CHIEF EDITOR DR. SYED MUBIN AKHTAR

KARACHI PSYCHIATRIC HOSPITAL

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Karachi Psychiatric Hospital held a Press Conference on World Epilepsy Day 2016

IN Patient Activities

Patients are Playing and Praying in ward

Under the supervision of recreation and Islamic Therapists

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8 Completely Wrong Assumptions about Addiction

By Jerry Nelson
~ 2 min read

Think of words to describe the typical drug addict. Honest, courageous and insightful are not the first you think of.

However, given the opportunity, many addicts end up developing these very qualities and contribute to society in a way no one ever imagined possible.

The recovery successes happen in spite of obstacles placed in front of them. The always-present threat of relapse to the pervasive stereotype addicts encounter along the way are hurdles that have to be overcome on the path to sober living.

Do drugs fry your brain? Already an addict, forever an addict? What's this about an "addiction gene"?

The topic of addiction is filled with myths, lies, half-truths and misinformation. Much of this may have been designed to frighten kids away from drug use, but some of it has gotten embedded in drug lore. The myths haven't succeeded in winning the war on drugs. What they have done is made it difficult for addicts to attempt therapy.

Programs such as Beachway Therapy Center include educational components to help laymen learn the difference between addiction myth and fact. Some of the more frequent myths heard include:

Myth 1: There exists an addiction gene

Studies by institutions such as the University of Utah show there isn't an addiction gene — or set of genes. There's no genetic way to determine if a given individual will become an addict. Yet if a person's parents are addicted, it doesn't mean their offspring will become addicts. It just means that children of addicts are at a higher risk.

Myth 2: Pot is a gateway drug

The dependence rate for alcohol is higher than that for marijuana. There is limited objective data that pot serves as a trigger to move someone to harder drugs of choice. Yes, teen use of marijuana should be discouraged, but the true "gateway" drugs are prescript opioids and drugs like OxyContin, Vicodin and Adderall. These all have powerful addictive characteristics and are available to teens.

Myth 3: Addiction is life-long

This places an impassioned and irrational weight on recovered and recovering addicts. Addiction is a disorder that lies on a spectrum. As in depression, everybody is different.

There are many instances where addicts have tried for years to defeat their addiction, other cases are different. Short-term users who put history behind them and move on to lead productive lives.

Myth 4: Drugs fry your brain

The 1987 anti-drug commercial used a frying egg to demonstrate "your brain on drugs." Drug abuse can be harmful to the cerebellum, but it is an oversimplification to say that drug abuse constantly causes persistent and critical brain damage.

This myth gives rise to the idea that recovered addicts are "damaged" and opens the door for employer discrimination, as well as discrimination by health care providers and law enforcement. However, some drugs, such as neurotoxins like methamphetamine, MDMA and cocaine do increase the risks.

Myth 5: Hit "rock bottom."

If a person waits until the hit "rock bottom" before seeking help, it may be too late — some people die before they bottom out. Each person has a different bottom with some being higher than others. For some individuals, getting arrested may be their "bottom". For others, it might just mean being confronted by family or friends or even doing poorly at work.

There is scant evidence that the level of consequences a person must accumulate before looking for help is related to success in recovery. However, it is always better to seek help earlier than later.

Myth 6: Addicts should be punished

Regardless of a person's age, income, employment status, gender or race, there's a popular misconception that once a person develops an addiction they are bad, weak or immoral.

The resulting hostility is totally unlike any other attitude towards chronic illnesses. It is true that many addicts do disgusting things. Addicts lie, cheat and steal to keep their habit going. The truth is, good people sometimes do bad things, and sick people need treatment.

Myth 7: Addiction is a choice

Willpower isn't enough. People do not choose to become an addict any more than they might choose to have cancer.

Myth 8: Just one type of substance

Formerly, experts believed that most addicts picked just one drug of choice and stuck with it. Today, the use of three or more substances — polysubstance — is the norm. Some individuals combine multiple substances to increase the intensity of the sought-for "high".

Is TMS Cost-Effective?

Psychiatric times

Dee Rapposelli

November 09, 2015 | Major Depressive Disorder, Depression, Transcranial Magnetic Stimulation

By Dee Rapposelli

Researchers looked into the efficacy and value of TMS for treatment-resistant depression.

RESEARCH UPDATE

Repetitive transcranial magnetic stimulation (rTMS) is known to be a safe, noninvasive, treatment for major depressive disorder (MDD), but how does cost compare with that of pharmacotherapy?

Australian researchers compared the cost-effectiveness of rTMS with pharmacotherapy in treatment-resistant patients with MDD (ie, those who have failed at least 2 courses of antidepressant therapy). They found that, although both pharmacotherapy and rTMS are clinically effective, rTMS is more cost-effective.

Considering that up to 40% of patients with MDD either do not respond to or tolerate pharmacotherapy and that up to 85% of patients who do respond can be expected to relapse within 15 years, exploration of methods that more economically sustain quality of life is worthwhile.

Although several studies have compared the cost of rTMS with that of electroconvulsive therapy, only one has compared the pharmacoeconomics of rTMS with that of pharmacotherapy for MDD. In that 2009 study, rTMS provides an incremental cost-effectiveness ratio of USD $34,999 per quality-adjusted life-year (QALY). (The willingness-to-pay threshold was set at USD $50,000 per QALY.)

To further explore the issue, the Australian research team used a 3-year Markov microsimulation model with 2-monthly cycles to compare costs and QALYs of rTMS and standard pharmacotherapy with a variety of commonly used antidepressant medications. These include

selective serotonin reuptake inhibitors, serotonin and norepinephrine reuptake inhibitors, tricyclic agents, noradrenergic and specific serotonergic antidepressants, and monoamine oxidase inhibitors. Data were also extracted from published literature, cost reports, and expert opinion. Incremental cost-utility ratios and univariate and multivariate probabilistic sensitivity analyses were applied.

rTMS, which induces an electrical current in a localized region of the cerebral cortex, requires a patient to present 3 to 5 times per week for 4 to 6 weeks of treatment. Treatment sessions last about 40 minutes. No anesthesia or muscle relaxants are needed. Patients can resume normal activities immediately after a session, although common adverse events include mild-to-moderate posttreatment headache and mild pain or discomfort at the treatment site.

Although use comes with a warning of potential seizure induction, no seizures were reported during clinical trials, and the postmarketing incidence rate, at less 0.1% per patient, is lower than that seen with standard pharmacotherapy. This, in itself, may render rTMS a superior choice for patients with concerns about adverse effects of pharmacologic agents regardless of cost concerns.

The Markov model showed that QALYs gained were 1.25 with rTMS vs 1.18 with antidepressant therapy, and costs were somewhat lower for rTMS in AUD: $31,003 vs $31,190 ($22,124 vs $22,260). The willingness-to-pay threshold in Australia is AUD $50,000 per QALY gained. Given that threshold, the findings showed that the probability that rTMS was dominant (ie, provided better patient quality-of-life at lower cost) was 32%, and the probability that rTMS was cost-effective compared with antidepressant therapy was 41%.

The bottom line

Sensitivity analyses confirmed the superiority of rTMS in terms of value for money compared with antidepressant medications, with multivariate analysis showing that the probability of rTMS being either dominant or cost-effective compared with antidepressant therapy exceeded 70%.

The findings confirmed those of the earlier study.

Stepped Approach Could Prevent Major Depression and Anxiety in Older Adults with Visual Impairment

By Kelly Young

Edited by David G. Fairchild, MD, MPH, and Lorenzo Di Francesco, MD, FACP, FHM

Stepped care may prevent older adults with visual impairment from developing major depression or anxiety, suggests a BMJ study conducted in Belgium and the Netherlands.

Over 250 adults aged 50 and older with vision impairment and subthreshold depression or anxiety were randomized to either usual care alone or with stepped care. Stepped care comprised the following treatment sequence (with each step lasting about 3 months): watchful waiting, guided self-help based on cognitive behavioral therapy, problem-solving therapy, and physician referral. If at 3 months, patients' anxiety or depression scores worsened, they moved to the next step.

At 24 months, the incidence of major depressive, dysthymic, or anxiety disorders was lower in the stepped care group (29% vs. 46%). Roughly six patients needed to be treated to prevent one case of depressive or anxiety disorder.

The authors conclude that their approach could help in the screening and treatment of older adults with visual impairment.

Creating a Family Health History

Why Create a Family Health History?

A Family Tree for Health

A family health history is a written record of a family's health. The history contains information about a family's medical conditions, lifestyle habits (for example, whether anyone in the family has smoked), and where and how family members grew up. It's like a family tree for health.

What a Family Health History May Reveal

You can use a family health history to see if you, your children, or your grandchildren might face an increased risk of developing serious health problems. These health problems might be common ones, such as heart disease, cancer, or diabetes. They could also be less common diseases that are passed from one generation to the next, such as hemophilia or sickle cell anemia.

People can't change the genes they inherit from their parents, but they can change things like diet, physical activity, and medical care to try to prevent diseases that run in the family. This is good news because many diseases result from a combination of a person's genes, lifestyle, and environment.

Actions That May Reduce Disease Risk

A health care professional can use a family health history to help assess a person's risk of certain diseases. The professional might recommend actions to lower the chance of getting those diseases.

Actions to reduce the risk of disease may involve

- lifestyle changes, such as eating healthier foods or exercising more
- getting certain medical tests
- taking medicines that are more effective based on your specific genes.

For example, a son with a family history of diabetes might be told to lose weight and exercise more. A daughter who is considering having a baby might get tested to see if she carries a gene for a rare condition that runs in the family.

How You and Your Family May Benefit

For older adults, a family health history might help explain why you have developed certain health conditions. But it is important to know that simply getting older increases the risk of many diseases, too.

Creating and sharing your family health history with your health care professional can help you be healthier. But perhaps the biggest benefit is providing information that may help your children and grandchildren live longer, healthier lives.

November is National Epilepsy Awareness Month

Learn about the many types of seizures.

A seizure is a short change in normal brain activity that can cause changes in awareness, behavior, or body movement. Because anyone can have a seizure, it's important to recognize seizure symptoms and to know how to help. There are over 30 different types of seizures. The signs of seizures depend on the part of the brain affected. Some seizures are mild and can go unnoticed. Others can cause the person to fall to the ground, or be unable to move or speak.

Epilepsy is not the only cause of a seizure. Seizures can happen from other problems, including:

- Brain injury.
- High fever.
- Alcohol or drug withdrawal.

Did you know?

There are actually over 30 types of seizures. Partial seizures are the most common type of seizure experienced by people with epilepsy.

There are two groups of seizures: Generalized seizures and partial seizures.

Generalized seizures

Generalized seizures affect both sides of the brain. These words are often used when describing generalized seizures:

- Tonic: Muscles in the body become stiff, forcefully.
- Atonic: Muscles in the body relax.
- Myoclonic: Muscles in the body jerk or twitch, usually on both sides of the body.
- Clonic: Periods of jerking spasms in muscles of the body, sometimes on both sides of the body.

Tonic-clonic seizures (or what used to be called grand mal seizures) are the most well-known type of seizure. Tonic-clonic seizures can make a person:

- Cry out.

- Lose consciousness.
- Fall to the ground.
- Have muscle jerks or spasms.

The person may feel tired after a tonic-clonic seizure.

Absence seizures (or what used to be called petit mal seizures) can cause rapid eye blinking or a few seconds of staring into space.

Partial seizures

Partial seizures are located in just one area of the brain. These seizures are also called focal seizures. There are at least three different types of partial seizures.

Simple partial seizures affect a small part of the brain. These seizures can cause jerking or a change in sensation, such as a strange taste or smell, or a "funny feeling" in the stomach.

Complex partial seizures can make a person with epilepsy confused or dazed. The person will be unable to respond to questions or direction for up to a few minutes. A person with this type of seizure may move around without purpose or direction.

Partial seizures that generalize begin in one part of the brain but then spread to both sides of the brain. In other words, the person first has a partial seizure, followed by a generalized seizure.

Epilepsy and Seizure First Aid Training

What do I do if I see someone having a seizure?

Not all seizures are emergencies and most will end within a few minutes. The first response is to remain calm, provide care, and comfort. Time the seizure, and check for a medical identification bracelet or other emergency information.

Learn about seizure first aid and what you can do to help during a seizure.

Read more about epilepsy and find the answers to common questions.

Alzheimer's Disease

U.S. Department of Health & Human Services

What is Alzheimer's disease?

What happens to the brain in Alzheimer's disease?

How many Americans have Alzheimer's disease?

How long can a person live with Alzheimer's disease?

What is dementia?

What is Alzheimer's disease?

Brain IlustrationAlzheimer's disease is an irreversible, progressive brain disorder that slowly destroys memory and thinking skills and, eventually, the ability to carry out the simplest tasks. In most people with Alzheimer's, symptoms first appear in their mid-60s. Alzheimer's disease is the most common cause of dementia among older adults.

The disease is named after Dr. Alois Alzheimer. In 1906, Dr. Alzheimer noticed changes in the brain tissue of a woman who had died of an unusual mental illness. Her symptoms included memory loss, language problems, and unpredictable behavior. After she died, he examined her brain and found many abnormal clumps (now called amyloid plaques) and tangled bundles of fibers (now called neurofibrillary, or tau, tangles).

These plaques and tangles in the brain are still considered some of the main features of Alzheimer's disease. Another feature is the loss of connections between nerve cells (neurons) in the brain. Neurons transmit messages between different parts of the brain, and from the brain to muscles and organs in the body.

Although treatment can help manage symptoms in some people, currently there is no cure for this devastating disease.

What happens to the brain in Alzheimer's disease?

Scientists continue to unravel the complex brain changes involved in the onset and progression of Alzheimer's disease. It seems likely that damage to the brain starts a decade or more before

memory and other cognitive problems become evident. During this preclinical stage of Alzheimer's disease, people seem to be symptom-free, but toxic changes are taking place in the brain. Abnormal deposits of proteins form amyloid plaques and tau tangles throughout the brain, and once-healthy neurons stop functioning, lose connections with other neurons, and die.

The damage initially appears to take place in the hippocampus, the part of the brain essential in forming memories. As more neurons die, additional parts of the brain are affected. By the final stage of Alzheimer's, damage is widespread, and brain tissue has shrunk significantly. Read more about what happens to the brain in Alzheimer's »

View Video: Alzheimer's Disease Process

This 4-minute captioned video shows the intricate mechanisms involved in the progression of Alzheimer's disease in the brain.

How many people have Alzheimer's disease?

Estimates vary, but experts suggest that more than 5 million Americans have Alzheimer's disease. Unless the disease can be effectively treated or prevented, the number of people with it will increase significantly if current population trends continue. That's because the risk of Alzheimer's increases with age, and the population is aging.

How long can a person live with Alzheimer's disease?

Alzheimer's is a slow disease that progresses in three stages—an early, preclinical stage with no symptoms, a middle stage of mild cognitive impairment, and a final stage of Alzheimer's dementia. The time from diagnosis to death varies—as little as 3 or 4 years if the person is older than 80 when diagnosed to as long as 10 or more years if the person is younger.

Alzheimer's is currently ranked as the sixth leading cause of death in the United States, but recent estimates indicate that the disorder may rank third, just behind heart disease and cancer, as a cause of death for older people.

What is dementia?

Dementia is the loss of cognitive functioning—thinking, remembering, and reasoning—and behavioral abilities to such an extent that it interferes with a person's daily life and activities. Dementia ranges in severity from the mildest stage, when it is just beginning to affect a person's

functioning, to the most severe stage, when the person must depend completely on others for basic activities of daily living.

The causes of dementia can vary, depending on the types of brain changes that may be taking place. Other dementias include Lewy body dementia, frontotemporal disorders, and vascular dementia. It is common for people to have mixed dementia—a combination of two or more disorders, at least one of which is dementia. For example, some people have both Alzheimer's disease and vascular dementia.

Other conditions that may cause memory loss or dementia include:

medication side effects

chronic alcoholism

tumors or infections in the brain

blood clots in the brain

vitamin B12 deficiency

some thyroid, kidney, or liver disorders

stroke

Parkinson's disease

Sleep disturbances

Some of these conditions may be treatable and possibly reversible. They can be serious and should be treated by a doctor as soon as possible.

Emotional problems, such as stress, anxiety, or depression, can make a person more forgetful and can be mistaken for dementia. For instance, someone who has recently retired or who is coping with the death of a spouse may feel sad, lonely, worried, or bored. Trying to deal with these life changes leaves some people confused or forgetful. The emotional problems can be eased by supportive friends and family, but if these feelings last for a long time, it is important to get help from a doctor or counselor.

Family-Based Intervention May Help Prevent Anxiety Disorders in Children

Aaron Levin
October 26, 2015

Children of anxious parents are at an increased risk of developing an anxiety disorder, but a new study suggests that a measure of prevention may be possible.

A cognitive-behavioral intervention aimed at families in which at least one parent had an anxiety disorder reduced the likelihood that children developed anxiety disorders, according to a study published September 24 in AJP in Advance.

Previous studies showed that the children of anxious parents are at a greater risk of developing an anxiety disorder, and parenting practices, such as overcontrol and overprotection, contributed to elevated anxiety. While anxiety prevention programs carried out in schools have been only modestly successful at reducing childhood and adolescent anxiety, less is known of the effects family-based interventions might have on the high-risk offspring of anxious parents.

"Prevention is always better than providing intervention after a disorder has been identified," child psychiatrist Paramjit Joshi, M.D., division chief of psychiatry and behavioral medicine at Children's National Medical Center in Washington, D.C., told Psychiatric News. "In that sense I think this is an important study and has clinical applicability."

The next step in the research is to examine the factors that predicted which children went on to develop an anxiety order and identify those who might be at greatest risk, says Golda Ginsburg, M.D.

University of Connecticut for the AJP in Advance study, Golda Ginsburg, Ph.D., a professor of psychiatry at the University of Connecticut Health Center, and colleagues randomly assigned 136 families to either the eight-week Coping and Promoting Strength program or a control condition using an informational pamphlet. All participating families had at least one parent who met DSM-IV-TR criteria for an anxiety disorder and at least one child aged 6 to 13 without an anxiety disorder.

As part of the intervention program, each family met individually with a trained therapist for 60 minutes a week for eight weeks. The first two sessions were for parents only, after which the children were included. As part of the program, parents learned about how to reduce modeling of anxiety, overprotection, and overall distress. The children were counseled to reduce risk factors like anxiety symptoms, social avoidance or withdrawal, or maladaptive thoughts. Families were shown how to identify signs of anxiety and strategies to cope with and reduce anxiety.

Participants in the information-monitoring group received a 36-page pamphlet containing information about anxiety disorders and associated treatments. Anxiety was assessed before the trial began, at the end of the intervention (or eight weeks after randomization), and at follow-ups six and 12 months later.

After finishing the program, children in the intervention group had lower symptom scores on average. Just three children (5 percent) in the intervention group met criteria for an anxiety

disorder by the end of the 12-month follow-up compared with 19 children (31 percent) in the information-monitoring group. At the one-year follow-up, youth in the control group also had higher anxiety symptom ratings than those in the intervention group.

"Among youth who received the intervention, those with high baseline anxiety symptom severity levels showed greater reductions in severity than those with low baseline levels, which suggests that the intervention is particularly helpful for youth with elevated anxiety symptoms," the study authors wrote.

"One interpretation could be that targeting only those with elevated symptoms would be more efficient," said Ginsburg in an interview. "However, we did not look at all moderators, and my next step is to look at what other factors predicted who went on to developing an anxiety disorder in order to also 'personalize' who might be at most risk and benefit most from prevention."

About 13 percent of the intervention cohort and 22 percent of the control families reported that their children received mental health services for anxiety during the study period, but the difference was not statistically significant. That narrow gap may have been either because the intervention's effect had no effect on treatment-seeking or because anxious young people typically seek care at low rates, the authors noted.

Reductions in the parents' modeling of anxiety and their global distress at the trial's end and at the six-month follow-up point mediated the intervention's effect on the severity of children's anxiety symptoms after one year, the authors said. "This finding clarifies potential mechanisms of the intervention's impact and suggests that targeting specific parenting behaviors (such as reducing anxious modeling) and lowering parents' overall distress levels (not anxiety specifically) were critical in reducing child anxiety symptoms."

While the study authors noted that the work needs to be replicated in a larger and more demographically diverse cohort, it may represent a step forward in efforts to reduce the number of children and adolescents with anxiety disorders.

"I think the important aspect of this study is that if we can provide an intervention for kids at risk for developing anxiety disorders given a family history of anxiety in a parent, then this speaks to the whole notion of early identification of at-risk children and preventative intervention," said Joshi.

The researchers recently received funding from the National Institute of Mental Health to follow this cohort of children for another seven years, said Ginsburg. "So we will also look at what holds up over time."

Sugars, Sweeteners & T2DM: The Bad, the Bad & the Ugly

Veronica Hackenthal, MD
Endocrinology Network

Do added sugars and artificial sweeteners play an equal role in type 2 diabetes risk?

In July 2015, the U.S. Food and Drug Administration (FDA) proposed the inclusion of "added sugars" to the percent daily value (%DV) on the Nutrition Facts label for packaged foods. Under the new proposal, "added sugars" would appear indented and below the "sugars" category that already appears on the label.

The decision comes after a review of recent scientific evidence by the 2015 Dietary Guidelines Advisory Committee showing that meeting daily nutrient needs can be difficult if added sugars exceed 10% of total daily calories. The committee also recommended that Americans cut down on empty calories and reduce their added sugar intake to fewer than 10% of daily calories. Currently, Americans obtain about 16% of their daily calories from sugars. To put that into perspective, a 20-ounce bottle of Coca-Cola contains about 16 teaspoons of added sugar, or 130% of the recommended daily intake.

The Institute of Medicine, American Heart Association, American Academy of Pediatrics, and the World Health Organization have also warned about the dangers of added sugars.

The FDA estimates that relabeling will cost about $2.3 billion, but cumulative benefits over 20 years could range from $21.1 billion to $31.4 billion.

The FDA proposal comes amid accumulating evidence suggesting that what you eat, rather than how much, plays a role in the development of chronic illnesses like diabetes and cardiovascular disease. For years, researchers had said that the main problem concerns the sheer number of calories consumed, as well as resulting obesity. Research now shows that the amount of empty calories—especially calories from added sugars—can contribute to the development of diabetes.

For example, a recent study that evaluated ten years of data on diabetes and nutritional food components in 175 countries has pointed to a direct, independent association between added sugars and diabetes. Results showed that increasing sugar intake by about one can of soda per day increased diabetes prevalence by 1.1% ($P<0.001$). The relationship remained even after adjusting for obesity, total calories, aging, income, sedentary behavior, alcohol use, and several other related factors.

A recent meta-analysis also looked at just this issue. It included 21 studies conducted in the US and UK, published through February 2014, and covering over 38,000 cases of incident T2DM. Results suggested that 11% of T2DM cases predicted to develop over 10 years in the US could be attributed directly to consuming sugar-sweetened beverages. Other types of beverages may not necessarily be safe, either. Results also showed that increasing consumption by one serving/day increased the incidence of T2DM by 13% for sugar-sweetened beverages, by 8% for artificially sweetened beverages, and by 7% for fruit juices, after adjusting for obesity.

Teasing out the role of artificial sweeteners in the development of metabolic disease, though, poses a tricky research question. That's because those at risk for metabolic disease or who are trying to lose weight may switch to artificially sweetened foods as a way to reduce sugar or calorie intake, which muddies the data.

One study in the UK aimed at clearing up this dilemma. The study included over 25,000 people who were free of diabetes at the start of the study. Researchers estimated sugary beverage consumption from participant-reported 7-day food diaries, and followed participants for over 10 years.

Results showed that consumption of artificially sweetened beverages increased the risk of T2DM by 22% (HR 1.22 [1.11, 1.33]), which was similar to the 21% increased risk of T2DM found with soft drinks (HR 1.21 [1.05, 1.39]). After adjusting for obesity, though, the association between artificially sweetened beverages and T2DM weakened (HR 1.06 [0.93, 1.20]). Further analyses showed that substituting artificially sweetened beverages for any type of sugar sweetened beverage failed to decrease the incidence of T2DM. This remained true even after adjusting for calorie intake and obesity.

Estimates showed that drinking one serving per day of water or unsweetened tea or coffee instead of sugar-sweetened beverages could decrease the incidence of T2DM by 14%-25%.

Research, mostly from animal studies, provides some evidence that artificial sweeteners may be metabolically active, increasing the risk of obesity, metabolic syndrome, and T2DM in and of themselves.

One explanation, based on rodent models, could be that artificial sweeteners interfere with learned responses to sweet foods. Upon ingestion of artificial sweeteners, the body senses the sweetness and gets ready to digest calories. The difference between anticipated and actual calories tricks the body, interfering with the glucoregulatory response controlling energy homeostasis. A second explanation holds that artificial sweeteners interfere with the gut microbiome, which contributes to glucose intolerance. One study exposed seven healthy human volunteers who did not usually eat artificial sweeteners to one week of the FDA maximum acceptable saccharin intake. Results showed that regular saccharin exposure in most participants increased glycemic responses to glucose load, as measured on oral glucose tolerance tests. Stool transplant from humans with altered glycemic responses to saccharin induced glucose intolerance in mice. A third theory holds that artificial sweeteners could interact with sweet-taste receptors expressed not just in the mouth but throughout the digestive system. This interaction could be detrimental to regulation of glucose absorption from the gut, and could also trigger insulin secretion.

Efired/Shutterstock.com Imitating nature can have its drawbacks. Sometimes the solution lies in the commonplace, like a simple glass of water.

• The FDA has proposed to include added sugars on the %DV label for packaged foods.

• Accumulating evidence suggests that consuming sugary beverages and foods plays a role in the development of diabetes.

• Artificial sweeteners may be metabolically active and increase the risk for obesity and diabetes.

• Substituting a glass of water for a sugary beverage may be one way to decrease the risk of diabetes.

Managing Behavioral and Psychological Symptoms of Dementia in the Era of Black Box Warnings

Rajesh R. Tampi, MD, MS

Psychiatric Times

Deena J. Tampi, MSN, MBA-HCA, RN

An in-depth look into the behavioral and psychological symptoms of dementia.

Behavioral and psychological symptoms of dementia (BPSD) are a heterogeneous range of psychological reactions, psychiatric symptoms, and behaviors that are unsafe and disruptive and impair the care of the individual. About one-third of community-dwelling individuals with dementia exhibit BPSD. The prevalence increases to approximately 80% in individuals residing in nursing facilities.

Jost and Grossberg found that behavioral symptoms were observed in 72% of individuals more than 2 years before the actual diagnosis of dementia. The prevalence of BPSD increased to 81% 10 months after the formal diagnosis of dementia. Unlike cognition, which declines over time in individuals with dementia, BPSD tends to fluctuate; agitation is the most persistent symptom.

Apathy is the most common problem in individuals with dementia; it tends to occur early in the course of illness and remains stable through the course of the illness. Delusions commonly seen in individuals with dementia include false beliefs (eg, of theft, infidelity, misidentification syndromes). Individuals with dementia who have a family history of depression appear to be at greater risk for a major depressive episode than those without a family history. Disinhibition occurs in approximately one-third of the patients. In patients with hallucination, the most common form is visual hallucination. Irritability and mood lability become more prevalent with the progression of dementia.

Findings indicate that BPSD occurs because of the complex interactions between anatomical, functional, and biochemical changes that occur in the brain. Certain genes can predispose individuals to BPSD, and premorbid personality may contribute to the emergence of certain types of behavioral symptoms.

BPSD is associated with greater overall burden of care for individuals with dementia. The emergence of BPSD often results in the referral of patients with dementia to a specialist's care. Paranoia, aggression, and sleep-wake cycle disturbances are associated with greater caregiver

burden, caregiver depression, and the risk of institutionalization. BPSD results in the worsening of activities of daily living, faster cognitive decline, and a poorer quality of life. It also adds to the overall direct and indirect cost of care for individuals with dementia after adjusting for the severity of cognitive impairment and other comorbidities.

Assessment

Assessment includes collateral information from caregivers and/or family members. Collateral information aids in determining the onset, the course, and the differential diagnosis of BPSD. It also helps identify risk and prognostic factors. Environmental factors and psychosocial stressors may be triggers for the onset or the worsening of symptoms. Underlying medical conditions, including pain syndromes, urinary tract infection, and dehydration, can result in exacerbation of symptoms. An evaluation for and management of these disorders will reduce the frequency and severity of BPSD and may mitigate the use of psychotropic medications.

Assessment tools such as the Neuropsychiatric Inventory, the Behavioral Pathology in Alzheimer Disease rating scale, the Consortium to Establish a Registry for Alzheimer Disease Behavior Rating Scale for Dementia, Dementia Behavior Disturbance scale, and the Neurobehavioral Rating Scale can be used to aid diagnosis of BPSD. These instruments help identify behaviors and rate their severity. They can also assist in tracking progression and in monitoring treatment response.

Management of BPSD

Nonpharmacological strategies should be the primary intervention for patients with BPSD. If the symptoms of BPSD are not well managed with nonpharmacological strategies, judicious pharmacotherapeutic trials can be tried. A decline in cognition can result in the emergence or worsening of BPSD. A trial of cholinesterase inhibitors and/or memantine may be initiated to delay cognitive decline.

Nonpharmacological management. Findings from a systematic review suggest that caregiver and residential care staff education and possibly cognitive stimulation therapy are effective in the management of BPSD. However, specialized dementia units were not consistently beneficial; changing the environment visually and unlocking doors reduced wandering.

Nonpharmacological interventions provided by caregivers can reduce the frequency and severity of symptoms and can reduce caregiver burden, with effect sizes similar to those associated with pharmacotherapy. Successful interventions included approximately 9 to 12 individual sessions

designed to meet the needs of the patient and his or her caregivers and were delivered in the patient's home using multiple components over 3 to 6 months with periodic follow-up.

One meta-analysis showed statistically significant results for nonpharmacological interventions on at least one measure of BPSD. These interventions included staff training in behavioral management strategies, mental health consultation and treatment planning, exercise, recreational activities, and music therapy or other forms of sensory stimulation. However, there were methodological limitations that suggest a possible risk of bias. Moreover, for many of the interventions, substantial resources from outside services were needed along with considerable time commitments from the nursing staff for the implementation of these strategies.

Pharmacological management. If possible, behaviors should be grouped into different clusters (eg, psychotic, mood). These clusters act as psychobehavioral metaphors of primary psychiatric disorders. If these behaviors are not well managed by individual medication trials, a judicious combination of medications can be tried (eg, an antidepressant with an antipsychotic).

Caution must be exercised when prescribing medications, given their adverse-effect profile and the vulnerability of the population being treated. Medication combinations, such as 2 antipsychotics or 3 or 4 different medication classes, should be avoided to minimize serious adverse events. The duration of effective medication trials should be about 3 to 4 months of clinical stability, followed by a trial of medication taper and discontinuation.

A meta-analysis by Ballard and colleagues demonstrated that compared with placebo, risperidone and olanzapine reduced aggression. Individuals treated with risperidone also had significant improvements in psychosis. A meta-analysis by Schneider and colleagues also found evidence for the efficacy of aripiprazole and risperidone in the management of BPSD.

In a randomized, double-blind, placebo-controlled trial, outpatients with Alzheimer disease and psychosis, aggression, or agitation were randomly assigned to olanzapine, quetiapine, risperidone, or placebo. There were no significant differences among the active agents regarding the time to discontinuation of treatment for any reason. The median time to discontinuation of treatment because of a lack of efficacy favored olanzapine and risperidone over quetiapine and placebo. The time to the discontinuation of treatment because of adverse events or intolerability favored placebo compared with the active agents.

Sertraline and citalopram were associated with a reduction in symptoms of agitation. No differences in behavioral symptoms were seen when trazodone was compared with haloperidol. Both SSRIs and trazodone appear to be well tolerated compared with placebo, typical

antipsychotics, and atypical antipsychotics. SSRIs and trazodone showed some benefit in the management of BPSD and were well tolerated.

Low-dose sodium valproate is ineffective in treating agitation in individuals with dementia, and high-dose divalproex sodium is associated with an unacceptable rate of adverse effects. In a literature review, Konovalov and colleagues found one study that showed statistically significant benefit of carbamazepine compared with placebo in the treatment of BPSD. However, most studies demonstrated no significant differences. In a majority of the studies, adverse effects were more frequent with the active medication.

Cholinesterase inhibitors provided modest improvements in neuropsychiatric and functional outcomes, with no differences in efficacy among the different drugs in this class. A meta-analysis showed modest improvements in symptoms with memantine. Moreover, it is well tolerated—the only adverse effects are confusion and sedation.

There is insufficient evidence to justify the use of benzodiazepines for managing BPSD. There have been only 5 controlled trials, which were of short duration and with a limited number of subjects. The efficacy data were limited and the adverse-effect profile was not benign.

The data for using pharmacotherapeutic agents in the management of BPSD are limited. There are no robust data on the efficacy of these drugs, but there are data on the adverse-effect profiles. Although limited, the best evidence for efficacy was found for antipsychotics, especially risperidone, olanzapine, and aripiprazole, and for SSRI antidepressants, namely sertraline and citalopram.

Antipsychotic use in elderly persons with dementia

Individuals at high risk for adverse effects of antipsychotics are those who are 85 years or older and who have vascular or mixed dementias or active cerebrovascular or cardiovascular diseases and significant impairments in activities of daily living. However, if the symptoms of BPSD are not sufficiently managed by other strategies, the use of antipsychotics may be justified.

When prescribing antipsychotic medications, it is sensible to use the lowest effective dose and for the shortest time to manage the symptoms. Close monitoring for adverse effects and their swift management in case they do arise will minimize serious adverse events.

Herrmann and Lanctôt completed a post hoc analysis of pooled results from randomized controlled trials of persons with dementia who were treated with risperidone and olanzapine. Their results indicate an increased incidence of cerebrovascular adverse events. Some of the increased incidence could be accounted for by nonspecific events other than stroke. There were a

greater number of persons with vascular and mixed dementias in the risperidone trials than in the olanzapine trials. In contrast to this post hoc analysis, other results show no increased risk of stroke in older individuals treated with risperidone and olanzapine compared with individuals treated with typical antipsychotics or untreated individuals with dementia.

FDA findings indicate that the use of atypical antipsychotics in individuals with BPSD is associated with increased mortality. The majority of placebo-controlled trials of olanzapine, aripiprazole, risperidone, and quetiapine showed 1.6- to 1.7-fold increases in mortality. Deaths were due to heart-related events (heart failure or sudden death) or infections (mainly pneumonia). The FDA asked the manufacturers of these drugs to include a black box warning in their labeling describing this risk and noting that these drugs are not approved for BPSD. Similar changes to the labeling for typical antipsychotics have also been added.

A meta-analysis by Schneider and colleagues found that the risk of death was greater among individuals who were treated with atypical antipsychotics. The sensitivity analyses did not show any evidence for differential risks for individual drugs, severity of dementia, sample selection, or the diagnosis. Wang and colleagues found that conventional antipsychotic medications were associated with a significantly higher adjusted risk of death than were atypical antipsychotic medications.

The risk of cerebrovascular adverse events was 1.3 to 2 times higher with active treatment compared with placebo; the risk of death was about 1.2 to 1.6 times higher. The risk is similar for typical and atypical antipsychotic agents. A higher than median dose of a drug, older age, a diagnosis of vascular dementia, and comorbid atrial fibrillation have been noted as risk factors for cerebrovascular adverse events. Older age, male sex, severe dementia, and functional impairment are associated with higher risk of death.

Conclusion

Common in individuals with dementia, BPSD is associated with poorer outcomes and greater burden of care. Data show that BPSD is caused by underlying neuroanatomical and neurochemical changes in the brain and a premorbid personality structure. Pharmacotherapy should be combined with nonpharmacological approaches for optimum results. The risk of prescribing medications for patients with BPSD (who are often elderly) should be carefully weighed with possible benefits. These medications should only be prescribed within the recommended dosage to reduce the risk of serious adverse events. Close monitoring of risk factors will also help mitigate serious adverse events.

When the Psychiatrist Has PTSD

Alisa G. Woods, PhD

Psychiatric Times

For PTSD in psychiatrists and other mental health care providers to be addressed, a major shift in medical culture and thinking is needed.

Dr John Bradford of Ottawa, Canada, had a long, successful career as a forensic psychiatrist, spanning several decades. Bradford's extensive experience with brutal people, such as sexual sadists and killers, led him to believe that he was resilient to trauma. The years of work, however, may have taken their toll. After watching highly disturbing video evidence of 2 women being assaulted as part of a high-profile case, Bradford began drinking excessively, and became depressed as well as suicidal. Despite 2 years of initial denial, he ultimately sought treatment for PTSD and admirably now speaks out about his experience. Although he still practices, Bradford avoids cases that involve graphic videos. His example illustrates that even seasoned professionals may fall prey to PTSD, and the toughest psychiatrists are not immune.

Michael F. Myers, MD, Professor of Clinical Psychiatry in the Department of Psychiatry & Behavioral Sciences at SUNY Downstate Medical Center in Brooklyn, New York, spoke at the American Psychiatric Association (APA) meeting in Toronto, Canada, in May 2015. His presentation "PTSD in Psychiatrists: A Hidden Epidemic" underscored the under-recognition of this prevalent condition in those who treat mental health conditions and discussed methods for increased recognition of PTSD as well as its treatment.

Ironically, mental health care providers may readily encourage their patients to confront psychiatric issues, but they are not always as willing to identify and address these problems in themselves, despite the frequent occurrence of PTSD in physicians. The lifetime prevalence of PTSD is between 8% and 9% in the general population, but is higher in physicians. For example, PTSD rates in physicians exposed to war range between 11% and 18% and are about 12% in those who practice emergency medicine. It makes sense that the prevalence of PTSD would be high in psychiatrists as well, owing not only to frequent traumatic encounters with those who have serious psychiatric conditions, but also to the vicarious transfer of their patients' experiences.

PTSD can strike at any professional stage, from the experienced psychiatrist such as Dr John Bradford, to physicians in training. In his talk at the APA meeting, Myers presented data from several studies that focused on medical students and PTSD. The hazing and overworking of medical students is a tradition that continues, possibly with the notion that extremely difficult conditions will prepare students for a doctor's life. The practice may however come with a cost. According to one study, 73% of medical students reported witnessing or experiencing mistreatment. In another study, 13% of 212 residents met the diagnostic criteria for PTSD based on a standard questionnaire; women were more often affected (20%) than men (9%). Lack of social support increased the risk of PTSD.

Myers cautioned that residents may experience PTSD because they are not psychologically prepared for the traumatic events they might witness. He discussed several clinical situations that could be disturbing to physicians in training, including observing severe injuries, treating battered infants, seeing amputations, or observing death in a young, healthy-seeming individual. Even being put-down and humiliated by a senior physician might trigger PTSD, something for traditionalists who think students need "toughening up" to consider.

According to Myers, circumstances that may precipitate PTSD include not only medical training but also traumatic events that took place before medical school. For example, immigrants who left war-torn countries or physicians who are specifically training to deal with traumatic situations may be vulnerable; military psychiatrists also may be particularly susceptible.

Adding to the list of those at risk provided by Myers, Dr Arthur Lazarus has identified several types of physicians who might be at elevated risk for PTSD. These include:

• Emergency physicians
• Physicians practicing in remote areas with limited medical services
• Physicians involved in malpractice litigation
• "Second victims," physicians who are exposed to trauma via their patients

Certainly psychiatrists would fall under the last category and may be members of the other categories as well. Understanding who is at risk can help predict and assess symptoms of PTSD in susceptible individuals.

Identification is of course the first step; however, treatment is crucial for improvement in the lives of the individual psychiatrists, as well as for those they affect professionally and personally. But if PTSD in psychiatrists is so deeply buried because of denial and a lack of social acceptance, what can be done about this problem?

Myers suggests several tactics. First, the culture of medicine needs to change. Physicians in training can be better prepared for possibly upsetting situations through debriefing, normalizing, and learning to accept the events. New institutional attitudes are needed, specifically encouraging physicians with PTSD symptoms to come forward and seek help early. The stigma and belief system that views physicians as being bulletproof to PTSD needs to change. Finally, coworkers should realize that some physicians will live chronically with residual symptoms. Again, acceptance is a key to improving the situation.

For the psychiatrist treating a colleague with possible PTSD, Myers recommends getting buy-in as a first start: for example, "see whether your patient is in agreement" when suggesting possible PTSD, and ask how the symptoms are affecting that person's ability to work. Reassurance that treatment is effective can be helpful, as well as noting that help through therapy or medication will positively affect the psychiatrist's professional and personal life.

But what if you are the psychiatrist or mental health care professional with PTSD? Myers believes that the signs can be there, but as psychiatrists "there can be a certain amount of denial at first. We practice differently: we don't listen as carefully; we shut the person down and reach for the prescription pad." To these individuals, he evokes the adage "physician, heal thyself." He suggests that psychiatrists should seek assistance and advice from a colleague and should not feel that they must abandon the profession. Treatment can actually maintain their ability to be effective psychiatrists.

Recognition naturally needs to be followed by treatment, including a complete assessment of PTSD symptoms as well as of comorbid symptoms. PTSD treatment may include medication, individual cognitive-behavioral therapy, and group therapy as needed. However, care does not stop at the individual with PTSD symptoms, since treatment of the family and marital therapy can also help. Balint groups—the presentation of clinical cases among physicians—help prepare people working together to deal with PTSD in psychiatrists specifically. Myers encourages the psychiatrist to "be an advocate and change maker."

Indeed, for PTSD in psychiatrists and other mental health care providers to finally be addressed, a major shift in medical culture and thinking is needed. Psychiatrists will hopefully learn to recognize signs of PTSD in their colleagues and also in themselves. The condition exists in psychiatrists and may even be common, and it needs to be treated.

When Reducing Antipsychotic Use in Dementia Patients Might Harm More Than Help

Joel Yager, MD reviewing Ballard C et al. Am J Psychiatry 2015 Nov 20.

Reducing antipsychotic use while adding social interaction reduced mortality, but medication reduction alone led to worse neuropsychiatric symptoms.

Health policymakers have advocated reducing antipsychotic prescriptions in demented patients because of links to increased morbidity and mortality. However, questions remain as to whether these adverse outcomes reflect underlying behavioral conditions for which the antipsychotics were prescribed or medication effects per se. To investigate how altering pharmacological and nonpharmacological interventions might affect demented nursing-home patients, investigators randomized 16 U.K. nursing homes for 9-month trials of various combinations of rigorous antipsychotic review, increased social interaction, and exercise.

Of the 277 patients (74% female), 18% were taking antipsychotics, and 87% had moderate or severe dementia. Antipsychotic review conformed to clinical guidelines that recommended treatment constraint, e.g., limiting newly initiated antipsychotics to 12 weeks followed by attempted discontinuation. Enhanced social interactions and exercise (for each, ≥1 hour weekly or 20% increase by study's end) followed manual-based programs.

Of the 20 patients on antipsychotics undergoing antipsychotic review, 50% stopped receiving medication (all had been medicated for ≥3 months); no medication was stopped in patients at homes not receiving review. Combining antipsychotic review with enhanced social interaction had a strong positive effect on reducing mortality, with most of the impact attributable to greater social interaction. Review alone did not alter agitation scores. Receiving antipsychotic review alone was associated with worse outcomes in neuropsychiatric symptoms, but this finding was mitigated in patients receiving enhanced social interaction. Exercise reduced neuropsychiatric symptoms but did not affect depression scores or mortality.

Six Months Is Long Enough for Adjunctive Antipsychotics After Resolution of Mania

Peter Roy-Byrne, MD reviewing Yatham LN et al. Mol Psychiatry 2015 Oct 13.

Continuing antipsychotics beyond 6 months may not further protect against mania recurrence and definitely causes more weight gain.

Augmenting a mood stabilizer with an atypical antipsychotic is often used for an acute manic episode, and continuation is recommended to prevent future episodes. Yet, atypical antipsychotics pose significant health risks from their metabolic and weight effects. These researchers investigated the optimal duration of continuation therapy. The 159 participants were in remission for 2 to 6 weeks from an acute manic episode and taking either lithium (n=85) or valproate (n=74) plus either risperidone (n=93) or olanzapine (n=66).

Participants were randomized to antipsychotic discontinuation immediately (with switch to placebo), at 24 weeks, or at 52 weeks. Time to relapse to any mood episode was similar for the 24- and 52-week groups and was significantly longer in the 24-week group than in the immediate-discontinuation group; 1-year episode rates were estimated at 65%, 65%, and 82%, respectively. Roughly two thirds of the relapses involved depressive episodes; yet, in secondary analyses, the benefit of longer treatment was significant only for protection against manic episodes. Subgroup analysis of risperidone and olanzapine recipients suffered from small sample sizes but suggested that risperidone was effective only in preventing mania whereas olanzapine was effective only in preventing depressive relapses. Antipsychotic-associated weight gain was significantly greater at 52 weeks than at 24 weeks.

Using the Internet to Increase Access to Evidence-Based Treatment for Obsessive-Compulsive Disorder

Psychiatric Times

Sapana R. Patel, PhD

Andrew B. Schmidt, LCSW, PhD

H. Blair Simpson, MD, PhD

Internet-based CBT has shown promise to improve access to therapy for patients with OCD, which is associated with a profoundly diminished quality of life and social isolation.

In recent years, the increasing number of computer and Internet users has greatly expanded the potential to access evidence-based care for the treatment of psychiatric disorders. Computer-

assisted and Internet-based treatments expand accessibility of treatment for individuals who may have economic, transportation, or other restrictions that limit access to face-to-face services. Furthermore, these programs have the advantages of increasing cost-effectiveness of evidence-based treatments by reducing contact time with a therapist, broadening client participation in therapy-based activities in real-world settings, and monitoring quality of care and client progress. Researchers have developed programs that range from self-directed therapy based on "self-help" models to therapist-supported interactive treatments with or without video conferencing. Much of the research on computer or Internet-based programs has been focused on anxiety disorders. Given the severe and disabling nature of obsessive-compulsive disorder (OCD), numerous efforts have been made to improve access to evidence-based treatment for those facing barriers to care.

OCD is associated with a profoundly diminished quality of life, social isolation, and a substantial economic burden on society; the lifetime prevalence of OCD is 2%. It is characterized by the presence of persistent and distressing thoughts and worries—obsessions—and repetitive or ritualized behaviors—compulsions. Most often the compulsions are carried out in direct response to the obsessions and serve to reduce anxiety and distress.

Evidence-based guidelines identify 2 types of effective treatments of OCD: medications, including SNRIs (eg, clomipramine) and SSRIs, and cognitive-behavioral therapy (CBT) consisting of exposure and response prevention (ERP). With ERP, patients gradually expose themselves to their fears or obsessions while refraining from engaging in rituals or compulsions. Exposures are conducted in a systematic fashion starting with situations that are least feared and gradually working toward situations that are most feared. Despite the documented efficacy of ERP, few patients receive this treatment in clinical practice, although many patients prefer CBT

to medication alone and CBT is superior to antipsychotic medications as an augmentation strategy for those experiencing residual symptoms. Barriers to care include uncertainty about where to seek treatment, lack of trained therapists, shame and stigma associated with mental health problems, time limitations, competing demands, and costs associated with seeking psychological care.

Multiple efforts have been made to improve access to CBT for patients with OCD; among these, an Internet-based CBT (I-CBT) for OCD, developed at the Karolinska Institutet in Sweden, is the most promising program developed to date. In I-CBT, individuals use an Internet-based treatment platform to learn about their illness and to engage in ERP under the confidential guidance of an expert therapist. I-CBT overcomes many barriers to care and allows individuals to receive evidence-based ERP treatment under the guidance of trained therapists without the traditional restrictions and requirements of face-to-face meetings.

In one trial, I-CBT was found to be far superior to a control treatment (response to treatment: 60% for I-CBT vs 6% for control); I-CBT also had low dropout rates (12%), comparable to rates seen in face-to-face treatment. Moreover, decreases in OCD severity with I-CBT are comparable to those with face-to-face ERP. I-CBT is a clear advance over other self-help programs because it is a single, integrated system of treatment supported by a therapist accessible anywhere via the Internet. This program has the potential to substantially affect public health and enable underserved OCD patients to access evidence-based care. However, I-CBT has never been tested outside of Sweden in diverse OCD patient populations and settings. It is exciting to see effects of I-CBT in Sweden, and we look forward to seeing how such a program might fare in the US. This type of innovation could change how we treat anxiety disorders, including OCD.

Study Shines a Light on Depression

Dee Rapposelli

Psychiatric Times

Researchers conducted a clinical trial comparing light therapy with antidepressant monotherapy. Here's what they found.

RESEARCH

Light therapy may be an effective treatment for major depressive disorder (MDD), says a multicenter team of researchers. Lam and colleagues conducted the first adequate-duration, placebo-controlled, randomized clinical trial comparing light therapy with antidepressant monotherapy (fluoxetine hydrochloride 20 mg/d) and combination light/antidepressant therapy.

The trial lasted 8 weeks and included 122 psychiatric outpatients, aged 19 to 60 years, who had a diagnosis of MDD of at least moderate severity as assessed by board-certified psychiatrists and confirmed via the Mini International Neuropsychiatric Interview and the Hamilton Depression Rating Scale. Of this total intent-to-treat population, 32 patients were allocated to receive light monotherapy, 31 to fluoxetine monotherapy, 29 to combination therapy, and 30 to placebo.

Light monotherapy consisted of exposure to an active 10,000lux fluorescent white light box for 30 minutes every morning plus ingestion of a placebo pill. Fluoxetine monotherapy included use of an inactive ion generator, which served as a sham light box. Patients allocated to combination therapy received both fluoxetine 20 mg/d and active light therapy, and those allocated to placebo only received a placebo pill and underwent exposure to the inactive ion generator.

Patients were regularly evaluated using the Montgomery-Asberg Depression Rating Scale (MADRS), Clinical Global Impression subscales for depression severity and improvement, and

the Quick Inventory of Depressive Symptomatology-Self report. Adverse events also were monitored.

More effective than fluoxetine alone

Light therapy, either as monotherapy or in combination with fluoxetine, was effective and well tolerated. Indeed, although combination therapy provided the most consistent effects, fluoxetine monotherapy was no more effective than placebo—although the researchers noted that the seemingly poor performance of fluoxetine monotherapy may have been a consequence of a smaller-than-planned sample size.

The mean change in MADRS score from baseline at 8 weeks was 16.9 (±9.2) in patients who received combination therapy, 13.4 (±7.5) in those who received light monotherapy, 8.8 (±9.9) in those who received fluoxetine monotherapy, and 6.5 (±9.6) in those who received placebo.

Response, defined as a 50% or more reduction from baseline in MADRS scores, was achieved by 76% of the group receiving combination therapy; 50% of the group receiving light monotherapy; 29% of those receiving fluoxetine monotherapy; and 30% of those receiving placebo. Remission, defined as a MADRS score of 10 or lower at the final visit, was achieved by 59% of the combination-therapy group, 44% of the light-monotherapy group, 19% of the fluoxetine-monotherapy group, and 30% of the placebo group. No significant differences in terms of adverse events were seen.

Mechanism of action to be determined

How light therapy ameliorates SAD—or depressive symptoms in general—is unknown, but the leading hypothesis has been that bright light may resynchronize circadian rhythms or restore neurotransmitter dysfunction. A monoaminergic effect also may be at play.

"We Should Live"- - Surviving After Catastrophic Death

Allen Frances, MD
Psychiatric Times

There is no one-size-fits-all solution to how people and cultures should respond to overwhelming stress, depression, and trauma.

Yutaka Ono has been a close friend of mine for 30 years and is one of Japan's most respected psychiatrists. Shortly after the massive earthquake and deadly tsunami that hit eastern Japan 4 years ago, he began regularly visiting Onagawacho--the hardest hit town. He became consultant to Yuri Sato, the brave, wise, and energetic public health nurse responsible for organizing the town's medical and psychosocial response to its massive tragedy.

On a recent visit to Onagawacho, Dr Ono introduced my family and his to Ms Sato and she provided an excellent slideshow explaining the mechanics of the tsunami, its devastating impact on her town, the loss of life, and the untiring efforts she and her staff had made to help the survivors endure and prevail in the face of unimaginable trauma and loss.

Ms Sato's presentation was understated and stoical until she reached the slide listing instructions now given to townspeople on what to do should there be a future tsunami.

Among these, I was struck by and asked about: "Escape to high ground immediately. Never go back to save people or possessions."

Ms Sato quietly explained that the tsunami's speed made it impossible to rescue others-- that each person had to be taught to do instinctively whatever it would take to simply save himself or herself. Going back in a futile attempt to save others would be wasteful of life. She added that it was particularly difficult to teach individualistic self-protection to people in Japan, because it so goes against prevailing values that cherish loyalty to others and self sacrifice for family and society.

Then, suddenly and unexpectedly, Ms Sato began to cry. She described how she had lost her own son because he had gone back to save his grandmother and both were caught in the giant wave as it rebounded to the sea. She said she felt very guilty for surviving and for having taught him to be so unselfish. With a different upbringing, he might have been more rational and still be alive. Soon all of us were crying uncontrollably. Life can be exquisitely cruel and confusing. Best intentions can have the worst outcomes.

I asked Ms Sato to write about her work in restoring Onagawacho and healing herself. Dr Ono has translated. Ms Saito writes:

"The massive tsunami caused by the Great East Japan Earthquake swallowed up most of my town and killed almost 1000 people, 10% of the population. Even now, after four and a half years, there are many whose remains still have not been found.

On that day, I evacuated to the roof of the town office building. Stupefied and aghast, I could only helplessly stare frozen at the approaching wall of seawater. The next day, as I walked

through the rubble, it felt like a scene from some out-of-this-world newsreel. I found the survivors squatting, huddled together in our gym which felt like a field hospital.

As public health nurses, our immediate task was to treat the injured and sick; collect and dispense medicines; and respond to the desperate conditions of the townspeople. We launched and managed an aid station and a welfare evacuation site for those in need of urgent nursing care; established countermeasures against infectious disorders; and arranged for emergency food supplies and sanitation. Housing was first in buildings that had survived on high ground -- eg, sports facilities, small public halls, and school buildings. Over several months, the survivors were then moved to temporary housing.

With the entire town disaster-stricken, I was overcome with a sense of doubt and anxiety. Questions continually spun around in my mind: "What is mental health care when all of us have suffered so greatly?" But with everyone in mourning, we were single-mindedly focused on not losing any more lives to suicide or accident.

We saw to the safety of patients with mental disorders and coordinated a treatment continuation system for them almost immediately after the tsunami.

People continued to be unable to accept the deaths of family members, relatives and friends -- and the fact that so many were still missing.

I often heard "I should have died," "Why did I survive?" or "I want my time to come soon." I also felt this way, but had too much work to do to linger on my own losses and feelings about them.

In such circumstances, unless a mechanism is set up within the local community that allows survivors to believe "we will live" and "we should live," people will drop out of society and languish on their own. We recognized that "community care" not "individual care" was our most important task, and we set out to build a new sense of community as soon as possible, after the more urgent needs of the immediate crisis had been met.

In this second stage of crisis management, we focused on "public health" as relates to housing, meals and work environment of the townspeople; resumed community organization activities; and provided support in the formation of new communities to partially replace what had been lost.

We resumed dialysis, provided maternity and infant checkups for pregnant women and newborns, gave mother and child vaccinations, and began special checkups for cancer patients and the mentally ill. Much of this health management was done in temporary housing. We also had to provide support to account for the changes in family composition caused by the tsunami-- many caretakers had themselves been killed.

In November 2011, we started the project "Onagawacho Counseling Center for the Mind, Body, and Life." The town was divided into seven areas and "expert staff of mind and body" (hereinafter referred to as "Koko-Kara Experts") were assigned to the sub-centers to provide health support and life support to the minds and bodies of the townspeople. This combined health management with reconstructing social bonds among the people who were isolated and desolate.

In each area, one Koko-Kara Expert (public health nurses, regular nurses, or counselors) was responsible for health consultations, home visits, and reorganizing a social structure in

cooperation with each neighborhood association and local officials (including the administrative community head, commissioned welfare and child commissioners, health promotion commissioners and diet improvement commissioners).

Our aim was not only mental health care but also reconstructing relationships and creating a new community. Building individual fitness requires restoring the physical and social environment. This is necessarily a gradual process, made difficult by the dilution of relationships in the community caused by the many deaths of townspeople and the fact that survivors were in temporary housing. The scattering of people from the same community in different shelters made their adjustment difficult.

We always kept in mind improvement in QOL (Quality of Life) by focusing not only on the "mind" and "body" but also the quality of "day-to-day life." Without mutual support in the community (families, colleagues and neighbors, etc), we cannot lift the spirits of others or protect and heal and care for the mind. "Skilled Listening Volunteers" were trained to increase the number of people who would listen to the stories of families, neighbors and friends.

We keenly sense the need to help create new relationships filled with "mutuality" and "thankfulness" by expanded communication-- circles of people to help people want to live. We opened cafes to promote social interaction and sponsored tea parties to legitimize having fun again.

Even after four and a half years, our challenges still have no end in sight.

We know there will be more tasks in the field of community mental health to help prevent mental disorders, alcohol problems, lonely deaths, and suicides."

Thanks so much Yuri and Yutaka for this information and inspiration.

We are a hardy species, evolved over millions of years to live in environments that were filled with catastrophes caused by natural disasters, famine, pestilence, and violence from fellow humans and other predators. Modern life provides some insulation for some of us, but traumatic stress remains widespread around the world and is a lurking risk for all of us.

We expected to encounter PTSD symptoms among the survivors of this horrifying experience, but Ms Sato said these have been rare. Much more common were depression, withdrawal, apathy, and guilt. Perhaps this reflects Japanese stoicism or the high average age of the survivors. But I think also that there is not a one-size-fits-all in the way people and cultures respond to overwhelming stresses. Our PTSD definition in DSM may be quite culture specific. Each person and each culture must be understood in it's own terms and context.

I will never forget the visit to the brave little town of Onagawacho and it's wonderful public health nurse, Ms Sato.

And I will never forget this sign on the wall of the town's cafe. It was drawn by a 94-year-old fisherman who had survived the tsunami. His calligraphy is beautiful, his poetic words haunting: "My life is study; my headstone will be my graduation certificate."

CDC Proposes Updated Guidelines for Opioid Prescribing

By Kelly Young
Edited by David G. Fairchild, MD, MPH, and Jaye Elizabeth Hefner, MD
The CDC has proposed 12 recommendations for clinicians prescribing opioids.
Among them:

- When initiating opioid therapy, immediate-release opioids at the lowest effective doses are recommended. Extra precautions should be taken when prescribing at or above 50 morphine milligram equivalents (MME) per day, and doses at or above 90 MME per day generally should be avoided.
- For treatment of acute, nontraumatic pain unrelated to major surgery, opioids usually should not be prescribed for more than 3 days.
- For patients with chronic pain, clinicians should discuss the benefits and harms of continued opioid treatment with patients at least every 3 months.
- Providers should use their state's prescription drug monitoring program to review the patient's history of controlled substance prescriptions when starting opioids for chronic pain and at least every 3 months during treatment. Urine testing should be performed before initiation and at least annually thereafter.
- Clinicians should avoid prescribing opioids for patients receiving benzodiazepines.

Revisiting an Old Friend

Brian Miller, MD, PhD, MPH
Psychiatric Times
Information about the performance of clozapine compared with other treatment strategies in usual practice may impact on its use in routine clinical settings. Here: findings from US national Medicaid data in a cohort of patients with treatment-resistant schizophrenia.
BRIEF COMMUNICATION
Despite evidence for superior efficacy in reducing psychotic symptoms, only a minority of patients with treatment-resistant schizophrenia in the US are prescribed clozapine. Rather than starting clozapine, clinicians often switch to a different, non-clozapine antipsychotic or combine antipsychotic medications. Information about the performance of clozapine compared with other treatment strategies in usual practice may impact on its use in routine clinical settings.
Stroup and colleagues compared the effectiveness and safety of initiating clozapine versus other antipsychotic treatment in a retrospective cohort of patients with evidence of treatment-resistant schizophrenia. The authors hypothesized that clozapine would be more effective in reducing the risk of psychiatric hospitalization, treatment discontinuation, and concomitant antipsychotic use.
Using a national (45 states) database from 2001 to 2009, the authors identified Medicaid enrollees aged 18 to 64 years with at least 2 outpatient or 1 inpatient claim for schizophrenia. All

subjects were in active treatment (ie, had filled a prescription for an antipsychotic within 30 days prior to study entry) and had not taken clozapine in the past year. Patients using long-acting injectable antipsychotic in the 60 days prior to study entry were excluded. Treatment resistance was defined as at least one psychiatric hospitalization, filling prescriptions for at least 2 antipsychotic medications, and a medication possession ratio of > 0.75 in the year before entering the study.

A cohort was assembled by 1:1 matching of patients with treatment-resistant schizophrenia who were eligible initiators of clozapine (N = 3123) versus standard antipsychotics (N = 3123). Intervention was defined as treatment with clozapine or another oral antipsychotic for which there were no filled prescriptions in the past year. The primary outcome measure was psychiatric hospitalization. Secondary effective outcomes were discontinuation of the initial antipsychotic medication (gap of > 30 days in prescription) and use of an additional antipsychotic medication. Safety outcomes included incident cases of acute myocardial infarction, stroke, self-injurious behavior, myocarditis, agranulocytosis, and intestinal obstruction. Data were analyzed using Cox proportional hazards models.

The majority of the cohort was male (52%) and Caucasian (56%) with a mean age of 39. Patients who initiated clozapine had significantly lower rates of psychiatric hospitalization than those who began taking other antipsychotic medications (hazard ratio [HR] = 0.78, 95% confidence interval [CI] = 0.69-0.88). They also had significantly lower rates of discontinuation of the index antipsychotic medication (HR = 0.60, 95% CI = 0.55-0.65) and first use of additional antipsychotic medication (HR = 0.76, 95% CI 0.70-0.82). Patients initiating clozapine also had a higher incidence of diabetes (HR = 1.63, 95% CI = 0.98-2.70); hyperlipidemia (HR = 1.40, 95% CI = 1.09-1.78) ; and intestinal obstruction (HR = 2.50, 95% CI=0.97-6.44)

The authors concluded that in a national cohort of patients with schizophrenia and evidence of treatment resistance, initiating clozapine was more effective than a standard antipsychotic on risk of hospital admission, antipsychotic discontinuation, and initiation of a new antipsychotic medication. They noted that their definition of treatment resistance required patients to be hospitalized and to initiate new medications; thus, the results are most applicable to this subgroup of patients (versus patients with schizophrenia started on clozapine following a period of stable symptoms in the community.)

<u>The bottom line</u>

Increased, judicious use of clozapine is warranted in routine clinical practice, together with vigilant monitoring to prevent and detect serious medical adverse events associated with initiation of this medication.

Disclosures:

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Convergence Science: Shaping 21st Century Psychiatry

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The Medici effect is upon us in biomedicine, and it's called convergence science.

THE FUTURE OF PSYCHIATRY

The Medici effect encapsulates the benefits of cross-pollination and interaction between individuals and teams from different fields in the pursuit of innovation. It is named after the wealthy Medici family who helped catalyze the Renaissance by bringing together poets, philosophers, scientists, painters, and other artisans to Florence, Italy. We believe the modern Medici effect is convergence science, and it is set to revolutionize health and medicine in the 21st century given the interplays occurring among physical, computer, and life sciences.

Convergence science is defined as the merging of distinct technologies, industries, tools, disciplines, or devices into a unified whole to create new pathways and opportunities. Convergence relies on a new integrated approach to solving problems too complex for any single discipline. Sharp and Langer1 described convergence as the "third revolution" in biomedicine after the development of molecular and cellular biology (the first revolution) and genomics (the second revolution). Based on a study of scientific progress at the Massachusetts Institute of Technology, convergence science was recommended as a way to blend diverse scientific disciplines. More than an interdisciplinary science, convergence integrates distant paradigms, systems, theories, and disciplines with problem-oriented research that crosses boundaries of academic, public, and private spheres.

Learning from oncology

Oncology may be the current biomedical frontier for convergence science. Indeed, a new academic journal has just been launched: Convergent Science Physical Oncology. The aim of the journal is to integrate physical science with cancer biology and clinical oncology to advance the understanding and treatment of cancer in patients. The National Cancer Institute's Center for Strategic Scientific Initiatives supports convergent approaches to cancer innovation. Their mission is to "create and implement exploratory programs focused on the development and integration of advanced technologies, transdisciplinary approaches, infrastructures, and standards to accelerate the creation of publically available, broadly accessible, multidimensional data, knowledge, and tools to empower the entire cancer research continuum for patient benefits."

This initiative has led to Cancer Nanotechnology Excellence and Integrative Cancer Biology Program Centers to be constructed to support convergent projects. Private companies (eg, IBM and NantHealth) are already developing data intensive systems to integrate, display, and analyze—via machine learning—data from all health providers, genomic and proteomic

analyses, imaging, and other medical devices, with the actionable health information available at the point of care, anywhere.

Initiatives supporting convergence science approaches for psychiatry

If convergence is an approach to complexity, then what appears promising for oncology should be even more useful for psychiatry. Indeed, this approach has already been harnessed for neuroscience. A number of large-scale convergence neuroscience initiatives are underway globally, and these initiatives are essential to making progress in neuroscience. These projects bring together researchers from a multitude of disciplines to understand the basic functioning of the human brain, and the dysfunction that occurs during illness.

One such initiative is the Brain Research Through Advancing Innovative Neurotechnologies (BRAIN) initiative funded by public agencies, private companies, and foundations. The express aim of this initiative is to develop new tools and technologies that will enable the research community to obtain a dynamic picture of the brain in action. With nearly 100 billion cells making 100 trillion connections, this is no small aim and will simply not be achievable with current tools and disciplinary approaches. One of the express themes of action for the BRAIN initiative is to "cross boundaries in interdisciplinary collaborations." The BRAIN initiative consists of teams of engineers, nanotechnologists, computational scientists, materials scientists, and neuroscientists to create the next generation of imaging tools or probes for brain activity.

Will convergence become the future of psychiatry?

Arguably, if the brain is so complex as to require a convergent approach, then the study of psychiatry (mind, brain, and behavior) is complexity on a whole different level. Although the challenge is evident, our approaches have been remarkably singular, focusing on the mind, the brain, or behavior in isolation, with too little integration across disciplines and even less assistance from the vast areas of science that are now poised to alter other areas of medicine.

What would a convergence science of mental illness look like?

A convergent approach to mental illness could begin with software engineers, informatics experts, behavioral scientists, and clinicians designing a new generation of devices to provide objective measures to augment patients' reports of their symptoms. Imagine devices developed to provide continuous assessment of mental state, similar to the glucose or heart rate monitors available today. Passive data such as voice analytics, facial expression monitoring, actigraphy, and engagement of social networks could indicate the onset of depression or mania. Closed loop brain stimulation could detect and correct abnormal neural circuit activity, as is used today for managing epilepsy.

Implanted devices for the management of hallucinations and obsessive thoughts may seem like science fiction, but with the engineering of wireless, miniaturized electrodes, a new era is emerging for interventional neurology as well as psychiatry. Envisage creating social prosthetics for children with autism, similar to the cochlear implants used routinely for nerve deafness. While today's tools such as Google Glass and Siri may not be up to the task, rapid progress in facial processing and computer engineering suggests that software could allow children with "social blindness" to read facial emotions in real time.

The application of convergence to psychiatry can be much more than smart software and social prosthetics. Convergence may include combining the current tools for diagnosis. The neurobiological mechanisms of psychiatric disorders are complex and often involve the interplay of changes in brain structure, function, neurochemistry, and neuropathology. Clark and colleagues have outlined the added value of multiple diagnostic modalities used in predicting the risk of transition into first episode psychosis. Karalunas and colleagues were able to refine subtyping of childhood ADHD by using biologically based behavioral dimensions (ie, temperament), a novel classification algorithm (ie, community detection analysis), and multiple external validators (ie, resting-state functional magnetic resonance imaging and cardiac measures of respiratory sinus arrhythmia and pre-ejection period). Diniz and colleagues have shown how plasma biosignatures and brain pathology relate to resistant cognitive impairment in late-life depression. They utilized comprehensive analysis of blood-based immune proteins, magnetic resonance imaging, and positron emission tomography with machine learning data analytics. While all of these examples draw from multiple data sources, the ultimate promise of convergence is the integration of biological, psychological, sociocultural, and environmental data into a more comprehensive, individualized portrayal of diagnosis (ie, precision medicine).

Early examples of convergence science for psychiatric disorders

Convergence science approaches could also aid in the development of novel treatment strategies for psychiatric disorders allowing interventions to be proactive as well as reactive. There is an increasing interest in electronic mental health interventions given enhanced computing power and the widespread availability and use of smartphones. Psychosocial treatments delivered via mobile health (mHealth)—the practice of medicine supported by mobile devices—have the benefit of reach, scalability, affordability, convenience, flexibility, and facilitation by a non-professional workforce. Concerns remain about acceptability to consumers, quality of interaction, and the potential for unsupervised and counterproductive therapy.

Massive open online interventions for mental health is a concept recently defined by Munoz and colleagues and describes mental health and substance abuse interventions, scientifically validated and available online to unlimited numbers of consumers. These researchers showed how a free, multifactorial smoking cessation program in English and Spanish could be used by 7607 participants worldwide. Their findings indicate a 50.3% quit rate over 12 months.

The field of socially assistive robotics in mental health, whereby robots assist patients through social interactions (eg, companionship, as therapeutic partner and/or coach), is receiving greater attention, particularly as computing power grows and artificial intelligence systems become more sophisticated, ubiquitous, and useful. A recent review by Rabbitt and colleagues has outlined potential benefits of socially assistive robotics (eg, providing therapy and monitoring where there are few mental health providers and reinforcing human-led therapy), as well as potential downsides (eg, poor quality user interface leading to frustration and cost considerations). Even in the near term, the development of monitors for the fidelity of psychosocial interventions could improve the quality of care.

Just as a glucose monitor in a contact lens provides continuous feedback for optimizing diabetes control; engineers, computer scientists, and psychiatrists can develop simple devices to assess the quality of psychiatric treatment as a path to improving outcomes. Examples in psychiatry include software and hardware for smartphones and wearable devices to monitor physical activity, sleep, social interactivity, calorimetry, and emotional tone of voice.

Implications of convergence science for psychiatry

If convergence science principles are part of the future of psychiatry, it may not be too soon to introduce this approach in the teaching of psychiatry. Additional skills may include an enhanced understanding of neuroscience, the "omics" (eg, genomics, proteomics, metabolomics), big data analytics, mHealth, economics, and policy. Yager suggests psychiatry training and careers are likely to undergo substantial change in the future to complement projected health burdens and innovations, postulating the development of additional psychiatric specializations in information technology and executive management (ie, combining clinical practice, administration, entrepreneurialism, and health services management).

The training of psychiatrists as clinical neuroscientists has recently been outlined, and indeed there are signs to suggest a resurgent interest in psychiatric neuroscience among medical students given the need for new discoveries and the promise of new research techniques. Interesting educational curricula to model include the National Institute of Mental Health (NIMH)-led, 4-day Brain Camp offered in the US to MD-PhD students; the National Neuroscience Curriculum Initiative; and the NIMH-funded, 5-day Summary Research Institute in Geriatric Mental Health.

Conclusion

The Medici effect is upon us in biomedicine, and it's called convergence science. We believe convergence science and its greater application to psychiatry is important to address the integration of mind, body, and behavior; the urgent need for improved quality and access to clinical care; the high burden of disease; and the promise of new technologies. As the Medici family catalyzed the Renaissance by bringing together poets, philosophers, scientists, and painters and other artisans, we believe a renaissance in psychiatry can be delivered by facilitating interactions, research, and innovations between clinical neuroscientists, molecular biologists, big data scientists, roboticists, imagers, public health experts, economists, and user interface and gamification experts.

Disclosures:

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Assessing ADHD in Preschool Children

E. Mark Mahone, PhD
Psychiatric Times

Do you know these 10 signs that may help you differentiate early signs of ADHD from the "typical" behavior of a 3- to 4-year-old?

Childhood ADHD is a major public health problem with prevalence estimates of over 5 million children in the US alone. Of particular concern is the progressive increase in diagnosis. For example, in 2013, the National Center for Health Statisticsestimated that 9.5% of children in the US aged 4 to 17 years (13.3% of boys, 5.6% of girls) have been diagnosed with ADHD. Moreover, nearly 2% of children aged 3 to 4 years had been diagnosed with ADHD—an almost 4-fold increase from 0.5% in 1997.

The need to identify ADHD earlier

Recently, a leading ADHD researcher, Stephen Hinshaw, warned that the movement toward universal pre-kindergarten might inadvertently lead to an "epidemic" of preschool children wrongly identified as having ADHD. This is largely due to the mismatch between biological readiness and earlier academic demands.

Increased prevalence of ADHD is of great concern because the disorder is associated with extraordinary societal costs. In 2011, for example, it was estimated that the annual incremental cost of illness for ADHD in the US was $143 to $266 billion, driven largely by high rates of psychiatric and learning disorders, injury, and mortality associated with the disorder.

An especially alarming trend is the identification of adverse outcomes among preschool children with ADHD. Even when children with ADHD are identified and treated in the preschool years, symptoms and functional difficulties continue throughout the elementary school years. A recent prospective study from the UK found that adults who were rated as hyperactive at aged 3 years incurred 17.6 times higher annual health care costs than concomitantly studied cohort of adults who had not been identified as hyperactive at aged 3 years.

The costly toll that ADHD takes on individual adjustment, family life, schools, health care, and social services underscores the importance of earlier identification and treatment. Biomarkers of ADHD, including neuroanatomic and functional anomalies, are associated with chronic cognitive and behavioral dysfunction. These brain-based changes develop early in life (even before formal diagnosis) and include delayed brain maturation (delay from to 2 to 5 years) by the time children with ADHD reach middle school age. Researchers are now seeking to identify biomarkers of ADHD as early as possible in order to facilitate improved outcomes. In one of the first

neuroimaging studies of preschoolers with ADHD (aged 4 to 5 years), Mahone and colleagues identified significant reductions in the caudate nucleus among children with ADHD—with greater reductions associated with more severe symptoms of hyperactivity.

Early signs

The emerging research literature now suggests that with careful and thorough assessment, ADHD can be accurately diagnosed in the preschool years. Given these considerations, the following signs are offered as examples of behavioral risk factors for ADHD in children aged 3 to 4 years that may help clinicians and parents differentiate early signs of ADHD from "typical" behavior.

1. Dislikes or avoids activities that require paying attention for more than a minute or two
2. Loses interest and starts doing something else after engaging in an activity for only a few moments
3. Talks a lot more and makes much more noise than other children the same age
4. Climbs on things when not supposed to
5. Can't hop on one foot (one time) by age 4 years
6. Nearly always restless—wants to constantly kick or jiggle feet, or twisting around in seat; "must" get up after being seated for only a few minutes
7. Has gotten into dangerous situations because of fearlessness
8. Warms up too quickly with strangers
9. Consistently aggressive with playmates; has been expelled from preschool or daycare for aggression
10. Has been injured (eg, stitches) because of moving too fast or running when not supposed to

If 2 or more of these symptoms are observed in young children, a referral should be made to a clinician with expertise in diagnosis and treatment of ADHD in the preschool years.

Disclosures:

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ان اقدامات میں تحقیق وترقی کے نجی اداروں میں جو کمپنیاں سرمایہ کاری کر رہی ہیں ان کو ٹیکس مراعات دینا، اہم ٹیکنالوجی کی تربیت کے لیے ملازمین کو بیرون ممالک بھیجنے کے لیے حکومتی امداد فراہم کرنا، نجی صنعتوں کی لیبارٹریوں کو مالی امداد کے ذریعے مضبوط کرنا۔ اعلیٰ ٹیکنالوجی کے شعبوں میں کاروبار کے لیے قائم ہونیوالی نئی کاروباری کمپنیوں کو لمبے عرصے کی ٹیکس چھوٹ دینا وغیرہ شامل ہیں۔ مثال کے طور پر 2001 میں میں نے بحیثیت وفاقی وزیر برائے سائنس و ٹیکنالوجی، تمام IT کی صنعتوں کو 15 سال کی ٹیکس چھوٹ دی تھی۔ نتیجتا IT کی معیاری تعلیم کے اداروں کے قیام کے لیے وسیع پیمانے پر سرمایہ کاری کی گئی تھی کہ سرکاری انجینئرنگ جامعات میں حکومت کی جانب سے اسی کروڑ روپے کی مالی امداد دی اور (endowments) قائم کئے گئے تاکہ انفامیشن ٹیکنالوجی کے شعبوں میں تحقیق وترقی میں تیزی سے اضافہ کیا جاسکے۔ اگلے ۱۵ سال میں ایک انقلاب برپا ہو گیا جس کا ثبوت یہ ہے کہ امریکہ کے مشہور اخبار نیویارک ٹائمز نے 10 اگست 2015 کو یہ خبر شائع کی ہے کہ پاکستان کے شعبہ IT کی سالانہ عالمی فروخت 2.8 ارب ڈالر تک پہنچ گئی ہے (2001ء میں یہ صرف 30 لاکھ ڈالر تھی)۔ اس خبر میں یہ بھی کہا گیا کہ پاکستان میں اب سالانہ دس ہزار IT کے ڈگری یافتگان فارغ التحصیل ہو رہے ہیں اور ان میں سے بہت سے فری لانس پروگرام کے طور پر کام کر رہے ہیں اور ملک کی سافٹ وئیر برآمدات 85 امریکی ڈالر سالانہ تک پہنچ گئی ہیں۔ اس طرح پاکستان کا شمار دنیا میں اس میدان میں تیسرے نمبر پر آگیا ہے (صرف امریکہ اور بھارت کے بعد)

سائنس پر سرمایہ کاری کے بہت دورس اثرات مرتب ہوتے ہیں۔ اس سے وابستہ دو مثالیں ہیں پہلی یہ کہ ایک چھوٹے سے ملک فن لینڈ جس کی آبادی صرف 55 لاکھ افراد پر مشتمل ہے (کراچی کی آبادی کا تقریبا ایک چوتھائی) اس کی ایک موبائل بنانے والی کمپنی کی برآمدات 2010 میں 50 ارب امریکی ڈالر تک پہنچ گئی تھیں یعنی پاکستان کی برآمدات سے دگنی تھیں۔ دوسری مثال سنگاپور کی ہے جس کی آبادی بھی لگ بھگ فن لینڈ کے برابر ہے لیکن اس کی برآمدات پاکستان کی برآمدات سے 20 گنا زیادہ ہیں۔ وجہ صرف اور صرف یہ ہے پاکستان کی حکومتوں میں تکنیکی سوجھ بوجھ کی صلاحیت، دور اندیشی اور بصارت کا شدید فقدان پایا جاتا رہا ہے۔

میں نے پچھلے ایک مضمون میں صدارتی نظام جمہوریت کے حق میں لکھا تھا جس پر جناب وجاہت مسعود صاحب نے اپنے 17 اکتوبر کے مضمون میں تنقید کی ہے۔ میں سب سے پہلے جناب وجاہت مسعود کی اس غلط فہمی کو دور کرنا چاہتا ہوں کہ جس صدارتی نظام جمہوریت کی میں تائید کر رہا ہوں یہ وہ نظام حکومت نہیں ہے جو پاکستان میں مختلف مارشل لاء آنے کے بعد رائج رہا ہے وہ آمریت تھی جمہوریت نہیں۔

لیے مالی ادارے نئے کاروبار کے لیے مالی معاونت اور تحقیق و ترقی کے نجی شعبوں کے لیے ساز گار ماحول کی فراہمی کی ضرورت ہوتی ہے جو کہ پاکستان میں ندارد ہے۔ بھارت کا سائنسی ترقیاتی بجٹ 200 ارب روپے ہے۔ آپ کو یقین نہیں آئے گا کہ پاکستان کا سائنسی ترقیاتی بجٹ اب صرف ایک ارب روپے رہ گیا ہے (2002 میں جس وقت میں وفاقی وزیر برائے سائنس و ٹیکنالوجی تھا یہ بجٹ ۶ ارب روپے تھا جسے کم کر دیا گیا ہے۔)

صنعتی اعتبار سے جدت طرازی اور ٹیکنالوجی میں ترقی انفرادی طور پر کمپنیوں کی سطح پر اور قومی پالیسی کی سطح دونوں پر اہم کردار ادا کرتی ہے۔ انفرادی کمپنیوں کی سطح پر یہ نہایت ضروری ہے کہ اعلیٰ تربیت یافتہ تکنیکی ماہرین اور انجینئر موجود ہوں جو کہ صنعتوں میں استعمال ہونے والی جدید مشینری کو استعمال اور برقرار رکھنے میں مہارت کے حامل ہوں۔ قومی پالیسی کی سطح پر ٹیکنالوجی کے میدان مین مقامی ترقی کے حصول کے لیے ٹیکنالوجی کی منتقلی کو غیر ملکی براہ راست سرمایہ کاری سے منسلک کیا جانا ضروری ہے کیونکہ کسی بھی پروجیکٹ کی منظوری کے لیے پلاننگ ڈویژن کی یہ اہم شرط ہونی چاہیے۔ بھارت نے یہ کئی دہائیوں پہلے ہی طے کر لیا تھا کہ غیر ممالک سے آنکھیں بند کر کے ٹیکنالوجی حاصل نہیں کریں گے بلکہ ہر پروجیکٹ میں اس کی منتقلی لازمی بنائیں گے اور ہمیں بھی اب یہ فوری طور پر کرنا چاہیے۔ جدت طرازی کے ماحول کی تشکیل کے لیے بہت سے مختلف پہلوئوں کو ہم آہنگ کرنا ہوتا ہے جبکہ پاکستان میں ان سب کا فقدان پایا جاتا ہے۔ ان میں سب سے اولین اہمیت کی حامل نرسری، پرائمری، ثانوی اسکولوں اور جامعات میں معیاری تعلیم کا انعقاد ہے۔ ایک نئے تعلیمی نظام کے ذریعے ہمیں طلبہ میں مسائل کو حل کرنے کی قابلیت ورجحان کی حوصلہ افزائی کرنی چاہئے بجائے اس کے کہ ایک ہی نقطہ نظر کو رٹوایا جائے جیسا کہ موجودہ تعلیمی نظام میں ہو رہا ہے۔ اسکول اور کالج کے اساتذہ کا اعلیٰ و معیاری تعلیم یافتہ ہونا ضروری ہے کیونکہ شخصیت کی بنیاد چھوٹی عمر ہی میں ڈالی جاتی ہے سنگاپور کی کامیابی کی ایک اہم وجہ یہ بھی ہے کہ وہاں اسکول کے اساتذہ کی تنخواہوں میں بے انتہا اضافہ کر دیا گیا تھا لہذا زیادہ سے زیادہ تعلیم یافتہ افراد اسکولوں مین بحیثیت استاد تعلیم دینے کی طرف متوجہ ہوئے۔ نہایت افسوس کی بات ہے کہ پاکستان کا شمار دنیا مین تعلیم پر سب سے کم خرچ کرنے والے ممالک میں نویں نمبر پر ہے۔ دوسرا اہم عنصر سائنسی تحقیق و ترقی پر سرمایہ کاری ہے۔ اعلیٰ تعلیمی کمیشن کے قیام کے بعد 2007 میں یہ رقم پاکستان کی GDP کے 0.63٪ تک پہنچ گئی تھی لیکن بعد میں یہ سرمایہ کاری کم ہو کر 0.3٪ ہی رہ گئی ہے جو کہ ہماری حکومتوں کی ناقص پالیسیوں کا منہ بولتا ثبوت ہے۔ تحقیق و ترقی پر سرمایہ کاری اور سماجی و اقتصادی ترقی کا آپس میں براہ راست تعلق ہے۔ چین، کوریا اور سنگاپور کی مثالیں ہمارے سامنے ہیں۔ تیسرا اہم عنصر جدت طرازی ہے جس کے لیے ایک مضبوط ماحولیاتی نظام ترتیب دینے کی ضرورت ہے اس کے لیے ایسی حکومتی پالیسیاں ترتیب دی جائیں جو کہ تحقیق و ترقی کے نجی شعبوں کو فروغ دیں۔ تمام ترقی یافتہ ممالک میں بیشتر تحقیق نجی شعبوں ہی میں ہوتی ہے اس کے لیے مناسب حکومتی پالیسیوں پر حکمت عملی اور نجی شعبے میں تحقیق و ترقی کے فروغ کے لیے اٹھائے گئے اقدامات کا بہت اہم کردار ہے۔

سائنس و ٹیکنالوجی اور جدت طرازی کی اہمیت

ڈاکٹر عطاء الرحمن

دور حاضر میں سائنس و ٹیکنالوجی اور جدت طرازی کسی بھی ملک کی سماجی و اقتصادی ترقی کے اہم عناصر ہیں۔ دنیا دو اقسام کے ممالک میں بٹ گئی ہے ایک وہ ممالک ہیں جو معیاری تعلیم، سائنس و ٹیکنالوجی کے حصول کے لیے وسیع پیمانے پر سرمایہ کار کر رہے ہیں اور جدت طرازی کے فروغ کے لیے موثر اقدامات کر رہے ہیں اور دوسرے وہ غریب ممالک ہیں جو نچلے درجے کی برآمدات پر بھروسہ کر رہے ہیں ان کی اقتصادیات کا انحصار بھی کم درجے کی زرعی برآمدات پر ہے۔ پاکستان کا شمار ان دوسرے قسم کے ممالک میں ہوتا ہے۔ اگر ہم کاروبار میں اضافہ اور جدت طرازی چاہتے ہیں تو ہمیں جدید ٹیکنالوجی اپنانا ہوگی۔ اس کے لیے یا تو ہم خود ٹیکنالوجی کو فروغ دیں یا پھر باہر سے ٹیکنالوجی منتقل کریں۔ بھارت میں ان دونوں طریقوں پر عمل کیا جارہا ہے۔ بھارت کا حالیہ فیصلہ کہ وہ نئے طیارے مغربی ممالک سے خریدنے کے بجائے اپنی صلاحیتوں کو بروئے کار لا کر خود بنائے گا، مہاتما گاندھی کی پالیسی کی عکاسی کرتا ہے۔ جن کی بنائی گئی خود انحصاری کی پالیسی پر بھارت کی حکومتیں عمل کرتی چلی آرہی ہیں۔ بھارت کی سب سے بڑی گاڑیاں بنانے کی کمپنی 1945 میں قائم ہوئی اس نے جدید ٹیکنالوجی اپنانے کا فیصلہ کیا اور اسکی پہلی گاڑی 1954ء میں تجارتی بنیادوں پر دستیاب ہوئی جو کہ جرمنی کی ایک بڑی گاڑیاں بنانے والی کمپنی کے اشتراک سے وجود میں آئی۔ 2008 میں اس نے دنیا کی سب سے سستی ترین گاڑی بنا کر دنیا کو حیران کر دیا جس کی قیمت ایک لاکھ روپے تھی۔ پاکستان میں اس طرح جدید اور اعلیٰ ٹیکنالوجی کی مصنوعات سازی کے شعبے کی جانب سے ایسی بصارت، ایسا لائحہ عمل کہیں نظر نہیں آتا۔ اسکی اہم وجہ یہ ہے کہ یکے بعد دیگرے نااہل حکومتیں قائم ہوئیں جنہوں نے کبھی بھی علم پر مبنی معیشت کی اہمیت اور اس کے دوررس نتائج کے بارے میں سوچا ہی نہیں۔

اگر ہم دنیا کے ساتھ قدم سے قدم ملا کر آگے بڑھنا چاہتے ہیں تو آسان راستہ ٹیکنالوجی کی منتقلی کا ہے۔ ٹیکنالوجی کی منتقلی کے لیے ہمیں نہایت اعلیٰ ہنر مند صلاحیتوں کے حامل کارکنوں کی ضرورت ہے۔ پاکستان ایسے افراد نہیں فراہم کر سکتا کیونکہ یہاں ایک ہزار سے زائد تکنیکی تربیت کے مراکز موجو ہونے کے باوجود انکا معیار نہایت پست ہے۔ ہمیں چاہیے کہ ان مرکز کو بین الاقوامی اداروں سے منسلک کروائیں تاکہ انکا معیار بھی عالمی معیار سے ہم آہنگ ہو سکے۔ ٹیکنالوجی منتقلی سے زیادہ مشکل اور محنت طلب کام نئی ٹیکنالوجی کی دریافت ہے۔ اس کے لیے اعلیٰ و معیاری جامعات کی تشکیل تحقیق و ترقی کے اداروں کا قیام، ٹیکنالوجی پارکوں کا قیام اور ان کے ذریعے نئے نظریات کو مصنوعات میں منتقل کرنے میں حکومتی معاونت، کاروبار کو پنپنے کے

فرانس میں مساجد کی تعداد چرچوں سے زیادہ ہے۔ ویسے برطانیہ کی حالت اس سے بھی زیادہ "خطرناک" ہے۔ وہاں تو مسلمانوں کی تعداد 30 گنا تیز رفتاری سے بڑھ رہی ہے۔ گزشتہ 30 برس میں تقریبا ایک لاکھ مسلمانوں کی آبادی ڈھائی لاکھ سے زیادہ ہو چکی ہے۔ اور ایک ہزار سے زیادہ مساجد ہیں جن میں سے بہت سی مساجد چرچ خرید کر بنائی گئی ہیں۔

اس دوسرے خطرے کے علاوہ ایک اور چیز جس نے مغرب کو پریشان کر رکھا ہے خصوصا فرانس جہاں شارلی ہیبڈو جیسا واقعہ پیش آیا اس کے بعد سے فرانس میں قبول اسلام کی شرح میں تیزی سے اضافہ ہو گیا ہے۔ اعداد و شمار سروے اور جائزوں پر انحصار کرنے والوں کی عقل اس معاملے میں خبط ہو گئی کہ 9/11 کے بعد سب سے زیادہ امریکیوں نے اسلام قبول کیا سب سے زیادہ گورے اور ان میں سب سے زیادہ خواتین تھیں یہی معاملہ شارلی ہیبڈو کا ہوا۔ جس مقصد کے لیے یہ کیا گیا تھا وہ تو حاصل نہیں ہوا یعنی مسلمانوں کو بدنام کرنا بلکہ قبول اسلام میں اضافہ ہو گیا۔ اب نیا حربہ آزمایا گیا ہے۔ دیکھیں اس کے اثرات کس قدر تیزی سے سامنے آتے ہیں۔ فرانسیسی مساجد کے ائمہ بتاتے ہیں کہ ہمارے پاس قبول اسلام کے لیے آنے والوں کی تعداد اور بڑھ رہی ہے۔ ایک فرانسیسی ریڈیو آر ٹی ایل کے مطابق گزشتہ سال پیرس کی مرکزی مسجد کی انتظامیہ نے 22 افراد کے قبول اسلام کی سند جاری کی تھی لیکن اس سال یہ تعداد 40 ہو چکی ہے۔ ایک نو مسلم نوجوان نے کہا کہ میں اس لیے مسلمان ہوا ہوں کہ دنیا کو بتا سکوں کہ اسلام کا چہرہ وہ نہیں جو بتایا جا رہا ہے۔ اسٹراسبورک اور آبرویلئیرز مین بھی قبول اسلام کی شرح میں 30 فیصد اضافہ ہوا ہے۔ لیون میں قبول اسلام کی شرح میں 20 فیصد اضافہ ہوا ہے۔ مغرب کے لیے خطرے کا باعث صرف قبول اسلام اور مساجد کی تعداد مین اضافہ نہیں ہے بلکہ جو لوگ اسلام قبول کر رہے ہیں وہ پڑھے لکھے اور معاشرے کے اہم لوگ ہیں۔ جن میں ڈاکٹر، اسکول ہیڈ ٹیچر اور پولیس افسران شامل ہیں۔ اس طرح شارلی ہیبڈو کے واقعے کے چند ہی روز میں ایک بزنس ڈائریکٹر ازابیلڈیٹک نے فیس بک پر قبول اسلام کا اعلان کر دیا تھا۔

اب ذرا 2007 کے ایک سروے کی رپورٹ بھی ملاحظہ فرمائیں جس سے پتا چل جائے گا کہ تارکین وطن کی آمد پر یورپ کو خوف کیوں پیدا ہوا۔ شرح پیدائش کے حوالے سے بھی رپورٹ ہے کہ فرانس میں 1.8 بچے فی خاندان ہے۔ برطانیہ میں 1.6 فیصد، یونان میں 1.3، جرمنی میں 1.3 اٹلی میں 1.2 اسپین میں 1.1 اور مجموعی طور پر پورے یورپی یونین کے 31 ممالک میں شرح پیدائش 1.38 بچے فی خاندان ہے۔ یہ صورت حال یورپی تعصب رکھنے والوں کے دماغوں کو کس قدر جھنجھوڑ رہی ہو گی اس کا اندازہ لگایا جا سکتا ہے۔ دوسرا پہلو یہ ہے کہ تاریخ کا جائزہ لے لیں تو اندازہ ہو گا کہ اسلحے اور طاقت کے استعمال کے بغیر دنیا میں اسلام کے قدم بڑھ رہے ہیں اور جتنا اس کے خلاف سازشیں ہوں گی یہ اتنا ہی پھیلے گا۔ آج، برقعے، ڈاڑھی، مساجد، قرآن سے شغف، حفاظ کی تعداد، ہر چیز میں اضافہ ہوا ہے۔ یہ کسی حکومت نے نہیں کی یہ صرف اللہ کی رحمت سے ہوا ہے۔

سانحہ پیرس چند پس پردہ حقائق

مظفر اعجاز

یہ سوال ہر ایک کی زبان پر ہے کہ اگر پیرس حملے میں مسلمان ملوث نہیں ہیں تو پھر یہ کس نے اور کیوں کرائے ہیں۔ اس دلچسپ اور معروف ترین سوال کا سادہ سا جواب ہے کہ صرف فرانس کے حالات یورپ کے حالات اور وہاں مسلمانون کی بڑھتی ہوئی آبادی اور اثر و نفوذ پر غور کر لیا جائے سب پتا چل جائے گا۔ 9/11 کے اسباب میں سے بہت سے ایسے ہی تھے کہ امریکا میں مسلمانوں کی تعداد بڑھ رہی تھی۔ مساجد میں اضافہ ہو رہا تھا۔ لیکن انہیں مسلمانوں اور مساجد کی تعداد مین اضافے سے کوئی خطرہ نہیں تھا۔ بلکہ خطرہ اس بات سے پیدا ہونے لگا تھا کہ سفید فام اور امریکی خواتین کی بڑی تعداد اسلام کی طرف راغب ہو رہی تھی۔ اب فرانس کا جائزہ لے لیں۔ یورپ کا ایک خاص مزاج ہے ہر کام سروے کی روشنی میں کرتے ہیں جدید تحقیق کی روشنی میں انڈے کھاتے ہیں، مرغی کھاتے ہیں، برڈ فلو کی خبر آجائے تو انڈہ، مرغی کھانا چھوڑ دیتے ہیں۔ میڈ کائو کی بیماری کے بعد تو پورے یورپ نے گائے کا گوشت کھانا چھوڑ دیا تھا۔ بھیڑوں میں بلکہ ان کے کھروں میں بیماری کی خبر سے لاکھوں بھیڑوں کو ضائع کروادیا کیوں کہ سروے اور جدید تحقیق آگئی تھی۔ اب اس تحقیق نے انہیں بتایا کہ اگر آپ اپنی ثقافت کو 25 سال سے زیادہ محفوظ رکھنا چاہتے ہیں تو آپ کے خاندانوں میں 2.11 بچے فی خاندان شرح پیدائش ہونی چاہیے جب کہ پورے مغرب میں اوسطا شرح پیدائش 1.65 بچے فی خاندان ہے۔ اور مسلمانوں میں یہ شرح ۳ فی صد فی خاندان سے زیادہ ہے۔ سروے اور تحقیق یہی بتاتی ہے کہ اگر 2.11 بچے فی خاندان کی شرح برقرار رکھی گئی تو یہ برعکس سفر شروع کر دیں گے اور یہ سفر 1.9 بچے فی خاندان سے شروع ہو جاتا ہے جب کہ مغرب میں 1.65 بچے کی شرح ہے اور اس صورت میں واپسی کا سفر 80 سے 100 برس میں مکمل ہو گا اور شرح پیدائش 1.3 یا اس سے کم ہو گئی تو واپسی ناممکن ہے۔ یہ تو ایک سبب مسلمانون سے خوف زدہ ہونے کا۔ اعداد شمار اور سروے کے مطابق صرف چند برس بعد پوری ثقافت معدوم ہو جائے گی اور صرف شرح پیدائش سے ہی مسلمانوں کا عددی غلبہ ہو جائے گا۔

دوسری بڑی خطرے کی بات یہ ہوئی کہ یورپ میں 1990 کے بعد سے سب سے زیادہ تارکین وطن مسلمان ممالک سے آئے ہیں۔ اس طرح فرانس میں اس وقت 20 سال یا اس سے کم کے مسلمان نوجوان اور بچوں کی شرح 30 فی صد ہے جب کہ پیرس اور مارسیلی جیسے بڑے شہروں میں یہ شرح 45 فی صد تک ہے۔ ذرا غور کریں جس معاشرے میں 45 فی صد نوجوان اور بچے مسلمان ہوں وہ اگلے 20 سال میں کہاں کھڑا ہو گا۔ ایک اور افتاد جس کے حوالے سے فرانسیسی وزیر داخلہ نے عندیا دیا کہ وہ پیرس حادثے کی روشنی میں فرانس میں مساجد بند کرنے پر غور کر رہے ہیں وہ یہ ہے کہ جنوبی

FREE WORKSHOP
On
Psychiatric Illness
Date: 27-02-2016

Karachi Psychiatric Hospital held the monthly workshop.

Topic: Epilepsy

02-02-2016

تحریک نفاذ اُردو، شام اُردو صحافی زاہد ملک کی کتاب ''ڈاکٹر عبدالقدیر خان و اسلامی بم'' کی تقریب پذیرائی کے موقع پر ڈاکٹر ظہیر احمد، ڈاکٹر سید مبین اختر، پروفیسر اظہار حیدری، نسیم انجم، علیم احمد، وسیم الدین بھوپالی، پروفیسر شاہین حبیب شرکاء سے مخاطب ہیں۔