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NIACINAMIDE AND VITAMIN B3 USED IN TREATMENT OF DEPRESSION

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ABSTRACT :-

The physiologically dynamic form of niacin, or vitamin B3, is niacinamide (also known as nicotinamide, 3pyridinecarboxamide). A deficiency of this vitamin results in the benign sickness pellagra, which has specific cutaneous symptoms. Since its discovery and isolation, this basic water-solvent nutrient has also been linked, when used as an effective specialist, to a wide range of dermatological beneficial benefits and systems. These include its obvious role as a suppressor of skin inflammation dynamics, an enhancer of the sphingolipid blend on the skin's surface, an enhancer of markers of epidermal separation and dermal expansion (with concurrent benefits to the layer corneum hindrance), and a mediator of photoimmune suppression and initiating growth.

Vitamin B3, nicotinamide, reduces depressed symptoms. According to this abstract, niacinamide may be able to treat depression by controlling the levels of adenosine triphosphate (ATP). According to the study, niacinamide not only raised SIRT1 activity but also helped mice exhibit depressive behaviors.

Treatment for depression with imipramine and tryptophan-nicotinamide together. Niacin consumption and depression are related According to this abstract, there is a U-shaped correlation between depression and niacin intake in the broader American population. The similar link was also discovered in men, Mexican Americans, White adults, those under 40, and those with BMIs under 30.

KEYWORDS :- Niacinamide, Nicotinamide, Vitamin B3, Depression, Antidepression, , Mental health, Dermatologicalbenefits .

INTRODUCTION :-

1. Overview According to WHO estimates, depression would rank as the second most common condition worldwide in terms of morbidity in ten more years. Currently, one in five women and twelve men suffer from depression. In addition to adults, depression affects 2% of schoolchildren and 5% of teenagers, with the majority of these cases going unreported. Despite the widespread belief that all psychological issues are depression, depression has been the most frequent reason for patients to visit a psychiatrist .[1–2].

2. Niacinamide is used to treat pellagra and other disorders associated to vitamin B3 deficiency. Acne, diabetes, cancer, osteoarthritis, aged skin, skin discolouration, and many other ailments are also treated with it, albeit the majority of these uses lack strong scientific backing. Niacinamide is not the same as niacin, L-tryptophan, nicotinamide riboside, inositol nicotinate, or NADH.They are not interchangeable.

3. vitaminB3 in the form of niacinamide is beneficial for maintaining healthy skin. In dermatology, nicotinamide has a long history of use. Despite its role in irritating illnesses and acne vulgaris, recent attention in its use has focused on its chemo-deterrent function in remedial applications and nonmelanoma skin malignant development (NMSC). We provide a current overview of nicotinamide's uses in dermatology and evaluate the evidence supporting these uses. Only human investigations are included. The water-soluble amide isotype of vitamin B3 is called nicotinamide (niacinamide), while the corresponding corrosive isotype is called nicotinic corrosive niacin. [3][4]

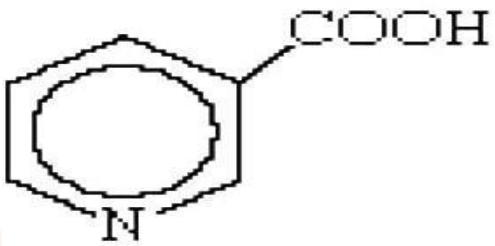

Figure No.1:-NIACIN

4. Another usage for niacinamide is in depression A widespread mental illness that causes a variety of psychological and emotional symptoms, depression affects 4.4% of people worldwide [5]. The intricate processes that underlie the pathophysiology of depression are still poorly understood. A protein deacetylase that aids in in vivo cellular control is called Sirtuin 1 (SIRT1). Research has revealed a possible link between it and depression [6] [7] [8]. It is controversial in animal modeling studies, nonetheless, whether its rise or fall contributes to depressed phenotypes. Chronic stress was shown to decrease SIRT1 activity in the dentate gyrus (DG) of the hippocampus in mice by Abe- Higuchi et al. [9], but chronic stress exposure was shown to increase SIRT1 activity in theDG of rats by Ferland et al. [10].

5. In the clinic, pellagra is the primary use of nicotinamide (NAM), a type of vitamin B3, which has been shown to be therapeutically helpful against a variety of diseases and ailments. Additionally, research indicates that NAM promotes the healing of bipolar disorder or depression.[11] [12]. According to Song et al., NAM functions mainly by promoting and inhibiting the synthesis and breakdown of monoamine- neurotransmitters. It may also help treat depression by acting as an antioxidant and boosting the levels of nicotinamide adenine dinucleotide (NAD+) [13]. The NAD+ co- substrate specificity

of a Sir2 enzyme can be changed by NAM, which is generated by sirtuin enzymes, by binding to a conserved pocket next to NAD+, as per earlier reports [14][15]. This inhibits the deacetylation of SIRT1.

Information of Depression:- The incidence, suffering, dysfunction, morbidity, and economic impact of depression make it an illness of significant public health concern. Women experience depression at a higher rate than men. One-year prevalence estimates for unipolar depressive episodes are 5.8% for men and 9.5% for women, according to the Global Burden of Disease report. The point prevalence is 1.9% for males and 3.2% for women. If current demographic and epidemiological transition trends continue, depression is predicted to become the second leading cause of disability-adjusted life years (DALYs), after ischemic heart disease, and to account for 5.7% of the overall burden of disease by 2020. [16].

Researchers in India have traditionally focused on depression as an illness because of its morbidity. Numerous writers have attempted to investigate its prevalence, nosological problems, psychosocial risk factors, such as life events, comorbidity, psychoneurobiology, treatment, outcome, prevention, disability, and burden, as well as symptomatology in the cultural context. Additionally, some research have attempted Research conducted in India on a range of depressive disorders is the main topic of this review. In order to do this, a comprehensive web search was conducted utilizing a variety of keyword combinations, including depression, life events, prevalence, classification, cultural concerns, result, prevention, disability, and burden. [17] [18].

TYPES OF DEPRESSION:- like many other disorders, depression can take many different forms.:-

1. A variety of symptoms that impair one's capacity to work, sleep, eat, and engage in formerly enjoyable activities are indicative of major depression. A person may experience these incapacitating depressive episodes once, twice, or multiple times during their lifetime.
2. Dysthymia, a milder form of depression, is characterized by persistent, chronic symptoms that prevent you from feeling well or from working at your best. Major depressive episodes are occasionally experienced by people with dysthymia.
3. Compared to other types of depression, manic-depressive or bipolar disorder is not nearly as common. There are cycles of mania or euphoria and depression. Most of the time, the mood changes gradually, but occasionally they are spectacular and quick. One may experience any or all of the additional symptoms of a mental disorder while in the depressed cycle. Any or all of the symptoms described under mania may be experienced throughout the manic cycle. Mania frequently has negative effects on judgment, thinking, and social behavior that can lead to embarrassment and major issues. [19] [20]

DEPRESSION SYMPTOMS:- Not all manic or sad people have all of the symptoms. Some people may have a few symptoms, while others may have several. Additionally, people may experience symptoms of varying intensities. Depression is characterized by:- [21] [22] [23] [24]

I. a persistently depressed, anxious, or empty mood;

II. feelings of pessimism and hopelessness;

III. guilt, worthlessness, and helplessness; and

IV. a loss of interest or enjoyment in once-enjoyed pastimes and activities, such as sex,

V. sleeplessness, excessive sleeping or waking up early,

VI. hunger and/or weight loss, or overeating and weight gain,

VII. a reduction in energy, exhaustion, slowing down,

VIII. suicidal thoughts or attempts,

IX. agitation, restlessness,

X. trouble focusing, remembering, or making decisions,

XI. chronic physical symptoms that don't go away, like headaches, digestive issues, and chronic pain .

CAUSES OF DISEASES AND FACTORS OF GENETIC CAUSES OF DEPRESSION:-

1. Functional polymorphisms (differences in DNA sequence that affect the expression and/or function of the gene product) in the loci encoding the serotonin transporter (SLC6A4), serotonin 2A receptor (5HTR2A), tyrosine hydroxylase (TH), the limiting enzyme for dopamine synthesis, tryptophan hydroxylase 1 (TPH1), and catechol-o-methyltransferase (COMT) (dopamine catabolism) [25] have been the focus of the majority of published genetic association studies of mood disorders

2. Although it has long been recognized that depression can run in families, it was unclear until recently whether a person's predisposition to certain disorders was inherited or if environmental factors, such as the environment, were the real cause. Researchers studying depression have shown that depressive disorders can be inherited to a certain degree. A susceptibility to depression seems to be inherited. This implies that we might inherit a propensity to develop clinical depression if we have close relatives who suffer from it. It does not imply that depression is our fate [26] [27]

3. .Bipolar disorder is strongly influenced by genetics. About half of people with bipolar illness have a parent who has experienced clinical depression in the past. A kid of a parent with bipolar disorder has a 25% probability of experiencing clinical depression of any kind. The likelihood that a kid will likewise have bipolar disorder ranges from 50% to 75% if both parents have the disease. Compared to those without such siblings, those with bipolar disorder may have an 8–18 times higher risk of developing bipolar disorder and a 2–10 times higher risk of developing major depressive disorder [28]

SIDE EFFECTS :-

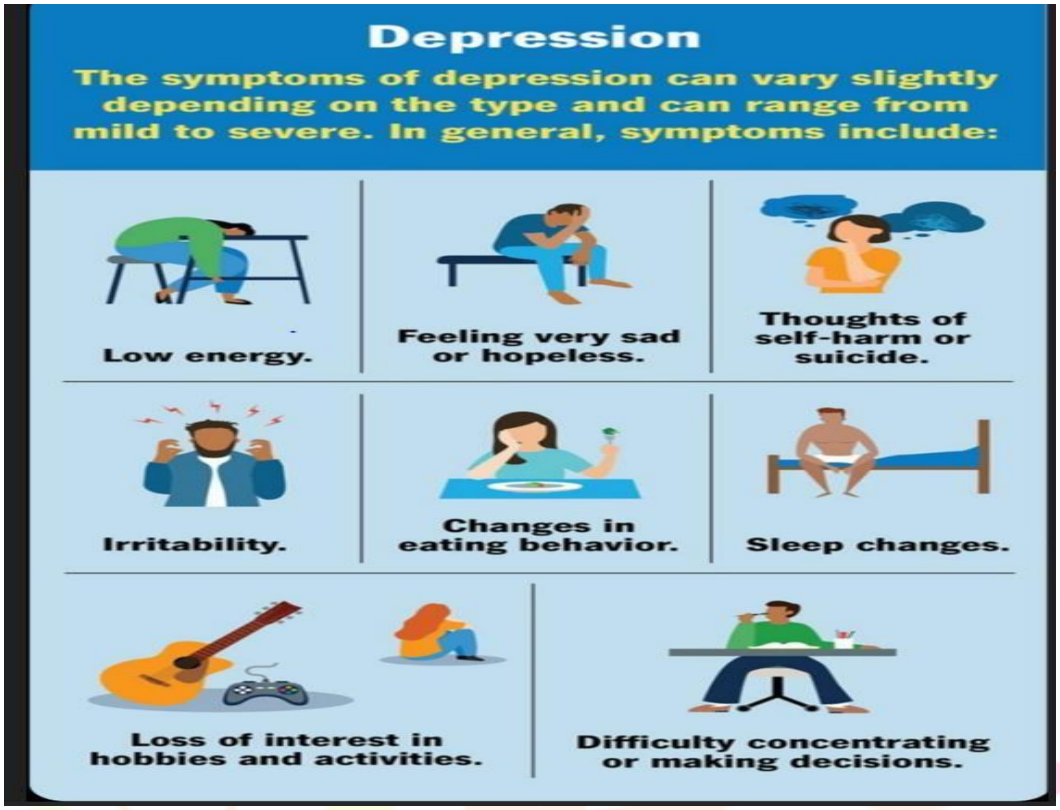

Figure No.2:- Side effects of Depression

TREATMENT:-

Therapy Both medicine and psychotherapy are effective treatments for mild depression. A combination of psychotherapy and medication may be necessary for moderate to severe depression [29]

DRUG TREATMENT:- The initial antidepressant is effective for 50–65% of patients In terms of effectiveness or response time, no antidepressant medication is better than another. The presence of medical and mental co-morbidity, past responsiveness, and comparing the patient's symptoms to the side effect profile can all help guide the decision. Consideration may also be given to relative costs (e.g., generics). Citalapram (Celexa ®) and generic fluoxetine are the favored agents at UMHS [30]. Antidepressant-treated patients should be continuously monitored for any potential worsening of depression or suicidality, particularly at the start of treatment or when the dosage is changed. It is thought that antidepressants' actions on neurotransmitters and neurotransmission are what give them their therapeutic effects. According to a scientific theory called the Monoamine Hypothesis, depression results from underactivity.

According to a biological theory known as the Monoamine Hypothesis, depression results from

monoamines including dopamine, serotonin, and norepinephrine not being as active in the brain.

Tricyclic antidepressants and monoamine oxidase inhibitors (MAOIs) were unintentionally found to be useful in treating depression in the 1950s. Joseph Schildkraut published his paper "The Catecholamine Hypothesis of Affective Disorders" in 1965 as a result of these discoveries and further supporting data. Schildkraut linked depression to low neurotransmitter levels. Studies on other mental illnesses, such schizophrenia, have also linked these conditions to low levels of specific

neurotransmitter activity [31,32,33]. For more than 25 years, the idea has been a primary area of study in disciplines including pathophysiology and pharmacology.

By blocking the monoamine oxidase enzyme, monoamine oxidase inhibitors (MAOIs) prevent the breakdown of the monoamine neurotransmitters dopamine, serotonin, and norepinephrine. This results in higher levels of these neurotransmitters in the brain as well as an increase in neurotransmission [34].

Figure No.3:-Treatment of depression

INFORMATION OF NIACINAMIDE:-

Nicotinamide is acquired from diet, and a lack of it can result in pellagra, which can lead to dementia, dermatitis, and the runs. Nicotinamide can be found in foods such as meats, liver, yeast, dairy products, vegetables, beans, nuts, seeds, green vegetables, cereals, coffee, tea, and baked bread.[35]

Nicotinamide addresses a viable therapy approach for skin breakout vulgaris and provides substantial soothing capabilities without the risk of bacterial resistance and underlying side effects. Nicotinamide, also known as niacinamide, is a form of vitamin B3, a basic water-solvent supplement found in a variety of foods. Although it has the same nutritional potential as nicotinamide, the amide form of nicotinic corrosive (niacin), it lacks the same gastrointestinal, hypolipidemic, and vasodilatory effects.

Furthermore, nicotinamide has demonstrated little adverse effects and toxicity at oral dosages of up to 3 grams daily for up to five years. Nicotinamide is the precursor to both the phosphorylated subsidiary NADP and nicotinamide adenine dinucleotide (NAD).[36]

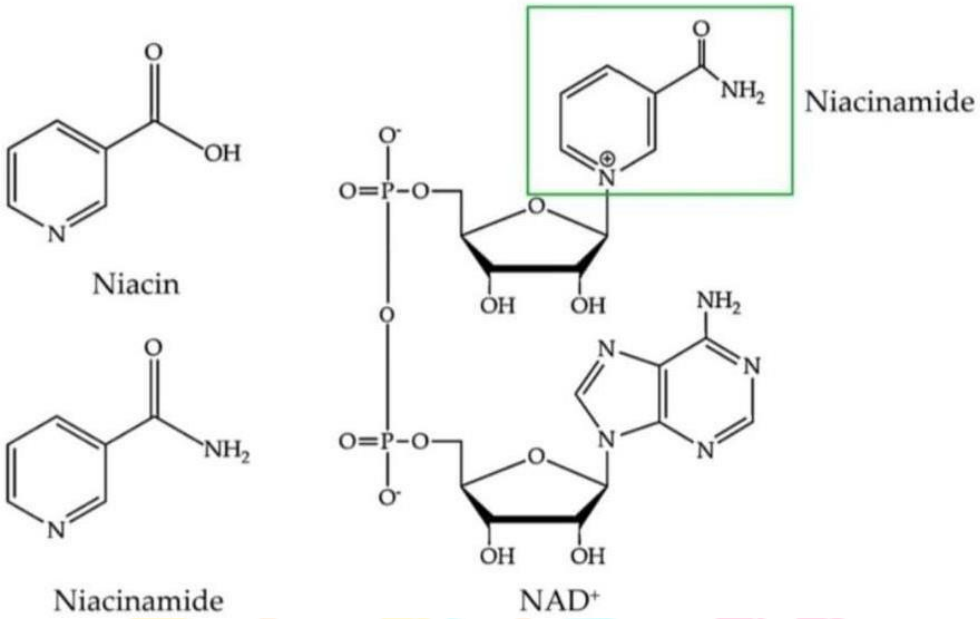

Figure No. 4 . The molecular structures of Niacin , Niacinamide and the NAD+

HISTORY OF NIACINAMIDE:-

1. The vitamin B3 amide niacinamide is sometimes referred to as Pellagra-Preventive, or PP for short. The name has some significance. The first American case of pellagra was identified in 1902, and forty years of pellagra plague ensued, with precisely three million cases and one hundred thousand deaths reported in states south of the Potomac and Ohio streams. Patients taking pellagra experienced a variety of debilitating side effects, primarily a variety of skin wounds. Sadly, this led to the public banishment of thousands of victims, all of whom were from regular, low-class, rural households that fed themselves on boring staple foods like fatback, molasses, and cornmeal.

2. The long-standing clinical opinion that pellagra was an infectious disease was altered by Joseph Goldberger, a Hungarian refugee who established a solid reputation for himself as a leading authority on clinical disease transmission. He showed how simple dietary supplements might be used to both prevent and treat pellagra. After thirteen years of work, in 1927, Goldberg persuaded the American Red Cross to provide dried yeast to victims of the Mississippi floods, thereby averting another devastating epidemic. Nicotinic acid and its derivatives (including niacinamide) were not identified as the subtle »PP« component until after 1937. By 1945, Goldberger's ancestry proved remarkably resilient; government-sponsored education had permanently altered the poor dietary habits of the South, and pellagra had been eradicated in the US.[37] [38]

SYNTHESIS:-Endogenous Synthetic Niacin is combined with dietary Trp via the kynurenine pathway (KP) (Figure 2), which primarily occurs in the liver and, less importantly, in the liver (especially[39] [40] [41].

STRUCTURE:-

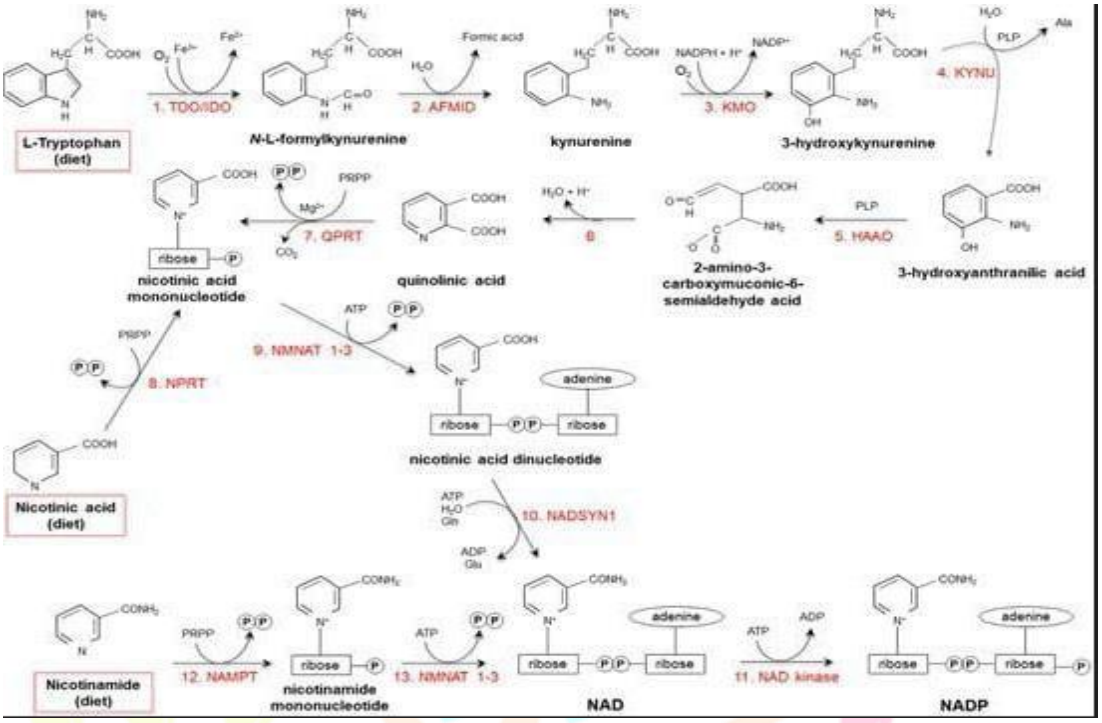

Figure No 5:-Structure of endogenous synthesis

Figure 3:- A novel combination of nicotinamide, nicotinic corrosive, and tryptophan that result in NAD(P).

(1) The pyrrole moiety of tryptophan (Trp) is oxidized by two iron porphyrin metalloproteins, tryptophan 2,3 dioxygenase (TDO, in the liver) and indolamine-pyrrole 2-3 dioxygenase (IDO, in extrahepatic tissues), which then frames N-Lformylkynurenine. Following the hydrolytic removal of the formyl bunch that delivers kynurenine by arylformamidase (AFMID), a mitochondrial flavo-protein that uses O2 as a substrate and NADPH as a cofactor (3) hydroxylates kynurenine to kynurenine-3 3-hydroxykynurenine monooxygenase (KMO). 2-amino-3carboxymuconic-6-semialdehyde corrosive is created when (4) kynureninase B (KYNU, a vitamin B6 subordinate chemical) and (5) 3-hydroxyanthranilic dioxygenase (HAAO, a nonheme iron-subordinate dioxygenase) are active. This unsound substance (6) instantly condenses and changes to form quinolinic corrosive; after that, (7)Phosphoribosyltransferase quinolinic (QPRT) decarboxylates quinolinic corrosive and converts it entirely to nicotinic corrosive mononucleotide. damaging The "rescue pathway" also delivers nicotinic corrosive mononucleotide through the action of (8) nicotinic corrosive phosphoribosyltransferase (NPRT). NAD synthetase (NADSYN1) and (9) nicotinamide/nicotinic corrosive mononucleotide-adenylyltransferases (NMNAT13) cause the age of NAD, which is subsequently (11) phosphorylated to produce NADP. Through the actions of (12) nicotinamide phosphoribosyltransferase (NAMPT) and (13) nicotinamide/nicotinic corrosive mononucleotideadenylyltransferase (NMNAT1-3), NAD can also be directly obtained from nicotinamide. Red edges: NAD(P) dietary antecedents. PLP stands for pyridoxal phosphate; PRPP for 5-phosphoribosyl- 1pyrophosphate; Ala for alanine; Gln for glutamine; Glu for glutamate. [42]

The rate-restricting protein is tryptophan 2,3 dioxygenase (TDO), which catalyzes the primary response. The efficiency of this anabolic pathway is influenced by a few physioneurotic, hormonal, and nutritional factors. Response rate is slowed by

deficiencies in vitamin B1, Trp itself, riboflavin, iron, heme, and vitamin B6, which are essential cofactors for specified catalysts. In general: (I) a diet high in protein (particularly the use of foods high in leucine, such as sorghum or maize) reduces the biosynthesis of niacin; (ii) a diet high in unsaturated fat increases it, while soaked unsaturated fats have no effect; (iii) the proportion of people who consume fewer carbohydrates that contain starch in relation to diets high in sucrose is higher; (iv) calorie restriction severely suppresses biosynthesis. [43]

The best-characterized KP modulators among substances are thyroxine, glucorticoids, and estrogens. TDO action is enhanced by estrogens; in women who are pregnant or using oral contraceptives, protein action is tripled. By triggering TDO through a component that is amplified by glucagon and inhibited by insulin and adrenaline, glucocorticoids energize union once more. Thyroxine's effects on TDO movement are still debatable because some studies found no effect, while others suggested a favorable activity.

[44] Due to individual differences, it has been determined that Trp is entirely replaced by niacin in healthy human beings, with a typical transformation efficacy of 60:1. Therefore, niacin equivalents (NE; 1 mg NE = 1 mg niacin or 60 mg Trp) are used to convey niacin admissions: Dietary Recommendations for Adults should take 16 mg of NE per day for men and 14 mg per day for women, with an acceptable upper admission level of 35 mg per day, since flushing is the primary adverse effect. [45]

THE MECHANISMS OF ACTION:- 1) The niacin is multifaceted, with several components that have not been fully explained.3 Lipids and apolipoprotein B (apo B)-containing lipoproteins can be reduced by niacin through modifying lipolysis in adipose tissue or fatty material amalgamation in the liver, which deteriorates apo B.[46] Niacin inhibits diacylglycerol acyltransferase-

2.3 in hepatocytes. This action limits the amount of fatty substances that are accessible to very low thickness lipoproteins (VLDL) by preventing the last stage of fatty oil union in hepatocytes.3 This movement also leads to decreased production of low thickness lipoproteins, which are the catabolic effect of VLDL, and intracellular debasement of apo B.[47]. A high-thickness lipoprotein (HDL) catabolismreceptor is also suppressed by niacin, increasing HDL levels and half-life.

METABOLISM:- The metabolites niacinamide, niacinamide N-oxide, nicotinuric corrosive, N1- methyl-2-pyridone-5-carboxamide, N1-methyl-4-pyridone-5-carboxamide, and trigonelline have been identified in human urine, although the writing does not clearly illustrate how niacin is digested.[48] The physiological function of niacin is as a precursor to the coenzymes of nicotinamide nucleotides, NADP and NAD. As quinolinic corrosive, a middle-of-the-road in the oxidative pathway of tryptophan digestion, can lead to the nicotinamide nucleotides, the conventional opinion is that tryptophan can 'fill in' for niacin when dietary admittance of the nutrient is low. In certain clinical situations, pellagra can be caused by disturbed tryptophan digestion even when niacin intake is clearly enough.[49]

PHARMACOLOGY ACTIVITY OF NIACINAMIDE:-

1. .Anti-inflammatory Properties: Studies like "Nicotinamide: A B vitamin that improves inflammatory skin conditions" that were published in the Journal of Clinical and Aesthetic Dermatology have shown that niacinamide can prevent inflammation. [50]

2. Antioxidant Effects: "The role of nicotinamide in oxidative stress and aging" discusses the function of niacinamide in cellular energy production and redox processes. [51]

3. Skin Barrier Function: "Nicotinamide increases biosynthesis of ceramides as well as other stratum corneum lipids to improve the epidermal permeability barrier" has research on howniacinamide improves ceramide formation. [52]

4. .Anti-aging benefits:-Research such as “Topical niacinamide reduces fine lines and wrinkles”[53]

5. Photoprotection: The article "Nicotinamide enhances repair of UV-induced DNA damage and reduces photodamage" from Photodermatology, Photoimmunology & Photomedicine goes into detail on niacinamide's photoprotective qualities. [54]

6. Serbm Regulation: "Nicotinamide: A potential treatment for acne" discusses how niacinamide affects sebum production. [55]

7. .Potential effects on depression:-The possible impact on "Nicotinamide and its effects on mood and depression" examines the possible mood-regulating benefits of niacinamide. [56]

NIACINAMIDE USE IN DEPRESSION:-

The use of complementary therapies, such as vitamins and supplements, to treat mental health issues like depression has grown in popularity in recent years. Niacin, a kind of vitamin B3, is one supplement that has drawn interest due to its possible function in treating depressive symptoms. In order to determine whether niacin is a safe and effective treatment for depression, let's examine the studies on the subject in more detail. By making more serotonin available in your body, a neurotransmitter that controls mood, appetite, and sleep, niacin may help alleviate depression indirectly. Although there is a dearth of research on this subject, a number of case studies and firsthand accounts have documented that consuming niacin has helped with a variety of mental health issues**.** **[**57**]**

Extreme sadness and hopelessness are hallmarks of depression, a mental condition that can interfere with day-to-day functioning. According to some depressed individuals, vitamin B-3 has been helpful. Some claim it lowers feelings of despair and hopelessness, and others say it made their depression completely go away. Depression has many different causes and therapies.

Scientific studies, however, have shown no proof that niacin can be used to treat depression at this time. Nonetheless, there is some evidence that suggests a B vitamin deficiency in depressed individuals. You should talk to your doctor about taking supplements or consuming foods that contain niacin if you're depressed. [58]

Additionally, there is evidence that those who suffer from depression can be lacking in B vitamins, such as niacin. However, niacin deficiency is not present in all depressed people.

Genetics, stressful life experiences, drugs, and hormones are just a few of the numerous possible reasons of depression. Similar to the causes, depression can be treated in a variety ofmethods. [59]

NIACINAMIDE BENEFITS FOR DEPRESSION CONDITION :-

1.Reduce depressive symptom.

2. Improve mood stability

3. Enhances emotional well being

4. Protects against neuronal damage
5. inflammation and oxidative stress
6. Supports brain health
7. Improves sleep quality
8. Reduces anxiety symptoms [60 – 67]

TREATMENT :-

A dose of about 20 mg may be sufficient to treat vitamin B-3 deficiency, but a significantly greater dosage may occasionally be required to treat severe depression. People with severe depression who respond to niacin therapy typically benefit from a significantly greater dose, ranging from 1,000 to 3,000 mg, according to internet testimonies. Food Matters, a 2008 nutrition documentary, claims that one woman's symptoms of sadness were alleviated after taking 11,500 mg daily

There is insufficient scientific evidence to back up these assertions or provide a precise dosage. It's crucial to start out slowly

and gradually raise the dosage if you choose to try niacin supplements. Since everyone responds to niacin differently, consult your doctor before starting any experiments. Overuse of this vitamin can have negative effects and be dangerous. [68]

Figure No 6 :- Capsule

VITAMIN B3`s EFFECTIVENESS FOR THE TREATMENT OF DEPRESSION:-

Ref	Condition	n	Protocol	Outcome	Type of Study
69	Various type of depression	15	Niacin was administered to each patient as a supplement to psychotherapy. Ten patients received 300–400 mg of niacin intravenously, and then oral niacin. Five additional patients did not get a first intravenous dose of niacin; instead, they received it orally. Before meals, all patients got progressively higher dosages of niacin until they reached 900 mg per day; however, one patient received 2,500 mg per day. For seven to ten days, all patients were kept on their highest daily dosage before the dosage was progressively reduced. Treatment with niacin lasted an average of two to six weeks.	After taking niacin together with psychotherapy, 14 out of the 15 patients showed both subjective and objective improvement.	Case series

70	Various type of depression	16	Eleven patients received divided dosages of 450–600 mg of niacin per day for the first week, followed by 900 mg per day for the next two weeks. Then the treatment was abruptly stopped. In the same way as the patients who received niacin, five patients received identical-looking placebo pills and no niacin. Patients were also administered nightly sedatives when needed.	No benefits wasobserved	Case series
71	Patients with a mixture of depressive and anxiety symptoms.	100	Patients received an elixir or tablet containing a combination of phenobarbital and niacin. There were 100 mg of niacin and 8 mg of phenobarbital in each tablet or 5cc of the elixir. Daily dosages of the combination were increased for all patients until day 15, when 900 mg of niacin and 72 mg of phenobarbital were achieved. The combination's daily dosages were lowered to 450 mg of niacin and 36 mg of phenobarbital between days 16 and 21. After day 21, the combination was presumably stopped.	5.4% of patients stopped treatment, 47.2% claimed definite improvement, 34.0% reported some improvement, and 13.2% reported no change.	Case series
72	Unipolar depression.	27	Patients were divided into two groups at random: group 1 underwent two weekly unilateral electroconvulsive therapy (ECT) treatments (a minimum of eight); group 2.	On day 10, group 2 outperformed group 1 (Beck scores: group 2, 15.2, and group 1, 16.8). The results reached statistical significance by	Human pilot open- label trial.

			received daily doses of 1 g of niacinamide and 3 g of tryptophan. A total of 12 patients in group 2 and 13 patients in group 1 finished the experiment. The day before the trial started, as well as on days 3, 7, 10, 14, 17, 21, 24, and 28, the Beck self-rating scale for depression was employed. Group 1's mean baseline Beck score was 25.6, whereas Group 2's mean baseline Beck score was 24.4.	day 21 (groups 1 and 2 Beck scores were 8.8 and 3.7, respectively; $p<0.05$). On day 28, the scores for each group were nearly the same.	
73	Newly admitted depressed patients with primary affective disorder	11	For four weeks, patients were given a mixture of tryptophanniacinamide. During week 1, patients received 2 g L-tryptophan and 0.5 g niacinamide; at the beginning of week 3, this dosage was progressively increased to 6 g L-tryptophan and 1.5 g niacinamide. If diazepam was required for agitation or sleeplessness, it was given to every patient. Patients' mental health was assessed using a Clinical Global Impression Scale of Severity of Depression (CGI) and a modified Hamilton Depression Rating Scale prior to therapy as well as on days 7, 14, 21, and 28. Additionally, at the same times, the Beck Depression Inventory was filled out.	The mean scores of all patients across all inventory showed statistically significant improvements (i.e., reductions) ($p<0.01$). On day 0, the average Hamilton score was 33.7; on day 28, it was 20.5. On day 0, the Beck score was 33.1; on day 28, it was 20.9. On day 0, the CGI was 7.2; on day 28, it was 4.5. Three marked respondents (50% or more), four moderate respondents (25–49%), and four non-responders (<25%) were based on the Hamilton scale's percentage improvement.	Human pilot open-label trial.

CONCLUSION :-

Niacinamide, sometimes called 3pyridinecarboxamide or Nicotinamide, is the physiologically active form of niacin, or vitamin B3. Certain cutaneous signs are indicative of the benign illness pellagra, which is caused by a vitamin shortage. When utilized as an effective specialist, this fundamental water-solvent nutrient has been connected to numerous dermatological systems and advantages since its discovery and isolation. These include its well-known function as a suppressor of the dynamics of skin inflammation, an enhancer of the sphingolipid blend on the skin's surface, an enhancer of markers of dermal expansion and epidermal separation (with simultaneous benefits to the layer corneum hindrance), and a mediator of photoimmune suppression and growth initiation.

Nicotinamide, vitamin B3, lessens the symptoms of depression. According to this abstract, niacinamide may help alleviate depression by regulating adenosine triphosphate (ATP) levels. The study found that niacinamide improved mice's depressed behaviors in addition to increasing SIRT1 activity. Impramine and tryptophan-nicotinamide combined are used to treat depression. Depression and niacin intake are linked. This abstract suggests that, in the general American population, there is a U-shaped relationship between niacin intake and depression. Men, Mexican Americans, white adults, those under 40, and people with BMIs under 30 were also shown to have a comparable association.

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