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# A Systematic Review: Depression From The Perspective Of Science And Religion In An Emerging Society

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## Abstract

This aim of this review is to understand the depression from the scientific and religious perspectives and how to manage and overcome it.
Despite the enormous medical and societal importance of depression, the origins and mechanisms underlying depression development are not well understood conceptually. A number of hypotheses have been put out to explain the development of depression, and biochemical, immunological, and physiological research have supported these hypotheses. In addition to the well-known "monoamine," "cytokine," and "stress-induced" (hypothalamus-pituitary-adrenal (HPA) axis and stress theories) depression models, altered brain neural plasticity, neurogenesis, and circadian rhythm desynchronosis (the chronobiological model) phenomena have also been put forth as potential causes of depression. From the religious perspectives. The ancient Greeks believed depression was a result of fluid imbalances of blood, phlegm, yellow and black bile. Meanwhile, early Christianity just threw the blame of the devil for man's suffering, which resulted from their internal battle to fight the temptations of sin. To some depression is an illness or an attitude of Life. Some however, when they are depressed resort to drinking of alcohol or committing suicide, but suicide is even a greater sin.
Depression comes and goes when attacked in the right way. Any crooked way brings punishment both on the patient and the other around him, including the society as a whole.

**Keywords :** depression, scientific, psychiatry, genetic and psychological.

## Introduction

The ancient Greeks believed depression was a result of fluid imbalances of blood, phlegm, yellow and black bile. Meanwhile, early Christianity just threw the blame of the devil for man's suffering, which resulted from their internal battle to fight the temptations of sin. Finally, Renaissance rolled around and scholars, such as Robert Burton, began to recognize the depression as a disease, but since then, attitudes and misconceptions have changed throughout history until we reached modern times when we came to understand there's something far more complicated than a simple chemical imbalance behind this darkening disorder.

## Depression from a scientific perspective.

One of the most prevalent types of psychiatric disease is depression. The World Health Organization estimates that a DD affects roughly 350 million individuals. Worldwide, the prevalence of this ranges from 8 to 12 percent in most nations, with Japan having the lowest frequency at 3 percent and the highest at 16.9 percent [1]. According to predictions, Depression will overtake ischemic heart disease as the second-leading global cause of disability by the year 2020 [2].
DD has a number of detrimental effects that are relevant to medicine and society, and they have a substantial negative impact on people's quality of life and capacity for adaptation. Suicide attempts may result from persistent somatic or neurological

problems combined with long-term, severe depression.

Despite the enormous medical and societal importance of depression, the origins and mechanisms underlying depression development are not well understood conceptually. A number of hypotheses have been put out to explain the development of depression, and biochemical, immunological, and physiological research have supported these hypotheses. In addition to the well-known "monoamine," "cytokine," and "stress-induced" (hypothalamus-pituitary-adrenal (HPA) axis and stress theories) depression models, altered brain neural plasticity, neurogenesis, and circadian rhythm desynchronosis (the chronobiological model) phenomena have also been put forth as potential causes of depression.

There is compelling evidence from family and twin studies that genetic factors contribute to the risk of depression. For instance, a meta-analysis of twin study data reveals that the heritability rate for depression is 37% (95% CI: 31% – 42%). based on family research, first-degree descendants of patients with depression have a two- to three-fold higher risk of developing depression [3]. Additionally, it has been demonstrated that heritability plays a significant role in severe forms of depression [4,5]. Whether DDs are inherited maternally or paternally affects the severity of the illness [6,7].

Numerous research have been conducted all over the world in an effort to identify the genes involved in the development of depression since 1978, when the first study devoted to finding potential candidate genes connected to DDs was published [8]. More than 100 candidate genes have been examined to determine any potential connections between their alleles and the likelihood of depression onset or its symptoms based on the information currently known on the probable neurobiological mechanisms underpinning DDs. The research into DD pathogenesis has produced conflicting outcomes Independent of the current explanations for the pathophysiology of depression, the development of DNA microchip technology has made it possible to undertake genome-wide associations studies (GWASs) to explore for risk factors of depression onset. However, GWASs using sizable datasets, including tens of thousands of patients in meta-analyses and thousands of patients with various kinds of DDs, have been unable to pinpoint any particular loci that are relevant for propensity to DDs. Additionally, the basic mechanisms underlying the pathophysiology of the disease of DDs have not been well described by these research. The fact that the genetic relationships and underlying mechanisms have not been adequately identified suggests that depression is a complex, multifactorial, diverse psychiatric illness. The coordinated activity of numerous genes and their interactions with various environmental factors and each other are thought to play a role in the predisposition to DDs. Additionally, it's possible that each gene alone contributes only a little amount to the pathophysiology of the illness [9].

The main explanations for the emergence of depression are covered in this overview, along with the genetic data that backs them up. The results of GWASs and the potential role of epigenetic variables in the risk of developing DDs are covered in the section's conclusion. As was mentioned above, meta-analyses have linked more than 20 genes to the start of DD. The majority of the time, these connections include genes unrelated to the basic hypotheses of depression ethnopathogenesis. Without any indications of the genetic risk factors for depression, association studies seemed to be linked to the switch to genome-wide methods of association analysis.

Families with members who have had several depressive episodes, a severe duration of the condition, or a young age at clinical onset were examined in the first stage of GWASs with a focus on patients with uncommon monogenic types of depression. Table 1 provides a summary of the findings from these research. These studies have found connections across extensive genomic regions, even those as long as entire chromosomes, and the candidate gene identification appears to be preliminary. The DD candidate genes that were previously identified in these genomic areas were the main basis for this finding.

Using SNP panels of DNA markers, Table 1 maps the loci connected to predisposition to various kinds of depression in family studies.

| Clinical phenotype | Chromosome | Candidate gene |
|---|---|---|
| Recurrent depression with early onset | 15q25.3–26.2 | *NTRK3* (neurotrophin 3 receptor) |
| Recurrent depression, depression-predominant bipolar disorder | 12q23 | NA |
| Depression with early (31 years of age) onset; depression with anxiety | chromosomes 3centr, 7p and 18q | NA |
| Recurrent depression without symptoms of bipolar disorder | 1p36, 12q23.3-q24.11 and 13q31.1-q31.3 | NA |
| Depressive disorder | chromosomes 17 and 8 | *SLC6A4* (solute carrier family 6 member 4) |

Over the past ten years, GWASs have been employed more frequently to pinpoint the genes responsible for complex traits. In this approach, groups of people

who share a particular trait of interest have hundreds of thousands to several million SNPs spread across their whole genome. It is feasible to establish a connection between the allelic variant in a specific region of the genome and the trait under study by analysis of the genotype-phenotype relationships. There is no preliminary hypothesis to explain how polymorphic variants of genes contribute to the emergence of an interest pathology, which is the main distinction between GWASs and candidate gene studies employing the case-control technique. To get statistically significant results, a study must, however Its algorithm calls for extremely large samples of sick and healthy individuals. It can be very challenging to achieve clinical homogeneity in very large samples, particularly when studying psychiatric diseases because there is almost never an instrumental method for evaluating the patient's condition and there is always a subjectivity factor affecting the diagnostic accuracy in the pertinent international classifications.

Numerous research have looked for regions linked to MDD or specific depressive symptoms. Table 2 provides a summary of the findings. This table largely focuses on research that examined depression risk as a disease rather than endophenotypes (such as clinical onset age, intensity of specific symptoms, and patient reactions to medication). The most statistically significant findings from the examined papers are also included in Table 2.

**Table 2** Major depressive disorders (MDDs) and recurring depressive disorders: Genome-wide association studies (RDDs).

| | Clinical Phenotype | chromosome | | Depressive gene | |
|---|---|---|---|---|---|
| | RDD, MDD | rs17077450 | $1.83 \times 10^{-7}$ | Near gene *DSEL* (dermatan sulfate epimerase-like) | The protein encoded by this gene is involved in metabolism of dermatan sulfate and chondroitin sulfate |
| | RDD, MDD | rs12462886<br>rs110634 | $1.73 \times 10^{-6}$<br>$6.78 \times 10^{-7}$ | No<br>*ATP6V1B2* (ATPase H+ Transporting V1 Subunit B2) | Gene desert<br>Encodes a protein that is a noncatalytic subunit of vacuolar ATPase complex VI |
| [28] | MDD | rs545843 | $5.53 \times 10^{-8}$ | *SLC6A15* (solute carrier family 6 member 15) | Encodes a protein that is a potassium-dependent transporter of uncharged amino acids that can play a role in the transport of neuromediator precursors in neurons |
| [29] | MDD | rs1558477<br>rs2522840 | $2.63 \times 10^{-7}$<br>$4.38 \times 10^{-6}$ | *ADCYAP1R1* (ADCYAP receptor type I)<br>*PCLO* (Piccolo Presynaptic Cytomatrix Protein) | The protein encoded by this gene is a receptor for pituitary adenylate cyclase-activating protein 1, which is involved in adenylate cyclase activation<br>The protein encoded by this gene is part of the presynaptic cytoskeletal matrix involved in the formation of active synaptic zones and transport of synaptic vesicles |
| | MDD | rs11579964<br>rs7647854 | $4 \times 10^{-6}$<br>$5 \times 10^{-6}$ | *NVL* (Nuclear VCP-Like)<br>*C3or70* (chromosome 3 open reading frame 70) | Encodes the AAA–ATPase superfamily protein NVL, whose different protein isoforms have been localized to distinct regions of the nucleus and have different functional properties<br>Unknown |
| [30] | DD symptoms | rs8020095<br>rs161645 | $3 \times 10^{-6}$<br>$8 \times 10^{-8}$ | *GPHN* (gephyrin)<br>*NUDT12* (Nudix Hydrolase 12) | Encodes the tubulin-binding protein gephyrin, which is involved in glycine receptor "anchorage" of the cytoskeleton; it is needed for the localization of $GABA_A$ receptors in the postsynaptic membrane.<br>Encodes a protein that regulates the concentration of individual nucleotides according to ambient conditions |
| [31] | MDD | rs8050326<br>rs11152166 | $3 \times 10^{-7}$ | *IRF8* (Interferon Regulatory Factor 8) | Encodes the transcriptional factor of Interferon |

| | | | | | |
|---|---|---|---|---|---|
| | | | $3 \times 10^{-6}$ | *CCBE1*(Collagen And Calcium Binding EGF Domains 1) | Regulatory Factor Family (IRF), that regulates the expression of genes stimulated by type 1 IFNs Encodes a protein that participates in extracellular matrix remodeling |
| | MDD with late onset age | rs7647854 | $5 \times 10^{-11}$ | *C3orf170* (chromosome 3 open reading frame 170) | Unknown |
| [32] | DD | rs10485715 | $8 \times 10^{-9}$ | *BMP2* (Bone Morphogenetic Protein 2) | Encodes a protein that is a secreted TGF-beta superfamily ligand that is important in bone and cartilaginous tissue formation |
| | DD in hepatitis C patients | rs1863918 | $8 \times 10^{-8}$ | *ZNF354C* (Zinc Finger Protein 354C) | Encodes a protein that is a transcriptional factor that binds to 5′-CCACA-3′-type sequences |
| | MDD\symptoms | rs9825823 | $7 \times 10^{-10}$ | *FHIT* (Fragile Histidine Triad) | Encodes a P1-P3-bis(5′-adenosyl) triphosphate hydrolase, an enzyme involved in the metabolism of purines |
| | MDD | rs12552 | $6.1 \times 10^{-19}$ | *OLFM4* (olfactomedin 4) | Encodes a protein that is an antiapoptotic factor that promotes tumor growth; |
| | | rs1432639 | $4.6 \times 10^{-15}$ | *NEGR1* (neuronal growth regulator 1) | Encodes a protein that serve as cell - adhesion molecules and regulate cellular processes as neurite outgrowth and synapse formation |
| | | rs12129573 | $4.0 \times 10^{-12}$ | *LINC01360 (long intergenic non- protein coding RNA 1360)* | Unknown |
| | | chr5_103942055_D | $7.5 \times 10^{-12}$ | Unknown | Unknown |

[26] published the results of the first GWAS using a sizable representative population (1738 DD cases, 1802 controls). No connection with any of the SNPs in this analysis reached the level of genome-wide significance. The rs2715148 gene showed the highest level of significance (p = 7.7 107). Additionally, 10 additional SNPs were found in this genomic area close to the PCLO gene to be significantly linked with DD (p = 105-10-6). They were assigned to the 167 kb sector that contained PCLO [26]. The PCLO protein is a key component of brain monoaminergic neurotransmission and is found in the cytoplasmic matrix of the presynaptic active zone. [30] demonstrated a link between the rs2522833 SNP in PCLO and DD in a population-based study from the Netherlands [30], supporting a potential function for this area in the beginning of depression. [29] discovered a strong statistically significant relationship between the development of DD and the rs2715148 SNP in PCLO in females (P = 5.64 107). Another SNP related with the prevalence of DD in men was discovered by this investigation in the LGSN gene (rs9352774, P = 2.26 104). This gene produces a protein that is linked to GS-I and, to a lesser extent, GS-II glutamine synthetases and is actively expressed in the human crystalline lens. Both the retina and the nervous system's glutamate exchange may be influenced by this protein. In a GWAS conducted by [27], glutamate was discovered to play a role in DD. In a potential regulatory area of HOMER1, which encodes proteins implicated in glutaminergic processes through interaction with the metabotropic glutamate receptors mGluR1 and mGluR5, they discovered a connection between DD and the rs7713917 SNP (P = 5.87 105).

We underline that, due in large part to the genetic makeup of complex variables predisposing to depression, the relationships found in the majority of GWASs did not reach a genome-wide significance threshold. We suggest that SNP markers having a probability value close to the genome-wide threshold level should also be taken into account when adjusting the genome-wide significance level. A genome-wide significance level has been reached in several investigations. In a recessive model assessing the impact of this polymorphism on the risk of DDs, [28] were the first to report a link between DDs and the rs1545843 SNP in SLC6A15 (solute carrier family 6, neutral amino acid transporter, member 15). Variable rs1545843 alleles have been shown to have different levels of SLC6A15 expression in the hippocampus of epileptic patients. This gene encodes the neutral amino acid transporter. The risk allele was shown to be associated with in vivo neuronal integrity, smaller hippocampus volume, and reduced SLC6A15 expression in the hippocampus by the authors, who also provided additional data to substantiate the association's relevance. In addition, Slc6a15 expression was shown to be lower in the hippocampus of mice with increased vulnerability to chronic stress.

[28] revealed a wealth of evidence in favor of the link between DD and SLC6A15. However, later GWAS failed to find any notable relationships with this gene. Only one gene, PCLO, appears to be related with DD in two GWASs, yet the results of GWASs are frequently not replicable.

A meta-analysis of GWAS data was carried out by the Psychiatric Genomics Consortium (PGC). The PGS study gathered and looked at individual genotypic and phenotypic data from patients from several research sites, in contrast to standard meta-analyses, which summarize the statistical data for each constituent analysis studied. The PGS released the findings of its genome-wide comparison analysis of 9519 samples from a control group of nine European groups and 9240 samples taken from DD patients. However, none of the SNPs discovered in earlier research reached a genome-wide significance level in the PGS study. The SNPs with the highest significance values were rs11579964 (P = 1.0 107) and rs7647854 (P = 6.5 107), which were located close to CNIH4, NVL, and WDR26, respectively. The aforementioned relationships were not supported by a later replicative study that was undertaken with

an independent sample (50,695 controls and 6783 patients with MDD).

As a result, no locus has been demonstrated to be significantly related with a DD across the entire genome. Additionally, associations found in other samples have not been replicated. The specific characteristics of the GWAS approach, which has concentrated on polymorphic sites with a high minor allele frequency (>5%) in the association analysis, may be the cause of this lack of significance and reliability. These common polymorphic variants are probably not pathogenically necessary on their own, but they might be in disequilibrium with rare variants of genes linked to the pathogenesis of DD. These uncommon variations can be unique to particular populations. A common polymorphic site may therefore be associated with a disease in one sample, and this relationship may be due to a disequilibrium linkage between this polymorphic site and a rare but pathogenically significant mutation in that sample. However, the pathogenically important site might not be present in a different sample; as a result, no correlation between frequent polymorphism and the occurrence of DD will be discovered. Additionally, it has been suggested that uncommon genetic variations with a frequency of less than 1% have a significant role in the development of other mental illnesses such schizophrenia and autism [33, 34].

To get around these issues, switching from microarray-based polymorphic DNA marker analysis to low-coverage DNA sequencing may offer a fresh approach to the challenge of DD-associated genetic variation identification. The first study of this kind was carried out as part of the CONVERGE Project [35] and involved genome sequencing in >9000 Chinese females with an average coverage of 1.7; Out of this group, 5000 of the female patients had melancholic depression, which is regarded as a more severe form of the illness. In this analysis, two loci were identified that showed a relationship at a 108 level: one was on the SIRT1 5′ side (SNP rs12415800), and the other was in an LHPP intron (SNP rs35936514). An independent sample of melancholy Chinese women verified this connection, and the overall significance values for the two samples were 2.53 1010 for SIRT1 and 6.45 1012 for LHPP. The two linked SNPs are both prevalent (minimum allele frequencies were 45.3 and 26.2 percent, respectively), but neither is present in the microarrays that are frequently used for SNP marker typing, suggesting that past GWASs may have overlooked them. Further analysis of the data from this project revealed that frequent SNPs were responsible for 20–30% of the DD risk dispersion, suggesting that the heritability of DD is evenly distributed across all chromosomes with DD-associated SNPs preferentially localized in both the coding and 3′-untranslated regions of genes. Particularly in the genes that are highly expressed in neural tissue, DD patients displayed an enhanced incidence of rare mutations in gene coding areas [36]. Importantly, this study included only females with a severe form of DD from a particular ethnic group (Han Chinese), which is sufficiently homogeneous and exhibits a greater heredity level. In this design, samples were included with greater rigor, and characteristics such the patients' sex, clinical DD variance, clinical onset age, and other variables that may affect the risk of disease and its progression were taken into account. The risk of DD development, however, may not be affected by these parameters; for instance, it was recently demonstrated that the clinical onset age had no impact on the results of the association study in the Chinese CONVERGE sample [37].

Ethnicity was crucial, according to a different study [38]. A combined examination of the findings from the CONVERGE inquiry of Chinese subjects and studies carried out by the PGC in various European groups were included in that paper. These investigations discovered a collection of SNPs particular to each ethnic group while also detecting SNPs that influence the risk of DD onset in both of the aforementioned ethnic groups. In both ethnic groups, it was shown that females and individuals with recurrent depression had the biggest genetic contribution.

GWASs have tried to incorporate environmental variables [31]. When they included case-control pairs of patients in the study—a process known as propensity score matching—they included stress-provoking events as a consideration. Through this methodology, they were able to compare DD patients and healthy controls subjected to comparable stressors and to lessen the heterogeneity of the samples with relation to the stress component. It appears that depression has a very complex genetic makeup, involving several loci that have diverse phenotypic effects and exhibit intricate inter-locus interactions. Genetic structure studies point to the necessity of moving from the study of individual SNPs to that of groups of SNPs, and then to incorporate a polygenic risk score, as employed in schizophrenia genetics research [39].

A method for examining gene networks made by combining signals from several SNPs and subsequent

functional analysis of the signaling and metabolic pathways have both been successfully applied to tackle similar issues. The capacity of comparative analysis of weak signals from several loci is increased by this method. This kind of analysis is demonstrated in the paper by [40]. The authors searched for and analyzed DD-linked SNPs and genes with these SNPs using data from a GWAS of samples from European cohorts in order to identify signal pathways connecting these genes to one another [40]. The pathogenesis of DD was revealed to include five of the resultant signal pathways. The following SNPs were linked to several DD-associated SNPs: rs3213764 in ATF7IP; rs2301721 in HOXA7; rs6720481 in LRRFIP1; and rs2229742 in NRIP1. Three of these were claimed to be correlated in some way with the negative regulation of gene expression (GO: 0016481, GO: 0045934, and GO:0010629).

For sampling, [32] and [41] proposed an alternative strategy. They created a questionnaire that the respondents were to fill out in order to diagnose DDs. Without a clinical diagnosis from a psychiatrist, depression was identified based solely on the questionnaire responses of the respondents. Despite doubts about the validity of the diagnosis, a wide range of phenotypic qualities were covered in the questionnaire, and respondents were unable to link any of them to any particular illnesses. They were able to significantly increase the sample size thanks to information from biobanks or mass genotyping services like 23 and me. As an illustration, the study by [41] included >450,000 people, and their analysis of the questionnaire data allowed them to identify depression in roughly 120,000 of the participants. This quantity of samples reduces the issues brought on by DD diagnostic errors by an order of magnitude compared to the PGC studies or CONVERGE Project samples. Given the breadth of the population examined, the scientists were able to locate 17 SNP markers in 15 loci with significance levels >5 108. Despite the fact that both research looked at samples with European origin, the DNA markers found were not the same as those connected to DD in the PGC investigations. As a result, the issue of the reproducibility of results from GWASs needs to be resolved.

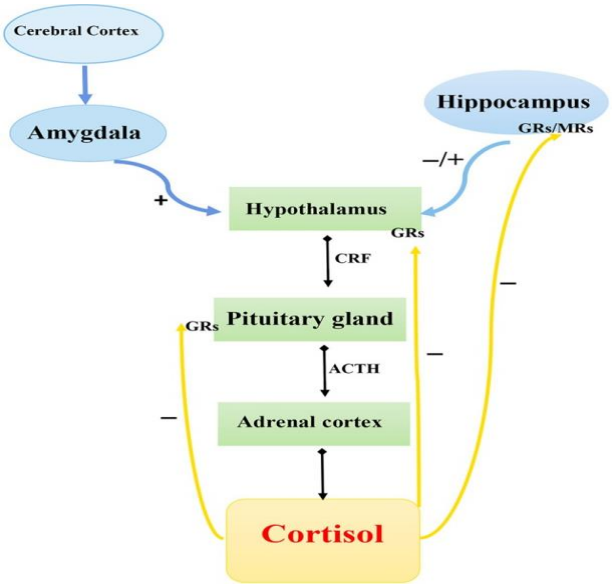
A meta-analysis of GWA trials could be used to address this issue. 44 independent loci were found to be statistically significant (P 5 108) in this meta-analysis. Of these loci, 6 shared loci with schizophrenia, 30 are novel, 14 were significant in a previous study of MDD or depressive symptoms. As a result, on the one hand, the larger sample sizes in the meta-analysis enable the validation of prior GWAS results for the previously identified loci linked to MDD. On the other hand, by expanding the sample size, it improves the study's power and makes it possible to discover novel loci connected to MDD. Simple count genetic risk score (SC-GRS), odds ratio weighted genetic risk score (OR-GRS), direct logistic regression genetic risk score (DL-GRS), polygenic genetic risk score (PG-GRS), and explained variance weighted genetic risk score are some of the methods that were suggested for calculating genetic risk score (GRS) (EV-GRS). Polygenic risk score (PGRS) is currently the approach that is most frequently employed [42]. This method has been applied to establish a shared genetic foundation for linked illnesses, to build risk prediction models, and to acquire evidence of a genetic effect even when no individual markers are significant [43]. There are already various methods for statistically analyzing GWAS data that focus on DNA marker combinations rather than individual DNA markers. Recently, multiple articles using PGRS for MDD and other mental diseases have been published [45,46,47]. It was shown that the PGRS might be used to assess the cumulative impact of a number of polymorphic gene variants to the development of endophenotypes of MDD. The MDD was split into two subcategories by [46] using the PGRS, one of which is similar to schizophrenia.

## Depressive illnesses exacerbated by stress.

Strong proximal indicators of the onset of depression include long-term stress and stressful life experiences that occur early in life. The reaction to stress suggests stability or the preservation of homeostasis, but chronic activation of the stress system increases the risk of obesity, heart disease, depression, and other problems, which can have negative or even deadly effects [11]. The mobilization of the body's reserves during exposure to stress of various etiologies is carried out by the hypothalamic-pituitary-adrenal axis and its three main components, the pituitary gland, the pituitary gland, and the adrenal cortex. These three organs are responsible for the adaptation to altered environmental conditions. The HPA system functions as follows: (Figure 1).

When under stress, neurons in the paraventricular nuclei of the hypothalamus secrete corticotropin-releasing hormone (CRH), which acts on the hypophysis to trigger the release of adrenocorticotropic hormone (ACTH), which in turn

stimulates the release of corticosteroids from the adrenal cortex, particularly cortisol. Cortisol, the final hormone produced by the HPA axis, binds to mineralocorticoid receptors (type 1) and glucocorticoid receptors (type 2) to form hormone-receptor complexes. These complexes are then transported into the cell nucleus where they interact with specific DNA regions, the glucocorticoid-response elements, to activate the expression of genes that are hormone-dependent [12].




Schematic diagram of hypothalamic-pituitary-adrenal axis. CRF, corticotropin-releasing factor, ACTH, adrenocorticotropic hormone, GRs, glucocorticoid receptor, MRs, Minerial ocorticoid.

Receptor → Secretion → stimulation → inhibition

The "stress-induced" explanation of DD onset is predicated on the idea that the HPA system's hyperactivity may be a key factor driving the onset of depression following stress. This theory is supported by several instances of the HPA system behaving abnormally during depressive episodes. First, the most powerful triggers for the onset of depression are stressful life events [13, 15]. Second, increased levels of cortisol (the human endogenous glucocorticoid), corticotropin (ACTH), and urine are typically observed in depressed patients [16]. Additionally, depressed individuals have larger suprarenal and hypophysis glands [17] and/or impaired corticosteroid receptor function [18]. In 50% of depressive persons, the HPA axis is shown to be overactive, and antidepressants taken regularly can help to reduce this activation [19].

Only a small subset of these genes have been extensively researched in relation to DDs, despite the fact that a sizable cohort of genes is likely involved in the normal operation of the HPA axis. The genes that code for the substances that cortisol and other stress-related glucocorticoid hormones are most likely to affect in DDs. In case-control studies, polymorphic variants of these genes were examined, and in certain instances, relationships between these variants and the onset of DD were found. For instance, correlations between the start of DD and polymorphic regions of the GR (NR3C1) and mineralocorticoid receptor (MCR; NR3C2) genes have been documented [20–22]. Additionally, postmortem research by [21] has demonstrated that the hippocampus of depressed patients exhibits decreased MCR mRNA expression.

Additionally, associations between DD and the CRH receptor-encoding genes CRHR1 and CRHR2 were discovered [23]. Significant correlations between depression and the SNV rs242939 in the CRHR1 gene have been observed by [23]. These authors also demonstrated that individuals with MDD exhibited the highest frequency of the G-G-T haplotype relative to controls (rs1876828, rs242939, and rs242941) [23]. The SNVs rs4076452 and rs16940655 of the CRHR1 gene were linked to DD [24]. Additionally, [25] found links between recurrent depression and the CRHR1 gene's rs242939 polymorphism.

We point out that some of the above mentioned connections were taken into account by [10] in their meta-analyses. These investigators failed to discover any conclusive relationships between DDs and polymorphisms in the genes responsible for the operation and control of the HPA axis [10].

## Brain metabolic responses that cause depression, according to science.

Although tragic life experiences, birth, retirement, and relationship issues are frequently cited as depression reasons, a chemical imbalance is thought to be the root cause of this complicated condition. A genetic susceptibility, drugs, stressful life experiences, and other medical conditions can all contribute to this chemical imbalance. Many people are unaware that depression has a biological foundation in addition to social and psychological repercussions. Here are a few of the biological causes of this mental illness that are known to exist. [48]

### Linked to Serotonin

As a neurotransmitter, or "feel-good" molecule, serotonin helps the brain's various regions communicate with one another. Some studies contend that serotonin regulates mood, and that low serotonin levels in the brain can cause mood disorders as well as other problems like panic attacks and obsessive-compulsive disorder. Serotonin deficiency is one of several scientific elements that are connected to depression, even though it can originate from many imbalances in the brain and body. [48]

## Deficiency in Serotonin

The difficulty of serotonin to reach receptor sites, a shortage of receptor sites able to accept the serotonin, or a tryptophan deficiency are some issues related to serotonin (the chemical serotonin is made from). One or more of these metabolic irregularities, according to researchers, can cause melancholy as well as other issues including anxiety disorder, obsessive-compulsive disorder, panic attacks, and even excessive rage.

Another idea concerning the origins of depression focuses on the continual process of brain cell renewal, which is thought to be mediated by serotonin. When new brain cells are suppressed, a person may experience depression. He thinks that SSRIs (serotonin reuptake inhibitors), which are popular antidepressant drugs designed to replace and enhance serotonin levels, aid in stimulating the generation of new brain cells, which in turn lessens depression.

## What the Hippocampus Does

The brain's hippocampus, which resembles a seahorse, controls memory and emotions. An international investigation found that individuals with chronic, inadequately managed depression had hippocampal shrinkage. A person's hippocampus gets smaller the longer they are depressed. Therefore, it has been determined that the condition of depressed people can be addressed by increasing the hippocampus's growth with new neurons. With the right care, they can live happier and healthier lives.

## Genetic Connection

A more complex type of sadness is hereditary depression. According to research, a child who has a depressed parent has a three times greater chance of developing depression than a child who doesn't. While the cause of the genetic link is yet unknown, a study has discovered that those with the serotonin transporter gene are more susceptible to depression. Each person possesses two copies of their genes, one from each parent. These genes might be either short or long. Your risk of developing depression increases with the length of your serotonin transmitter gene.

As has been stated previously, depression is a complex condition, and even in this day and age, determining what causes it can be challenging. Although the precise cause is unknown, there are a number of ideas and studies pertaining to this mental disorder that assist us comprehend the biological elements that contribute to the disease.

Low levels of serotonin, the neurotransmitter in charge of joy and exhilaration, were thought to be the root of the problem by scientists. They believed scientists had it all figured out since certain depression patients who were given serotonin-boosting medications began to feel better. However, when they looked more closely, they discovered that the hippocampus of these patients was abnormal.

The brain's seahorse-shaped structure, which controls memory and emotion, was often smaller in depressed people. Even worse, the hippocampus got smaller the longer they were depressed, ultimately reducing its capacity. Patients were happier and healthier because new neurons were supporting the hippocampus's growth.

When you consider that depression is genetically inherited from one or both of your parents, the complications only increase. Your risk of developing depression increases with the length of your serotonin transmitter gene, especially when stressful or upsetting life events are involved. According to the National Alliance on Mental Health, about 50% of adults in the United States who are depressed actually receive any therapy each year, which equates to about 25 million people in the US and 350 million worldwide. Without therapy, depression tightens its grip on millions of people's minds, exacerbates symptoms, and finally progresses to severe levels that only get worse with time.

When they arrive on the depression's embankment, the waters are dark, the sky is overcast, and the setting sun only produces seasickness.

## Depression and Religion

Depression has a miserable array of misguided stigmas and criticism surrounding its diagnosis. Some believe depression is a prolonged bad mood or just someone’s negative outlook on life, but it’s so

much more than that and science has proven it. The ancient Greeks believed depression was a result of fluid imbalances of blood, phlegm, yellow and black bile. Meanwhile, early Christianity just threw the blame of the devil for man's suffering, which resulted from their internal battle to fight the temptations of sin. Finally, Renaissance rolled around and scholars, such as Robert Burton, began to recognize the depression as a disease, but since then, attitudes and misconceptions have changed throughout history until we reached modern times when we came to understand there's something far more complicated than a simple chemical imbalance behind this darkening disorder.

Almost every individual has in one time of life been depressed. But what do we really mean when we hear depression being mentioned? As there are many human beings, so are there many opinions. To some depression on may occur to them when they are in a low-mood. others may feel depressed when they lose a loved object. To some depression is an illness or an attitude of Life. All these are many views about depression. But what does depression mean? This in what the memoir is all abouts and attempt to study it from early infancy to old age, bringing out also its effects on the individual in question. As human beings we are bound to be depressed at one time of our existence because we are not machines. The only way out then is to know how to free ourselves from depression whenever we fall into the trap. Some however, when they are depressed resort to drinking of alcohol or committing suicide, but suicide is even a greater sin.

## The Nature of Depression

Human beings being what they are cannot think of a well-balanced mood, this gives rise to the fact that our day-to-day mood is not constant. 0ur bodily functions such as temperature changes every now and then and we cannot say that they are not movable. As far as we live on and experience day and night come and go, so does our mood change by conforming to the feelings at a given time. From this we can arrive at the fact that there are some tines when our mood is down. Depression takes place when our mood is down. There are however, other words used when we are trying to say that some body is depressed. We may say that the person is 'fed up', he is in the blues', he is gloomy or that he is in the 'hands of misery', Also he can be described as 'feeling low', lifeless or being at his 'low ebb' or flat'. So, when all these words are being applied to a person's mood, we automatically know that he is depressed. There have been many views about depression. Some have regarded depression as a state or dejection accompanied powered sensitivity to certain stimuli reduction of physical and mental activity, and difficulty in thinking; an unwarranted condition of prolonged sadness or downcast. [51] For some depression is a state of sadness or futility. But some have understood depression as being a basic psycho-biological affective reaction which like anxiety, becomes abnormal when it occurs in inappropriate circumstances and when it persists for undue length of time. What is depression really all about. Following Rubinstein, can say that depression is a temporary emotional state that normal individual experiences or a persistent state that nay be considered a psychological disorder, characterized by sadness and low self-esteem. [52] Depression can be seen also as an extreme sadness, usually without personal loss, often accompanied by feelings of worthlessness and reduced activity level. All these have boiled down to the fact that depression is a feeling of pessimism, worthlessness, hopelessness. From this point let us consider the different types or divisions of depression. Though there has been arguments whether there are types of depression, we can still say that there are two major types of it. The one type of depression we call reactive depression or Neurotic or exogenous depression. The causes of this type of depression mainly come externally. There is also the second type of depression called Endogenous or Psychotic depression. This is of the internal. But this dualist view of depression has become more and more confusing. Mitchell would maintain that this dualism is a complicated when experience suggested that some depression on showed aspects of both. Reactive and Endogenous depressions. [53] However, some referred to this as Mixed depression" There is some danger any way calling certain depressions endogenous because it would all mean that there are no known external causes and so miss factors which would be used in the treatment or the prevention on of a recurrence, In spite of all these anyway, we can still group certain class of depression as belonging to the endogenous class. Let us now consider the Reactive or Neurotic Depression.

## Depression and Suicidal Behavior

Surely all of us experience times in our lives when we simply cannot face another day; when life Just does not seem worth the agony and pain it forces us to endure. Perhaps we have Just lost a parent or an

intimate friend; a lover has disappointed us; we have Just failed a course or been fired from a job we liked; we have just lost our property to the government without reason or denied of our right as citizens. All these trigger us to nurse the feeling of suicide. Yet however "appropriate" and strong these feelings of utter hopelessness and the fleeting impulse to end it all, most of us do not give in to the impulse. Eash suicidal person has the wish to die, the wish to die, the wish to kill the wish to be killed. Suicide has been seen as an aggressive act in which anger is directed both outwards against others and inwards against the self. Suicide is not meant for a particular group of people; every person may be suicidal candidate at some moment of his existence. In the realm of suicide there is neither profession nor class distinction. The word 'suicide' really means killing self but within suicidal behavior, the motive of self-destruction is not always present or, if it is, it exists in varying degrees. suicide is most succinctly defined in terms of two basic characteristics; intention and outcome. ‘true' suicides involve people who intend to kill themselves and who actually do so. Although outcomes are obvious the individual either survives or dies. intention is not easily recorded. Suicidal acts and attempted suicide of self- destructive intent are more common among older people and more often men than women. men tend to take their lives in a violently dramatic way leaving no doubt as to their intention (guns, drowning, car accidents), while women are more inclined to be passive in taking their lives (sleeping pills, overdose of drugs, carbon mono oxide)

"The thought of suicide is a great consolation.
By means of it one gets successfully through many bad night"

The suicidal attempts were associated with many types of personality disorder-infantile, psychopathic and hysterical as well neuroses, mental sub normality, depression and schizophrenia. According to caplet and lebovici. the precipitating factors were also manifold, including difficulties of many types like that of fustrarion in love; pregnancy; difficulties in adjusting to compulsory military service and so on. as a person becomes seriously depressed with marked ideas of guilt, self-reproach and despair, there is an increasing risk of suicidal intent. Thus, from all these we can say that physiological, social or psychological factors play part in bringing about suicide. At least half of the suicides in later life are committed by people who have some sort of psychiatric illness.

Complex personality tests are of little benefit in detecting those who will kill themselves. Although the majority of severely depressed people consider suicide, people often take their own lives when they are not depressed. research into the problem of suicide has been however carried on analyzing suicidal notes, reconstructing the dead person's history,

studying the precipitating factors in so far as they can be discovered, and by studying evidence supplied by persons who made unsuccessful attempts to kill themselves. some cases quite clearly indicate lethal intention; a Jump from the roof of a high building or the firing of a bullet through the brain. Sometimes however, the intention is far from clear.

Harari H, and Kaplan R.f. would maintain;

Some experta are convinced that suicidal people
do not particularly want to die; they just have
stopped caring about whether or not they are alive.

So the risk of suicide increases with depression. patients who are severely may attempt to kill themselves not merely because they have nothing to live for but also of their delusions.

As we have mentioned above, self-destructive intent is more common among older people and more often men than women. Actual committing of suicide is also more common among men than women. But women may have the lead when we talk of suicidal attempts. Also, investigations have proved it right that Christians enjoy less suicidal rate among their members than the other religions. More lettered commit more than the unlettered. Suicidal gestures also are most likely to happen with younger women of a hysterical or acting our personality. suicidal rates are higher for women in the middle age but the rate for men continues to rise. There should really be strong social prohibitions against suicide and against killing others whether by neglect or deliberately. This drastic removal of oneself through suicide may also be the supreme form of punishment directed towards someone else to whom he is dear. As Symonds would maintain;

"Anyone who attempts suicide is going to have a solicitous fuss made over him, and if suicide is successful, there are those who will mourn at the funeral"

Some however see suicide as a means of escaping from aggressive impulses towards others which assume such dangerous implications. but suicide should be considered as an indirect way of hurting others and of inflicting the supreme punishment on the self. One hurts oneself by cutting off at the roots all hopes and ambitions and enterprises of his life.

On the other hand, if suicide is attempted as a means of escaping from the worries of life or from

psychological disorder like depression, anxiety, frustration etc, it would be said to be a greater sin. As Diggony would maintain;

"If suicide is attempted it will simply add more conflict, count as an error, and increase the exasperation".

This would mean that an empirical description of a suicidal impulse shows that it is a mistake in terms of the ends the impulse is seeking to satisfy. Suicide should not arise as a result of slough or want of manliness. suicide is really a cowardly act. The period that immediately follows a suicidal attempt shows a depressive or neurotic reaction with signs of increased tension such as restlessness, anxiety, sleep disturbances, nail biting, increased irritability and over sensitivity. Some of the cases also showed deterioration in social behavior and alcoholism. Suicidal behavior or real suicide should not be considered as 'escape' from depression.

## Reactive Depression

As we mentioned above this type of depression mainly has its arousals external1y. This has a certain clinical feature and can occur at any stage of 1ife. It can occur in early infantile period, childhood stage, adolescence period, adulthood or later adulthood periods and lately it can also occur in old age. We can say that adverse circumstances are probably the most common cause of this type of depression. Many instances spring to mind and it can occur at any age. This type of depression can even attack child who has Just come out of the womb of his mother. In this type also a situation rather than an accident can be an arouser. So, we do not have to be mindful of accidental things only, we must be able to check the situations we find ourselves always. Iet us consider now what we really mean by these adverse circumstances or the external events that are the arousals of reactive depression. The external causes say include any type of loss especially the loss of one 's parents which is more serious at the early infantile period. he infant who for some reason or another is separated from his mother is likely to get depressed and can indeed feel so for a long period of time. At first with tears and anger he demands the mother back and seems hopeful that he will succeed in getting her. But when he fails, he despairs that the effort to get his mother back has failed and from this point he would detach the mother entirely. here Seems to forget his mother so that if the mother is still alive and she comes back for him, he remains curiously interested in her, and may even see not to recognize her. This is phase of detachment. Again, going to school may well be an arousal of depression in the child. In school, he dislikes his new environment and yearns to be back at home with mother. This is 'school phobia because any time he hears school mentioned, he develops some fear. Some children who fall into this trap have resorted to feel sick at least every morning during school days. Again, he may develop the idea of doing his morning duty late so that he may be late for school or if possible, miss school entirely. Broken love affairs are also very common cause of reactive depression. Many lovers have threatened to take their Lives if their love go unrequited. Again, we can experience depression through disappointment. Most people can cope with disappointment any way but sone cannot, treating this point in a short and simple way, a great Indian philosopher-Tagore said that we should always expect nothing so that we will not

be disappointed. [53] his follows that we shall not experience depression that is caused by disappointment. Also parting with a dear friend can cause a reactive depression in an individual. We do not want to part with our friends even for a minute not talking of one month, a year or worse still indefinite years. Sone individual a feel depressed throughout the parting period and they can only feel alright when the friend is back again. For them to say good-bye is to die a little. We have other causes like the loss of job which may occur at adolescence, adulthood and old age periods. In old age we normally talk of retirement from work and this produces adverse effect on the old man whose social development has been tampered with. We can talk also of unemployment. All these causes reactive depression to the affected Individual. We have also other cases like the loss of status and self-esteem we like always to hold high. We have failure to live up to expectation, guilt over past actions. Also, the prospect of dying, all these can make us have reactive depression. There are really some who do not want to die, even when they are old, they Cherish to live on rather than to die Such people feel depressed at the slightest illness. We have the case of a situation rather than an accident being an apparent cause or reactive depression. Thus, a woman became depressed when her husband was put on night work. The depression however did not arise from sympathy with the husband over the hardship he had to put up with. the result of his night work was that he was around the house a great deal during the day. In this case now, she has centered her affection on the husband but the night work has come to disturbs the relationship. The result she gets is therefore depression. Also, failure

in examination can cause reactive depression to a very serious student, for him, he may think himself as being the least in intelligence of all the students. Take for instance a student who wants to enter into a Higher Institution but has failed its entrance examination for six times. He will automatically feel he has no brain and the next step after being depressed or during depression period would be to give it up. for him, there is no need for trial. He may even give up schooling. We have other cases like failure in an interview of any kind. Also lost of property can trigger off reactive depression. This is the more reason why government should steer clear of people's properties. A man has the right to his property and once this right is tampered with, he may react in a depressive way. Also not getting a desired promotion at work can cause depression. Having outlined some few causes of reactive depression, let us now look at endogenous depression.

## Endogenous Depression

This is a type of depression that Grows within. In this case, we cannot Say that this particular external object or that particular external act is responsible for this type of depression. It is strictly assumed to be caused internal1y.For watts, it is a disorder of the emotional control Centre for which we can see no obvious cause, and many patients suffering from that type of depression show a dramatic response to physical methods of treatment. [54] This type of depression as we mentioned above in also called Psychotic and this tend to have different clinical features and occurs later in life. In this type of depression there is therefore a hereditary organic factor which makes the patient physically disposed to this type of reaction. And this type of depression is more severe than the Reactive depression. It is far more common than most people realize. 1t is frequently overlooked, yet it causes an immense amount of human suffering both for the patient and for the patient's family. Patients suffering from this type of depression often feel perplexed by what has happened to them. for them, they have found no cause why this type of depression should fall upon them. For example, when they are explaining to the doctor about their situation, from their words or complaints you will see that they are really surprised - I have no worry, but I feel awful, just cannot understand myself.[54] This is the one way they have expressed how surprised they are to be in that state of endogenous depression. In our everyday life, you will see people whom you may qualify as being self-sufficient but they are still utterly miserable. So, this explains how Endogenous depression affect us. Certain cases of this type of depression have been traced back to the family history. It is likely that a positive family history of depression could be evidence of this type of depression. it could represent a clear-cut genetical inheritance by the patient from his family. Similarly, a history of previous depression in an individual patient may carry the same mixed Interpretation. that is to say that a history of previous depressions may have variable effects on the patient at this particular moment. But this type of depression is simple to treat once it has been recognized, but if missed, the patient may suffer it for months or even years and he may resort to end it all in suicide. For better attention to the patient, correct diagnosis and treatment need to be employed. Delusions however are signs of a very severe Endogenous depression. Most cases of Endogenous depression make a good response to anti-depressant drugs. From this point now let us consider how a patient react to these types of depression. Depression is an exaggeration of the mood that all of us experience for brief periods of time. The person sinking into a deepening depression begins to be pre-occupied with feelings of failure, sinfulness, worthlessness and despair. He cannot be reasoned with or told to cheer up for his woes in an internal event that does not correspond to reality as others see it. The depressed may not hallucinate, but he may descend to Such a stuporous level of mental and physical inactivity that he may be bed-ridden and require force-feeding. He feels that he has failed utterly. His ability to associate with other people and be committed to his work or studies gradually declines. He may turn to friends for consolation, but he blames his troubles entirely on himself. He is tired and apathetic most of the time, and even negativistic. Depressed persons do inhibit outward expressions of anger and they tend to blame themselves for their difficulties. The depressed person also blames his environment for falling to provide rewards for adaptive behavior. Once people become depressed sad, withdrawn, Inactive their main source of reinforcement that are maladaptive (weeping, complaining, self -criticism). But because 1t is tiresome to be around a depressive patient who refuses to cheer up, the depressed person 's behavior eventually, alienates even those who are close to him, producing further reduction in reinforcement, increased social isolation and unhappiness.

Depressed person always likes being alone and it is normal for depressed people to wish to be by

themselves. They try always to isolate themselves from others. They feel negatively about others around them and for them they cannot produce anything positive. Depressed person is very indifferent to unit activities, slow in speech and movements, falls asleep at times outside bedtime. He can even spend most of his time sleeping, and this entails less production in any field of work he happens to belong.

A depressed person has also difficulty in concentration. That is why depressed students scoreless mark at school. The more serious thing is that they will not link their scoring less mark to their state of being depressed but they will conclude that they are the least in intelligence among the students.

Also depressed person can feel a sense of 'nothing'. For Caplan and Leborici, the depressed person has the feelings of nihilism, apathy and insomnia. [55] He feels he is not loved by anyone and therefore not wanted, there is an unwarranted condition of prolonged dejection, the state of being sad and downcast. He automatically retires to the world of his own.

Depressed person also feels gloomy, his thinking is sluggish and he speaks in a monotone. His attitude is one of uncertainty and doubt. He feels unworthy and incompetent, lacks interest and initiative. Nothing pleases him and nothing in worth doing for him. He may even lack the initiative to plan what to do to his abnormal activities. If he is a tennis player, tennis playing would mean nothing for him at this moment of depression. He can lack even all sexual urge so that there is nothing you can do to arouse him.

On a higher level, the attitude of the patient who is depressed in that of morbidity and being in a state of despair. He despairs even at the sight of a new day. Some patients feel so guilty and unworthy and unwanted that they refuse to partake of food; others feel so despondent that they never speak and when spoken to, mutter unintelligently. from his answer you will see that his whole body has been affected by this state of depression he finds himself. This renders him for the time being hopeless, so that he feels sorry for himself, thus the situation is impossible to cone out from.

He may also at this moment lose his dearth of ideas, and almost in complete inaction. He is there and just there. He is alive but life still need to be pumped into his empty shelf; he needs to be awakened from this terrible state. Some patients of depression also show some delusional trends, especially of sin, of being worthless, and of guilt. Certain patients however, remain reasonably well oriented; others partiall1y disoriented. For Thorpe & Co.

"Depression is accompanied by morbidity, dearth of ideas, abulia, stupor, and clouding of consciousness. The patient is mute and negativistic, he neither speaks nor respond when spoken to. He is totally unresponsive that he must be tube-feed and otherwise cared for; he will not even attend to his bowel and bladder needs". [56]

This really can be seen aa the highest effect of depression to the patient. This has gone to the extent that he cannot even attend to his bowel and bladder needs. In this case, the patient may be completely and may remain almost motionless for long periods of time until moved or disturbed. Thorpe and Co. would also say that at this point of depression, hallucination and illusions are frequently present, delusion is only occasionally evidenced. [56] The patient's face frequently presents a mask-like appearance. He is sad faced and possesses a stooped posture. Also, his physical symptoms often developed and this usually include stomach disorders, loss of weight, sleeplessness, constipation, weakness and fatigue,

## Depression as Masked

When we are depressed, or someone is depressed, it is usually pretty obvious to those around him that he is not well; the mood of depression is often so 'catching' that other people, aware of their own reactions to him, can 'feel' that he is depressed. Not everyone who says he is depressed is really suffering from a depression, and not everyone who is depressed is able to convey the fact either to himself or to those around him. This however boils down to the fact that depression may even go unrecognized although the depressive process is still there causing discomfort and distress to the sufferer and disrupting the lives of those around him especially his family and friends.

It is really good to have a good knowledge of depression and know exactly when we are depressed. We must not also deny the fact that depression can show itself in uncommon ways. But a good knowledge of depression will help us to pin point even the uncommon ways. Just think of how easy it would be for you to go to your doctor and say 'Doctor, I am feeling depressed. Recognize that depression exists as an entity and this recognition will help you to realize when you are depressed. You must also be in touch of your inner feelings you can then use them to convey your experience clearly to others.

If you do not know what you are suffering because you were not in touch with your inner feelings, and you do not have the capacity or vocabulary to express that into words which you can then use to convey your experience clearly to other, you may go to the doctor and simply say, I just do not feel well, I have got bad nerves and leave it to your doctor to determine what is wrong with you to tell you that you are depressed. If the doctor is not able to diagnose your depression also, he may diagnose something else like neurosis, hysteria or whatever. The first and commonest cause of masked or unrecognized depression is the inability to recognize and express the depression.

What happens in dynamic terms when a person becomes depressed and his psyche as it were, recognizes that this is so? He will automatically resort to use all types of defense mechanisms to defend himself like displacement, denial and conversation. These are the commonest type of defense mechanism and how can they operate?

Displacement

This means that the person's attention is diverted from something which is threatening, on to something else which is less threatening and more easily acceptable. As column would hold:

"Displacement as a defense mechanism is an unconscious shift of emotion and symbolic meaning from one person or object to a substitute. Typically, it involves discharging hostility onto a safer person than one which aroused it".

Also, Otto & Co. maintain that displacement is the mental mechanism which transfers an emotional reaction to a substitute when it cannot be shown to one who causes it.

When people become depressed, the depression itself may release so much anxiety that the original depression is lost masked by the released emotion so that the doctor diagnoses 'anxiety state' rather than 'depressive state'. In displacement, a motive that be gratified in one form is directed into a new channel. Similarly, feelings of depression can be displaced or diverted into openly expressed irritability, resentment or anger. In the process once more the original depression is lost.

## Denial

Denial is also another type of defense mechanism that the individual uses to defend himself. This means quite simply, persuading oneself that something is not so in the hope that it will go away. If we tell ourselves that we are not depressed then perhaps the depression will go away. If we cannot see something, perhaps it is not there. This is a sort of flight from the actual fact that is depression. A Coleman would hold:

"One defense mechanism that many people learn to use for protecting the self from unpleasant or devaluating situations is refusal to face the situation". (24)

It all means then hat when an external reality is too unpleasant to face, we may deny that it exists. For e.g., the parents of fatally ill child may refuse to admit that there is anything wrong even though they are fully informed of the diagnosis and expected outcome. Because they cannot tolerate the pair that acknowledge reality would produce, they resort to the defense mechanism of denial, at least for a while. The patient of depression denies the impending situation, perhaps thinking a mistake had been made in the diagnosis. The denial of depression consists of falsifying depression either by convincing ourselves it does not exist or by feeling it in a distorted way.

Many adults still have the emotional child inside them that plays up when things go wrong. Rather than facing up to depression and trying to determine its causes so that perhaps it can be dealt with, they would rather run away from it, turning their backs on it, denying its existence, believing that, in so doing, they are in fact making it disappear, making it go away. But the fact is that they are trying to deny the reality of the underlying depression that is in them.

Conservation:

With conversion, the psychic energy of the depressed state is not only diverted and displaced to be covered up by other feelings, but also in a sense, it is converted into other experiences. For Hershey and Lugo

"A conversion defense also known as psychosomatic disorder, consists of organic symptoms with or without underlying organic disease produced primarily by emotional stress and tension"

So just as a seed is not covered over by the plant, but actually becomes the plant, so depression can become converted into bodily distress of which is the commonest expression.

It is vital to be aware that depression can be masked so as not to be taken in by the so-called smiling depression (that is a patient looking happy when he is really depressed), the psydosomatic disorder, cases of pain, of anxiety and excitement. It is easy to forget or fail to see the depression that lies beneath the surface. At the opposite extreme, there is the risk of seeing depression where none exists, but remembering that depression is a fairly common human existence, everyone should be less likely to

miss it when it really does exist. We should not try to evade or deny depression in order to protect the good image of our parents or friends.

## Conclusion

It is really better to avoid depression than to suffer it. Some of the depressive moods that often afflict us are not mainly caused by serious things like bereavement, emotional deprivation, lost of loved object, being amputated, etc. some of us fall in the arms of depression for no serious cause. At least Neurotic depression can be prevented by living a full life in the society, having friends and relations around during the downs of life. We know all well that isolation is one of the chief causes of depression and even suicide yet, we still try to isolate ourselves from people for no just cause.

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