

HEALTH AND STRESS

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ADVANCES IN MULTIPLE SCLEROSIS & OTHER STRESS-RELATED RESEARCH

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Can Stress Contribute To The Onset And Course Of Multiple Sclerosis?

Multiple sclerosis (MS) is a chronic progressive disease of the nervous system in which the myelin covering or white protective sheath surrounding nerves is attacked. This can cause varying degrees of intermittent disruption of nerve signal transmission, particularly in pathways that affect vision, sensation and the use of the arms and legs. Multiple sclerosis literally means "many scars" but multiple also describes the characteristic remissions and relapses of symptoms that usually recur over many years. The disease is referred to as disseminated sclerosis in Great Britain to indicate that MS lesions are scattered throughout the body without any persistent pattern.

The type of symptoms as well as their severity vary depending on the location of the lesions but most often include visual disturbances or uncontrollable eye movement (nystagmus), slurred and other speech disturbances, loss of balance, poor coordination, tingling sensations, problems with perception of pain, touch or temperature, loss of bladder or bowel control, and partial or complete paralysis of other muscles.

Extreme or unusual fatigue is present in 80% of MS patients. Physical exhaustion occurs after mild exertion but the cause of mental fatigue is not clear. It is not related to the degree of disability since it may persist during remissions or when other symptoms are minimal. Patients often feel fine for the first few hours of the day but then become progressively tired

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for no apparent reason. Sleep disturbances are common in MS and could be a factor since some patients report feeling much improved after a short nap.

This is quite different than the chronic fatigue of clinical depression that occurs in about half of all multiple sclerosis patients at some time during the course of their illness. Other mental or emotional complaints include laughing or crying despite the absence of any apparent reason for mirth or sadness and there may

also be unexplained and sustained euphoria. While multiple sclerosis can cause brain lesions these do not seem to be related to either the frequency or severity of fatigue or any of the above paradoxical behaviors.



MS was delineated in 1868 by Dr. Jean-Martin Charcot, a pathologist at Paris's Salpêtrière Hospital for indigents and women with mental disorders. He was particularly interested in a malady then called hysteria that was believed to be caused by a severely stressful event like an accident but later could become progressive and persistent. In order to study this he mastered the new technique of hypnosis, since he viewed the hypnotic trance as similar to a bout of hysteria. He became famous for his achievements, and, as depicted on the left, physicians flocked from all over to witness his results and to study under him.

One of Charcot's students was Sigmund Freud, who was so impressed that he named his oldest son Jean-Martin after Charcot. The very first patient Freud treated on his return to Vienna in 1868 was his former Nanny, who had multiple sclerosis. The disease was then called "creeping paralysis" and was considered to be a mental disorder due to hysteria. That same year, Charcot reported his findings on a young woman he had been studying for years. In addition to slurred speech and abnormal eye movements she had a peculiar tremor of her hands that became worse whenever she attempted to touch or pick up an object. On autopsy, he found what he called "*la sclérose en plaques*" (multiple sclerosis) affecting the optic nerves, cerebellum, brain stem and spinal cord and noted that these locations corresponded with her clinical signs and symptoms. For years, including when I went to medical school, the diagnostic criteria for multiple sclerosis were "Charcot's triad": slurred speech, nystagmus and intention tremor. We now recognize that MS patients may exhibit none of these symptoms and that they can be found in other conditions. Charcot also described amyotrophic lateral sclerosis (ALS), a progressive degenerative neurologic disorder that is fatal due to respiratory failure. It is called Lou Gehrig's disease in the U.S. but is still referred to as Charcot disease in Europe. While a progressive downhill course can be seen in multiple sclerosis it is usually characterized by periods of remissions and exacerbation that frequently do not result in severe disability.

What causes multiple sclerosis is not clear but most authorities believe that it is an autoimmune disorder in which immune system components attack nervous system structures as if they were some foreign invader or infection. Certain viruses are known to produce nerve inflammation and damage the protective myelin sheath and more than a dozen, including measles, herpes, Epstein-Barr and canine distemper viruses have been investigated but no definitive link has been demonstrated. MS flare-ups frequently recur in asymptomatic patients or symptoms worsen after various viral infections, presumably due to stimulation of the immune system. Hepatitis B and influenza vaccines that activate immune responses have also been implicated and several years ago, hepatitis B vaccination was temporarily halted in France because it appeared to be associated with an outbreak of MS. Researchers suggested that this could have been due to aluminum compounds in the vaccine that have been shown to trigger or intensify immune system responses. Although subsequent studies done by vaccine manufacturers failed to find any such problems, a recent report by Harvard researchers confirmed that people vaccinated against hepatitis B had triple the risk of developing multiple sclerosis over the following three years. (*Neurology 2004; 63:838-42*)

Multiple sclerosis is most often diagnosed between the ages of 20 and 40 and its onset is rare in children or people over 55. Women are affected 50% more frequently than men are and it is more common in Caucasians, particularly those of Northern European ancestry. The incidence of MS increases progressively as you move away from the Equator and some studies have found it to be as much as five times greater in North America and Europe compared to the tropics. In Australia, the incidence is also five times higher in temperate Tasmania than in subtropical Queensland although the ethnic background is similar. (*BMJ Jan. 2005 330:120*) However, it is not seen in Eskimos, Gypsies and Bantus, and its very low incidence in native Indians in North and South America, Japanese and other Asians suggests that there may be genetic influences.

Researchers very recently reported that month of birth for people in the Northern Hemisphere may be a factor. (*BMJ, Dec. 2004 10:1136, Jan. 2005*) An analysis of over 42,000 patients from Canada, the UK, Sweden and Denmark showed a 13% increase in risk of multiple sclerosis for those born in May compared with November and a 19% decreased risk for those born in November compared with May. The effect was most evident in Scotland, where MS prevalence is the highest. Studies have shown that low vitamin D levels during pregnancy could affect brain development and vitamin D deficiency during the first and second trimester would be most apt to occur during winter months because of less exposure to sunlight prior to delivery in May. It has also been noted that the progressive increased incidence of multiple sclerosis since it was first described correlates closely with the increased use of chemicals in industry and agriculture here and in Europe and this also holds true for other autoimmune disorders.

Unlike ALS, in which 10% of patients have a clear family history and/or evidence of a genetic defect, multiple sclerosis is not believed to be hereditary and its occurrence in successive generations is very rare. However, some patients are thought to have a genetic predisposition that could be race related. In one study of hospitalized patients, blood relatives had a much higher incidence of other degenerative nervous and mental diseases compared to relatives of a control group admitted for fractures. As a result, it has been suggested that perhaps MS is simply one manifestation of some familial neuropathic disorder that is expressed in different ways depending on which genes are involved.

Many patients link the onset of symptoms to some stressful or traumatic event and are convinced that stress worsened their symptoms or was responsible for their recurrence when they were in remission. In his initial 1868 description, Charcot noted that emotional stress, adverse social circumstances and particularly persistent grief seemed to precipitate the disease. However, a 22 year-old Englishman named Augustus D'Este may have been the first to note this relationship. On December 13, 1822, he experienced the sudden onset of a severe disturbance in vision shortly after attending the unexpected funeral of a close relative and friend. As he wrote in his diary, "I was obliged to have my letters read to me, and their answers written for me, as my eyes were so attacked, that when fixed on minute objects, indistinctness of vision was the consequence. Soon after, they completely recovered their strength and distinctness of vision." Although he remained asymptomatic for some time, subsequent diary entries leave little doubt that this was the onset of multiple sclerosis, since it later progressed to include this and other typical neurological complaints. Another recent article reported that the risk of a recurrence of the disease doubled in the four weeks following stressful events such as the death of a family member, close friend or even a pet. (*Journal of Neurology Neurosurgery and Psychiatry 2004; 75:60*)

The role of stress in MS has been controversial since it is largely based on anecdotal reports. In one study, 100 multiple sclerosis patients and 73 controls with other neurological and non-neurological diagnoses were asked to rate the degree of stress they had experienced in the two years prior to the onset of symptoms. Four out of five MS patients reported high

degrees of stress during this period compared to only half of the control group. In a subsequent study, no difference was found in life change event stress scores in the month prior to a relapse of MS compared to baseline values or other four-week periods. However, when these patients were interviewed two years later, 60% believed that stress had adversely affected their condition and almost half felt that it had been responsible for a relapse. Another paper on the relapsing-remitting form of the disease showed that patients reporting more stress were at greater risk for an attack.

Critics point out that patients who suffer a relapse are more likely to seek an explanation and hence report stressful events during the preceding weeks. This is quite common in other conditions like cancer and heart attacks in which patients frequently attribute their illness to psychological factors. As previously emphasized, association does not mean causation, as is evident from the several hundred risk factors for heart attacks. Some suggest that so-called "psychological stress" and neurological relapse may even be "different temporally disseminated manifestations of the same disease process." (*BMJ 2004;328: 287*) Others claim that although reports of the significant role of stress in worsening multiple sclerosis abound there are also instances where stress seems to have had a beneficial effect. In addition, few articles offer a plausible rationale or mechanism of action to explain these purported relationships other than to cite effects on the immune system. The problem here is that immune system function can be measured in many different ways and that the same stressor can cause an increase in some components, a decrease in others, or have little effect either way. Stress can be assessed using various techniques ranging from daily hassles to antecedent life change events but it is doubtful that a broken shoe lace or getting stuck in a traffic jam on the way to an important appointment has the same effect on the immune system as the death of a spouse or loved one. Finally, although the stress of bereavement has been demonstrated to lower immune system defenses and cancer patients have been shown to have reduced immune system defense, this hardly proves that stress causes cancer. It is therefore not surprising that some studies have failed to find any consistent relationship between stress and recurrence or worsening of symptoms in MS patients.

In an attempt to make some sense out of all this, researchers recently reported on a review of all papers published in peer reviewed journals from 1965 to February 2003 containing the terms "stress", "trauma" and "multiple sclerosis". Of the 20 studies identified, 14 satisfied the strict criteria required to exclude possible confounding influences. A meta-analysis of these pivotal studies led to the conclusion that there was a consistent association between stressful life events and subsequent exacerbation or worsening of multiple sclerosis. However the data did not allow the linking of specific stressors to these setbacks. It was emphasized that these findings should not be interpreted as implying that MS patients are responsible for any deterioration in their status, which is not uncommon in cancer. Nothing could be worse for someone suffering from a serious disease than to be additionally burdened with the guilt that failure to improve or a downhill course is due to some weakness in their character, such as an inability to cope with stress. The authors expressed the hope that investigation of the psychological, neuroendocrine, and immune mediators of stressful life events on exacerbations might lead to new behavioral and pharmacological strategies that could provide more precise and useful information on the mechanisms that mediate their effects. (*BMJ 2004;328:731-736*) This reference is a rich source of citations, one of the most impressive being a prior publication of the senior author. Using sophisticated imaging techniques, he and coworkers had shown a clear correlation between the development of new MS brain lesions and increased stress levels as assessed by both major life change events and daily hassle scores. This was a careful prospective study that obviated any possible errors that might be due to recall bias. (*Neurology 2000;55: 55-61*)

As noted, multiple sclerosis is believed to be an autoimmune disorder in which the immune system attacks normal nervous tissues as if they were foreign. A similar disease known as experimental autoimmune encephalomyelitis (EAE) that is also characterized by relapses and remissions reminiscent of MS can be induced by injecting animals with spinal cord tissue from other species. The resultant lesions show large amounts of immune system T-helper white cells like those seen in humans and the disease will not occur if these T-lymphocytes are absent due to removal or irradiation of the thymus. This animal model has made it possible to study the effects of agents that affect the immune system, including interferons, cortisone and related steroids as well as chemotherapy drugs used to treat cancer. Some of these are now approved for treating multiple sclerosis. Studies of the effects of stress on EAE have been inconclusive but it would obviously be difficult to reproduce much less measure the stress of depression in experimental animals. Tysabri, a monoclonal antibody that is administered intravenously once a month received accelerated FDA approval three months ago because of the consistent and significant improvement seen in patients after one year. More information may be gained by studying the effects of stress on the response to this or other new treatments that have fairly predictable results. The jury is still out, but based on results in rheumatoid arthritis, lupus and other autoimmune disorders, it seems likely that certain types of emotional distress will be shown to play a role in the onset and course of multiple sclerosis. Discovering how these effects are mediated may lead to advances in both our understanding and treatment of this unpredictable and disturbing disease.

Stress, Skin, Cervical And Breast Cancers

According to Johns Hopkins researchers, chronic stress can speed up the onset of skin cancer. They exposed mice to large amounts of UV light and the scent of fox urine, a powerful stressor for rodents. The stressful exposure began 14 days before irradiation and continued three times a week for the duration of the study. The first skin cancers appeared in eight weeks and after 21 weeks, 14 of the 40 stressed mice had at least one tumor. Non-stressed controls exposed only to UV light and housed in another room did not begin to develop tumors until 13 weeks. At the end of the study, 35% of the stressed mice had at least one tumor compared to only 7 % of controls. Most tumors in both groups were squamous cell carcinomas which, unlike basal cell cancers, can spread to other parts of the body. The study director noted that if these results were extrapolated to the problem of sun-induced skin cancers in humans that "stress-reducing techniques could help to ameliorate the development of these tumors." Fair-skinned people exposed to large amounts of UV light and patients previously diagnosed with squamous cell skin cancer, genetic diseases or organ transplants that predispose them to cancer are considered to be at high-risk and might benefit the most from stress reduction training. Chronic stress is known to suppress immune system defenses that recognize foreign or abnormal cells and target them for destruction. However, acute episodic stress can increase immune system resistance and plans are under way to investigate the mechanisms that provide this potential protection. (*Journal of the American Academy of Dermatology, December 2004*)

The ability of psychosocial stress to increase risk of cervical cancer was confirmed in a study of almost 300 low-income women receiving family planning services at health department clinics. Divorce, infidelity, an increase in the number of arguments, and psychological and physical partner violence were especially likely to be associated with an increase in precancerous lesions. (*Psychosomatic Medicine 2003 65:644-651*) Stress was also associated with increased risk of the most common type of uterine cancer in monkeys living in groups with an established social hierarchy. It has previously been shown that subordinate monkeys exhibit various indicators of increased stress such as elevated heart rate, higher levels of cortisol and increased cardiovascular disease. Researchers compared breast and uterine endometrial tissue from dominant and subordinate postmenopausal monkeys noting the type of cells present, the percentage of dividing cells and the number of

estrogen and progesterone receptors. Changes in these parameters, that are predictive of a predisposition to cancer, indicated greater risk of uterine endometrial cancer but not breast cancer in the subordinate group. It should be noted that only increased risk rather than an actual increase in uterine cancer was found. (*Menopause 2004 Jul-Aug;4:389-99*)

There are conflicting reports about the role of stress in the development and progression of breast cancer, possibly because of unknown genetic or other confounding factors, in a Finnish study of over 10,000 same-sex twins followed from 1982 to 1996. In 1975, and again in 1981 and 1990, subjects completed baseline questionnaires designed to assess known breast cancer risk factors as well as stressful life change events. A review of the 180 breast cancer cases that developed during the 15-year study period found that accumulated life stressors in the five years before the baseline assessment was associated with an increase in breast cancer. Severe life stresses (divorce/separation, death of a spouse, close friend or relative) were associated with a two-fold increase in breast cancer. The researchers confirmed these findings in studies of twin pairs in which one twin developed breast cancer and the other did not. Confounding factors like weight change, smoking, or alcohol use that often accompany stressful life events were excluded and results were also adjusted to eliminate the influence of known breast cancer risk factors such as nulliparity or late age at the first term pregnancy. (*Am J Epidemiol 2003,57:415-23*)

The unexplained rise in breast cancer in recent decades has been attributed to an increase in job stress in working women. It has been established that the earlier a woman becomes pregnant the less likely she is to develop breast cancer because pregnancy reduces levels of prolactin, a pituitary hormone that stimulates breast tissue growth and promotes breast cancer in experimental animals. Statistics show that as more and more career oriented women enter the work force they tend to remain single, or marry and decide not to have children, or do so when they are much older. Single career females also have fourteen times more deadly ovarian cancer than a matched group of homemakers. However, a recent report that reviewed data from over 37,000 women who were followed for eight years found no increased breast cancer in women who had rated their stress at work as "severe" compared to others who described their job stress as "minimal". Critics claim that the follow-up period may not have been long enough and that stress levels could also have changed during the study. (*Am J Epidemiol 2004 Dec. 60:1079-1086*)

Stress, Heart Attacks, Hypertension, Stroke And Sudden Death

Type A behavior is as significant a risk factor for heart attacks as smoking, elevated blood pressure or cholesterol. As suggested in a prior Newsletter, Type A's may be addicted to their own adrenaline surges and unconsciously seek ways to get the "high" they provide. As a result they often create deadlines and challenges when none need exist, such as leaving an assignment to the last minute or reaching a certain destination by a specific time when driving for long periods. Swedish doctors recently reported that the stress of facing a high-pressure deadline at work dramatically increased the likelihood of having a heart attack within the next 24 hours. They monitored the number of first heart attacks in over 3500 workers aged 45-70 at the start of the study. Participants completed questionnaires about specific life event changes within the past 12 months, provided detailed information on stressors at work and were also interviewed and examined. During the follow-up period, 1381 men and women experienced the first onset of a non-fatal myocardial infarction. Men were six times more likely to have a heart attack if they had taken on increased responsibilities at work, particularly when these were viewed as stressful. Although only 8% of the study group had experienced a stressful situation at work the day before their heart attack, this was much higher than similar stresses during the same period that were not job related. An intense impending high-pressure deadline increased the risk of a heart attack within the next 24 hours by a factor of six in men and

three in women. In contrast, after other known risk factors were taken into account, no link was found between accumulated stressful life events in the preceding year and increased risk of heart attack. (*J Epidemiol Community Health* 2005 Jan; 59:23-30)

The large Swedish Malmo Preventive Project study did find that chronic stress was associated with increased stroke as well as heart disease. Between 1974 and 1980, more than 13,000 men and women reported in periodic questionnaires that they had experienced significant chronic stress during the previous five years. Their medical records were compared with those of 10,000 controls without this complaint and both groups were followed for two decades. The results revealed that those reporting chronic stress were 14% more likely to develop heart problems or stroke than non-stressed controls regardless of other risk factors. The most significant effect was seen in men with chronic stress, who were twice as likely to suffer a fatal stroke than controls. (*Eur Heart J.* 2004 25:867-73) Acute stress and especially anger was also associated with risk of non-fatal stroke in an Israeli study of 200 patients who were interviewed within a few days after their attack. They were asked to rate their moods and recall notable events hour by hour for the day leading up to the start of their symptoms. Events and emotions in the two hours just before the stroke were then compared with what had been reported for the corresponding two-hour period the day before. It was found that 43 patients had experienced significant anger or negative emotions during the two hours before the stroke, but that only six had reported such symptoms on the previous day. This effect was more common in males and was most pronounced in patients younger than 69. According to the lead author, severe anger and similar emotional triggers could increase the risk of stroke risk 14-fold over the following two hours (*Neurology* 2004;63:2006-201)

It has long been recognized that sudden death is often precipitated by a severely stressful event. This has been attributed to ventricular fibrillation resulting from increased secretion of adrenaline and similar compounds from the adrenal medulla and while it occurs most often in patients with existing heart disease it has also been seen in healthy young adults with no coronary atherosclerosis. Such hormonal surges also cause blood pressure elevations that can result in a fatal stroke. British researchers now report that sudden cardiac death from emotional stress may be triggered by erratic brain signals to the heart. Nerve connections from the brain stem to the left and right side of the heart regulate heart rate in response to physical or mental activity. These electrical stimuli normally travel across the heart in a smooth and even fashion that results in a regular rhythm regardless of rate. However, if the balance between right and left sympathetic nerve stimulation is disrupted the propagation of the electrical wave becomes irregular and the resultant short circuits can result in various rhythm disturbances. In a very recent report, volunteers with a history of heart disease who had their brain stem activity monitored by PET scanning while undergoing mental stress tasks showed a significant degree of asymmetry between left and right sympathetic output. Sophisticated ECG recordings revealed concomitant ventricular repolarization abnormalities that are known to be associated with serious arrhythmias. The study authors suggest that more severe mental or emotional stress could result in sudden death via this mechanism, especially in patients with known coronary heart disease. (*Brain* 2005 128:75-85)

Stress, Infertility And Miscarriages?

Women who fail to conceive despite repeated careful attempts for years and the absence of any known abnormalities are apt to receive advice such as "Just relax and you'll get pregnant", "Don't think about it too much", "You're trying too hard", "Take a vacation" or "Just adopt and you'll get pregnant". These suggestions are based on the assumption that stress causes infertility and are supported by numerous anecdotal reports. On the other hand, it can be difficult to distinguish between cause and effect since months of doctor's visits, continuous monitoring and costly medications without any results can be very

stressful. This is often intensified by feelings of self-blame due to the implication that you are causing the problem because of an inability to relax or cope with stress in addition to being overly concerned. Many infertile women also tend to feel like outsiders at social functions when the conversation inevitably turns to children related topics.

Infertility can significantly diminish sexual drive and relationships as each encounter becomes more of a scheduled duty rather than a spontaneous expression of love. Men with diminished sperm counts similarly often experience temporary impotence even though there is no relationship between virility and sperm production and high stress levels can also decrease sperm production. In one German study, rapists who had impregnated their victims and were now on death row were asked to give semen samples. All had sperm counts of zero although they had obviously been fertile at the time of the rape. Stress can cause spasm in the fallopian tubes and hormonal disturbances that result in irregular or no menstruation and double the likelihood of dysmenorrhea. However, there is no solid proof that stress causes infertility. (*Occup Environ Med* 2004; 61:1021-6)

The evidence is much stronger with respect to miscarriages. Studies have shown that when pregnant mice are exposed to loud noise levels there is a rise in cortisol that suppresses the production of progesterone, a hormone that is crucial to maintaining a healthy pregnancy. Along with this is a fall in progesterone-induced blocking factor (PIBF) which helps prevent the immune system from attacking the placenta as if it were foreign. When PIBF levels are low the blood supply to the placenta is impaired, which can cause rejection of the fetus. A recently reported study that monitored 854 pregnant women confirmed the stress-low PIBF link to miscarriage. All subjects had blood tests to measure hormones and completed questionnaires to assess stress levels. Researchers found that the 55 women who miscarried were much more likely to have reported increased stress and that they also had lower progesterone and PIBF levels. Progesterone supplementation has long been used to sustain pregnancy in patients with a prior history of miscarriage. These studies may help identify others at increased risk and also improve progesterone dosage to provide optimal protection. (*New Scientist*, Nov. 17. 2004)

Does DHEA or Vitamin C Protect Against Damage Due To Stress?

Dehydroepiandrosterone (DHEA), which is produced in the adrenal cortex, is a precursor of many other steroid hormones. DHEA levels are highest around age 20 to 25 but fall progressively with aging so that they are only about 20 -25% of peak values in senior citizens. Since DHEA can also be obtained by extracting a sterol from wild yams it is viewed in the U.S. as a natural substance that can be sold without a prescription. As a result it has been touted as a virtual "fountain of youth" that can boost sex drive, enhance energy, mood and memory, improve immune system function, increase muscle mass and promote weight loss. While scientists are understandably skeptical about these and other extravagant and unproven claims, there is now evidence that DHEA may serve some protective function during stressful situations.

Researchers studied 25 young military recruits who took part in a survival school that mimicked the environment in a POW camp. Five days prior to the ordeal, blood and saliva samples were taken to measure DHEA and cortisol levels and participants completed questionnaires to assess stress related to their new environment. The survival training included mental and physical stressors such as food and sleep deprivation and irritating interrogations. Following a 30-minute interrogation period, levels of cortisol and DHEA were again measured. DHEA-cortisol ratios during this period were significantly higher in subjects who reported fewer symptoms of anxiety and tension and who also exhibited superior military performance. These results support previous studies showing that DHEA blocks the brain damage due to increased levels of cortisol and similar glucocorticoids released during stress. (*Arch Gen Psychiatry* 2004, 61:819-25)

Ascorbic acid (vitamin C) is released from the adrenal cortex during stress and was used to assess the severity of various stressors prior to the advent of accurate hormonal assays. Stress increases vitamin C requirements and high doses of vitamin C are often claimed to provide numerous benefits ranging from the prevention and treatment of colds to retarding the aging process. Animal studies have shown that vitamin C can reduce the secretion of cortisol during chronic stress as well as the typical signs that are usually associated with emotional and psychological stress. To test this in humans, 120 healthy people were subjected to significant mental stress by performing a public speaking task as well as mathematical problems under time pressure. Half of the participants received 1000 mg. of vitamin C three times a day before and during the testing period and the other half served as a control group. Dietary intake of vitamin C was similar in both groups. Researchers reported that cortisol levels, blood pressure and other signs of stress were all significantly higher in controls compared to those taking vitamin C. The vitamin C group also reported feeling less stressed. Vitamin C is the most commonly consumed dietary supplement but the amounts used in this study were 50 times the official U. S. recommendation and it is not known if smaller doses would provide similar results. (*Psychopharmacology 2004, 59:319-24*)

Can Acne And Baldness Be Due To Stress?

Acne, wrinkles and hair loss are believed by many to be caused or aggravated by stress but there are few supportive scientific studies. In one report, 15 female and 7 male university students with acne filled out questionnaires to rate stress levels and were followed for a month during a non examination period as well as three days before and 7 days after important examinations. Researchers confirmed that students reporting much more stress related to examinations also had a worsening of their acne, even when factors like changes in diet and hours or quality of sleep were taken into consideration.

The reason for this is not clear but acne forms when oily secretions from sebaceous glands beneath the skin plug up the pores. Male hormones can increase these secretions, which explains why acne tends to be a particular problem in adolescent males when testosterone production surges. The culprit appears to be dihydrotestosterone (DHT), a metabolite that increases sebum production and oily skin and has been implicated in male and female baldness. It has now been discovered that corticotropin releasing hormone (CRH) from the hypothalamus that initiates the pituitary response to stress is also released in sebaceous glands causing oily skin that sets the stage for acne. In addition, studies in mice suggest that CRH could contribute to the hair loss seen in male pattern baldness that is generally attributed to androgens. (*Arch Dermatol 2003; 139:897-900*)

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