

23rd March



پاکستان
قریب ارادہ

Pakistan Day

Karachi Psychiatric Hospital

BULLETIN

(Psychiatric Research Articles)

CHIEF EDITOR:
DR. SYED MUBIN AKHTER

MARCH
and APRIL

2018

MONTHLY PSYCHIATRIST MEETING

5th March 2018



*At
Karachi Psychiatric Hospital*

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A Free Smartphone App Reduces Symptoms of Post-Traumatic Stress Disorder

Joel Yager, MD reviewing Kuhn E et al. J Consult Clin Psychol 2017 Mar.

In a randomized, controlled trial, significant improvements seen at 3 months of treatment persisted at 6 months.

Although smartphone apps are widely used in mental health practice, few studies have meaningfully evaluated their utility. In this first-ever rigorous evaluation of a mobile app for symptoms of post-traumatic stress disorder (PTSD), investigators used advertisements to recruit adults meeting screening criteria for PTSD.

The 120 participants (mean age, 39; women, 69%; white race, 67%) were randomized to 3 months of treatment with PTSD Coach, a free app for Android or Apple devices developed by Department of Defense and Veterans Affairs, or to a waiting list. Not intended to replace professional care, the app offers psychoeducation, symptom assessment checklists, evidence-based self-management tools (e.g., relationship exercises, stress inoculation training, and grounding), and a “find support” section that includes personalized information and emergency services.

Attrition was low (overall, 14%). Participants used the app an average of 1 to 2 days per week. At the end of treatment, clinically significant improvement was seen in 47% of app users vs. 26% of waiting-list controls. App users showed greater improvement in PTSD symptoms, depressive symptoms, and psychosocial functioning (but not coping self-efficacy). Improvements generally persisted 3 months later.

COMMENT

The generalizability of this study is limited by its complete reliance on self-report measures in a convenience sample. Also, using digital devices for mental health treatment might evoke some degree of placebo response. Nevertheless, the potential utility of the app, as an adjunct to conventional PTSD treatment, bears further study. In the meantime, clinicians in routine practice might encourage patients to download and try the app (<http://www.ptsd.va.gov/public/materials/apps/PTSDCoach.asp>).

Unloved in Childhood: 10 Common Effects on Your Adult Self

By Peg Streep

When a child's emotional needs aren't met in childhood, her development and personality are shaped in specific ways. While it's true that everyone's childhood experience is different—one daughter may have an emotionally absent and dismissive mother who pays no attention to her, another might have a thoroughly enmeshed one who also ignores her needs but for different reasons, while a third daughter might be seen as only an extension of a mother high in narcissistic traits—there are nonetheless broad and reliable statements which can be made about the effect of these experiences. They are invaluable to understanding how your childhood shaped your personality and behaviors.

In the years before and since I wrote *Mean Mothers*, I've had the opportunity to hear from literally hundreds of women who have shared their stories. They reveal common themes, on the one hand, and unique, individual variations, on the other. As an unloved daughter myself, these stories amplify and expand the discussions offered by psychological research.

Here, in no particular order, are the most common—and the most lasting—effects these childhood experiences have on daughters. Their influence lasts long into adulthood, sometimes even into the sixth or seventh decade of life, unless they are addressed through therapy and self-knowledge.

1. Insecure attachment

A loving and attuned mother raises a child who feels understood and supported; she learns that relationships are stable and caring, that the world is a place of opportunity to be explored, that people take care of you. She has a secure base.

The child of an emotionally unreliable mother—sometimes there and sometimes not—understands that relationships are fraught and precarious, and that nothing is guaranteed. She grows up anxiously attached, hungry for connection but always waiting for the other shoe to drop.

The child with a mother who's withheld or combative learns to armor herself, to be as self-reliant as she can be; she is avoidant in her attachment style. While the securely attached daughter seeks out intimacy, her avoidant counterpart wants no part of it; the anxiously attached daughter seeks it out but can never find her footing since she's terrified of rejection.

These patterns of attachments arc into adulthood and affect friendships and romantic liaisons alike.

2. Undeveloped emotional intelligence

A child learns what she's feeling through dyadic interaction; a mother's gestures and words teach the baby to self-soothe when she's stressed or uncomfortable. Later, the mother will play a key role in helping her daughter articulate her feelings, name them, and learn to manage her fears and negative emotions.

The insecurely attached daughter doesn't learn to regulate her emotions; she's either engulfed by them or walled off from them. Both insecure styles of attachments get in the way of naming emotions and using them to inform thought—key aspects of emotional intelligence.

3. Impaired sense of self

A mother's face is the first mirror in which a daughter catches a glimpse of herself. The attuned and loving mother's face reflects acceptance, communicating, "You are you and you are just fine as you are." The unloving mother's face reflects supposed flaws and inadequacies; if the daughter is shunned or ignored, she absorbs the lesson that she's not worth dealing with or, if she's constantly criticized, she thinks she'll never be good enough.

Few unloved daughters see themselves with any clarity at all, especially if they've been scapegoated in the family.

4. Lack of trust

To trust others, you must believe that the world is essentially a safe place and the people in it well-intentioned, if sometimes imperfect. With an emotionally unreliable mother or one who is combative or hypercritical, the daughter learns that relationships are unstable and dangerous, and that trust is ephemeral and can't be relied on. Unloved daughters have trouble trusting in all relationships but especially friendship.

5. Difficulties with boundaries

The attuned mother teaches her baby that there's healthy space and breathing room even in close relationships; she doesn't intrude into her baby's space, forcing her to interact when she's not ready. Her behavior reflects the understanding that there's an area of overlap but that each person in the dyad is whole unto herself.

The avoidant daughter sees any overlap as too close and intrusive; she prefers to interact on more superficial levels so that her independence is never threatened. This tends to be a response to either a mother's intrusiveness or unreliability. The anxious daughter doesn't understand healthy space and mistakes a friend's or partner's need for boundaries as rejecting. She wrongly believes that being subsumed is a synonym for love.

6. Choosing toxic friends and partners

We all seek out the familiar (see the shared root with the word *family*?) which is just dandy if you have a secure base, and definitely less than optimal if you're an unloved daughter. The chances are good that, initially at least, you'll be attracted to those who treat you as your mother did—a familiar comfort zone that offers no comfort. Until you begin to recognize the ways in which you were wounded in childhood, the chances are good that you'll continue to recreate the emotional atmosphere you grew up with in your adult relationships.

7. Dominated by fear of failure

No one likes to fail, of course, but a securely attached daughter is unlikely to see a setback or even a failure as defining her self-worth or as proof positive of some basic flaw in her character. She'll be bruised but she's more likely to understand her failure as a consequence of having set the bar high in the first place.

That's absolutely not true of the unloved daughter who will take any rejection or failure as a sign that her mother was right about her after all. She remains highly motivated to avoid failing at any cost, often to her own detriment; many unloved daughters are chronic under-achievers as a result.

8. Feelings of isolation

Because the culture stubbornly believes that all mothers are loving and that mothering is instinctual, the unloved daughter mistakenly believes she's the only child on the planet to find herself in this predicament. As a result, she feels isolated and afraid, and is likely to continue to self-isolate because of her deep shame. She's not likely to tell anyone. More than anything, she wants to belong to the tribe—those girls who hug their moms and laugh with them.

9. Extreme sensitivity

Fear of rejection often dominates the daughter's inner world because she's afraid of more proof and evidence that her mother is right and that she really is worthless and unlovable. Her sensitivity is only increased by the likelihood that her mother and others accuse her of being "too sensitive"—the most common "explanation" of verbal abuse offered up by abusers.

10. Conflicted

What I call the *core* conflict—the daughter's continuing hardwired need for her mother's love and support versus her growing recognition of how her mother has wounded her—can dominate a daughter's life well into adulthood. It feeds her confusion, insecurity, and inner turmoil.

The first step of the long path to healing is recognition.

Healthy Sex Life at Home May Boost Job Performance, Satisfaction

By Rick Nauert PhD

New research suggests that maintaining a healthy sex life at home boosts employees' job satisfaction and engagement at the office.

Oregon State University researchers believe their finding emphasizes the value of a strong work-life balance.

In the study of work and sex habits of married employees, investigators found that those who prioritized sex at home unknowingly gave themselves a next-day advantage at work.

Researchers discovered a "day after" effect, where individuals were more likely to immerse themselves in their tasks and enjoy their work lives.

"We make jokes about people having a 'spring in their step,' but it turns out this is actually a real thing and we should pay attention to it," said Keith Leavitt, Ph.D., an expert in organizational behavior and management.

"Maintaining a healthy relationship that includes a healthy sex life will help employees stay happy and engaged in their work, which benefits the employees and the organizations they work for."

The study also showed that bringing work-related stress home from the office negatively impinges on employees' sex lives.

In an era when smart phones are prevalent and after-hours responses to work emails are often expected, the findings highlight the importance of leaving work at the office, Leavitt said.

When work carries so far into an employee's personal life that they sacrifice things like sex, their engagement in work can decline.

The researchers' findings appear in the *Journal of Management*. Co-authors are Dr. Christopher Barnes and Trevor Watkins of the University of Washington and Dr. David Wagner of the University of Oregon.

Sexual intercourse triggers the release of dopamine, a neurotransmitter associated with the reward centers in the brain, as well as oxytocin, a neuropeptide associated with social bonding

and attachment. That makes sex a natural and relatively automatic mood elevator and the benefits extend well into the next day, Leavitt said.

To understand the impact of sex on work, the researchers followed 159 married employees over the course of two weeks, asking them to complete two brief surveys each day.

They found that employees who engaged in sex reported more positive moods the next day, and the elevated mood levels in the morning led to more sustained work engagement and job satisfaction throughout the workday.

The effect, which appears to linger for at least 24 hours, was equally strong for both men and women and was present even after researchers took into account marital satisfaction and sleep quality, which are two common predictors of daily mood.

“This is a reminder that sex has social, emotional, and physiological benefits, and it’s important to make it a priority,” Leavitt said. “Just make time for it.”

Twenty years ago, monitoring sleep or daily step counts or actively practicing mindful meditation might have seemed odd, he said, but now they are all things people practice as part of efforts to lead healthier, more productive lives. It may be time to rethink sex and its benefits as well, Leavitt said.

“Making a more intentional effort to maintain a healthy sex life should be considered an issue of human sustainability, and as a result, a potential career advantage,” he said.

U.S. employers probably won’t follow the lead of a town councilman in Sweden who recently proposed that local municipal employees be allowed to use an hour of their work week for sex. The councilman’s hope is to boost the town’s declining population as well as improve employee moods and productivity.

But employers here can steer their employee engagement efforts more broadly toward work-life balance policies that encourage workers to disconnect from the office, Leavitt said. The French recently enacted a law that bars after-hours email and gives employees a “right to disconnect.”

“Technology offers a temptation to stay plugged in, but it’s probably better to unplug if you can,” he said.

“And employers should encourage their employees to completely disengage from work after hours.”

Cooking and Mental Health

Neil Petersen

Cooking at home is the best of both worlds, according to new research: it's healthier and cheaper than eating out! The downside, of course, is that you have to cook.

The study asked 437 Seattle-area adults about their eating habits, including how often they ate out and how often they stayed in to cook their own meals. The data revealed that people who frequently ate home-cooked meals tended to score higher on an index of healthy eating, while those who ate out the most often tended to score lower.

Not only that – people who ate out frequently spent an average of \$330 on food every month, compared to only \$273 for their home-cooking counterparts. In other words, cooking at home seems to be both cheaper and healthier than eating out frequently. The researchers also found that eating more home-cooked meals was associated with variables like being married, being unemployed, and having children, but not with income or education levels.

Cooking for yourself may have some less tangible benefits, too. Besides easing the burden on your pocketbook, it could be good for your mental health. For example, a 2016 study found that teens with more cooking skills tend to have better mental health, less depression and stronger family ties.

Of course, this study didn't look at causation, so it's probably not as simple as saying that home-cooked meals can prevent depression! It's quite possible, for instance, that being less depressed makes it easier to learn to cook, or that having a more stable family life lowers teens' risk for depression and makes them more likely to acquire cooking skills. But there definitely does appear to be some sort of link between cooking skills and mental health.

That's why some researchers have suggested a role for cooking classes in the education system. A 2015 study that implemented a class where students learned how to cook new foods suggested that cooking classes in schools could teach a whole range of skills like teamwork, cultural awareness and social engagement.

Cooking classes have also been explored as a mental health intervention for adults. In one study, researchers from University of Northern Colorado ran a six-week cooking and nutrition class for 18 people with severe mental illnesses. A survey at the end of the study suggested that the class helped participants improve their diets and become more confident when both cooking and grocery shopping.

Ultimately, the big picture for all these studies is the same: if you're looking for a cheap, practical way of improving your life, putting in the time to regularly prepare your own food can't hurt. Never underestimate the power of a home-cooked meal!

Exercise and Depression in Youth

By Karen Dineen Wagner, MD, PhD

Child Adolescent Psychiatry, Depression

CHILD & ADOLESCENT PSYCHIATRY

Following the holidays and the start of the New Year, adults often make renewed efforts to exercise. Physical and mental health can be affected positively by an increase in physical activity. Exercise has shown some benefits of improvement in depression for adults, but is there a relationship between exercise and depression in children and adolescents? Some recent studies shed light on this issue.

Physical activity and depression

The relationship between physical activity and major depression in middle childhood was assessed in a prospective study. Six-year-old children ($N = 795$) in a community in Norway were followed up at age 8 and 10 years. Semistructured clinical interviews of parents and children were conducted to evaluate symptoms of major depression at each assessment. Physical activity was assessed using an accelerometer that was worn for 7 days during the assessment periods.

Higher levels of moderate to vigorous physical activity at age 6 predicted fewer major depressive symptoms 2 years later. Similarly, moderate to vigorous physical activity at age 8 predicted fewer major depressive symptoms 2 years later. There was no association between sedentary behavior and depression. On the basis of these findings, the investigators concluded that increasing moderate to vigorous physical activity in children may prevent future symptoms of depression.

The relationship between physical activity and depression was also recently studied in adolescents during the course of 11 years. Adolescents ($N = 1160$) with a mean age of 13 years completed questionnaires at 4 points, up to age 21. Depression was assessed by self-report using the Children's Depression Inventory. Physical activity was assessed by self-report using the Leisure Time Exercise Questionnaire in which adolescents assessed their activity over the past 7 days and recorded the number of episodes of mild, moderate, and strenuous activity.

It was found that physical activity decreased over time and symptoms of depression increased over time. Higher initial symptoms of depression were associated with greater decreases in physical activity over time. The authors suggest that treatment strategies target symptoms of depression at about age 13 to prevent a decrease in physical activity and an increase in depressive symptoms.

Role of exercise in the treatment of depression

Given the association between physical activity and depression in youth, would exercise add benefit to the treatment of depression in youth? In the largest controlled trial to date, Carter and colleagues evaluated preferred intensity exercise for depressed adolescents. Adolescents ($N = 87$) who were in treatment for depression were randomized to either 12 sessions of aerobic exercise at preferred intensity along with treatment as usual or treatment as usual only.

The exercise consisted of circuit training with 8 exercise stations. The duration of each session was 1 hour. Exercise was defined as preferred intensity because the participants chose the order of the different exercises, chose the intensity at which they exercised, chose when to rest, and were not obligated to exercise at higher levels or attend the sessions.

The primary outcome measure to assess depression was the Children's Depression Inventory. No significant difference was found between the preferred intensity exercise plus treatment as usual group and the treatment as usual only group at 6 weeks after the intervention. However, at the 6-month follow-up, the group that received the preferred intensity exercise had a significantly greater improvement in depressive symptoms than the group that received only treatment as usual. The investigators conclude that preferred intensity exercise in addition to treatment as usual may have benefit for adolescents receiving treatment for depression.

From an adolescent's perspective, what aspects of exercise are beneficial to improve depressive symptoms? Carter and colleagues interviewed 26 adolescents with depression who had participated in the preferred intensity exercise program. The valued aspects of the intervention were as follows:

- The importance of choice: being able to select exercise intensity and choice of whether to attend
- Shared experience: being with other adolescents who were experiencing similar problems and concerns
- A sense of achievement: doing something and feeling better about themselves
- Routine: having a consistent routine in their lives

→ Being distracted: being able to focus attention on exercise and distract themselves from other problems and concerns

→ Feeling healthier: as a result of participating in exercise

The perceived changes reported by the adolescents were improved sleep; increase in energy; improved motivation to engage with peers, family, and school activities; improved mood; improvement in self-efficacy; better social interactions; and a more positive attitude toward exercise.

In light of these findings, clinicians may want to add exercise to the treatment armamentarium for depressed youth.

Disclosures:

Dr. Wagner is Professor and Chair of the department of psychiatry and behavioral sciences at the University of Texas Medical Branch at Galveston. She is the Child and Adolescent Psychiatry columnist for Psychiatric Times.

Geriatric Psychiatry Research Update: March 2017

By Mark L. Fuerst

Geriatric Psychiatry, Alzheimer, Cognitive Disorders

The results of 3 new studies in geriatric psychiatry show that mentally stimulating activities may decrease the risk of mild cognitive impairment, targeted noninvasive brain stimulation may combat specific memory impairments, and sleep apnea may increase the risk of dementia in older men without the apolipoprotein E ε4 allele.[1-3] Scroll through the slides for the latest findings and take-home messages.

Study 1. Mentally Stimulating Activities May Decrease the Risk of Incident Mild Cognitive Impairment (MCI) in Elderly Persons: A prospective, population-based cohort study included 1929 cognitively normal persons (median age, 77 years at baseline). During a median follow-up of 4 years, playing games, engaging in craft activities, using a computer, and participating in social activities were associated with a decreased risk of incident MCI. For those who carried the apolipoprotein E ε4 allele, there were significant associations between decreased MCI risk and computer use or being socially active.[1]

Clinical Implications for Study 1: Encourage elderly patients to participate in simple, accessible activities that stimulate the brain.

Study 2. Targeted Repetitive Transcranial Electromagnetic Stimulation (rTMS) May Improve the Precision of Memory Recollection: The hippocampal posterior-medial (HPM) network has been implicated in the recollection of highly precise contextual and spatial information. Sixteen healthy adults underwent memory testing about 24 hours before and after HPM network-targeted high-frequency rTMS. The same intensity stimulation was delivered to a site outside the HPM network, which amounted to a sham intervention. Active stimulation led to “highly consistent” improvements in precision of memory recollection, while sham stimulation results were “at chance.”[2]

Clinical Implications for Study 2: Noninvasive brain stimulation can enhance reactivation of precise memory details that are often disrupted in those with brain injuries or dementia.

Study 3. Sleep Apnea May Increase the Risk of Dementia in Older Men Without the Apolipoprotein E (APOE) ε4 Allele: Some 7547 men without dementia or other active neurological conditions that affect cognition were interviewed at baseline for sleep apnea. The effect of self-reported sleep apnea on dementia risk depended on their *APOE* ε4 status. When the

allele was absent, there was a 66% higher risk of developing dementia; whereas there was no additional risk for participants with the *APOE ε4* allele.[3]

Clinical Implications for Study 3: Ask older patients about sleep apnea problems, given the potential association with future cognitive dysfunction.

Parkinson's Disease

Parkinson's disease is a brain disorder that leads to shaking, stiffness, and difficulty with walking, balance, and coordination.

Parkinson's symptoms usually begin gradually and get worse over time. As the disease progress, people may have difficulty walking and taking. They may also have mental and behavioral changes, sleep problems, depression, memory difficulties, and fatigue.

Both men and women can have Parkinson's disease. However, the disease affects about 50 percent more men than women.

One clear risk factor for Parkinson's is age. Although most people with Parkinson's first develop the disease at about age 60, about 5 to 10 percent of people with Parkinson's have early-onset[”] disease, which begins before the age of 50. Early-onset forms of Parkinson's are often, but not always, inherited, and some forms have been linked to specific gene mutations.

What Causes Parkinson's Disease?

Parkinson's disease occurs when nerve cells, or neurons, in an area of the brain that controls movement become impaired and/or die. Normally, these neurons produce an important brain chemical known as dopamine. When the neurons die or become impaired, they produce less dopamine, which causes the movement problems of Parkinson's. Scientists still do not know what causes cells that produce dopamine to die.

People with Parkinson's also lose the nerve endings that produce norepinephrine, the main chemical messenger of the sympathetic nervous system, which controls many automatic functions of the body, such as heart rate and blood pressure. The loss of norepinephrine might help explain some of the non-movement features of Parkinson's, such as fatigue, irregular blood pressure, decreased movement of blood through the digestive tract, and sudden drop in blood pressure when a person stands up from a sitting or laying-down position.

Many brain cells of people with Parkinson's contain Lewy bodies, unusual clumps of the protein alpha-synuclein. Scientists are trying to better understand the normal and abnormal functions of

alpha-synuclein and its relationship to genetic mutations that impact Parkinson's disease and Lewy body dementias.

Although some cases of Parkinson's appear to be hereditary, and a few can be traced to specific genetic mutations, in most cases the disease occurs randomly and does not seem to run in families. Many researchers now believe that Parkinson's disease results from a combination of genetic factors and environmental factors such as exposure to toxins.

Symptoms of Parkinson's Disease

Parkinson's disease has four main symptoms:

- Tremor (trembling) in hands, arms, legs, jaw, or head
- Stiffness of the limbs and trunk
- Slowness of movement
- Impaired balance and coordination, sometimes leading to falls

Other symptoms may include depression and other emotional changes; difficulty swallowing, chewing, and speaking; urinary problems or constipation; skin problems; and sleep disruptions.

Symptoms of Parkinson's and the rate of progression differ among individuals. Sometimes people dismiss early symptoms of Parkinson's as the effects of normal aging. In most cases, there are no medical tests to definitively detect the disease, so it can be difficult to diagnose accurately.

Early symptoms of Parkinson's disease are subtle and occur gradually. For example, affected people may feel mild tremors or have difficulty getting out of a chair. They may notice that they speak too softly, or that their handwriting is slow and looks cramped or small. Friends or family members may be the first to notice changes in someone with early Parkinson's. They may see that the person's face lack expression and animation, or that the person does not move an arm or leg normally.

People with Parkinson's often develop a parkinsonian gait that includes a tendency to lean forward, small quick steps as if hurrying forward, and reduced swinging of the arms. They also may have trouble initiating or continuing movement.

Symptoms often begin on one side of the body or even in one limb on one side of the body. As the disease progresses, it eventually affects both sides. However, the symptoms may still be more severe on one side than on the other.

Many people with Parkinson's note that prior to experiencing stiffness and tremor, they had sleep problems, constipation, decreased ability to smell, and restless legs.

Diagnosis of Parkinson's Disease

A number of disorders can cause symptoms similar to those of Parkinson's disease. People with Parkinson's-like symptoms that result from other causes are sometimes said to have parkinsonism. While these disorders initially may be misdiagnosed as Parkinson's, certain medical tests, as well as response to drug treatment, may help to distinguish them from Parkinson's. Since many other diseases have similar features but require different treatments, it is important to make an exact diagnosis as soon as possible.

There are currently no blood or laboratory tests to diagnose nongenetic cases of Parkinson's disease. Diagnosis is based on a person's medical history and a neurological examination. Improvement after initiating medication is another important hallmark of Parkinson's disease.

Treatment of Parkinson's Disease

Although there is no cure for Parkinson's disease, medicines, surgical treatment, and other therapies can often relieve some symptoms.

Medicines for Parkinson's Disease

Medicines prescribed for Parkinson's include:

- Drug that increase the level of dopamine in the brain
- Drug that affect other brain chemicals in the body
- Drugs that help control nonmotor symptoms

The main therapy for Parkinson's levodopa, also called L-dopa. Nerve cells use levodopa to make dopaminie to replenish the brain's dwindling supply. Usually, people take levodopa along with another medication called carbidopa. Carbidopa prevents or reduces some of the side effects of levodopa therapy-such as nausea, vomiting, low blood pressure, and restlessness-and reduces the amount of levodopa needed to improve symptoms.

People with Parkinson's should never stop taking levodopa without telling their doctor. Suddenly stopping the drug may have serious side effects, such as being unable to move or having difficulty breathing.

Other medicines used to treat Parkinson's symptoms include:

- Dopamine agonists to mimic the role of dopamine in the brain
- MAO-B inhibitors to slow down an enzyme that breaks down dopamine in the brain
- COMT inhibitors to help break down dopamine
- Amantadine, an old antiviral drug, to reduce involuntary movements
- Anticholinergic drugs to reduce tremors and muscle rigidity

Deep Brain Stimulation

For people with Parkinson's who do not respond well to medications, deep brain stimulation, or DBS, may be appropriate. DBS is a surgical procedure that surgically implants electrodes into part of the brain and connects them to a small electrical device implanted in the chest. The device and electrodes painlessly stimulate the brain in a way that helps stop many of the movement-related symptoms of Parkinson's, such as tremor, slowness of movement, and rigidity.

Other Therapies

Other therapies may be used to help with Parkinson's disease symptoms. They include physical, occupational, and speech therapies, which help with gait and voice disorders, tremors and rigidity, and decline in mental functions. Other supportive therapies include a healthy diet and exercises to strengthen muscles and improve balance.

For More Information About Parkinson's Disease

National Institute of Neurological Disorders and Stroke

1-800-352-9424 (toll-free)

braininfo@ninds.nih.gov

www.ninds.nih.gov

Michael J. Fox Foundation for Parkinson's Research

1-800-708-7644 (toll-free)

www.michaeljfox.org

Parkinson's Disease Foundation

1-800-457-6676 (toll-free)

info@pdf.org

www.pdf.org

Parkinson's Disease Information Page

What research is being done?

Current research programs funded by the National Institute of Neurological Disorders and Stroke are using animal models to study how the disease progresses and to develop new drug therapies. Scientists looking for the cause of PD continue to search for possible environmental factors, such as toxins, that may trigger the disorder, and study genetic factors to determine how defective genes play a role. Other scientists are working to develop new protective drugs that can delay, prevent, or reverse the disease.

More information about Parkinson's Disease research is available at:
Focus on Parkinson's Disease Research

Information from the National Library of Medicine's MedlinePlus

Parkinson's Disease

Definition

Parkinson's disease (PD) belongs to a group of conditions called motor system disorders, which are the result of the loss of dopamine-producing brain cells. The four primary symptoms of PD are tremor, or trembling in hands, arms, legs, jaw, and face; rigidity, or stiffness of the limbs and trunk; bradykinesia, or slowness of movement; and postural instability, or impaired balance and coordination. As these symptoms become more pronounced, patients may have difficulty walking, talking, or completing other simple tasks. PD usually affects people over the age of 60. Early symptoms of PD are subtle and occur gradually. In some people the disease progresses more quickly than in others. As the disease progresses, the shaking, or tremor, which affects the majority of people with PD may begin to interfere with daily activities. Other symptoms may include depression and other emotional changes; difficulty in swallowing, chewing, and speaking; urinary problems or constipation; skin problems; and sleep disruptions. There are currently no blood or laboratory tests that have been proven to help in diagnosing sporadic PD. Therefore the diagnosis is based on medical history and a neurological examination. The disease can be difficult to diagnose accurately. Doctors may sometimes request brain scans or laboratory tests in order to rule out other diseases.

Treatment

At present, there is no cure for PD, but a variety of medications provide dramatic relief from the symptoms. Usually, affected individuals are given levodopa combined with carbidopa. Carbidopa delays the conversion of levodopa into dopamine until it reaches the brain. Nerve cells can use levodopa to make dopamine and replenish the brain's dwindling supply. Although levodopa helps at least three-quarters of parkinsonian cases, not all symptoms respond equally to the drug. Bradykinesia and rigidity respond best, while tremor may be only marginally reduced.

Problems with balance and other symptoms may not be alleviated at all. Anticholinergics may help control tremor and rigidity. Other drugs, such as bromocriptine, pramipexole, and ropinirole, mimic the role of dopamine in the brain, causing the neurons to react as they would to dopamine. An antiviral drug, amantadine, also appears to reduce symptoms. In May 2006, the FDA approved rasagiline to be used along with levodopa for patients with advanced PD or as a single-drug treatment for early PD. In March 2017, the FDA approved safinamide tablets as an add-on treatment for individuals with PD who are currently taking levodopa/carbisopa and experiencing "off" episodes (when the person's medications are not working well, causing an increase in PD symptoms).

In some cases, surgery may be appropriate if the disease doesn't respond to drugs. A therapy called deep brain stimulation (DBS) has now been approved by the U.S. Food and Drug Administration. In DBS, electrodes are implanted into the brain and connected to a small electrical device called a pulse generator that can be externally programmed. DBS can reduce the need for levodopa and related drugs, which in turn decreases the involuntary movements called dyskinesias that are a common side effect of levodopa. It also helps to alleviate fluctuations of symptoms and to reduce tremors, slowness of movements, and gait problems. DBS requires careful programming of the stimulator device in order to work correctly.

Prognosis

PD is both chronic, meaning it persists over a long period of time, and progressive, meaning its symptoms grow worse over time. Although some people become severely disabled, others experience only minor motor disruptions. Tremor is the major symptom for some individuals, while for others tremor is only a minor complaint and other symptoms are more troublesome. It is currently not possible to predict which symptoms will affect an individual, and the intensity of the symptoms also varies from person to person.

A New Medication for Tardive Dyskinesia

Joel Yager, MD reviewing Hauser RA et al. Am J Psychiatry 2017 Mar 21.

Valbenazine, a vesicular monoamine transporter 2 inhibitor, had a favorable benefit vs. harm profile for reducing tardive dyskinesia.

Until very recently, no medication was FDA-approved for tardive dyskinesia, although several, including tetrabenazine (a vesicular monoamine transporter 2 [VMAT2] inhibitor approved for treating hyperkinetic symptoms in Huntington disease), have been used off label. In an industry-sponsored, multisite, phase-3, double-blind trial, researchers randomized 234 patients with tardive dyskinesia (mean age, 56; men, 54%) to 6 weeks of valbenazine (a newly FDA-approved VMAT2 inhibitor) at 40 mg, valbenazine at 80 mg, or placebo.

Patients had stable schizophrenia, schizoaffective disorder, or mood disorder; tardive dyskinesia had lasted at least 3 months. Exclusion criteria included medical instability and other prominent neurological disorders (e.g., parkinsonism, akathisia, or truncal dystonia). Stable medications for psychiatric and general medical conditions were continued. Almost 90% of patients completed the trial.

Based on assessment of patients' videos, valbenazine at 6 weeks produced significant improvement compared with placebo on the Abnormal Involuntary Movement Scale (AIMS) at both the 80-mg dose (mean AIMS reductions, 3.2 vs. 0.1 with placebo; number needed to treat for one to benefit [NNT], 4; number needed to harm [NNH], 13) and the 40-mg dose (mean AIMS reductions; 1.9 vs. 0.1; NNT, 7; NNH, -32 [the negative value indicates fewer adverse effects with valbenazine than placebo]). Psychiatric symptoms remained stable. Treatment-emergent adverse effects, occurring in $\leq 5\%$ of any group, were primarily somnolence, akathisia, and dry mouth.

COMMENT

The authors are awaiting results from a 42-week extension study, a 52-week study, and a ≤ 72 -week rollover study. Comparisons of valbenazine to tetrabenazine would also be of interest. Although newer antipsychotics have reduced the rate of tardive dyskinesias, cases still occur. New treatment options are likely in the offing for this troubling condition.

Which Drugs Work for Diabetic Peripheral Neuropathy Pain?

By Kelly Young

Edited by Susan Sadoughi, MD, and André Sofair, MD, MPH

Duloxetine, venlafaxine, pregabalin, oxcarbazepine, tricyclic antidepressants, atypical opioids (e.g., tapentadol), and botulinum toxin are all more effective than placebo in reducing pain associated with diabetic peripheral neuropathy, according to a *Neurology* review.

Researchers examined 50 new trials of various diabetic peripheral neuropathy medications, along with 50 studies that were part of a 2011 review.

Ineffective treatments for pain included dextromethorphan, gabapentin, typical opioids (e.g., oxycodone), topical capsaicin, and mexiletine. The evidence wasn't strong enough to make recommendations on treatments' effects on quality of life.

Most of the trials lasted less than 3 months, so the review couldn't address long-term harms. The authors write: "This is particularly important for atypical opioids, which we found were effective in short-term studies, as new guidelines ... now recommend against the use of opioids for chronic pain conditions given lack of evidence for long-term benefit and increasing evidence of serious risks."

The Secret Life of Bipolar Disorder

BY Chris Aiken, MD

Bipolar Disorder, Cultural Psychiatry, Psychotherapy

TABLE. “The Rule of 3” hinting at soft bipolarity in a clinically depressed individual⁷

- > 3 Major depressive episodes
- 3 Failed marriages
- 3 Failed antidepressants
- 3 First-degree relatives with affective illness
- 3-Generation family history of affective illness
- Eminence in 3 fields in the family
- 3 Simultaneous jobs
- Proficiency in 3 languages (for US-born citizens)
- 3 Distinct professions (exercised simultaneously)
- 3 Comorbid anxiety diagnoses
- Triad of past histrionic, psychopathic, or borderline diagnoses
- Triad of trait “mood lability,” “energy activity,” and “daydreaming”
- Flamboyance expressed in a triad of bright colors
- 3 Substances of abuse
- 3 Impulse control behaviors
- Simultaneous dating of 3 individuals

TABLE. “The Rule of 3” hinting at soft bipolarity in a clinically depressed individual

Hollywood depictions of bipolar disorder often feel stilted, with characters that seem molded from the *DSM* criteria. Claire Danes changed all that in her gritty portrayal of an FBI agent with bipolar disorder in the Showtime series *Homeland*. Her source material? YouTube: “There was a lot of footage of people who recorded themselves when they were in manic states. I think they were probably up in the middle of the night and lonely and, you know, needed to talk.”

The Internet can enrich our understanding of bipolar disorder as well. In his column last month, Dr. Phelps drew from the vivid texts of bloggers with hypomania. The Internet is also the source

of a remarkable new study that recruited no fewer than 71,247 people to complete a lengthy online survey. About a quarter of them had a mood disorder; all were Brazilian. In addition to the usual psychiatric rating scales, the survey asked a host of personal questions that offer a rare glimpse into the bipolar life.

People with bipolar disorder are active—in mind, body, and spirit. Compared with controls and those with unipolar depression, they change religions, hairstyles, and sexual partners more often. They read more books. They curse more and have more “provoked” car accidents. Their clothing is more extravagant and, even in this age of body art, they are more likely to get tattoos and piercings than those with unipolar depression and controls.

There’s a special knack for starting relationships that often goes along with bipolar disorder. This can be an asset in an otherwise rocky life. In previous research, we learned that bipolar patients have broader social networks than do those with other diagnoses. In the Brazilian data, they had multiple marriages (≥ 3) and multiple sexual partners (≥ 60) more often than those with unipolar depression and controls. *Pathologic love* was also more common among those with bipolar disorder, particularly among the women.

Pathologic love is not a symptom I could find in the *DSM*, but from the paper’s description it looks similar to a chief complaint I often encounter in practice: “Have you ever been so in love with or obsessed about someone that nothing else mattered to you, you felt you could not live without this person, felt bad when away from this person, and tried to monitor his/her activities?” Prior to this research, we knew surprisingly little about the sexual lives of bipolar patients. Although couples with bipolar disorder rank sexuality as their most important marital concern, a 2016 review concluded that “the overwhelming majority of articles that look at couples in which one partner is bipolar exclude the topic of sexuality.”

These new data help fill in that gap, though that wasn’t the primary aim. The authors actually set out to test a controversial hypothesis put forth by Hagop Akiskal: that behavioral markers can help distinguish bipolar and unipolar depression. In 2005, Akiskal derived the “rule of 3’s” by comparing these markers in 1000 outpatients with bipolar II disorder and unipolar depression. His list is also revealing (**Table**), and it’s significant that this unorthodox perspective can now claim validation across 2 cultures.

As colorful as these data are, it’s important to keep in mind that only a minority of bipolar patients endorsed these soft signs (5% to 30%). The greater world is also full of people with artistic gifts, exuberant lives, and sailor’s tongues who *don’t* have bipolar disorder. Soft signs of bipolarity are only useful when they occur alongside a history of cyclical depressions. In that context they can prompt us to gather more history, interview the relatives, and use antidepressants with caution (if at all). Should you encounter these signs outside of the office setting, you can safely put any screening questions aside and move on to what’s likely to be a lively conversation with an interesting person.

Disclosures:

Dr. Aiken is the Director of the Mood Treatment Center and an Instructor in Clinical Psychiatry at the Wake Forest University School of Medicine. He does not accept honoraria from pharmaceutical companies but receives honoraria from W.W. Norton & Co. for *Bipolar, Not So Much*, which he coauthored with James Phelps, MD.

Anxiety Therapy Better for Depression Than CBT?

By Nancy A. Melville
WebMD Health News

Metacognitive therapy (MCT), a psychotherapeutic approach that targets persistent rumination and negative thought processes, has a large effect size when used to treat depression and may offer a viable alternative to mainstay cognitive-behavioral therapy (CBT), results from a new randomized control trial show.

"The results of this trial of MCT for depression are encouraging with large and statistically significant reductions both for depressive and anxious symptoms following 10 sessions of treatment. These improvements were sustained at 6 months follow-up," the authors, led by Roger Hagen, PhD, Norwegian University of Science and Technology, Trondheim, Norway, write.

The study was published online January 24 in *Frontiers in Psychiatry*. Adrian Wells, PhD, is the senior author of the study.

Anxiety Treatment

With the most common approaches to the treatment of depression, such as CBT or antidepressants, recovery rates are often low and relapse rates are high, so MCT is being eyed as a potential alternative.

MCT was originally developed in the 1990s for the treatment of generalized anxiety disorder. It focuses on shifting maladaptive rumination, or "overthinking," which can occur with psychiatric disorders.

As opposed to analyzing and challenging thoughts, as is practiced with CBT, MCT focuses on reducing the thought process that drives persistent ruminating thoughts, said Dr Hagen.

"In CBT, we focus on thought content, with [the clinician] and the patient working together to examine the validity of the content, asking, for instance, 'What is really the evidence that you are stupid?'"

"With MCT, we try to instead focus on reducing rumination and changing maladaptive metacognitions, such as, 'I have to analyze why I became depressed,' or 'I need to find out what is wrong with me in order to snap out of my depression,'" he told *Medscape Medical News*.

Edge Over CBT?

Although numerous studies have shown the benefits of MCT for patients with anxiety, only a few, including a study published in 2009, have looked at the approach in depression.

The lead author of that study, Jennifer Jordan, PhD, senior research fellow and clinical psychologist with the Department of Psychological Medicine at the University of Otago, Christchurch, New Zealand, told *Medscape Medical News* that the 2009 findings suggest "MCT may have an advantage over CBT, given that there is increasing recognition that neuropsychological impairments seen in the acute state of depression often don't remit with mood recovery with standard treatment, as was previously assumed."

For the current study, investigators randomly assigned 39 patients with depression to receive 10 sessions of MCT, either immediately or after a 10-week waiting period.

The sessions focused on increasing meta-awareness by identifying thoughts that triggered rumination and worry; challenging beliefs about uncontrollability of rumination and dangers of rumination; and modifying positive beliefs about rumination and worry.

The results, assessed using the Beck Depression Inventory, showed that those who received MCT had significant improvements in symptoms of depression and anxiety compared to the wait-list group (Cohen's $d = 2.51$ and Cohen's $d = 1.92$, respectively).

The analyses of clinical significance showed that 70% to 80% of the total sample of patients achieved recovery, as measured by the Hamilton Rating Scale for Depression-17. This result, the researchers note, is consistent with results from previous uncontrolled studies of MCT.

A core focus of the MCT approach is what the therapy's developers call the cognitive attentional syndrome (CAS), consisting of persistent rumination, threat monitoring, and ineffective coping. With depression, a key component can include repeated analysis of negative feelings and past failures, the authors note.

"Depression is understood as an extension of low mood resulting from a problem of overthinking (eg, worry and rumination) and withdrawal of active coping. (eg, social withdrawal and reduction in activity)," they write.

Specific strategies of MCT to tackle symptoms of CAS include verbal reattribution and behavioral experiments, such as an attention training technique, detached mindfulness, and postponement of rumination.

"The CAS is driven by metacognitions, both positive: 'To ruminate about my problems can help me find a solution,' and negative: 'My rumination is uncontrollable,'" Dr Hagen said.

"In therapy, we try to help the patient to see that rumination is not uncontrollable and that there are no benefits in using rumination to self-regulate."

Delivered by Experts

Another recent study that compared MCT with CBT showed interesting differences in some cognitive functions, but not mood. For that study, 48 patients with depression were randomly assigned to receive 12 weeks of treatment with either MCT or CBT.

After the 12 weeks, those in the MCT group demonstrated significantly greater improvements in executive functioning, including spatial working memory and attention tasks, compared to the CBT group.

Importantly, an earlier randomized study conducted by Dr Jordan and her colleagues showed no difference between CBT and MCT in terms of depression scores.

"It was noteworthy that MCT delivered by our team was no less effective than CBT, given the extent of training that we had in MCT was far less than our training and experience with CBT, which is the core training model in clinical psychology training in New Zealand," said Dr Jordan.

She added, however, that an important limitation of some MCT research, including the current study, is that the treatment was administered by experts who developed the therapy or who are working directly with the developers.

"The effect sizes in the studies published by the core MCT researchers with Adrian Wells are incredibly large," Dr Jordan said.

"This speaks to the promise of MCT when administered by those with a high degree of training and supervision by the originator(s).

"However, there is an allegiance literature that suggests that it can be difficult to achieve similar levels of effectiveness as the therapies are disseminated. Thus, there is a need for further independent research."

One Size Doesn't Fit All

Philip Muskin, MD, professor of clinical psychiatry at Columbia University Center for Psychoanalytic Training and Research, in New York City, agreed that the findings are encouraging but that larger studies are needed.

"It's a small study with a robust finding, and it's an interesting approach because it goes beyond the process of thinking with CBT," he told *Medscape Medical News*.

"MCT appears to be unique in delving more into how one's mind is working – asking not, 'Where did this come from in your childhood?' but, 'How is your mind working to process it and how is that affecting you?'"

He noted that the approach of addressing rumination in depression is important.

"We know that people who are depressed do ruminate about negative things. Patients will tell you that at night they have racing thoughts, which makes you think they may be bipolar, but they're not – they're just lying in bed and going over the whole day and all of the things they think they did poorly."

Because some patients experience such symptoms more than others, the proposal of an alternative to CBT for patients who do not respond to that approach could be highly useful, Dr Muskin added.

"The reality is there are patients who either don't want to take antidepressant medications or don't respond well to certain psychotherapies, so we want as many treatments as we can.

"Everyone is different, and you want to have choices of a treatment that is best – one size doesn't fit all."

The authors, Dr Jordan, and Dr Muskin have disclosed no relevant financial relationships.

Potential Benefits of Testosterone Therapy in Older Men

Thomas L. Schwenk, MD reviewing Roy CN et al. JAMA Intern Med 2017 Feb 21.

In randomized trials, hemoglobin levels rose in anemic men, and bone density increased. In three studies, researchers evaluated effects of testosterone therapy in older men. Two of these were from the Testosterone Trials (T Trials), a series of seven linked studies, in which 788 men (mean age, 72) with total testosterone levels <275 ng/dL were randomized to testosterone gel (to maintain testosterone levels of 300–800 ng/dL), or placebo for 1 year. Sexual and physical function outcomes were published earlier (NEJM JW Gen Med Mar 15 2016 and N Engl J Med 2016; 374:611).

In one T Trial, 16% of men had anemia (hemoglobin level, ≤ 12.7 g/dL); anemia was unexplained in half of these men. Increases in hemoglobin levels of at least 1.0 g/dL occurred in significantly more testosterone recipients than placebo recipients (53% vs. 17%); this effect occurred in men with and without known causes of anemia. The authors imply that low serum testosterone actually might be responsible for some cases of unexplained anemia.

In another T Trial, bone outcomes were assessed in 211 men at baseline and after treatment. Testosterone recipients had significantly greater increases than placebo recipients in several measures of bone density and strength, but the study was too small and too short in duration to assess fracture risk.

Finally, in a retrospective U.S. cohort study, researchers examined adverse cardiovascular events in $\approx 44,000$ men with total testosterone levels <300 ng/dL; about 9000 men received testosterone therapy (which increased median testosterone level from 212 ng/dL to 318 ng/dL), and 35,000 did not. During median follow-up of 3.4 years, adverse cardiovascular events occurred less often in testosterone recipients than in nonrecipients (17 vs. 24 events/1000 person-years; adjusted hazard ratio, 0.67).

COMMENT

These results add a few new pieces to the puzzle of whether testosterone therapy benefits hypogonadal older men. However, the two randomized trials were too small and too short for researchers to assess clinical outcomes or harmful side effects. And the cohort study — with results that differ from those of several other observational studies — was subject to confounding by unmeasured variables. In the absence of results from larger, longer trials, detailed shared decision making still is strongly recommended.

2017 Diabetes Care Standards Issued

By Amy Orciari Herman

Edited by Jaye Elizabeth Hefner, MD

The American Diabetes Association has released its 2017 "Standards of Medical Care in Diabetes." An overview of the group's guidance on pharmacologic therapy for type 2 diabetes is available in the *Annals of Internal Medicine*.

Among the recommendations:

- Metformin is still the preferred first-line regimen and should be prescribed for most patients at the time of diagnosis, provided it's not contraindicated. Starting at 500 mg once or twice daily may help minimize gastrointestinal side effects, followed by gradual titration up to 2 g/daily.
- If metformin is contraindicated or poorly tolerated, clinicians should use a patient-centered approach to choose from the other available agents. Tables detailing all FDA-approved diabetes medications, including their costs, are provided.
- Dual therapy should be considered for asymptomatic patients with hemoglobin A1c levels of 9% or greater.
- Insulin may be advisable for symptomatic patients or those with an HbA1c of 10% or greater or blood glucose of 300 mg/dL or greater.

Coffee, Tea, and the Heart

Joel M. Gore, MD reviewing Miller PE et al. Am J Med 2017 Feb .

Tea may be heart-healthy, and coffee may have a neutral effect, according to a large observational study.

A majority of Americans passionately consume coffee and tea. Using data from the population-based Multi-Ethnic Study of Atherosclerosis (MESA; N=6814; mean age, 62; 53% women), investigators prospectively studied the impact of coffee, tea, and caffeine on cardiovascular health.

MESA participants completed food-frequency questionnaires with items on coffee and tea (black or green); coffee caffeination was not queried. Daily caffeine intake from all beverages and foods was calculated. Of the participants, 25% reported not drinking coffee, 24% reported <1 cup daily, and 51% reported ≥ 1 cups daily. For tea, 58% reported no tea drinking; 29% reported <1 cup daily, and 13% reported ≥ 1 cup daily.

At follow-up (median, 11.1 years), the incidence of all cardiovascular events and heart cardiovascular events was 10.8 and 7.5 per 1,000 person-years. Consuming ≥ 1 cup of tea daily was associated with a lower incidence of future cardiovascular events compared with no tea intake. Compared with no coffee intake, consuming <1 cup daily was associated with a higher incidence of such events; regular drinking had a neutral effect. In multivariate analyses, coffee intake was not associated with coronary artery calcium progression. In contrast, drinking ≥ 1 cup of tea daily or having higher caffeine consumption was linked to reduced progression of coronary artery calcium.

COMMENT

In this healthy population, regular tea drinking was associated with slower progression of coronary artery calcium and a lower rate of cardiovascular events. Habitual coffee intake was not associated with coronary artery calcium progression and appeared to have a neutral effect on cardiovascular endpoints. These observational findings require further validation and confirmation in well-controlled trials. In the meantime, the results will reassure the multitude of Americans who consume caffeine daily. What individuals put in their beverages was not reported: holding the cream and sugar seems prudent.

Sudden unexpected death in epilepsy: measures to reduce risk

Mclean B and Colleagues

Abstract

This review looks at the strategies that may help to reduce the risk of sudden unexpected death in epilepsy beyond that of trying to achieve seizure cessation, which is not possible for up to 30% of patients with epilepsy. These strategies include seizure safety checklists, mobile phone technology, telehealth and various devices currently available or in development. We highlight interventions where there is evidence of benefit, and draw attention for the need both to involve patients with epilepsy in risk reduction and to improve communication with those at risk.

Probiotic in Yogurt May Improve Depressive Symptoms

Nancy A. Melville

Lactobacillus, a probiotic bacteria found in live-culture yogurt, appears to reverse symptoms of depression in mice, new research shows. In addition, investigators have discovered a specific mechanism suggesting a direct link between the health of the gut microbiome and mental health.

In a recognized preclinical model of depression, investigators examined the gut microbiome of mice before and after they were exposed to chronic stress. The major change they found was a loss of *Lactobacillus* and an increase in circulating levels of kynureneine metabolites, which are known to drive depression. With the loss of *Lactobacillus* came the onset of depressive symptoms. But after supplementation with *L. reuteri* to restore *Lactobacillus*, kynureneine metabolism normalized, and so did the animals' behavior.

"A single strain of *Lactobacillus* is able to influence mood," lead investigator Alban Gaultier, PhD, of the University of Virginia School of Medicine, Charlottesville, said in a release.

"This is the most consistent change we've seen across different experiments and different settings we call microbiome profiles. This is a consistent change. We see *Lactobacillus* levels correlate directly with the behavior of these mice," said study researcher Ioana Marin, a PhD student.

The study was published online March 7 in *Scientific Reports*.

Gateway to New Depression Treatments?

The authors hypothesized that *Lactobacillus* suppresses kynurenine and keeps levels of the depression-driving metabolite in check. When *Lactobacillus* is depleted, levels of kynurenine increase.

To test this theory, the researchers conducted an experiment to elevate kynurenine levels in mice while administering *Lactobacillus*. They found that improvement in depressive symptoms were diminished in this setting.

"Mechanistically, we identified that *Lactobacillus*-derived reactive oxygen species may suppress host kynurenine metabolism by inhibiting the expression of the metabolizing enzyme IDO1 in the intestine. Moreover, maintaining elevated kynurenine levels during *Lactobacillus* supplementation diminished the treatment benefits. Collectively, our data provide a mechanistic scenario for how a microbiota player (*Lactobacillus*) may contribute to regulating metabolism and resilience during stress," the investigators write.

The investigators believe the findings should hold true in people.

"Some of the same strains of *Lactobacillus* used in the study are present in humans as well as mice. In addition, kynurenine metabolism imbalances have been shown to be associated with depression in humans," Dr Gaultier told *Medscape Medical News*.

Dr Gaultier said the discovery may open the door for new treatments for depression as well as other disorders, such as anxiety.

"The big hope for this kind of research is that we won't need to bother with complex drugs and side effects when we can just play with the microbiome. It would be magical just to change your diet, to change the bacteria you take, and fix your health — and your mood," he said.

Although Dr Gaultier knew of no other studies suggesting that symptoms of stress or depression could be improved with *Lactobacillus*, studies in humans have suggested benefits with other probiotics.

Paradigm Shift in Neuroscience

As reported by *Medscape Medical News*, a small pilot study of healthy men suggested benefits with a probiotic strain of *Bifidobacterium longum*. In preclinical studies, *B longum* was shown to be a "putative psychobiotic" that yielded benefits in stress-related behaviors in mice.

In this placebo-controlled clinical study, men received the *B longum* probiotic daily for 4 weeks. They then received a matching placebo capsule for another 4 weeks.

The results showed reductions in the stress hormone cortisol and a blunted increase in subjective anxiety in response to acute stress.

At the time, senior author Gerard Clarke, PhD, of the APC Microbiome Institute at University College Cork, in Ireland, said that the concept that the gut microbiome is a key regulator in the brain and in behavior represented a "paradigm shift in neuroscience."

In commenting on the current study, Dr Clarke, who was not involved in the research, noted the findings build on the evidence with valuable insights on mechanisms behind the gut-brain relationship.

"This field urgently requires such mechanistic insights that can help expedite the translation of these and associated findings," he told *Medscape Medical News*.

The role of the kynurenine pathway in the relationship is especially notable, he said.

"In many ways, the neurobiological features of depression, such as stress and inflammation, create the perfect storm for increased kynurenine pathway metabolism," said Dr Clarke.

"Although earlier studies implicated the gut microbiome as an important regulator of this metabolic cascade, we weren't sure which particular members of the consortium of bacteria in the gut were most important for these host-microbe interactions."

"This study is important, as it demonstrates that *Lactobacillus* might be critical for restraining excessive production of kynurenine, and they also show that supplementation with *L reuteri* can help apply the brakes to this runaway metabolic train under pathological conditions."

The broader possible implication of dietary probiotic supplementation with *Lactobacillus* obtained through live-culture yogurt as a means of improving depression is feasible, Dr Clarke added, noting the findings from his study on *B longum*.

A Note of Caution

"A note of caution is advisable, as another candidate psychobiotic we tested (*L rhamnosus* [JB-1]) with quite a strong preclinical signal did not translate well," Dr Clarke said.

"Interestingly, this was also a *Lactobacillus* strain but with a mechanism of action based on communication via the vagus nerve rather than regulation of kynurenine production," Dr Clarke said.

Dr Clarke noted that many aspects of microbiome-gut-brain axis are intriguing, but more research on humans is needed.

"We still need more clinical studies to confirm that the important preclinical observations, like the one made here, will translate to humans," he said.

"The field also needs more mechanistically oriented studies, and that is one of the reasons the study reported here is an important addition to the literature."

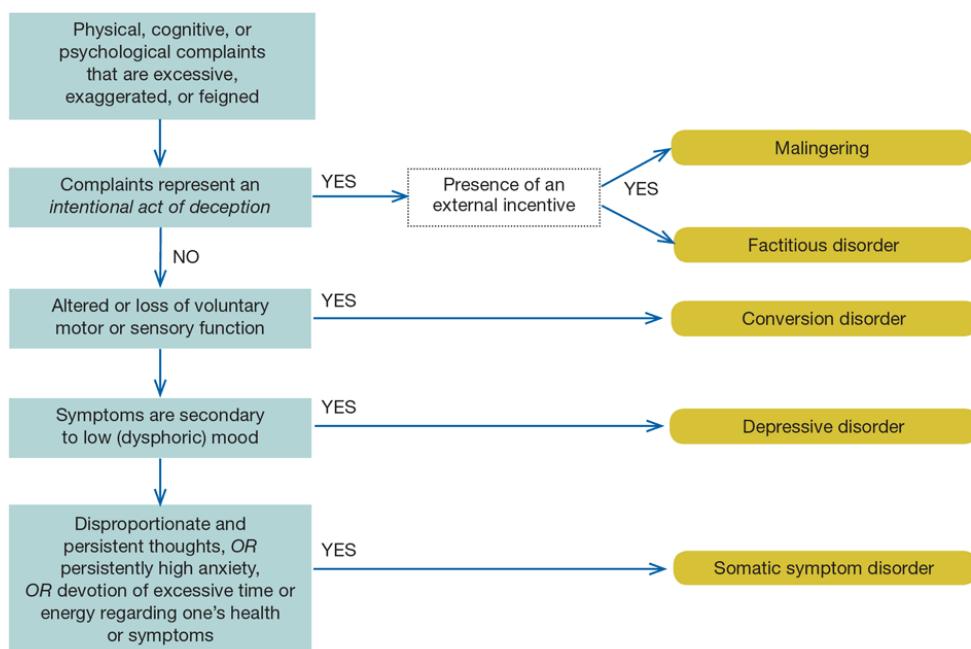
The study received funding from the National Multiple Sclerosis Society and the National Institute of Mental Health. Dr Clarke is currently funded by the Irish Health Research Board, the Health Service Executive, and the Air Force Office of Scientific Research. He is also on the editorial board of Scientific Reports.

Challenges in Assessing and Managing Malingering, Factitious Disorder, and Related Somatic Disorders

By Phillip K. Martin, PhD and Ryan W. Schroeder, PsyD

Special Reports, Forensic Psychiatry, Munchausen Syndrome, Somatoform Disorder

Figure. Differential diagnosis of malingering, factitious disorder, and selected related disorders



Malingering, factitious disorder, and related somatic disorders present with unique diagnostic and treatment challenges. Reporting of symptoms that are excessive, nonexistent, or exaggerated beyond available medical evidence is a central feature of each condition, and this can make the clinical differentiation of these disorders a daunting task. Treatment is similarly difficult because, by the very nature of these conditions, a patient's self-report cannot be relied upon *prima facie* and traditional treatment approaches often do not address the underlying impetus for the reported symptoms. Management of such patients is an unwelcome undertaking for many mental health providers, and many non-psychiatric physicians prefer to avoid it altogether. Psychiatry and other mental health services, therefore, may provide a unique role by recognizing and addressing these conditions in their own patients and by providing useful consultation to providers of other specialties in instances of noncredible symptom report.

Malingering

Criteria. As documented in DSM-5, malingering is not a mental disorder but is, instead, a condition that may be a focus of clinical attention. While listed under a general heading of "Nonadherence to Medical Treatment," malingering is not simply nonadherence. Rather, malingering is defined as an intentional production of grossly exaggerated or feigned symptoms motivated by an external incentive, such as obtaining financial compensation or evading criminal prosecution. Thus, while malingering should be considered whenever the veracity of a patient's self-report is called into question, a dubious symptom report, in and of itself, is not sufficient to diagnose malingering. Similarly, attempts to obstruct or derail evaluation or treatment due to poor participation, nonadherence, or vague or inconsistent reporting are not enough to determine the presence of malingering. To determine that a patient is malingering, the following conditions must be met:

- Symptoms are feigned or grossly exaggerated
- Excessive symptom production must be intentional
- The symptom production is motivated by an external incentive (eg, avoiding work or military duty or criminal prosecution, or obtaining financial compensation or drugs)

DSM-5 supportive indicators. Both DSM-IV-TR and DSM-5 provide 4 conditions under which malingering "should be strongly suspected." These include medicolegal context, discrepancy between self-report and medical findings, poor patient cooperation, and antisocial personality disorder. While these conditions are included to potentially aid clinicians in flagging cases in which malingering should be considered, it is important to be aware that these supportive features are neither necessary nor sufficient to determine malingering.

Some argue that the previously listed indicators—particularly antisocial personality disorder and uncooperativeness during an evaluation—should be ignored because they do not adequately distinguish malingers from nonmalingers. For example, many malingers are not uncooperative; indeed, they may appear very cooperative and compliant if they believe that such

behavior will help to manipulate their providers into believing their symptoms.³ Thus, these proposed indicators should not be viewed as diagnostic criteria or central features of malingering.

Is it really malingering? Caution is recommended when you are unsure whether a determination of malingering is actually appropriate. It is not uncommon for patients with depression, anxiety, or chronic pain to report symptoms or to demonstrate signs that exceed those expected for their medical or psychiatric conditions. In some patients, such displays are unintentional and may reflect a transfer of psychological symptoms to physical symptoms, a heightened preoccupation and concern with physical or psychological symptoms, or an increased perception of symptom intensity relative to other patients with similar afflictions.

Beyond keeping in mind that some displays of symptom magnification may be unintentional or not motivated by external incentives (and, therefore, not malingering), remember that a diagnosis of malingering can have serious negative consequences for patients. Malingering is not just a clinical term used by physicians; it is also a forensic term used by attorneys and it can have legal implications. As such, some forensic clinicians have indicated that the term malingering be reserved for cases where the evidence for the diagnosis is incontrovertible. In cases where it is unclear whether a patient is malingering, it may be more appropriate to describe the patient's behavior with terms such as unreliability (presentation of inaccurate information), nondisclosure (withholding of information), deception (attempts to distort or misrepresent information), or atypical (presentation of unusual information).

Factitious disorder

Similar to malingering, a diagnosis of factitious disorder also requires conscious and intentional falsification of physical or psychological symptoms. Thus, both etiologies should be considered in any case where a volitional attempt to deceive medical providers via exaggeration or feigning of symptoms is suspected. Despite these similarities, the 2 conditions differ in regards to patients' motivation to deceive. Malingering requires that deception be motivated by an external incentive. A diagnosis of factitious disorder requires that the deception occur even in the absence of an external incentive. This suggests that individuals with factitious disorder are motivated by an internal incentive, where deceptive behaviors might serve the purpose of gaining nurturance, attention, or sympathy from family, friends, or medical providers.

While the main tenets of factitious disorder remain fairly similar across DSM-IV-TR and DSM-5, a prior criterion that required that the motivation for deceptive behavior be "to assume the sick role" is now absent from DSM-5. This change is likely a reflection of the challenges in determining the presence or absence of specific internal incentives. Importantly, clinicians may now make the diagnosis without needing to make inferences regarding a patient's internal motivation to deceive (eg, assuming a sick role) so long as an external incentive is not apparent and malingering has been excluded as a cause of the deception.

Factitious disorder imposed on another

Factitious disorder imposed on another (formerly factitious disorder by proxy) occurs when one volitionally falsifies the psychological or physical signs or symptoms of another person in the absence of an external incentive. In some instances this may take the form of an individual falsely reporting or exaggerating another's symptoms to receive sympathy or attention. In more deleterious instances, individuals may actually induce physical or psychological harm or injury to another.

For example, in Munchausen syndrome by proxy, a parent might surreptitiously cause medical issues in a child (such as poisoning the child to the point of sickness) and then repeatedly take the child to a pediatrician for evaluation of the symptoms to gain professional attention and personal nurturance from the issue.

Certainly ethical and legal issues can arise due to this type of behavior, and it is essential that clinicians be aware of relevant state laws and institutional policies, as both may vary by location. When the victim is a child, mandatory reporting laws are likely applicable and efforts should be made to protect the child from further harm.

Differentiating malingering and factitious disorder from related somatic disorders

A number of substantive changes to the diagnostic labels and criteria for somatoform disorders appear in DSM-5. These disorders are now referred to as somatic symptom and related disorders. This DSM diagnostic category includes factitious disorder as well as conditions such as somatic symptom disorder, illness anxiety disorder, and conversion disorder (functional neurological symptom disorder). The latter disorders can be difficult to clinically differentiate from malingering and factitious disorder because patients with these disorders also report symptoms that are in excess of, inconsistent with, or incompatible with known manifestations of true medical illness.

For example, patients with somatic symptom disorder—the condition that most closely resembles the condition previously referred to as somatization disorder—may express concern, report disruption of daily life, or seek out medical intervention for their somatic symptoms to an extent that is excessive given the actual severity of any true medical condition. However, such patients differ from those with malingering or factitious disorder in that they do not intentionally exaggerate or falsify their symptoms for the purpose of an external or internal incentive (Figure). Rather, patients with somatic symptom disorder truly believe that their symptoms are real, are genuinely distressed by their purported symptoms, and often lack insight into the psychological processes underlying their symptoms.

Similarly, a diagnosis of conversion disorder is appropriate when patients present with a clearly neurologically incompatible loss or alteration in motor or sensory function that cannot be attributed to an intentional act of deception. DSM-5 does not require clinicians to determine that symptoms are unintentionally produced to diagnose conversion disorder. This seems to imply that when discriminating between conversion disorder, malingering, and factitious disorder, a greater degree of confirmatory evidence is required for malingering/factitious disorder, and that in the absence of such evidence (ie, evidence of feigning), conversion disorder is likely a more appropriate diagnosis in cases of medically unexplained neurological symptoms.

Standardized assessment of excessive illness behavior

In many cases, psychiatric providers can determine that symptoms are excessive, exaggerated, or feigned based on clinical history and examination findings. When feigning or exaggeration of symptoms is suspected but not confirmed or in cases where differential diagnosis of these conditions is still questionable, psychiatric providers may consider referring patients to a clinical neuropsychologist or psychologist for additional workup. Clinical neuropsychologists assess cognitive functioning to detect true cognitive changes. At the same time they often employ standardized and well- validated tests that are sensitive to patient attempts to exaggerate or feign cognitive impairment.

Common validity tests administered by neuropsychologists include Test of Memory Malingering, Word Memory Test, Medical Symptom Validity Test, and Rey 15 Item Test. These tests were designed to appear challenging to an examinee but, in actuality, are easily performed even by individuals with rather severe cognitive impairment. Similarly, both neuropsychologists and clinical psychologists commonly utilize emotional and personality measures such as the Minnesota Multiphasic Personality Inventory – 2nd edition and the Personality Assessment Inventory to identify invalid reporting of both psychological and somatic symptoms. These measures often include validity scales that directly assess for honest and accurate responding as well as clinical scales that assess for underlying personality characteristics that might be linked to or directly causing the exaggerated symptoms. Such standardized assessment provides an objective approach for helping to determine the veracity and nature of a patient's reported symptoms.

Treatment and management

A major hurdle in diagnosing and treating patients with disorders characterized by medical and psychiatric deception is that key distinguishing features of the disorders—those relating to intent and motivation—are not readily observed in most clinical settings.⁵ Determining whether a patient's deception is motivated by external versus internal factors can be difficult even in the presence of secondary gain, as external incentives (eg, financial gain, avoidance of work) may not always be the primary operant or may work alongside internal motivators (eg, sympathy from a spouse or co-workers). Even when psychological or neuropsychological testing

unequivocally documents that a patient is presenting excessive or exaggerated symptoms, testing might not provide full insight into the motivation behind the documented exaggeration. Thus, testing should be viewed as an empirical method to determine whether a patient's complaints are valid (ie, are the complaints accurately reported and not exaggerated?), but testing should not be viewed as a means to solely or specifically diagnose malingering.

Because differential diagnosis of malingering, factitious disorder, and related somatic disorders is often difficult even when there is documentation of symptom exaggeration, it is recommended that clinicians try to extend beyond categorical thinking about the conditions and instead try to understand the function of the deceptive behavior (eg, avoiding work to avoid stress caused by a difficult co-worker) when treating and managing individuals with such presentations.⁵ This approach may allow for a bridge to treatment in patients whose deception is rooted in poor coping or potentially remediable psychological problems.

Patients may find discussions regarding stress and coping strategies to be more palatable than confrontations about their deception or assertions that "it's all in your head." In cases where illness deception is potentially affected by stress, depression, or anxiety, both psychotherapeutic and pharmacological interventions may be warranted and helpful. Documentation of both true and falsified symptoms can be beneficial in justifying clinicians' diagnosis and treatment, and in providing information to other providers who work with the deceptive patient.

When a patient's excessive report or falsification of symptoms is likely to result in overuse of medical services (eg, over-prescribing of medications, repeated surgeries), it is often helpful for mental health clinicians to recommend that other providers adopt relatively conservative treatment approaches to minimize iatrogenic effects and unnecessary health care expenditures. When you suspect malingering, true symptoms may sometimes exist. If falsified symptoms can be disentangled from non-falsified symptoms, treatment of true symptoms may be possible in some cases.

Disclosures:

Dr Martin is a Neuropsychology Post-Doctoral Fellow and Dr Schroeder is Assistant Professor and Board-Certified Clinical Neuropsychologist in the department of psychiatry and behavioral sciences at the University of Kansas School of Medicine in Wichita, KS. The authors report no conflicts of interest concerning the subject matter of this article.

Hot Topics of 2016: In and Around Psychiatry

By Allan Tasman, MD

It's been a really interesting year for psychiatry, to say the least. Because of that we decided to poll our editorial board and some of our most widely read contributors and their colleagues to get a very unscientific survey of this year's most meaningful issues. I have a few of my own that I'll save till the end, but here are some of our contributors' responses.

Genomics

Drs. Ronald Pies, Barbara Schildkrot, and Paul Summergrad all nominated the genomics and proteomics research that has given rise to incredible advances in our understanding of major psychiatric disorders like schizophrenia, bipolar disorder, and major depression. One study, cited by Ron Pies, identified genes that seem to commonly affect all 3 of these disorders and among other things relate to control of brain cell communication and immune responsiveness. Another report, mentioned by both Schildkrot and Summergrad, focuses on the role of the C4A gene in the development of schizophrenia and discusses the implication of the genetic findings and potential approaches this suggested disease mechanism has for treatment.

One compelling aspect of these examples is the fact that the results come from exceptionally large groups of subjects. I recently heard a talk by one of the researchers describing a study with 40,000 patients. He noted that the state of the art level of expectation of significance in current genomic research now is 5×10^{-8} . That's an incredible change from commonly used research standards of 10^{-2} .

Psychotomimetic agents for treatment

Drs. James Knoll, Steve Koh, and Michael First all mentioned work being done with ketamine for rapid-onset antidepressant effect, and its possible use in quickly ameliorating suicidal ideation. Ketamine, which got its very negative reputation in the 1960s, seems to work by an as-yet unknown mechanism. This research is important not only in the study of ketamine itself, but because, as James Knoll wrote, it offers an opening into studying other psychotomimetic agents, which have gone unexplored in recent decades.

Neuromodulation, imaging, biomarkers

Dr. Helen Lavretsky's vote goes to the rapidly expanding field of neuromodulation research, and she refers us to 2 important books on the subject. The field offers a promise of mood and cognitive benefits for treatment of a variety of disorders. I can personally vouch for the importance of further work in this area, since in my own lab, we have used both RTMS and neurofeedback training in several studies that found benefits in the treatment of ADHD or autism.

Other important areas of neuroscience research were mentioned by Dr. Jerald Kay. One study he notes from *Nature*, relates to the Human Connectome Project. Several hundred healthy young adults were studied, and 97 previously unidentified new specific areas in the cortex were found. He also highlights a study in which the relationship between early life stress and amygdala hyper-reactivity is investigated. While the lasting biological effects of early life stress have been known for decades, this study predicts with more than 80% accuracy the likely response to antidepressants in this population. It emphasizes the rapidly intensifying area of research aimed at identifying biomarkers associated with either specific diseases or treatment interventions. The study also suggests the potential utility of combining biological markers with other historical and/or psychological information to predict treatment response.

Opiate addiction

Several contributors voted for the long overdue attention to the epidemic of opiate addiction. I had written about this in an editorial earlier this year. In that column, I said that I was glad this issue was finally being addressed but chastised both the government and large medical organizations like the AMA that ignored the problem, about which psychiatrists had been long aware, which had spiraled out of control years ago. Dr. Thomas Kosten cites an article by Nora Volkow and colleagues that offers convincing evidence to a broad medical readership that standard prescribing practices for opiates are not tenable. They also highlight both prescribing practice changes and policy changes, including much better education starting at the medical school level and research aimed at developing new potent, but nonaddictive pain medications and nonpharmacological pain-treatment strategies. Nearly all her recommendations are embodied in the FDA and CDC guidelines for opiate prescribing.

Physician-assisted suicide

Several of our contributors mentioned social issues or phenomena. Dr. Cynthia Geppert notes the rising trend for states in the US to pass laws that legalize physician-assisted suicide. She cites the important Pies article published in *Psychiatric Times* about a new Canadian law with frightening prospects for catastrophic outcomes. Unlike other laws in the US or abroad, the Canadian law codifies physician-assisted suicide not only for a life-limiting illness but also for what the bill calls “intractable” mental illnesses—to include not only dementia but also disorders such as depression or PTSD.

The most, but not only, appalling component of the law is that it applies to minors as well as the elderly or others with terminal physical illnesses. Dr. Geppert writes, “ . . . ask yourself if you believe we can define what intractable means in psychiatry? Do we really know when a patient’s case is futile? Are depressed adolescents truly capable of a rational decision to die?” Of even greater concern, she notes that this law was changed without long study, rigorous research, or important debate, which usually precede major changes in mental health public policy.

Violence

Dr. Renato Alarcon focuses our attention on the global epidemic of violence and its psychological impact. He writes,

The August 16 photograph of Omram Daqueesh, the 5-year-old boy from Aleppo, Syria, looking dusty, bleeding copiously, stunned and weary, unable to understand the why and how of deadly airstrikes over his hometown, was the most dramatic representation of a human crisis of universal proportions. As a reflection of cruelty, fanaticism, neglect, and opportunism, this development showed the global impact of violence as an emotional and behavioral trait almost gone out of control. Violence and its many faces, its presence in multiple forms (war, crime, abuse, exploitation, homelessness, murder) practically all over the world (and the US is not certainly an exception), has become in 2016 the most formidable challenge to American and world psychiatry's discourse, role, and actions as a professional, academic, and scientific endeavor. . . . psychiatry has witnessed this year a seemingly unstoppable, world-wide dehumanizing process. Studying its nature, educating society and fighting against the emotional wounds of violence to correct its erosive course, must gain prominence as the main objectives of our discipline now and in the future.

Parity and politics

In the government action arena, I was very pleased to see the October announcement of the release of the final report from the Obama administration's Federal Parity Task Force. The report outlines action steps to strengthen insurance coverage for mental health and substance use disorders. The comprehensive and lengthy set of action items and proposed regulatory changes are encouraging and long overdue. My enthusiasm is tempered by the November elections and the likelihood that the incoming administration will not take the needed actions to implement the recommendations nor support ongoing funding.

And, speaking of the election, I feel compelled to say a few words. Dr. Steve Moffic wrote that he thought the important issue for us about the presidential election is that we should re-evaluate the strictures placed on members of the American Psychiatric Association by the "Goldwater Rule." This APA policy was put into place following the 1964 presidential election when a number of psychiatrists publically stated their belief that Barry Goldwater had a mental illness. The rule prohibits saying such a thing about a person who we have not personally evaluated nor without that person's permission. Personally, I think it's a good rule, although I agree that there may well be value in revisiting it. I wrote earlier in the campaign about my views of the very low value of untrained people wildly throwing around diagnoses, and you can read it again if you wish.

To me, the important take-home message of this campaign has less to do with evaluating the fitness of the candidates, but more what the election's outcome tells us yet again about the role of empathy in politics. Whichever side you were on in the election, what's clear is that decisions

were made by many people not on their thoughts about the candidates. Nor did voters choose based on the strength of intellectual arguments. Their decisions in most cases, I believe, were based on their feelings about themselves and their own lives. The frustration, fear, and rage we saw, which led to an outpouring of votes for Donald Trump, was not invented by either Donald Trump or Bernie Sanders; it was already there. And like an empathic listening psychotherapist, the candidates who empathized with those feelings in the electorate, whether actual or feigned empathy, formed an alliance that led to lots and lots of votes and an electoral college victory (although at the time that I wrote this, Hillary Clinton was leading the popular vote).

Good psychotherapy does not primarily produce change through intellectual discourse but through engaging the emotional valance for certain cognitions in our patients—in my book, this is what won the election. Being a CNN and MSNBC addict (though I haven’t had much trouble going through withdrawal), I rarely heard any reference to those affects from the Democrats. The Republicans, at least since Nixon, have been masters at this, as is, for better or worse, the new President-elect.

There are lots of reasons anyone wins an election, including this one, but I think the politico-therapeutic alliance is a major factor that rarely gets much attention until the election postmortems. And then, the lessons are often forgotten. No one has to be well versed in Maslow’s hierarchy of needs pyramid to know that economic security, which supplies food and shelter, and safety/security for self and family are the most basic and primal. Bill Clinton, he of “I feel your pain,” and James Carville, he of “It’s the economy, stupid” (his exhortation to the campaign staff), and just last month, Donald Trump, are textbook examples. No one paying attention to this issue should have been surprised that Trump won, which in my view was due in large part to his bonding with voters around their affects stemming from economic insecurity (no job, no food or shelter, which leads to fear and insecurity about safety, which then often leads to rage).

One very important difference between therapy and politics, however, is that psychotherapists are well aware of the power of affects and their potentially harmful impact on the individual’s thinking and behavior. Because of this, we do our best to be exquisitely sensitive about modulating our interactions with our patients so that the individual is not overwhelmed and therefore maladaptive responses are kept in check. When political candidates for national office (this year we saw this at both the presidential and congressional level) not only empathize with strong dysphoric affects, but also mirror those intense affects in their own words and affects, what can be easily understood as fostering loss of usual constraints on maladaptive actions, it can unleash extremely worrisome consequences. And, to our great dismay, we have seen, both during the election campaign and its aftermath, the alarming rise in reported incidents of hateful speech and actions, often but not only by adolescents and young adults, directed against women and members of minority ethnic and racial groups.

And just a bit more

I can't end this list of important issues from 2016 talking about politics, so just let me mention 2 other topics from the research world that made my own list. From *Science* magazine last February comes a report highlighting the role that maternal immune activation (as in Zika but other infections too) influences fetal brain development. This has been known for a long time, but has not been much of a subject for psychiatric research outside of decades-old explorations of a link between schizophrenia (or more recently autism) and maternal infection. Understanding the pathophysiology of this effect has tremendous implications for understanding the potential impact of prenatal maternal immune activation on many psychiatric illnesses.

Finally, I have to highlight the explosion of interest in the gut microbiome and its role in both physical and psychiatric disorders such as depression. While this field of research is really in its infancy, it has been known for ages that there are intimate connections between the gut and the brain. Now we are wondering what role our gut bacteria play in these processes. It is really like beginning to explore a whole new galaxy, and I'll bet there is a great deal that pertains to mental functioning and psychiatric disorders awaiting discovery.

Well, it's been an exhausting year, and if you're still reading, this column has probably taxed your attention a bit, too. I hope everyone enjoys the holiday season, has time for at least a short break, and has a safe, healthy, productive and, more importantly, peaceful year.

Postpartum Depression Screening During Well-baby Care Benefits Moms

(http://www.medscape.com/viewarticle/885358?scr=wnl_edit_tp&uac=268994AZ)

By Will Boggs MD
September 08, 2017

Reuters

NEW YORK (Reuters Health) - Postpartum depression screening during well-child care can improve early detection of depression and maternal outcomes, researchers from the Netherlands report.

"This study not only strengthens current evidence that screening is an effective way to reduce depressive symptoms, but also demonstrates that screening can improve secondary outcomes, like general maternal functioning and parenting aspects, in this case, maternal self-efficacy," Dr. Angarath I. van der Zee-van den Berg from University of Twente, Enschede, the Netherlands, told Reuters Health by email.

About 7% of women develop major depression within 3 months after childbirth, when many are no longer followed by their obstetricians. Screening with the Edinburgh Postnatal Depression Scale (EPDS) can improve early detection, but only if it can be feasibly administered.

Dr. van der Zee-van den Berg's team investigated whether repeated EPDS screening for Postpartum Depression Screening in the Well-child care (WCC) setting, followed by routine care for screen-positive mothers, improves maternal and child outcomes, compared with usual care in the same setting.

The intervention group included 1,843 mothers, and the usual-care groups included 1,246 mothers, according to the September 7 Pediatrics online report.

Significantly fewer mothers in the intervention group were depressed at 9 months Postpartum (0.6% with major depression, 3.0% with major or minor depression), compared with the usual-care group (2.5% with major depression, 8.4% with major or minor depression), reflecting a 72% lower adjusted risk of major depression and a 60% lower adjusted risk of major or minor depression.

The intervention group had significantly better scores than the usual-care group for parenting, anxiety symptoms, and mental health functioning.

The groups did not differ in physical functioning, and the intervention's effect on the socioemotional development of the children (as measured by the Ages and Stages Questionnaire-Social Emotional) was negligible.

Among mothers who could recall having experienced a depressive period in the 9 months since giving birth, 60% of both the intervention and the usual-care groups reported having consulted their family practitioners about their depression or having received further treatment (38% and 37%, respectively).

"Implementing screening is a simple and effective way to reduce their burden of Postpartum depression provided that diagnosis and treatment can be offered (with or without referral)," Dr. van der Zee-van den Berg's said. "We would also like to emphasize the supporting and normalizing role of WCC, which can prevent the need for further treatment, especially in mild cases."

"In setting where WCC is less widely available, screening preferably should be implemented by other healthcare professionals who see mothers frequently in the first year Postpartum," she said. "Professionals performing the screening need to feel competent in discussing depressive symptoms and guiding mothers to further treatment. Adequate training is required and should underline the importance of paying attention to the interaction between mother and child."

"Future studies should further clarify if screening can improve child outcomes as well," Dr. van der Zee-van den Berg's added. "Other instruments and long-term outcomes at different stages of age may be needed to demonstrate the benefits. The effects are likely to increase when incorporating specific attention for the interaction of depressed mothers and their children in the trajectory after screening."

Dr. Jennifer Doering from University of Wisconsin-Milwaukee College of Nursing, who has investigated various aspects of perinatal and postpartum depression, told Reuters Health by email, "We have a significant amount of evidence suggesting that providers cannot accurately tell a woman is depressed simply by interacting with her during a healthcare encounter.

Evidence-based screening tools like the EPDS significantly improve the ability of providers to detect women who can benefit from further evaluation for diagnosis and treatment."

This study adds to this body of literature and uses a study design that is stronger than past studies that have found weaker effects of screening," she said.

"Many health providers believe that any screening is better than no screening," Dr. Doering said. "In health care setting where WCC is less widely available, even screening one affords providers

an opportunity to detect clinically significant depression symptoms before symptoms become chronic.”

While this study found little effect of depression screening on child outcomes, studies that show the most adverse outcomes associated with postpartum depression occur in cases where depression symptoms are chronic and go undetected and untreated,” she added. Apart from a 6-week visit or a repeat pregnancy, women may not regularly interface with an obstetrical provider where perinatal depression screening may primarily take place.”

“Screening within well-child care is an opportunity to slow or stop the trajectory towards chronic depression and optimize mother and child outcomes,” Dr. Doering concluded.

Source: <http://bit.ly/2xdWeMt>
 Pediatrics 2017.

Prevention of Alzheimer’s Disease: Lessons Learned and Applied

[\(http://www.medscape.com/viewarticle/884615\)](http://www.medscape.com/viewarticle/884615)

**Galvin JE
 J Amer Geriatr Soc. 2017 Aug 2.**

Summary

Alzheimer disease (AD) prevalence in the United States currently exceeds 5 million, and the Alzheimer Association estimates a US prevalence of 16 million by the year 2050 unless an effective treatment is developed. Increasing age is still the leading risk factor for AD, with a prevalence of 24% by age 82 years. Other nonmodifiable risk factors include female sex, positive family history, and presence of the apolipoprotein E ε4 allele.

At present, only four FDA-approved medications are available for memory and behavioral AD symptoms, Disease-modifying treatment trials have been unsuccessful, although several new trials are underway and research continues. Prevention strategies are therefore essential and are now facilitated by advances in diagnostic criteria, biomarker development, and greater understanding of the biophysiological underpinnings of AD.

Risk factor prevention should target diabetes mellitus and insulin resistance, obesity, metabolic syndrome, hypertension, hypercholesterolemia, cerebrovascular disease, depression,

psychological and physiologic stress, traumatic brain injury, sleep-disordered breathing, smoking, alcohol abuse, high blood pressure, renal disease, alcohol and tobacco use, high cholesterol, coronary heart disease, sedentary life style, and diet. These potentially modifiable risk factors, when combined, account for more than 50% of AD risk, based on observational studies, and many of these risk factors do not appear to affect amyloid or tau proteins. In sporadic and genetic forms of AD, pharmacologic trials of antiamyloid therapies are ongoing.

Modifiable factors appearing to protect against AD include cognitive reserve and mental activity, educational attainment and lifelong learning, cognitive leisure activities, physical activity and exercise, social engagement, mindfulness and wellness activities, optimism and purpose in life, healthy diet, and omega-3 intake. A review of 19 studies suggests that certain brain-stimulating activities may help reduce AD risk. These include crossword puzzles, card games, computer use, arts or crafts, taking classes, group discussion, and listening to music.

Depending on the type of exercise and its intensity, physical activity may lower AD risk by up to 65%. Underlying mechanisms may include reduction in blood vessel disease, better pulmonary function, increased cell survival, and anti-inflammatory effects.

Up to 30% of AD may be preventable by well-balanced, healthy lifestyle choices, including regular exercise, social engagement, and a healthy diet including recommended servings of fresh fruits and vegetables, whole grains, and lean proteins, and avoiding processed foods.

Viewpoint

Although clear explanations are lacking regarding why some people and not others develop AD as they age, targeting known risk factors may help prevent or forestall the disease. Healthy lifestyle modification including increased physical activity, healthy diet, social engagement, and mentally challenging activities may be effective.

Future research into AD prevention should concentrate on persons at increased risk because of genetic or other vulnerabilities, and on better management of chronic health conditions and lifestyle choices.

Conventional clinical trials require large-sample, long-duration, randomized designs and may therefore be difficult to conduct. By contrast, a precision-medicine approach using a single subject may more rapidly determine whether personalized prevention plans may optimize person-centered outcomes. Several different pathways may trigger AD, suggesting that several different strategies may prevent or forestall AD. Even if ineffective alone, these precision strategies can reduce comorbidities and thereby substantially increase the probability that amyloid- or tau-specific therapies will be effective. The usually slow, progressive course of AD suggests that patient-specific approaches may have a chance to be effective.

MOOD SCALE

Geriatic Depression Scale Short Form English Scoring

Choose the best answer for how you have felt over the past week:

- 1. Are you basically satisfied with your life? YES / NO**
- 2. Have you dropped many of your activities and interests? YES / NO**
- 3. Do you feel that your life is empty? YES / NO**
- 4. Do you often get bored? YES / NO**
- 5. Are you in good spirits most of the time? YES / NO**
- 6. Are you afraid that something bad is going to happen to you? YES / NO**
- 7. Do you feel happy most of the time? YES / NO**
- 8. Do you often feel helpless? YES / NO**
- 9. Do you prefer to stay at home, rather than going out and doing new things? YES / NO**
- 10. Do you feel you have more problems with memory than most? YES / NO**
- 11. Do you think it is wonderful to be alive now? YES / NO**
- 12. Do you feel pretty worthless the way you are now? YES / NO**
- 13. Do you feel full of energy? YES / NO**
- 14. Do you feel that your situation is hopeless? YES / NO**
- 15. Do you think that most people are better off than you are? YES / NO**

Answers in **bold** indicate depression. Although differing sensitivities and specificities have been obtained across studies, for clinical purposes a score > 5 points is suggestive of depression and should warrant a follow-up interview. Scores > 10 are almost always depression.

مغرب زدگان

(شاہنواز فاروقی)

(جسارت)

مغربی دنیا نے جدید ذرائع ابلاغ کے پروپیگنڈے کو جادو بنایا ہے۔ اس جادو کی قوت کا یہ عالم ہے کہ اس کے ذریعے جھوٹ کو تجویز اور دن کو رات ثابت کیا جاسکتا ہے۔ اس کا ایک ثبوت یہ ہے کہ عراق کے صدر صدام حسین کے پاس بڑے پیانے پر تباہی پھیلانے والے ہتھیار نہیں تھے مگر مغربی ذرائع ابلاغ نے اس سلسلے میں اتنا شور چیزیا کہ ساری دنیا کو یقین ہو گیا کہ صدام حسین کے پاس کچھ نہ کچھ ضرور ہے۔ اس یقین کی آڑ میں امریکا اور اس کے اتحادی برطانیہ نے عراق کی اینٹ سے اینٹ بجادی۔ جب عراق کی اینٹ سے اینٹ بجائی جا پکھی تو معلوم ہوا کہ صدام حسین کے پاس تو کچھ بھی نہیں تھا۔ مگر اس بات کو مغرب کے ذرائع ابلاغ نے روپورٹ کرنے کی طرح روپورٹ ہی نہ کیا۔ چنانچہ عراق کا خطرناک ہتھیاروں سے پاک ہونا ٹھیک طرح سے "خبر" بھی نہ بن سکا۔

مغرب کے ذرائع ابلاغ کے جادو کی دوسرا بڑی مثال ملالہ کی "ایجاد" ہے۔ ملالہ کی موجودگی اپنی اصل میں باشتبی کی موجودگی تھی۔ مگر مغرب کے ذرائع ابلاغ نے دیکھتے ہی دیکھتے کو "دیو" بنا کر کھڑا کر دیا۔ یہاں تک کہ ملالہ نوبل انعام کی مستحق قرار پائی۔ ملالہ نے اگر لڑکیوں کی تعلیم کے لیے بچپاں لڑکیوں کے لیے ایک اسکول بھی کھولا ہوتا تو کہا جاسکتا کہ اس نے علم کی خدمت کی، مگر ملالہ کی گردہ میں تو ایک اسکول کی شہرت بھی نہ تھی۔ اس سے بہتر تھا کہ مغربی دنیا شیما کرمانی کو نوبل انعام دے دیتی۔ شیما کرمانی کو اور کچھ نہیں تو بھارت کا کلاسیکل رقص آتا ہے اور انہوں نے گزشتہ چالیس سال میں سیکڑوں لڑکیوں کو رقصاص بنایا ہے۔ یہ بات مذہبی اعتبار سے کتنی ہی ناقابل قبول کیوں نہ ہو مگر شیما کرمانی نے رقص کے شعبے میں کچھ تو کیا ہے۔ بد قسمتی سے ملالہ کو تور رقص بھی نہیں آتا۔ مگر اس سے کیا ہوتا ہے۔ مغرب کے حکمران اور مغرب کے ذرائع ابلاغ ملالہ کے ساتھ تھے اور انہوں نے کچھ نہ کرنے کے صلے میں ملالہ کو نوبل انعام سے نواز دیا۔ ہمیں حیرت ہے کہ جس مغرب کو ملالہ کی غیر موجود خدمات نظر آگئیں اس مغرب کو کبھی عبدالستار ایڈھی کی چالیس پچاس سالہ خدمات نظر نہ آئیں۔ خیر یہ اچھا ہی ہوا رنہ جو مغرب ملالہ کو کچھ نہ کرنے پر ایک عدد نوبل انعام سے نواز سکتا ہے وہ مغرب ایڈھی صاحب کو کم از کم پچاس نوبل انعام دینے پر مجبور ہو جاتا۔ ملالہ کا نام ایڈھی صاحب کے ساتھ لینا ایڈھی صاحب کی توہین ہے مگر مغرب کا جادو یہ ہے کہ ملالہ اور ایڈھی صاحب کو ساتھ کھڑا کیا جائے تو یہ ایڈھی صاحب باشتبی اور ملالہ دیو قامت نظر آتی ہیں۔ اس لیے کہ ملالہ کے ہاتھ میں دنیا کا سب سے بڑا انعام ہے اور ایڈھی صاحب کے ہاتھ کچھ بھی نہیں ہے۔ خیر مغرب نے ملالہ کو عظیم بناؤ لا تو یہ مغرب کا جرم ہے۔ مگر ملالہ کو تو معلوم ہے کہ وہ کچھ بھی نہیں ہیں۔ مگر مغرب کے پروپیگنڈے کے جادو نے ملالہ کو بھی یقین دلادیا ہے کہ وہ یقیناً ایک عظیم شخصیت ہیں۔ اس کا ایک ثبوت "الدن" میں ہونے والے لاہور لٹریری فیسٹیول سے ملالہ کا خطاب ہے۔ اس خطاب میں ملالہ نے کہا کہ مجھ پر تنقید کی جاتی ہے کہ میں نے پاکستان میں کیا کیا ہے؟۔ ملالہ نے کہا کہ سیکورٹی اسباب کی وجہ سے ہم بتانہیں سکتے کہ ہم پاکستان میں کس کے ساتھ مل کر کیا کر رہے ہیں؟۔ تسلیم کہ اس وقت ملالہ پاکستان میں کچھ نہ کچھ

کر رہی ہوں گی مگر سوال یہ ہے کہ انہوں نے نوبل انعام ملنے سے قبل پاکستان میں کیا کیا؟ کوئی اسکول کھولا؟ کوئی کالج قائم کیا؟ کسی یونیورسٹی کا نگہ بندیار رکھا؟ کوئی کوچنگ سینٹر قائم کیا؟ چار بچوں کو ٹیوشن پڑھایا؟ اگر ملالہ نے ان میں سے کوئی کام بھی نہیں کیا تو وہ مغرب کی "ڈارنگ" کیسے بنیں؟ انہیں نوبل انعام کیوں دیا؟ کیا اس لیے کہ کل مغرب انہیں پاکستان میں اپنے مقاصد کے لیے استعمال کرے؟ یہاں سوال تو یہ بھی ہے کہ ملالہ اس وقت پاکستان میں جو کچھ کر رہی ہیں اگر وہ پاکستان کی خدمت ہے تو اس پر پرداز لانے کی کیا ضرورت ہے؟ اور اگر اس پر پرداز لانے کی ضرورت ہے تو وہ خدمت کیسے ہے؟ لیکن یہاں سب سے اہم بات اس امر کی نشاندہی ہے کہ مغرب کے پروپیگنڈے نے ملالہ کو بھی یقین دلادیا ہے کہ اس نے کچھ کیا ہی ہو گا جس کی وجہ سے وہ مغرب میں ہاتھوں ہاتھی جا رہی ہے۔ لیکن اندر میں ہونے والے لاہور لٹریری فیسٹیول میں صرف ملالہ ہی نے تماشا نہیں کیا، پاکستان کی معروف انگریزی ناول نگار کاملہ شمسی نے بھی اپنارنگ دکھایا۔ انہوں نے فرمایا کہ ہندوستانیوں اور پاکستانیوں کے لیے آزادی کی کہانی سنانا دشوار ہے۔ اس لیے کہ اس کہانی میں ناقابل یقین تشدد اور دکھ ہے۔ انہوں نے کہا کہ 1971ء کی کہانی سنانی بھی مشکل ہے کیوں کہ اس کہانی میں اقتیت نے اکثریت پر تشدد کیا اور 1971ء کے واقعات پاکستان میں تاریخ کی کتابوں کا حصہ نہیں ہیں۔

اس حقیقت سے انکار ممکن نہیں کہ قیام پاکستان کے بعد ہونے والے مسلم کش فسادات میں لاکھوں مسلمان شہید ہوئے لیکن اس کے باوجود قیام پاکستان انسانی تاریخ کے عظیم ترین واقعات میں سے ایک ہے اور قیام پاکستان کی مسرت اس دکھ سے بہت بڑی ہے جو کہ لاکھوں مسلمانوں کے قتل عام سے پیدا ہوا۔ اس مسرت کے دکھ سے بڑے ہونے کی ایک وجہ یہ ہے کہ مسلمان دلی، یوپی، مشرقی پنجاب ہر جگہ اقلیت میں تھے، چنانچہ وہاگر چاہتے بھی تو ہندوؤں اور سکھوں کے ساتھ جھگڑا مول نہ لیتے۔ مطلب یہ کہ قیام پاکستان کے بعد جو کچھ ہوا وہ ہندوؤں اور سکھوں کا کیا درہ اتھا۔ چنانچہ مسلمانوں کی اجتماعی نفسیات احساس جرم کی نفسیات نہیں تھی اور نہ ہے۔ یہی وجہ ہے کہ اہل پاکستان آج بھی اپنی آزادی کو بہت بڑی نعمت سمجھتے ہیں۔ بلاشبہ 1971ء کے واقعات کا ہماری تاریخ کی کتابوں میں نہ ہونا بہت ہی افسوس ناک بات ہے۔ لیکن حقیقی تاریخ صرف پاکستان میں نہیں پوری دنیا میں نصابی کتب سے غائب ہے۔ مثلاً سفید فاموں نے امریکا پر قبضے کے لیے 10 کروڑ ریانڈنڈیز (Red Indians) مار ڈالے۔ مگر امریکا میں یہ بات اسکول کیا یونیورسٹی کی سطح پر بھی نصاب کا حصہ نہیں۔ آسٹریلیا میں سفید فاموں نے اپنا بقہہ مستحکم کرنے کے لیے آسٹریلیا کے اصل باشندوں یعنی ایب اور بینڈز (Aborigines) کا قتل عام کیا اور 45 لاکھ افراد کو مار ڈالا۔ آسٹریلیا میں یہ تاریخ کسی بھی سطح پر تعلیمی نصاب کا حصہ نہیں۔ ہندوستان میں اعلیٰ ذات کے ہندو ہزاروں سال سے کروڑوں شودروں اور دلوں پر ظلم کر رہے ہیں مگر یہ ظلم بھارت میں کہیں بھی نصاب کا حصہ نہیں۔ آخر کاملہ شمسی اور ان جیسے لوگ ان حقائق کا تذکرہ کیوں نہیں کرتے؟ آخر یہ لوگ کیوں نہیں کہتے کہ جس طرح پاکستان کے حکمران پاکستان کی نئی نسلوں کو صحیح تاریخ نہیں پڑھا رہے اس طرح امریکا، آسٹریلیا اور بھارت کے حکمران طبقات بھی اپنی نئی نسلوں کو اصل تاریخ نہیں پڑھا رہے۔

فیسٹیول سے خطاب کرتے ہوئے مرزا وحید نے قیام پاکستان کو "Partition" (قرار دیا اور فرمایا کہ 1998ء میں "Partition" جو ہری ہو گیا) کیوں کہ 1998ء میں پاکستان نے اپنی دھماکے کر ڈالے۔ پاکستان کے سیکولر اور لبرل عناصر کبھی پاکستان کے لیے "آزادی" کا لفظ استعمال نہیں کرتے۔ وہ ہمیشہ آزادی کو "Partition" یا " تقسیم" کہتے ہیں۔ تقسیم کا مطلب یہ ہے کہ قیام پاکستان سے پہلے "بھارت ماتا" ایک "وحدت" تھی مگر مسلمانوں نے پاکستان بنانے کے لئے کردیے۔ ظاہر ہے کہ یہ بھارت اور اس کے رہنماؤں کا "بیانیہ" ہے۔ اس کے برعکس آزادی کا لفظ پاکستان سے محبت کرنے والوں کی نفسیات کا عکس ہے اور یہ جنوبی ایشیا کے مسلمانوں کی تاریخ کا اصل ترجمان ہے۔ بد قسم سے مرزا وحید نے بھی قیام پاکستان کے لیے آزادی کے بجائے " تقسیم (Partition)" کا لفظ استعمال کیا۔ مگر یہ ان کا پہلا جرم ہے۔ ان کا دوسرا جرم یہ ہے کہ انہوں نے فرمایا کہ

1998ء میں تقسیم (Partition) نیوکلیئر ہو گیا۔ یہ ایک صریح جھوٹ ہے۔ اس کی وجہ یہ ہے کہ اس خطے میں ایسی ہتھیاروں کی دوڑ پاکستان نے نہیں بھارت نے شروع کی۔ اس نے 1974ء میں ایسی دھماکا کر دیا تھا۔ مگر مرزا وحید کو 1974ء تو یاد نہ آیا۔ انہیں یاد آیا تو 1998ء۔ اتفاق سے 1998ء میں بھی پاکستان نے ایسی دھماکوں میں پہل نہیں کی تھی۔ پہل بھارت نے کی تھی پاکستان نے صرف اس کا جواب دیا۔ فیضیوں سے مدھر جعفری نے بھی خطاب فرمایا۔ انہوں نے کہا کہ بھارتی پنجاب اور پاکستانی پنجاب کے کھانے ایک سے ہیں البتہ ان کے نام مختلف ہیں۔ ایک اور فرق یہ ہے کہ پاکستان گوشت خور ہیں اور بھارتی پنجاب کے پنجابی سبزی خور ہیں۔ البتہ سبزی خور علاقوں زیادہ ترقی یافتہ ہیں۔ بھارت کے لوگ جب پاکستان کے حوالے سے بات کرتے ہیں تو وہ کہتے ہیں ہمارا سب کچھ ایک جیسا ہے۔ بس "تقسیم کی لکیر" نے ہمیں ایک دوسرے سے جدا کر رکھا ہے۔ مطلب یہ کہ "تقسیم کی لکیر" کو مٹا دیا جائے تو ہم پھر سے ایک ہو جائیں گے۔ بد قسمتی سے مدھر جعفری نے بھی یہی کہا ہے۔ انہوں نے پاکستان کے تشخص یا identity کو "کھانوں" کی سطح پر لا کھڑا کیا حالاں کہ پاکستان کا حقیقی تشخص مذہبی، تہذیبی اور تاریخی ہے۔ مزے کی بات یہ ہے کہ ایک جانب مدھر جعفری کہہ رہی ہیں کہ ہمارے کھانے ایک جیسے ہیں اور دوسری جانب انہیں یہ " واضح" کرنے میں بھی دلچسپی ہے کہ "سبزی خور" گوشت کھانے والوں سے زیادہ ترقی یافتہ ہیں یعنی سیکولر اور لبرل عناصر اور اہل بھارت جب چاہتے ہیں پاکستان اور بھارت کو ایک جیسا دلخواہ دیتے ہیں اور جب چاہتے ہیں دونوں کے "فرق" پر اصرار کرنے لگتے ہیں۔

KARACHI PSYCHIATRIC HOSPITAL

KARACHI ADDICTION HOSPITAL



Established in 1970

Modern Treatment With Loving Care

بِالْحَلَقِ عَمَلُه - جَدِيدٌ تَرِّيْنَ عَلاجٌ

Main Branch

Nazimabad # 3, Karachi

Phones # 111-760-760
0336-7760760

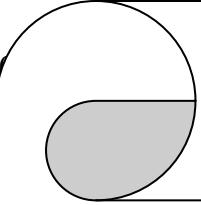
Other Branches

- **Male Ward:** G/18, Block-B, North Nazimabad, Karachi.
- **Quaidabad (Landhi):** Alsyeed Center (Opp. Swedish Institute)
- **Karachi Addiction Hospital:**
(1) D-71, Nawab House, Block B, North Nazimabad, Karachi.
(2) Rimpala Plaza M.A Jinnah Road, Karachi.

E-mail: support@kph.org.pk

Skype I.D: kph.vip

Visit our website: <www.kph.org.pk>



MESSAGE FOR PSYCHIATRISTS

Karachi Psychiatric Hospital was established in 1970 in Karachi. It is not only a hospital but an institute which promotes awareness about mental disorders in patients as well as in the general public. Nowadays it has several branches in Nazimabad ,North Nazimabad and in Quaidabad. In addition to this there is a separate hospital for addiction by the name of **Karachi Addiction Hospital**.

We offer our facilities to all Psychiatrists for the indoor treatment of their patients under their own care.

Indoor services include:

- 24 hours well trained staff, available round the clock, including Sundays & Holidays.
- Well trained Psychiatrists, Psychologists, Social Workers, Recreation & Islamic Therapists who will carry out your instructions for the treatment of your patient.
- An Anesthetist and a Consultant Physician are also available.
- The patient admitted by you will be considered yours forever. If your patient by chance comes directly to the hospital, you will be informed to get your treatment instructions, and consultation fee will be paid to you.
- The hospital will pay consultation fee DAILY to the psychiatrist as follows:

Rs 700/=	Semi Private Room Private Room
Rs 600/=	General Ward
Rs 500/=	Charitable Ward (Ibn-e-Sina)

The hospital publishes a monthly web journal in its website by the name ‘The Karachi Psychiatric Hospital Bulletin’ with latest Psychiatric researches. We also conduct monthly meetings of our hospital psychiatrists in which all the psychiatrists in the city are welcome to participate.

Assuring you of our best services.

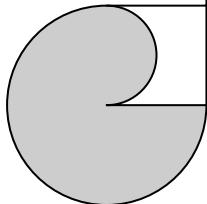
Shadab Nizam

(C.E.O)

Contact # 0336-7760760

UAN # 111-760-760

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Our Professional Staff for Patient Care

❖ Doctors:

- 1. Dr. Syed Mubin Akhtar**
MBBS. (Diplomate American Board of Psychiatry & Neurology)
 - 2. Dr. Major (Rtd) Masood Ashfaq**
MBBS, MCPS (Psychiatry)
 - 3. Dr. Javed Sheikh**
MBBS, DPM (Psychiatry).
 - 4. Dr. Akhtar Fareed Siddiqui**
MBBS, F.C.P.S
 - 5. Dr. Salahuddin Siddiqui**
MBBS
 - 6. Dr. Sadiq Mohiuddin**
MBBS
 - 7. Dr. Seema Tahir**
MBBS
 - 8. Dr. Muhammad Ali**
MBBS
 - 9. Dr. Naseer Ahmed Cheema**
MBBS
 - 10. Dr. Munawar Ali**
MBBS
 - 11. Dr. Farooq-e-Azam**
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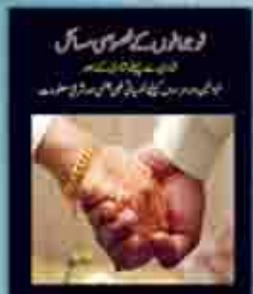
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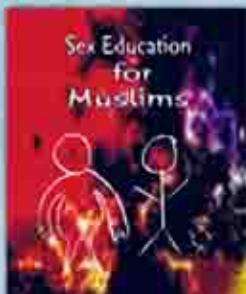
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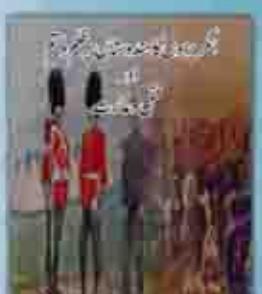
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The same book has been translated into Urdu under the title of
"جنسی امور کے تصوری سائل"

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