the University of Alabama and the University of Trieste (Italy), along with BioResearch S.A. (which manufactures SAME) sponsored a symposium on SAME. The purpose of the meeting was to gather all the data together on using SAME as a treatment for neuropsychiatric disorders. Among the papers presented were the results of a study done at the University of California at Irvine on 18 patients hospitalized for depression. In this study, intravenous SAME was compared to oral imipramine (Tofranil). The researchers found that 67% of the SAME patients had 50% or greater improvement by the 14th day of the study, compared to only 22% of the patients given imipramine.

A larger study by DeVanna and Rigamonti confirmed these results in a placebo controlled, double-blind study using oral SAME. In this study, patients with major depression were given 1,600 mg of SAME per day. In order to reduce the "placebo effect", DeVanna and Rigamonti gave the patients a placebo for a week before beginning the real trial. Patients who felt better after taking the sugar pill were excluded from the study.

Using four different depression scales to measure response, the researchers found that by day 10, SAME had decreased depression 27% versus imipramine's 18% on the Hamilton Rating Scale for Depression. On day 20, the anti-depressant effect of SAME and imipramine were similar, although SAME had a clear advantage on the anxiety scale. On day 42, imipramine surpassed SAME. More patients dropped out of the study due to side effects from imipramine than SAME.

SAME has also been compared to desipramine (Norpramin), amitriptyline (Elavil), and chlorimipramine in placebo-controlled, double-blind studies. According to one meta-analysis of these studies, 92% of patients responded to SAME, compared to 85% for the tricyclics. SAME has also been compared to amoxapine (Asendin), maprotiline (Ludiomil), and trazadone (Desyrel).

DEPRESSION CAUSED BY ORGANIC DISEASE

SAME has been tested for depression caused by a variety of diseases, including Parkinson's Disease (PD), fibromyalgia, cancer, cardiovascular disease, and rheumatoid arthritis. And researchers have used SAME successfully in conjunction with drug and alcohol withdrawal.

PARKINSON'S DISEASE (PD)

The incidence of depression in PD patients is about 46%. It is interesting to note that in one recent study, 32% of PD patients had a lifetime history of depression. Unfortunately, there is only one published double-blind, placebo-controlled study on using SAME for depression in PD patients. That study, conducted in Italy (where SAME is manufactured) shows a definite improvement in depressive symptoms. Importantly, SAME did not affect L-Dopa treatment. (Ed. note: L-Dopa is the precursor to dopamine, which is the standard treatment for PD). The most significant side effect was that three patients complained of elation during the first days of treatment.

L-dopa (the treatment for PD) depletes SAME. In rodents, SAME bounces back in the brain after L-dopa, but doesn't in the liver. It has been suggested that L-Dopa may cause damage to organs such as the liver, where SAME disappears after L-Dopa treatment. This theory has never been proven or disproven.

It has been suggested by one research group that since L-Dopa depletes SAME, excess SAME maybe the cause of PD. The researchers attempted to prove their theory by injecting huge amounts of SAME directly into the brains of rodents so as to produce PD-like symptoms. One problem with the study is that such huge amounts of any substance injected directly into an organ can cause severe damage. Many different substances can create PD symptoms by depleting dopamine in the brain. Manganese chloride is one of those substances. Iron is another. Another is MPTP, which is used by researchers in studies to create PD in animal models.

The one published study on giving SAME to PD patients does not support the theory that SAME is detrimental to PD patients. On the contrary, the results of that study show a beneficial effect. The fact that no patient had to increase their dose of L-Dopa suggests that SAME does not interfere with L-Dopa therapy.

New and exciting research is being published on the real cause of PD. Scientists are once again focusing on the role of serotonin in dopamine production. Researchers at Sandoz have shown that the part of the brain affected by PD-the substantia nigra-contains serotonin-related receptors. This part of the brain is always associated with dopamine, and since the discovery of L-Dopa, research into PD has centered around dopamine. But researchers have shown that serotonin raises dopamine, and dopamine lowers serotonin in certain parts of the brain. This see-saw relationship between serotonin and dopamine ensures that neither substance gets too high. Since dopamine can become toxic to neurons, the interplay between serotonin and dopamine can't be ignored.

L-DOPA AND SEROTONIN

In a recent study from the National Institute of Neuroscience in Japan, researchers demonstrated that the serotonin inhibitor, para-chlorophenylalanine (PCPA) decreases dopamine activity, which L-Dopa does not restore. However, intravenous serotonin does restore dopamine activity after PCPA therapy. This study implicates a completely new pathway in dopamine production and maintenance that L-Dopa does not affect. It opens a new avenue of PD research. It does not mean that PD patients can cure themselves by taking serotonin- PD is an extremely complicated disease which involves free radicals, among other things-but it does open up exciting possibilities.

Research shows that L-Dopa quits working after about 4 years. Some of the new research on the interaction between dopamine and serotonin seems to indicate that the ultimate failure of L-Dopa may relate to its depletion of serotonin. In a study in *Neuroscience Letters* in 1993, it was shown that PD patients have significantly decreased levels of dopamine and serotonin in their cerebral spinal fluid. Patients treated with L-dopa have even less serotonin than untreated patients (although they have increased dopamine). The depression that a lot of PD patients have may be a result of the loss of serotonin, both from the disease itself and from L-Dopa treatment. It is important to note that the drug Selegiline (Deprenyl), which is often used in PD patients to keep L-Dopa effective longer, increases serotonin.

The Foundation looks forward to further research into the relationship of dopamine, L-Dopa, serotonin, and SAME which plays a role in converting serotonin to melatonin, and converts homocysteine to sulfur-related antioxidants.

RHEUMATOID ARTHRITIS (RA)

Fifty-nine RA patients participated in a study to measure the effect of SAME on depression caused by RA. All the patients were experiencing major depression. They were given 200 mg SAME per day by injection. Compared to placebo, patients receiving SAME improved significantly on the Hamilton Rating Scale for Depression (HAM-D).

OSTEOARTHRITIS

A two-year study involving 108 patients (97 by the end of the study) was published in the *American Journal of Medicine* in 1987. It not only showed how SAME alleviates pain, but also how it alleviates depression in people with osteoarthritis. Participants in the study were given 600 mg of SAME per day the first two weeks, and 400 mg/day thereafter.

FIBROMYALGIA

Fibromyalgia is one of those mysterious syndromes that can either occur by itself, or accompany other diseases such as lupus and chronic fatigue immune dysfunction syndrome. The main feature of fibromyalgia is persistent pain-not necessarily in the joints, but deep in muscles-that occurs for no apparent reason. The symptoms of fibromyalgia include tenderness at several points of the body. In addition, people with fibromyalgia frequently have fatigue, sleep disturbances, numbness, joint swelling, and other symptoms. It is often treated with tricyclic antidepressants.

It has been suggested by several researchers that SAME might be a good substitute for tricyclic antidepressants for the treatment of fibromyalgia. Several studies have proven them right. In a study in the *Scandinavian Journal of Rheumatology*, 800 mg of SAME per day for six weeks improved "clinical disease activity", pain, and morning stiffness. Mood improved when measured by the Face Scale, but there was no significant improvement on the Beck Depression Inventory.

An earlier study did find, however, significant improvement on the HAM-D and the Scala di Autovalutazione per la Depressione in 11 of 17 fibromyalgia patients taking SAME.

Another study compared SAME to transcutaneous electrical nerve stimulation (TENS). Fifteen patients with primary fibromyalgia were given one 200 mg injection of SAME in the morning, plus a 200 mg tablet at noon and in the evening. The study lasted 6 weeks. During the first 2 weeks, patients in the SAME group had a significant decrease on two depression scales. During the last 2 weeks, a significant reduction on a third scale occurred. The TENS patients did not do as well. But when 5 of them switched to SAME, their depression scores also decreased. No side effects were reported.

A study published in *Current Therapeutic Research* found that a 200 mg injection of SAME plus 400 mg orally twice a day significantly decreased depression in fibromyalgia patients beginning on day 7. This coincided with a decrease in physical symptoms.

CARDIOVASCULAR DISEASE, CANCER AND OTHER ILLNESSES

A group in Italy tested the effects of SAME on depression caused by different illnesses. Of the 55 patients tested, 40 were inpatients. All patients had moderate-to-major depression. Inpatients were given two 200 mg injections of SAME. Outpatients took two 400 mg tablets of SAME for 4 weeks. Besides cardiovascular disease and cancer, patients were suffering from alcohol-related liver disease, insulin-dependent diabetes, posttransfusion hepatitis, obesity, cerebro-vascular disorder, bronchial asthma, viral pneumonia, endocrine diseases, psoriasis, herniated disk, and congenital hip dislocation. Scores on the Beck's Depression Inventory were significantly improved in the patients receiving SAME. Side effects were minimal, and none of the people treated dropped out of the study because of them.

The authors point out that SAME may be particularly beneficial in treating depression in heart disease patients because tricyclics, MAO inhibitors, and second-generation antidepressants (such as Prozac, etc.) are contraindicated in these patients.

There have been conflicting reports that some heart medications cause depression. In an effort to clear up the controversy, a researcher in Denmark recently reported the results of an analysis he did of 17,636 prescriptions. He found a correlation between angiotensin-converting enzyme (ACE) inhibitors, calcium channel blockers, and prescriptions for anti-depressants. Diltiazem (Cardizem) seemed particularly problematic. People who take these drugs should be aware that they may cause depression.

THE DANGERS OF ANTIDEPRESSANT DRUGS

"Six depressed patients free of recent serious suicidal ideation developed intense, violent suicidal preoccupation after 2-7 weeks of fluoxetine (Prozac) treatment. This state persisted for as little as 3 days to as long as 3 months after discontinuation of fluoxetine. None of these patients had ever experienced a similar state during treatment with any other psychotropic drug."

This report, from Harvard Medical School, set off a fire storm that raged in the pages of the American Journal of Psychiatry for two years. The issue of whether or not Prozac causes suicidal impulses is still not settled. One thing is for certain though-antidepressants turn up in drug-related overdoses almost as frequently as sedatives, which are used (along with alcohol) most frequently to overdose. The obvious reason is that people taking antidepressants are more likely to attempt suicide. Another, more subtle, reason is that antidepressants can take 4-6 weeks to work. A study in the British Medical Journal (which set off another fire storm) found that people who had been taking antidepressants for less than 30 days, and people taking high doses (which usually occurs in the beginning of therapy) were more likely to commit suicide.

Studies show that people who overdose on the older, tricyclic antidepressants are more likely to die than those who take the newer selective serotonin-reuptake inhibitors (SSRI). This makes Prozac, Paxil and others a safer choice-particularly for older people who can't metabolize the drugs well. The problem is that SSRIs don't work for everyone, and when they do, they sometimes have intolerable side effects.

The side effects of tricyclic antidepressants make up a long list. Dry mouth, weight gain/loss, constipation, blood sugar increase/decrease, insomnia/drowsiness, nausea, and sweating are some of the milder side effects. The SSRIs are noted for their inhibition of libido, anxiety, nausea, heart palpitations, and other central nervous system and gastrointestinal effects.

All antidepressants pose risks of life-threatening events including stroke, heart failure, and liver disease. Tricyclics cause liver damage through inhibition of Cytochrome p450 enzymes which are used by the liver to detoxify drugs. Recently, the MAO inhibitor, moclobemide was accused (in Lancet) of causing fatal liver cholestasis (stoppage of bile). The manufacturer, Hoffmann-La Roche, responded that the death was more likely caused by Prozac, which has been associated with liver abnormalities. The natural anti-depressant, SAME, has been shown to be liver-protective in numerous studies.

The side effects of antidepressants relate to their interaction with certain receptors. No one knows exactly what antidepressants do to receptors. Furthermore, receptors that respond to antidepressants are located throughout the body-not just in the brain. For example, the gut problems associated with SSRIs probably relate to a serotonin receptor known as 5-HT₃ found in the gut. At present, a dozen different types of serotonin receptors have been found-with more on the way. It will be years before scientists understand exactly what antidepressant drugs do in the body.

Patients who have been taking antidepressants for more than two months should never suddenly stop taking them suddenly because severe withdrawal reactions have been reported. Garner, et al. reviewed some of the data on withdrawal from tricyclic antidepressants in *The Annals of Pharmacotherapy*. Some clinicians believe that withdrawal occurs because antidepressants down-regulate, and possibly reconfigure, receptor sites so that when the drug is removed, the body is left "crippled"-unable to respond with its own biochemicals.

Never combine different types of anti-depressants; anti-depressants and tryptophan; or anti-depressants and SAME without first consulting a physician.

SAME causes very few, if any, side effects. It has been given intramuscularly, intravenously, and orally with good results. It has been given to hundreds of patients with different types of depression-including patients debilitated from physical illness. It has been given to recovering drug and alcohol addicts to reduce depression and control drug cravings. Several authors have referred to it as the antidepressant for the '90s. The Foundation agrees. SAME has everything going for it that an antidepressant should: The Life Extension Foundation believes that people should try this non-toxic anti-depressant before resorting to more toxic antidepressant drugs. Anyone currently taking antidepressant drugs MUST consult their physician before switching to SAME. Never discontinue any anti-depressant, or start therapy, without first consulting your physician.

DOSAGE

The effective oral dose of SAME for depression is 800-1600 mg/day. The Foundation recommends that people begin by taking two 400 mg tablets per day-once in the morning, and once in the afternoon. A third tablet should be added at noon, if the

depression does not improve within 2 days. A fourth tablet in the evening may be necessary for some people. Severely depressed people have been given 1600 mg from the first day without significant adverse effect beyond dry mouth and nausea.

PART 1 of SAME (S-adenosylmethionine)

PART 3 of SAME (S-adenosylmethionine)

PART 4 of SAME (S-adenosylmethionine)

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