Mental Wellness Task Force Lunch and Learn Series
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Perceiving something as stress will cause an elevation of several hormones (cortisol, norepinephrine, epinephrine). The elevation of these hormones can have an effect on the function of the brain which are:

1. You have difficulty thinking clearly and focusing. You may act impulsively (action without consideration of its consequences)
2. You become depressed and have the 'blues (often with a loss of brain cells in the hippocampus)
3. Your ability to remember things may decrease at a younger age than would occur if you could cope with stress (often with a loss of brain cells in areas of the brain associated with memory)

Why do I mention the loss of brain cells? Because there is emerging evidence that keeping the concentration of your stress hormones low results in an increase in the number of brain cells!

Issues:

- Childhood physical abuse history is associated with depression
- Compared to subjects who had never been bullied, those exposed to bullying in school were at a significantly increased risk of having been diagnosed with depression between the ages 31-51 years
- Job strain, high job demand with perception of inadequate reward, a low role in making decisions at work, are associated with a reduced quality of health including an increased risk of heart disease and depression

A rapid elevation of norepinephrine when experiencing an acute stressor (unanticipated, short duration) will interfere with the ability to focus and think clearly. There are ways to rapidly lower the concentration of stress hormones. However, because it is difficult to think clearly when experiencing acute stress you often don't remember to do one of the 3 things that can rapidly lower the concentration of stress hormones.

Therefore, I recommend that you put sticky notes up to remind you or that you share this information with friends and colleagues so that when they see you are experiencing stress they can remind you to do one of these things and you can do the same for them.

Techniques to calm your mind and improve the quality of your mental and physical health when experiencing an **ACUTE STRESS** (one that is unanticipated, has sudden onset, and short duration):

- Deep breathing
- Thinking of funny things
- Chanting

DEEP BREATHING FOR CALMING YOURSELF WHEN EXPERIENCING AN ACUTE STRESSOR

The following focuses on a technique that you will be able to use to calm your mind, reduce the concentration of stress hormones in your blood, and contribute to an enhancement of your health.

You use this technique when something happens that you find disturbing and you feel an increased amount of anxiety and stress. This techniques will help to rapidly calm you, help you to think more clearly, and to focus on what you are doing. It is even helpful for children who are having difficulty keeping calm.

Q: What is abdominal or diaphragmatic breathing?
A: This is a way that you can rapidly increase the amount of oxygen in your blood to a much greater extent than occurs when you take a breath by expanding the wall of your chest. When you take an abdominal breath and increase the amount of oxygen in your blood, your brain will detect the increased oxygen and will respond by decreasing the concentration of stress hormones in the blood.

The diaphragm separates the abdominal and chest cavities. Pushing the wall of your abdomen out causes the diaphragm to drop further than when you merely expand your chest wall to take in air. This increases the space that the lungs can expand into and increases the amount of air and oxygen that is inhaled. As we age, however, most of us take more shallow breaths and get less oxygen into our blood than we are capable of.

Q: What is the difference between abdominal/diaphragmatic breathing and chest breathing?
A: Breathing from the chest delivers approximately a teacup of air to the lungs (about 500 ml), breathing from the abdomen can deliver as much as 8 times the amount of air to the lungs. This makes a huge difference in the amount of oxygen in the blood

How we breathe also has an impact on our nervous system. Less oxygen stimulates the production of shorter, more “restless” beta waves in the brain. More oxygen stimulates the longer, slower alpha waves associated with relaxation and calm mind states. For this reason, breathing from the abdomen is helpful in eliciting relaxation and protecting the body from the harmful effects of stress.

Q: Abdominal breathing is difficult. How do I learn to breathe this way?
A: You’re right. It is hard to change the way you breathe. Practice! You’ve had a lifetime of breathing incorrectly; it will take a while to learn to breathe correctly. Breathing is generally easier to practice initially when lying down, so practice at home lying down or sitting up.

Use the following to learn how to increase the volume of air flowing into the lungs. The essential point is that by pushing the abdominal wall out, the diaphragm will drop, increasing the space that the lungs can expand into. This maximizes the flow of air into the lungs and of oxygen into the blood.

1. Put your right hand on your abdomen, right at the navel, and put your left hand on your chest, right in the center. You may find it helpful to close your eyes.
2. Take a deeper inhalation than usual and focus on the rising of the abdomen as the lungs fill with air and the diaphragm flattens down, causing the belly to rise. You should feel your stomach rising about an inch as you breathe in, and falling about an inch as you breathe out.
3. Most of the movement should be in the lower hand; the other hand on the chest moves only slightly.
4. The trick to shifting from chest to abdominal breathing is to make one or two full exhalations (pushes air out from bottom of lung, creating a vacuum that will pull in an abdominal breath on your next inhalation), pause, then inhale slowly.
5. Nostril breathing is generally recommended but if you are more comfortable breathing through your mouth, do so.
6. You should not take more than five deep breaths. If you feel a little dizzy, take fewer deep breaths.

It is important to emphasize that one needs to practice this technique to increase its effectiveness. Remember, it is impossible to take deep breaths if you are holding your stomach in.

Important:

- Use this technique of breathing when you experience something that is upsetting to you. Do your regular breathing at other times.

- If you experience an acute stressor that lasts for several hours, only use abdominal breathing intermittently, not all the time. Take 3-5 deep breaths to calm yourself and then breathe regularly. When you again feel anxious, take 3-5 deep breaths.

HUMOR FOR CALMING YOURSELF WHEN EXPERIENCING AN ACUTE STRESSOR

Another way to rapidly calm yourself when experiencing an acute stressor is to find something to laugh about. Of course when you are being stressed it is difficult to think of something funny, so it is a good idea to have something ready. To do this find some calm time and think of some things that make you laugh. It may be an episode of a TV show, a movie, or things you have experienced. Select 3-5 memories that make you laugh and store them away in your mind. You can call the place in your mind where you keep your funny memories your “funny relaxer”. Then

when something is upsetting you and causing stress and you don't feel like doing deep breathing, go to your "funny relaxer" and lighten up as you chuckle to yourself.

CHANTING FOR CALMING YOURSELF WHEN EXPERIENCING AN ACUTE STRESSOR

Another technique that we have found to be effective to help reduce the response to an acute stress is to say a brief relaxing chant to yourself. The chant can be a religious phrase or just a few words, such as "I am a good person", or, "All will be well", or "I will be well".

After deciding on the words you will use you set them to a simple tune, one that you find pleasant or one that has a religious feeling to it, such as the tune of a Gregorian chant.

First chant your phrase quietly. Do this several times. Then do it without making any sound. While you are doing this feel calm, relaxed, and comfortable. Practice this for several days, quietly saying your chant to yourself while being in a relaxed state. We want your mind to associate the chant with a relaxed feeling.

Now, when you experience an acute stress say the chant silently to yourself. Notice how this relaxes and calms you and allows you to focus.

At different times of experience with stress you may decide to take a few deep breaths, go to your "funny relaxer", or do your chant. All may be effective for you, or just one or two of them. Decide what works best for you. Use what is effective to calm you.

DEPRESSION

Depression can have varying degrees of severity, duration, and causation. The information I am going to discuss apples only to mild and moderate depression, not severe depression.

Superficial description of the types of depression:

Mild depression usually causes symptoms that impact on our daily activities. People are less interested in doing things they previously enjoyed, unusual irritability, reduced motivation in work, home or social activities are common. However function continues, perhaps not as well as normal.

Moderate depression can cause difficulties with social, work and domestic activities. The characteristics described for mild depression are worse. Simple things start to require real effort or just get neglected.

Severe depression causes considerable distress or agitation, loss of self-esteem or feelings of uselessness and guilt. Unlikely to be able to continue with work, social and domestic activities. Suicide is a distinct and major danger.

I'd like you to imagine how you feel when you have a cold

- Effect on the body-Muscle and joint aches
- Effect on the brain
  - Tired
  - Down in the dumps (feeling blue)
  - Loss of appetite

The feelings of tiredness, having the blues (depression) and loss of appetite are due to substances (called cytokines) that are released by your immune system in response to the infection.

The cytokines are transported to the brain and cause the effects mentioned above.

We are now learning that depression, in some people, is caused by cytokines released from cells of the immune system that travel to the brain. Interestingly, when everything is working well the brain tells the adrenal gland that the immune system has been activated and the adrenal then releases cortisol to shut off the production of cytokines and you feel fine. However, if one has experienced high levels of stress early in life (physical, mental, or sexual abuse) the control mechanism may not work properly and cytokine production stays high leading to prolonged feelings of depression.

In addition, the etiology of clinical depression is undergoing further reevaluation based on studies indicating that sustained elevated levels of glucocorticoids can damage neurons that are important to the etiology of depression. High levels of chronic stress may have this effect on the brain. Thus, depression may be a disease in which neurodegeneration is a critical component. A possible mechanism of the action of antidepressant medication is stimulation of the regeneration of neurons damaged by glucocorticoids.

Remaining married in late adulthood affords men unique and robust protection against elevated levels of blood markers associated with inflammation.

Elevated blood markers associated with inflammation are associated with poorer physical function in older adults with various comorbidities, as assessed by a common battery of clinical assessments. Chronic subclinical inflammation may be a marker of functional limitations in older persons across several diseases/health conditions.

Low childhood SES and a harsh early family environment appear to be related to elevated blood markers associated with inflammation in adulthood.

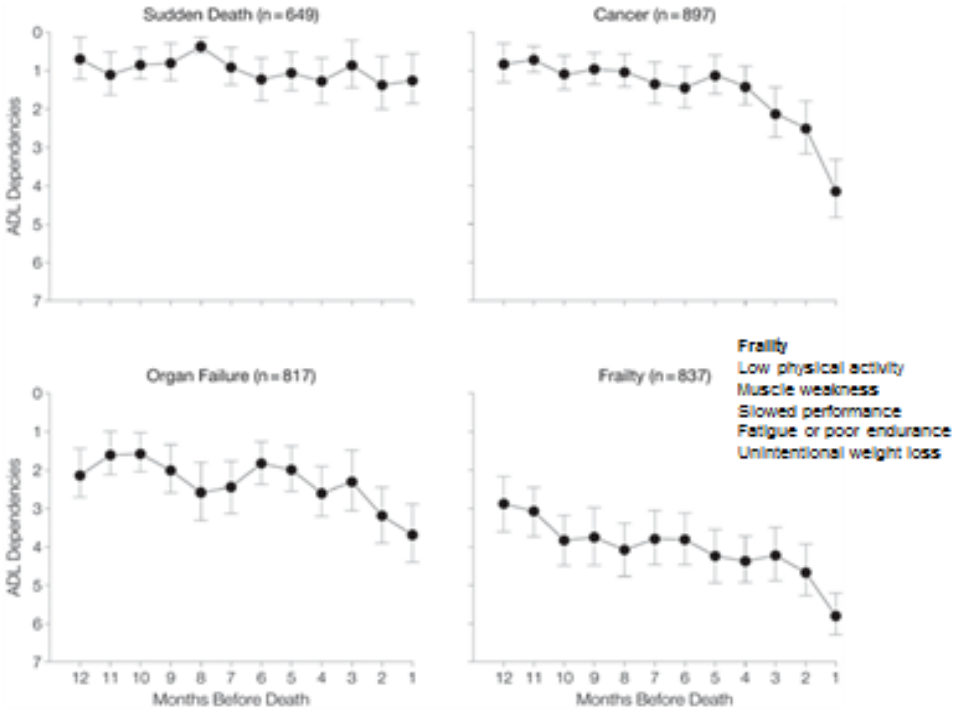
MILD AND MODERATE DEPRESSION RESPOND TO TREATMENT WITH ANTIDEPRESSANT MEDICATION

CONSIDERABLE EVIDENCE SUGGESTS THAT THE REASON THE ANTIDEPRESSANT MEDICATION WORKS IS DUE TO A PLACEBO EFFECT (WATCH: http://www.cbsnews.com/news/treating-depression-is-there-a-placebo-effect/)

MY RECOMMENDATION FOR INDIVIDUALS WITH MILD OR MODERATE DEPRESSION IS TO INCREASE THE AMOUNT OF PHYSICAL ACTIVITY THEY ENGAGE IN (WALK MORE) AND INCREASE THE AMOUNT OF TIME THEY SPEND WITH PEOPLE (VOLUNTEER SOMEWHERE, JOIN A CLUB THAT DOES SOMETHING OF INTEREST TO YOU, SEE IF THERE IS A WALKING GROUP IN YOUR NEIGHBORHOOD YOU CAN JOIN, BECOME A MENTOR IN A PUBLIC SCHOOL SYSTEM, READ TO CHILDREN AT YOUR LOCAL LIBRARY……).

REDUCE YOUR FEELING LONELY

HOW DO YOU WANT TO DIE?



Participants were asked if they needed help or were unable to perform each of the following 7 activities of daily living (ADLs): walking across a small room, bathing, grooming, dressing, eating, transferring from bed to chair, and using the toilet.

For those interested I am providing some literature related to the possible effect of stress as a cause of depression

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http://www.ncbi.nlm.nih.gov/pubmed
Following each PMID number below is the title of the publication and the abstract.

PMID: 22423117

Depression: an inflammatory illness?
Major depressive disorder (MDD) is associated with significant morbidity and mortality. Findings from preclinical and clinical studies suggest that psychiatric illnesses, particularly MDD, are associated with inflammatory processes. While it is unlikely that MDD is a primary 'inflammatory' disorder, there is now evidence to suggest that inflammation may play a subtle role in the pathophysiology of MDD. Most of the evidence that links inflammation to MDD comes from three observations: (a) one-third of those with major depression show elevated peripheral inflammatory biomarkers, even in the absence of a medical illness; (b) inflammatory illnesses are associated with greater rates of MDD; and (c) patients treated with cytokines are at greater risk of developing major depressive illness. We now know that the brain is not an immune privileged organ. Inflammatory mediators have been found to affect various substrates thought to be important in the aetiopathogenesis of MDD, including altered monoamine and glutamate neurotransmission, glucocorticoid receptor resistance and adult hippocampal neurogenesis. At a higher level, inflammation is thought to affect brain signalling patterns, cognition and the production of a constellation of symptoms, termed 'sickness behaviour'. Inflammation may therefore play a role in the aetiology of depression, at least in a 'cohort' of vulnerable individuals. Inflammation may not only act as a precipitating factor that pushes a person into depression but also a perpetuating factor that may pose an obstacle to recovery. More importantly, inflammatory markers may aid in the diagnosis and prediction of treatment response, leading to the possibility of tailored treatments, thereby allowing stratification of what remains a heterogeneous disorder.

PMID: 12450959

Childhood trauma associated with smaller hippocampal volume in women with major depression. Smaller hippocampal volume has been reported only in some but not all studies of unipolar major depressive disorder. Severe stress early in life has also been associated with smaller hippocampal volume and with persistent changes in the hypothalamic-pituitary-adrenal axis. However, prior hippocampal morphometric studies in depressed patients have neither reported nor controlled for a history of early childhood trauma. In this study, the volumes of the

hippocampus and of control brain regions were measured in depressed women with and without childhood abuse and in healthy nonabused comparison subjects. METHOD: Study participants were 32 women with current unipolar major depressive disorder-21 with a history of prepubertal physical and/or sexual abuse and 11 without a history of prepubertal abuse-and 14 healthy nonabused female volunteers. The volumes of the whole hippocampus, temporal lobe, and whole brain were measured on coronal MRI scans by a single rater who was blind to the subjects' diagnoses. RESULTS: The depressed subjects with childhood abuse had an 18% smaller mean left hippocampal volume than the nonabused depressed subjects and a 15% smaller mean left hippocampal volume than the healthy subjects. Right hippocampal volume was similar across the three groups. The right and left hippocampal volumes in the depressed women without abuse were similar to those in the healthy subjects. CONCLUSIONS: A smaller hippocampal volume in adult women with major depressive disorder was observed exclusively in those who had a history of severe and prolonged physical and/or sexual abuse in childhood. An unreported history of childhood abuse in depressed subjects could in part explain the inconsistencies in hippocampal volume findings in prior studies in major depressive disorder.

PMID: 18602762

The link between childhood trauma and depression: insights from HPA axis studies in humans. Childhood trauma is a potent risk factor for developing depression in adulthood, particularly in response to additional stress. We here summarize results from a series of clinical studies suggesting that childhood trauma in humans is associated with sensitization of the neuroendocrine stress response, glucocorticoid resistance, increased central corticotropin-releasing factor (CRF) activity, immune activation, and reduced hippocampal volume, closely paralleling several of the neuroendocrine features of depression. Neuroendocrine changes secondary to early-life stress likely reflect risk to develop depression in response to stress, potentially due to failure of a connected neural circuitry implicated in emotional, neuroendocrine and autonomic control to compensate in response to challenge. However, not all of depression is related to childhood trauma and our results suggest the existence of biologically distinguishable subtypes of depression as a function of childhood trauma that are also responsive to differential treatment. Other risk factors, such as female gender and genetic dispositions, interfere with components of the stress response and further increase vulnerability for depression. Similar associations apply to a spectrum of other psychiatric and medical disorders that frequently coincide with depression and are aggravated by stress. Taken together, this line of evidence demonstrates that psychoneuroendocrine research may ultimately promote optimized clinical care and help prevent the adverse outcomes of childhood trauma.

PMID: 18250257

Influence of child abuse on adult depression: moderation by the corticotropin-releasing hormone receptor gene. CONTEXT: Genetic inheritance and developmental life stress both contribute to major depressive disorder in adults. Child abuse and trauma alter the endogenous stress response, principally corticotropin-releasing hormone and its downstream effectors,

suggesting that a gene x environment interaction at this locus may be important in depression. OBJECTIVE: To examine whether the effects of child abuse on adult depressive symptoms are moderated by genetic polymorphisms within the corticotropin-releasing hormone type 1 receptor (CRHR1) gene. DESIGN: Association study examining gene x environment interactions between genetic polymorphisms at the CRHR1 locus and measures of child abuse on adult depressive symptoms. SETTING: General medical clinics of a large, public, urban hospital and Emory University, Atlanta, Georgia. PARTICIPANTS: The primary participant population was 97.4% African American, of low socioeconomic status, and with high rates of lifetime trauma (n = 422). A supportive independent sample (n = 199) was distinct both ethnically (87.7% Caucasian) and socioeconomically (less impoverished). MAIN OUTCOME MEASURES: Beck Depression Inventory scores and history of major depressive disorder by the Structured Clinical Interview for DSM-IV Axis I Disorders. RESULTS: Fifteen single-nucleotide polymorphisms spanning 57 kilobases of the CRHR1 gene were examined. We found significant gene x environment interactions with multiple individual single-nucleotide polymorphisms (eg, rs110402, P = .008) as well as with a common haplotype spanning intron 1 (P < .001). Specific CRHR1 polymorphisms appeared to moderate the effect of child abuse on the risk for adult depressive symptoms. These protective effects were supported with similar findings in a second independent sample (n = 199). CONCLUSIONS: These data support the corticotropin-releasing hormone hypothesis of depression and suggest that a gene x environment interaction is important for the expression of depressive symptoms in adults with CRHR1 risk or protective alleles who have a history of child abuse.

PMID:15759588

Early adversity and the prospective prediction of depressive and anxiety disorders in adolescents. The current study was a prospective exploration of the specificity of early childhood adversities as predictors of anxiety and depressive disorders in adolescents. Participants were 816 adolescents (414 males, 402 females) with diagnostic information collected at age 15; information on early adversities had been collected from the mothers during pregnancy, at birth, age 6 months, and age 5 years for a related study. Adolescents with "pure" anxiety disorders were compared with adolescents with "pure" depressive disorders (major depressive disorder, dysthymia), and these groups were compared to never-ill controls. Analyses controlled for gender and maternal depression and anxiety disorders. Results indicated that adolescents with anxiety disorders were more likely than depressed youth to have been exposed to various early stressors, such as maternal prenatal stress, multiple maternal partner changes, and more total adversities, whereas few early childhood variables predicted depressive disorders. Even when current family stressors at age 15 were controlled, early adversity variables again significantly predicted anxiety disorders. Results suggest that anxiety disorders may be more strongly related to early stress exposure, while depressive disorders may be related to more proximal stressors or to early stressors not assessed in the current study.

PMID:15488250

Adverse childhood experiences and the risk of depressive disorders in adulthood.

BACKGROUND:

Research examining the association between childhood abuse and depressive disorders has frequently assessed abuse categorically, thus not permitting discernment of the cumulative impact of multiple types of abuse. As previous research has documented that adverse childhood experiences (ACEs) are highly interrelated, we examined the association between the number of such experiences (ACE score) and the risk of depressive disorders. **METHODS:**

Retrospective cohort study of 9460 adult health maintenance organization members in a primary care clinic in San Diego, CA who completed a survey addressing a variety of health-related concerns, which included standardized assessments of lifetime and recent depressive disorders, childhood abuse and household dysfunction. **RESULTS:**

Lifetime prevalence of depressive disorders was 23%. Childhood emotional abuse increased risk for lifetime depressive disorders, with adjusted odds ratios (ORs) of 2.7 [95% confidence interval (CI), 2.3-3.2] in women and 2.5 (95% CI, 1.9-3.2) in men. We found a strong, dose-response relationship between the ACE score and the probability of lifetime and recent depressive disorders ($P<0.0001$). This relationship was attenuated slightly when a history of growing up with a mentally ill household member was included in the model, but remained significant ($P<0.001$). **CONCLUSIONS:**

The number of ACEs has a graded relationship to both lifetime and recent depressive disorders. These results suggest that exposure to ACEs is associated with increased risk of depressive disorders up to decades after their occurrence. Early recognition of childhood abuse and appropriate intervention may thus play an important role in the prevention of depressive disorders throughout the life span.

PMID: 22494534

Clustering of depression and inflammation in adolescents previously exposed to childhood adversity.

BACKGROUND:

There is mounting interest in the hypothesis that inflammation contributes to the pathogenesis of depression and underlies depressed patients' vulnerability to comorbid medical conditions. However, research on depression and inflammation has yielded conflicting findings, fostering speculation that these conditions associate only in certain subgroups, such as patients exposed to childhood adversity.

METHODS:

We studied 147 female adolescents. All were in good health at baseline but at high risk for depression because of family history or cognitive vulnerability. Subjects were assessed every 6 months for 2.5 years, undergoing diagnostic interviews and venipuncture for measurement of two inflammatory biomarkers, C-reactive protein (CRP) and interleukin-6 (IL-6). Childhood

adversity was indexed by parental separation, low socioeconomic status, and familial psychopathology.
RESULTS:
Multilevel models indicated that childhood adversity promotes clustering of depression and inflammation. Among subjects exposed to high childhood adversity, the transition to depression was accompanied by increases in both CRP and IL-6. Higher CRP remained evident 6 months later, even after depressive symptoms had abated. These lingering effects were bidirectional, such that among subjects with childhood adversity, high IL-6 forecasted depression 6 months later, even after concurrent inflammation was considered. This coupling of depression and inflammation was not apparent in subjects without childhood adversity.
CONCLUSIONS:
These findings suggest that childhood adversity promotes the formation of a neuroimmune pipeline in which inflammatory signaling between the brain and periphery is amplified. Once established, this pipeline leads to a coupling of depression and inflammation, which may contribute to later affective difficulties and biomedical complications.

PMID: 16316783

Cytokines sing the blues: inflammation and the pathogenesis of depression. Increasing amounts of data suggest that inflammatory responses have an important role in the pathophysiology of depression. Depressed patients have been found to have higher levels of proinflammatory cytokines, acute phase proteins, chemokines and cellular adhesion molecules. In addition, therapeutic administration of the cytokine interferon-alpha leads to depression in up to 50% of patients. Moreover, proinflammatory cytokines have been found to interact with many of the pathophysiological domains that characterize depression, including neurotransmitter metabolism, neuroendocrine function, synaptic plasticity and behavior. Stress, which can precipitate depression, can also promote inflammatory responses through effects on sympathetic and parasympathetic nervous system pathways. Finally, depression might be a behavioral byproduct of early adaptive advantages conferred by genes that promote inflammation. These findings suggest that targeting proinflammatory cytokines and their signaling pathways might represent a novel strategy to treat depression.

PMID: 18391129

Elevated inflammation levels in depressed adults with a history of childhood maltreatment.

CONTEXT: The association between depression and inflammation is inconsistent across research samples. OBJECTIVE: To test whether a history of childhood maltreatment could identify a subgroup of depressed individuals with elevated inflammation levels, thus helping to explain previous inconsistencies. DESIGN: Prospective longitudinal cohort study. SETTING: New Zealand. PARTICIPANTS: A representative birth cohort of 1000 individuals was followed up to age 32 years as part of the Dunedin Multidisciplinary Health and Development Study.

Study members were assessed for history of childhood maltreatment and current depression. MAIN OUTCOME MEASURES: Inflammation was assessed using a clinically relevant categorical measure of high-sensitivity C-reactive protein (>3 mg/L) and a dimensional inflammation factor indexing the shared variance of continuous measures of high-sensitivity C-reactive protein, fibrinogen, and white blood cells. RESULTS: Although depression was associated with high levels of high-sensitivity C-reactive protein (relative risk,1.45; 95% confidence interval,1.06-1.99), this association was significantly attenuated and no longer significant when the effect of childhood maltreatment was taken into account. Individuals with current depression and a history of childhood maltreatment were more likely to have high levels of high-sensitivity C-reactive protein compared with control subjects (n = 27; relative risk, 2.07; 95% confidence interval, 1.23-3.47). In contrast, individuals with current depression only had a nonsignificant elevation in risk (n = 109; relative risk, 1.40; 95% confidence interval, 0.97-2.01). Results were generalizable to the inflammation factor. The elevated inflammation levels in individuals who were both depressed and maltreated were not explained by correlated risk factors such as depression recurrence, low socioeconomic status in childhood or adulthood, poor health, or smoking. CONCLUSIONS: A history of childhood maltreatment contributes to the co-occurrence of depression and inflammation. Information about experiences of childhood maltreatment may help to identify depressed individuals with elevated inflammation levels and, thus, at greater risk of cardiovascular disease.

PMID:12181102

Physical activity reduces the risk of subsequent depression for older adults. Previous studies assessing protective effects of physical activity on depression have had conflicting results; one recent study argued that excluding disabled subjects attenuated any observed effects. The authors' objective was to compare the effects of higher levels of physical activity on prevalent and incident depression with and without exclusion of disabled subjects. Participants were 1,947 community-dwelling adults from the Alameda County Study aged 50-94 years at baseline in 1994 with 5 years of follow-up. Depression was measured using criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (Washington, DC: American Psychiatric Association, 1994). Physical activity was measured with an eight-point scale; odds ratios are based upon a one-point increase on the scale. Even with adjustments for age, sex, ethnicity, financial strain, chronic conditions, disability, body mass index, alcohol consumption, smoking, and social relations, greater physical activity was protective for both prevalent depression (adjusted odds ratio (OR) = 0.90, 95% confidence interval (CI): 0.79, 1.01) and incident depression (adjusted OR = 0.83, 95% CI: 0.73, 0.96) over 5 years. Exclusion of disabled subjects did not attenuate the incidence results (adjusted OR = 0.79, 95% CI: 0.67, 0.92). Findings support the protective effects of physical activity on depression for older adults and argue against excluding disabled subjects from similar studies.

PMID:16575423

Antidepressant effects of exercise: evidence for an adult-neurogenesis hypothesis? It has been hypothesized that a decrease in the synthesis of new neurons in the adult hippocampus might be linked to major depressive disorder (MDD). This hypothesis arose after it was discovered that antidepressant medications increased the synthesis of new neurons in the brain, and it was noted that the therapeutic effects of antidepressants occurred over a time span that approximates the time taken for the new neurons to become functional. Like antidepressants, exercise also increases the synthesis of new neurons in the adult brain: a 2-3-fold increase in hippocampal neurogenesis has been observed in rats with regular access to a running wheel when they are compared with control animals. We hypothesized, based on the adult-neurogenesis hypothesis of MDD, that exercise should alleviate the symptoms of MDD and that potential mechanisms should exist to explain this therapeutic effect. Accordingly, we evaluated studies that suggest that exercise is an effective treatment for MDD, and we explored potential mechanisms that could link adult neurogenesis, exercise and MDD. We conclude that there is evidence to support the hypothesis that exercise alleviates MDD and that several mechanisms exist that could mediate this effect through adult neurogenesis.

PMID:18078985

The effects of physical activity counseling on mood among 75- to 81-year-old people: a randomized controlled trial. To examine the effects of physical activity counseling on mood among older people unselected for their depressive symptomatology. METHODS: Data are from "Screening and Counseling for Physical Activity and Mobility in Older People" project (SCAMOB), conducted in Finland during 2003-2005. SCAMOB was a 2-year single-blinded randomized controlled trial among 624 participants 75 years and older randomized into physical activity counseling group and control group. Depressive symptoms were assessed at baseline and after 24 months using Center for the Epidemiologic Studies Depression Scale. RESULTS: Among all the study participants, no effect of intervention was observed. However, among subgroup with minor depressive symptoms at baseline, a significant treatment effect was observed, where depressive symptoms decreased in the intervention group and increased in the control group. CONCLUSIONS: These findings suggest that physical activity counseling may reduce depression among those with minor depressive symptoms, which warrants for future studies.

PMID:18062765

Intervention study of exercise for depressive symptoms in women. Clinical depression affects millions of women annually. Exercise has been studied as a potential antidepressant, with most studies supporting its efficacy. Exercise also has the potential to reduce the risk for physical comorbidities that occur with depression. However, less is known about the types of exercise

programs to which women with depressive symptoms will adhere. Our objectives were to (1) compare two exercise programs, varying in their degree of structure, on improvements in physical activity and (2) compare the two exercise interventions on depressive symptoms, body composition, and fitness. METHODS: Women with depressive symptoms (physician diagnosed and confirmed with the Beck Depression Inventory) residing in the greater Boston area were recruited for this 3-month intervention study. Continuous enrollment took place between November 2005 and November 2006. Women were randomly assigned to either a clinic-based or home-based exercise intervention, with assessments at baseline and 3-months. RESULTS: Participants (n=32) were predominantly minority (81.4%) and, at baseline, had moderate symptoms of depression (Beck Depression Inventory [BDI], mean=25.6, SD=9.3), and were sedentary (mean=35.8 min/week of moderate and vigorous activity, SD=31.4). Gain scores for depressive symptoms (clinic-based mean=-11.7, home-based mean=-9.7) and physical activity (clinic-based mean=65.4, home-based mean=39.0) indicate strong improvements across time. Intent-to-treat analyses on 3-month data show that both interventions were associated with improvements in time spent in physical activity and depressive symptoms. Neither intervention impacted body composition or fitness. CONCLUSIONS: Both exercise programs were associated with reductions in depressive symptoms and increased physical activity participation, suggesting that even a home-based program can benefit women with depressive symptoms.

PMID:10547175

Effects of exercise training on older patients with major depression. Previous observational and interventional studies have suggested that regular physical exercise may be associated with reduced symptoms of depression. However, the extent to which exercise training may reduce depressive symptoms in older patients with major depressive disorder (MDD) has not been systematically evaluated. OBJECTIVE: To assess the effectiveness of an aerobic exercise program compared with standard medication (ie, antidepressants) for treatment of MDD in older patients, we conducted a 16-week randomized controlled trial. METHODS: One hundred fifty-six men and women with MDD (age, > or = 50 years) were assigned randomly to a program of aerobic exercise, antidepressants (sertraline hydrochloride), or combined exercise and medication. Subjects underwent comprehensive evaluations of depression, including the presence and severity of MDD using Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria and Hamilton Rating Scale for Depression (HAM-D) and Beck Depression Inventory (BDI) scores before and after treatment. Secondary outcome measures included aerobic capacity, life satisfaction, self-esteem, anxiety, and dysfunctional cognitions. RESULTS: After 16 weeks of treatment, the groups did not differ statistically on HAM-D or BDI scores (P = .67); adjustment for baseline levels of depression yielded an essentially identical result. Growth curve models revealed that all groups exhibited statistically and clinically significant reductions on HAM-D and BDI scores. However, patients receiving medication alone exhibited the fastest initial response; among patients receiving combination therapy, those with less severe depressive symptoms initially showed a more rapid response than those with initially more severe depressive symptoms. CONCLUSIONS: An exercise training program may be considered an alternative to antidepressants for treatment of depression in older persons. Although antidepressants may facilitate a more rapid initial therapeutic response than exercise,

after 16 weeks of treatment exercise was equally effective in reducing depression among patients with MDD.

PMID: 21148807

Exercise and pharmacotherapy in patients with major depression: one-year follow-up of the SMILE study. *OBJECTIVE:* To examine a 1-year follow-up of a 4-month, controlled clinical trial of exercise and antidepressant medication in patients with major depressive disorder (MDD). *METHODS:* In the original study, 202 sedentary adults with MDD were randomized to: a) supervised exercise; b) home-based exercise; c) sertraline; or d) placebo pill. We examined two outcomes measured at 1-year follow-up (i.e., 16 months post randomization): 1) continuous Hamilton Depression Rating Scale score; and 2) MDD status (depressed; partial remission; full remission) in 172 available participants (85% of the original cohort). Regression analyses were performed to examine the effects of treatment group assignment, as well as follow-up antidepressant medication use and self-reported exercise (Godin Leisure-Time Exercise Questionnaire), on the two outcomes. *RESULTS:* In the original study, patients receiving exercise achieved similar benefits compared with those receiving sertraline. At the time of the 1-year follow-up, rates of MDD remission increased from 46% at post treatment to 66% for participants available for follow-up. Neither initial treatment group assignment nor antidepressant medication use during the follow-up period were significant predictors of MDD remission at 1 year. However, regular exercise during the follow-up period predicted both Hamilton Depression Rating Scale scores and MDD diagnosis at 1 year. This relationship was curvilinear, with the association concentrated between 0 minute and 180 minutes of weekly exercise. *CONCLUSION*: The effects of aerobic exercise on MDD remission seem to be similar to sertraline after 4 months of treatment; exercise during the follow-up period seems to extend the short-term benefits of exercise and may augment the benefits of antidepressant use.

PMID: 21282661

Exercise training increases size of hippocampus and improves memory.

The hippocampus shrinks in late adulthood, leading to impaired memory and increased risk for dementia. Hippocampal and medial temporal lobe volumes are larger in higher-fit adults, and physical activity training increases hippocampal perfusion, but the extent to which aerobic exercise training can modify hippocampal volume in late adulthood remains unknown. Here we show, in a randomized controlled trial with 120 older adults, that aerobic exercise training increases the size of the anterior hippocampus, leading to improvements in spatial memory. Exercise training increased hippocampal volume by 2%, effectively reversing age-related loss in volume by 1 to 2 y. We also demonstrate that increased hippocampal volume is associated with greater serum levels of BDNF, a mediator of neurogenesis in the dentate gyrus. Hippocampal volume declined in the control group, but higher preintervention fitness partially attenuated the decline, suggesting that fitness protects against volume loss. Caudate nucleus and thalamus volumes were unaffected by the intervention. These theoretically important findings indicate that aerobic exercise training is effective at reversing hippocampal volume loss in late adulthood, which is accompanied by improved memory function.