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Study of biochemical changes in the human body due to stress and depression at workplace in government sector in India

Pinkey Kumari Singh* Dr. R.K Singh** Dr. J.P Mishra*** Dr. Kumari Swarnim****

**Research Scholar, Dept. of Psychology, K.U Chaibasa,*
***Dept. of Chemistry BIT Sindri Dhanbad,*
****Vice chancellor, Ramchandra Chandravansi University, Palamu,*
***** Assistant Professor, department of zoology, Ranchi Women's College, Ranchi.*

I. Introduction

The word stress implies an experience of negative emotions that comes in the wake of anticipated physiological, biochemical, cognitive, and behavioral changes that work towards either changing the stressor or adjusting its effects. Stress has been defined as "the pattern of physiological reactions that prepares an organism for action.At some point, nearly everyone encounters stressful life events: the death of a loved one, the loss of a job, an illness, or a relationship downward.When genetic, biology, and stressful situations come together, depression can result. Stress has its own physiological consequences. It triggers a chain of chemical reactions and responses in the body. If the stress is short-lived, the body usually returns to normal. But when stress is chronic or the system gets overdrive, changes in the body and can be long-lasting.

It is often said that depression result from a chemical imbalance. Research suggests that depression doesn't spring from simply having too much or too little of certain brain chemicals. Rather, there are many possible causes of depression, including faulty mood regulation by brain, genetic vulnerability, stressful life events, medications, and medical problems. It's believed that several of these forces interact to bring on depression.

To be sure, chemicals are involved in this process, but it is not a simple matter of one chemical being too low and another too high. Rather, many chemicals are involved, working both insides and outside nerve cells. There are million, even billions, of chemical reactions that make up the dynamic system that is responsible for your mood, stress, perceptions, and how you experience life.

Biochemical responses to stress:

It is only during the past ten years that to talk of stress as a primary cause of disease has become even remotely respectable in medical circles. Early this century Cannon (1929) pioneered the study of the adrenomedullary response to stress, and attribute most of the effect he observed to 'adenine'. This way a crude extract of adrenal tissue containing mainly adrenaline and a smaller proportion of noradrenaline, although with greater equality in carnivores that herbivores being related to several diseases, including hypertension.

Selye (1971) was concerned with the adreno-cortical response to acute stress, especially physical trauma, although he later extended his work to the influence of long-term events on the development of cardiovascular disease. It was only when Dr. Lennant Levi (1971) and his co-workers started to put together the large amount

of diverse information available from catecholamines, but no consist rise in either lipid or glucose levels because of increased use of both these metabolic fuels (Taggart et al. 1972). The apparent increases in the pressures of urban living (Carruthers 1974) the influence of lifestyle on liability to develop heart disease (Friedman & Roseman 1959) and the availability of stress-blocking drugs, all combined to accelerate the growth of interest in this field.

The pre-dominant response observed, was an increase in noradrenaline secretion, which resulted in a rise in blood pressure and plasma lipid levels. Aversion, induced by watching violent films, caused a vagotonic reaction with slowing and increased adrenaline secretion (Carruthers & Taggart 1973).

Scientists found that the earliest response to stress happen in the brain within seconds of perceiving a 'stressor'. Chemicals which signal between nerve cells (neurotransmitters) are released. These include serotonin and adrenaline. Following this, stress hormones are released, which particularly affect areas of the brain key for memory and regulating emotions. Repeated stress changes how well these systems can control the stress response.

Researchers are also investigating these systems are involved in anxiety and depression, suggesting a biochemical link between stress and mental illness. Recent studies have shown that long-term stress can change the structure of the brain, especially in areas supporting learning and memory. It can affect both nerve cells (gray matter) and the connections between them (white matter). It is possible these changes, along with other factors, can increase the likelihood of developing mental illness.

For a long time, researchers suggested that hormones have receptors just in the peripheral tissues and do not gain access to the central nervous system (CNS) (Lupien and Lepage, 2001). However, observations have demonstrated the effect of anti-inflammatory drugs (which are considered synthetic hormones) on behavioral and cognitive disorders and the phenomenon called "Steroid psychosis" (Clark et al., 1952).

Hormonal involvement:

In biology terms, stress has been defined by various physiologic changes including activation of the pituitary adrenal axis, which leads to the liberation of adrenal steroids triggered by the release of adrenocorticotrophic hormones (ACTH) from the pituitary. This ACTH stimulation is controlled by the corticotropin releasing factor (CRF) present in the hypothalamus and released in response to various stressors. This over-activation can produce the psychopathology of anxiety such as disorders, depression and even damage to body organs in chronic several cases.

Neurotransmitters and Stress:

Gamma-aminobutyric acid

It is an important inhibitory neurotransmitter in the central nervous system. The role of GABA and benzodiazepine receptors has been well documented in stress disorders such as anxiety, epilepsy, insomnia, and convulsive disorder. Stress has been reported to alter the content of the GABA neurotransmission, which suggests the involvement of GABA in stress-induced behavioral and biochemical alterations. Melatonin is a secretory product primarily synthesized in the pineal gland and released into the blood stream and cerebrospinal fluid. Study revealed the involvement of GABAergic mechanism in the hypnotic action of melatonin. Stress induces the release of corticotropin releasing factor (CRF) and GABA from the amygdala and hypothalamus.

Dopamine:

Stress-induced changes in dopamine (DA) levels within terminal areas seem to involve mainly ventral tegmental area projecting cells. Finding from preclinical studies suggest an uneven response of dopamine in different stressful stimuli. Specifically, an acute and controllable/escapable physical stress was seen to cause an enhanced

dopamine efflux in the ventral striatum, whereas chronic and uncontrollable/inescapable exposure to the same stress attenuated dopamine release.

Norepinephrine:

Brain epinephrine serves globally as an alarm system that decreases neurovegetative functions, such as eating and sleeping, and this contributes to accompanying increase in autonomic and neuroendocrine responses to stress, including HP axis activation. Norepinephrine also activates the amygdala, the principal brain locus for fear-related behaviors, and enhances the long-term storage of aversively charged emotional memories in sites such as the hippocampus and striatum. Hence, it is quite evident that both noradrenaline and CRF are involved in behavioral responses to stress.

Serotonin:

Previous reports have suggested that stress affects the activity of central dopaminergic and serotonergic neurons. Interaction between serotonin and CRF have been demonstrated by various studies in different parts of the brain. Studies have proved that significant reduction in serotonin level increases the responsiveness to stress.

Stress and immunological changes:

Stress has been associated with impaired immune function and increased susceptibility to infectious disease. It is now believed that the nervous, endocrine, and immune system are also so intimately connected that they should be regarded as a single network rather than as three separate system. It is widely accepted that psychological stress and psychiatric illness can compromise immune function, and soluble mediators released by immune cells can affect the central nervous system, thus producing alterations in behavior.

How stress affects the body:

1. Stress can be defined as an automatic physical response to any stimulus that requires you to adjust to change. Every real or perceived threat to your body triggers a cascade of stress hormones that produces physical changes. We all know the sensations: your heart pounds, muscles tense, breathing quickness, and beads of sweat appear. This is known as the stress response.

2. The stress response starts with a signal from the part of your brain known as the hypothalamus. The hypothalamus joins the pituitary gland and the adrenal glands to form a trio known as hypothalamic-pituitary-adrenal (HAP) axis, which govern a multitude of hormonal activities in the body and may play a role in depression as well.

3. When a physical or emotional threat looms, the hypothalamus secretes corticotropin-releasing hormone (CRH), which has the job of rousing your body. Hormones are complex chemicals that carry messages to organs or groups of cells throughout the body and trigger certain responses. CHR follows a pathway to your pituitary gland, where it stimulates the secretion of adrenocorticotropic hormone (ACTH), which pulses into your bloodstream. When ACTH reaches your adrenal glands, it prompts the release of cortisol.

4. The boost in cortisol readies your body to fight or free. Your heart beats faster- up to five times as quickly as normal-and your blood pressure rises. Your breath quickens as your body takes in extra oxygen. Sharpened senses, such as sight and hearing, make you more alert.

5. A fast heartbeat and increased heart rate can also be symptoms of high stress levels. One study measure heart rate reactivity in response to stressful and non-stressful events, finding that heart rate was significantly higher during stressful conditions.

6. If the adrenal gland slacks off on cortisol production the result may be obesity, heart disease, or osteoporosis; too much of the hormone can cause women to take in masculine trait like hair growth and muscle development and lead to one of the greatest fears of all for aging men- baldness. High level of cortisol also may kill of brain cells crucial for memory.

Biochemical response to depression

Depression has been linked to problems or imbalances in the brain, specifically with the neurotransmitter serotonin, norepinephrine, and dopamine. It is very difficult to measure the level of neurotransmitter in a person's brain and their activity.

Patients with major depression shows increased cerebral blood flow and metabolism in the amygdala. Activation in the left amygdala persisted after recovery depression. During depression, amygdala activation correlated positively with depression severity and baseline plasma cortisol levels.

The neurotransmitter serotonin is involved in controlling many important bodily functions, including sleep, aggression, eating, sexual behavior and mood. Serotonin is produced by serotonergic neurons. Current research suggests that a decrease in the production of serotonin by these neurons can cause depression in some people, and more specifically, a mood state that can cause some people to feel suicidal. In the 1960s, the "catecholamine hypothesis" was a popular explanation for why people developed depression. This hypothesis suggested that a deficiency of neurotransmitter norepinephrine (also known as noradrenaline) in certain areas of the brain was responsible for creating depressed mood. Researchers has investigated linkages between stress, depression, and norepinephrine. Norepinephrine helps our bodies to recognize and respond to stressful situations. Researchers suggest that people who are vulnerable to depression may have a norepinephrinergic system that doesn't handle the effects of stress very efficiently. The neurotransmitter dopamine is also linked to depression. Dopamine plays an important role in controlling our drive to seek out rewards, as well as our ability to obtain a sense of pleasure. Low dopamine levels may, in part, explain why people with depression don't get the same sense of pleasure out of activities or people that they did before becoming depressed.

In addition, new studies are showing that other neurotransmitters such as acetylcholine, glutamate, and Gamma aminobutyric acid (GABA) can also play a role in depressive disorders. More research is necessary to understand their role in depression's biochemistry.

Neurohormones:

A neurohormone is a hormone produced by cells in the brain and released into the blood stream. Most of the research on the role of neurohormones in depression has focused on the functioning of the hypothalamic-pituitary- adrenal (HPA) axis. The HPA axis has sometimes been referred to as the body's stress axis. It involves a complex set of interactions between the hypothalamus (H), pituitary (P), and adrenal (A), glands, and plays a key role in several bodily process (e.g., digestion and immune system function), as well as the regulation of mood and emotions, and the physiological response to threat and stress.

In a stressful encounter, the HPA axis operates in three stages:

1. The first stage involves the release of Corticotropin Releasing Hormones (CRH) and Vasopressin in the hypothalamus following the presence of a stressor.
2. The release of these hormones triggers the second stage of the process- the stimulation of a part of the pituitary gland, which consequently, secretes adrenocorticotropic hormone (ACTH) into the blood stream.
3. In the third stage, ACTH, travelling in the blood stream, stimulates the adrenal glands, which produce glucocorticoid hormones, such as cortisol. Cortisol is often referred to as the stress hormones. Cortisol is released into the large muscle groups, which are important for short-term threat response and survival.

Stress and the Brain Function

For a long time, researchers suggested that hormones have receptors just in the peripheral tissues and do not gain access to the central nervous system (CNS) (Lupien and Lepage, 2001). The hippocampus area has both types of receptors, while other points of the brain have only glucocorticoid steroid receptors (de Kloet et al., 1999 [23]). The effects of stress on the nervous system have been investigated for 50 years (Thierry et al, 1968[115]). Some studies have shown that stress has many effects on the human nervous system and can structurally changes in different parts of the brain (Lupin et al., 2009 [65]). Chronic stress can lead to atrophy of the brain mass and decrease its weight (Sarahian et al., 2014[100]). However, it is now obvious that stress can cause structural changes in the brain with long-term effects on the nervous system (Reznikov et al., 2007[89]).

Stress and Memory

High concentration of stress hormones can cause declarative memory disorders (Lupien and Lepage, 2001[63]. Memory is one of the important functional aspects of the CNS and it is categorized as sensory, short-term, and long term. Short-term memory is dependent on the function of the frontal and partial lobes, while long-term memory depends on the function of large areas of the brain (Wood et al., 2000[121]). Various studies have shown that stress can cause functional and structural changes in the hippocampus section of the brain (McEwen, 1999[72]).These structural changes include atrophy and neurogenesis disorders (Lupien and Lepage, 2001[63]). Also, chronic stress and, consequently, an increase in plasma cortisol, leads to a reduction in the number of dendritic branches (Woolley et al., 1990[122]) and the number of neurons (Sapolsky et al., 1990[99]), as well as structural changes in synaptic terminals (Sapolsky et al., 1990[99]) and decreased neurogenesis in the hippocampus tissue (Gould et al., 1998[35]). Additionally, people with either Cushing's syndrome (with an increased secretion of glucocorticosteroids), or people who receive high dosages of exogenous synthetic anti-inflammatory drugs, are observed to have atrophy of the hippocampus and associated memory disorders (Ling et al., 1981[61]).Stress also has negative effects on learning process during stress. In summary, the adverse effects of stress on cognition are diverse and depend on the type, timing, intensity, and the duration (Sandi, 2013[95]). Importantly, it should be emphasized that different people may exhibit varied responses in cognition when exposed to the very stressful stimulus (Hatef et al., 2015 [39]).

Brain and neuroendocrine functioning and depression:

Brain and depression:

Depression is essentially a disorder of negative perceptions (cognitions) and emotions, typically in response to stressors that are interpreted as loss and inadequacy of the sense of self, but also negatively affecting thoughts about others, the world, and the future. It is in the brain, which is extensively governed and shaped by thousands of genes, and by experience and development, that scientist have looked at processes that control perception, emotional reactions, stress perception and coping with stressful situations. We focus mostly on neuroendocrine functioning in responses to perceived stress, and aspects of brain structure and function particularly relevant to depressive emotional states.

Neuroendocrine functioning (hypothalamic-pituitary-adrenal axis) and depression:

Stressful life events precede and presumed to precipitate a depressive episode in the great majority of cases of depression. Depression is associated with several abnormalities of the HPA axis associate with high levels of cortisol. Settler and Miller (2011) analyzed several hundred studies comparing depressed and nondepressed patients on indicators of various HPA axis hormones and found significantly higher levels of cortisol among depressed patients. Testing the hypothesis of abnormal HPA axis involvement in depression, individuals' early studies demonstrated that administration of a synthetic form of cortisol (dexamethasone) elicited different patterns of response in depressed patients and healthy controls. Healthy individuals typically display a

temporary of real cortisol but return to normal levels over a period of hours. However, patients displayed greater resistance to cortisol suppression and earlier 'escape' from suppression, consistent with abnormal levels and diminished homeostatic processes.

Neural aspects of depression:

Although earlier biological research on depression focused extensively on neurotransmitter systems, development of modern imaging technique, along with basic experimental animal research, has revolutionized research on understanding both normal and abnormal structural and functional properties of the brain. With respect to depression, much of the emphasis has been neural areas and functions that support perceptions and interpretations of the self and environment, and the areas and functions responsible for emotional reactions and emotional regulation. Thus, the topics of great interest in depression involve brain regions and processes that account for biased attention to negative stimuli, heightened emotional reactivity and reduced cognitive control. More specifically, subcortical areas of the brainthat comprise the limbic system (including the amygdala and hippocampus) that regulate emotion, motivation, learning and memory, and their reciprocal connectivity with the prefrontal cortex and related areas, have been particular focus of research on depression. The hippocampus is a small structure located under the cerebral cortex and serves many functions including those related to learning and memory. It is known to be highly susceptible to the damaging effects of excessive stress-related glucocorticoid, resulting in impaired learning and memory.

Neurotransmitters:

Neurotransmitters are chemicals that operate between neurons, facilitating the transmission of a massage or signal from one neuron to another. Neurotransmitters communicate information between neurons, they are partly responsible for how we think, feel, and behave. So, when we feel good-happy, elected or joyful-on a molecular level this corresponds to certain types of neurotransmitters (e.g., endorphins) being released by neuron and acting on others.

Serotonin:

Serotonin, also known as 5- Hydroxy tryptamine or 5- HT for short, is found throughout the body, including the brain and gut. In fact, only a very small percentage of 5-HT receptors are in the brain. It is sometimes referred to as the 'happy transmitter', as it appears to play an important role in feelings of happiness and well-being. However, there are some concerns that this description could be misleading. First, some people have pointed out that use of the term 'happy transmitter' was encouraged by drug companies to sell more of their antidepressants! Second, it has not been clearly established that serotonin levels are necessarily linked to positive emotions and moods. For example, serotonin plays an important role in the regulation of sleep, appetite, and sexual desire which, can all be affected when people get depressed.

Scientists have suggested severalways that serotonin may be associated with depression, including:

- The low production of serotonin in brain cells (potentially linked to a lack of tryptophan, a chemical involved in the making of serotonin).
- A reduction in the amount of serotonin reaching the receptors sites or cells.
- A lack of serotonin receptor sites themselves.

Noradrenaline (Norepinephrine):

Noradrenaline plays a crucial role in the body and brain's ability to become activated for action, and therefore, are associated with the brain's response to stress.Norepinephrine is depleted within the brain; it results in the returns of depressive symptoms. In the brain, norepinephrine increases arousal and alertness, promotes vigilance, enhances information and retrieval of memory, and focuses attention; it also increases restlessness

and anxiety. In the rest of the body, norepinephrine increases heart rate and blood pressures, triggers the release of glucose from energy stores, increase blood flow to skeletal muscle, reduces blood flows to the gastrointestinal system, and inhibits voiding of the bladder and gastrointestinal motility.

Dopamine:

Increasingly, scientists are looking at the potential role of dopamine in depression, and in particular, its link anhedonia. Short-term release of dopamine is generally associated with the experience of positive emotions and pleasurable feelings, such as happiness, joy, and elation. Considerable research has previously highlighted the important role of dopamine in pleasure, reward, and addictions. Dopamine could also be important in major depressive disorder. People with depression often exhibit reduced motivation, anhedonia. Dopamine dysregulation could mean that the brain is producing too little or too much dopamine. Low dopamine deficiency, can be caused by a variety of factors, including conditions such as Parkinson's disease, schizophrenia, and depression. It is also possible to have too much dopamine, effects of overly high dopamine levels include high libido, anxiety, difficult sleeping, and stress.

Non-monoamine neurotransmitter (GABA):

Although the monoamines have received the greatest amount of attention in depression, scientists are increasingly looking at the role of GABA (gamma amino butyric acid). GABA has an important role in regulating and fine turning our mood, thinking and behavior. It appears to play an important inhibitory, 'balancing' role on other neurotransmitters- such as the monoamines described above. People with depression have lower level of GABA than non-depressed people, with those more severely or chronically depressed having the lowest levels of GABA.

How depression affects the body:

1. Depression can be triggered by traumatic life events, poor diet, genetic conditions, blood sugar imbalances, medical illness, hormone imbalances, lack of exercise and meditations, drug and alcohol use, and digestive difficulties. These triggers are known to cause or contribute to neurotransmitter imbalances.

2. Normally, a feedback loop allows the body to turn off "fight-or-flight" defenses when the threat passes. In some cases, though, the floodgates never close properly, and cortisol levels arise too often or simply stay high. This can contribute to problems such as high bold pressure, immune suppression, asthma, and possibly depression.

3. Among the best-known culprits are two thyroid hormone imbalances. An excess of thyroid hormone (hyperthyroidism) can trigger manic symptoms. On the other hand, hypothyroidism, a condition in which your body produces too little thyroid hormones, often leads to exhaustion and depression.

4. Heart disease has also been linked to depression, with up to half of heat attacks survivors reporting feeling blue and many having significant depression. Depression can spell trouble for heart patients: it's been linked with slower recovery. Future cardiovascular trouble, and a higher risk of dying within about six months.

5. Increasingly, researchers are recognizing the role of the immune system and inflammatory response in depression. The immune system and inflammatory are one of our body's key protective systems; in the presence of an identified harmful or noxious stimulus (e.g., bacteria, injury)., affected cells 'signal' to the body (via a variety of substance, e.g., cytokines) that there is a problem.

6. Our brains and digestive systems are strongly connected, which is why many of us get stomachaches or nausea when we are stressed or worried. Depression can get you in your gut too causing nausea, indigestion, diarrhea, or constipation.

Effects of stress and depression at workplace in government sector:

There is growing evidence of the global impact of stress and depression. Mental health problems are among the most important contributors to the burden of disorder in Indian government sector. Five of the 10 leading causes of disability in India are mental health problems. There are as relevant in low-income countries as they are in rich ones, cutting across age, gender, and social strata. Furthermore, all predictions indicate that the future will see a dramatic increase in mental health problems. Stress and depressive disorders, for example, represent one of the most common health problem of government sector in India.

The impact of mental health problems in the workplace has serious consequences not only for the individual but also for the government sector in India.

1. **Opportunity barriers:**Face numerous barriers in obtaining equal opportunities – environmental, access, legal, institutional, and attitudinal barriers which cause social exclusion. For people with depression or stress, social exclusion is often the hardest barrier to overcome and is usually associated with feeling of shame, fear, and rejection.

2. **Absenteeism:**

- Increase overall sickness absence, particularly short periods of absence.
- Poor health (depression, stress, burnout).
- Physical conditions (high blood pressure, heart disease, ulcers, sleep disorder, skin rashes, headache, neckache and back neckache, low resistance to infections).

3. **Low work performance:**

- Reduction in productivity and output.
- Increase in error rates.
- Increased number of accidents.
- Deterioration in planning and control of work

4. **Staff attitude and behavior:**
- Loss of motivation and commitment.
- Burnout.
- Staff working increasingly long hours but for diminishing returns.
- Poor timekeeping.
- Labor turnover (particularly expensive for companies at top levels of management).

5. **Relationships at work:**

- Tension and conflict between colleagues.
- Poor relationships with clients.
- Increase in disciplinary problems.

Worker's health is a separate goal. Addressing mental health issues in the workplace means incorporating social responsibility in a firm's everyday practices and routines.

Workplace activities for mental health

1. Health education to raise awareness of factors affecting health and well-being.
2. Screening programs to detect risk factors or early signs of disease.
3. To consider the role of the workplace in promoting good mental health practices for employees.
4. To discuss the importance of work for persons with mental health problems.
5. How to handle an employee who becomes ill with a mental health problem, such as depression.
6. Vocational rehabilitation models/program for persons with long-term mental health problems.

Discussion:

Workers with depression experience cognitive symptoms, such as trouble concentrating, indecisiveness or forgetfulness up to 94% of the time during an episode, reducing their level of performance in the workplace to below the standard expected. A large proportion of the workforce having suffered from depression associate it with concentration difficulties (57%), indecisiveness (44%) and forgetfulness (33%). Employees with depression report on average 5.6 hours per week of lost time, representing a serious of productive capacity. 81% of lost productivity time (LPT) is due to reduced performance while on the job.

The WHO goes on to suggest that the available literature on the impact of treatment for all forms of depression on workers productivity costs suggests that the gain made in reduced absenteeism and improved productivity at work may offset the treatment costs. The organization approach focuses on an assessment of hazardous work conditions and an organizational resource to support worker's mental health. It is very difficult to tell which specific occupational factors contribute to the development and worsening of mood disorders.

Stress reduction programs, such as teaching affective coping skills and relaxation techniques, may help workers to manage work stress better and reduce their risk of depression. We should not wait until workers becomes aged and suffer from chronic problems. Rather, long-term intervention must be started from young adulthood or even adolescence. Significant number of new cases of depression and stress can be attributable to work stress and younger workers with depression are suffering from even more severe absenteeism. Adequate interventions can significantly improve employment outcomes and reduce workplace conflicts in depressed employed persons.Employer's experience expensive consequences of depressionlike- absenteeism, lower productivity, disability to work, accidents and the inappropriate use of work conditions.The promotion and prevention programs will attempt to create a climate that fosters motivation and commitment, reduces obvious stressful and depressiveagents, and promote harmony among co-workers.

Conclusion:

The issue of work and mental health illness has been explored from two different perspectives. The first emphasized mental health problems that may arise in employees who have an employment history. The second addressed the issues of making employment accessible to person who never had a job or have lost it due to serious mental illness. With respect to the impact of mental health problems at work, a major study suggested a

prevalence of 18.2% for any mental health problems. Work impairment is always higher in worker in government sector with comorbid psychiatric disorder (more than one disease at the same time). Further, most individuals with mental health problems do not receive professional help.

This study found that stress and depression symptoms are related to biochemical changes of workers at workplace in government sector in India. Psychological and biochemical factors within and outside of the workplace are important determinants of workplace among the depression and stress. With regards to the role of depression we found that depressed worker had four times the amount of work limitations as controls and 2.5 times the number of absences, these gaps narrowed but never close. Serios mental illness affects approximately 2% of the world's population. It results in persons having much difficulty in filling the role which they have set for themselves in life.

There many factors involved in addressing the importance of work for people with mental health problems, as well as identify effective practices that encourage employment, re-employment, and retention. Social support systems, mental health professionals and employers all have a significant role in helping individuals define options, make choices, learn to manage potentially disabling conditions, and avoid long-term hospitalization. Give the importance of work, and due to advances made in the prevention, treatment and rehabilitation of mental health problems, itseminent sense to address all aspects of the mental well-being of employee.

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