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Anti-Depressant activity of flowers and fruit extracts of *cassytha filiformis*.

P.Poli Reddy*, Aerpula Dharani, Edla Mounika, Pakala Gayatri, Bheema Naik Angothu, N.Shiva Krishna, and K.N.V.Rao

Nalanda college of Pharmacy, Cherla Pally, Nalgonda, TS-508001

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*Corresponding Author Email: polireddy0004@gmail.com

ABSTRACT
Anti-depressant activity of flowers and fruit extracts of *cassythafiliformis*. The present study attempts to study the anti-depressant activity of cassythafiliformis aqueous extract by using forced swim test. Animals were grouped into 4 treatment groups (N=4) and injected with normal saline (control), amitriptyline10 mg/kg (standard drug), cassythafiliformis aqueous extract of 100&200mg/kg (test drug) orally. Duration of immobility was observed for forced swim test by comparing with the control and standard drug. The result showed that in forced swim test the aqueous extract of cassythafiliformis at dose of 200mg/kg significantly reduced immobility time in acute studies and further reduced in chronic studies. However, the effect was increased in swimming and significantly changed in climbing in acute studies and increased in duration of swimming and climbing in chronic studies of forced swim test. The aqueous extract of cassythafiliformis possesses dose dependent anti- depressant effect. The current study confirms that antidepressants activity of selected dose (200 mg/kg).

KEY WORDS:
Anti- depressant activity, Forced swim test, Preliminary phytochemical constituents, Amitriptyline, Neurotransmitter.

1. INTRODUCTION:

Antidepressants are a class of medication used to treat major depressive disorder, anxiety disorder, chronic pain, and addiction. Common side effects of antidepressants include dry mouth, weight gain, dizziness, headeache, akathisia, sexual dysfunction, and emotional bunting. There is an increased risk of suicidal thinking and behaviour when taken by children, adolescents, and young adults. Discontinuation syndrome, which resembles recurrent depression in the case of the SSRI class, may occur after stopping the intake of any antidepressant, having effects which may be permanent and irreversible.

Research regarding the effectiveness of antidepressants for depression indults is controversial and has found both benefits and drawbacks. Meanwhile, evidence of benefit in children and adolescents is unclear. even though antidepressant use has considerably increased in children and adolescents in the 2000s. While a 2018 study found that the 21 most commonly prescribed antidepressant medications were slightly more effective than placebos for the short-term (acute) treatments of adults with major depressive disorder. Other research has found that the placebo effect may account for most or all the drugs' observed efficacy.

Research on the effectiveness of antidepressants is generally done on people who have severe symptoms, a population that exhibits much weaker placebo responses. meaning that the results may not be extrapolated to the general population that has not (or has not yet) been diagnosed with anxiety or depression.

1.1.1. Major depressive disorder:

The UK National Institute for Health and Care Excellence (NICE)'s 2022 guidelines indicate that antidepressants should not be routinely used for the initial treatment of mild depression, "unless that is the person's preference". The guidelines recommended that antidepressant treatment be considered:

- For people with a history of moderate or severe depression.
- For people with mild depression that has been present for an extended period.
- As a first-line treatment for moderate to severe depression.

- As a second-line treatment for mild depression that persists after other interventions.

The guidelines further note that in most cases, antidepressants should be used in combination with psychosocial interventions and should be continued for at least six months to reduce the risk of relapse and that SSRIs are typically better tolerated than other antidepressants.

American Psychiatric Association (APA) treatment guidelines recommend that initial treatment be individually tailored based on factors including the severity of symptoms, co-existing disorders, prior treatment experience, and the person's preference. Options may include antidepressants, psychotherapy, electroconvulsive therapy (ECT), transcranial magnetic stimulation (TMS), or light therapy. The APA recommends antidepressant medication as an initial treatment choice in people with mild, moderate, or severe major depression, and that should be given to all people with severe depression unless ECT is planned.

Reviews of antidepressants generally find that they benefit adults with depression. On the other hand, some contend that most studies on antidepressant medication are confounded by several biases: The lack of an active placebo, which means that many people in the placebo arm of a double-blind study may deduce that they are not getting any true treatment, thus destroying double-blindness; a short follow up after termination of treatment; non-systematic recording of adverse effects; very strict exclusion criteria in samples of patients; studies being paid for by the industry; selective publication of results. This means that the small beneficial effects that are found may not be statistically significant.

Among the 21 most commonly prescribed antidepressants, the most effective and well tolerated are escitalopram, paroxetine, sertraline, agomelatine, and mirtazapine. For children and adolescents with moderate to severe depressive disorder, some evidence suggests fluoxetine (either with or without cognitive behavioral therapy) is the best treatment, but more research is needed to be certain. Sertraline, escitalopram, and duloxetine may also help reduce symptoms.

A 2023 systematic review and meta-analysis of randomized controlled trials of antidepressants for major depressive disorder found that the medications provided only small or doubtful benefits in terms of quality of life. Likewise, a 2022 systematic review and meta-analysis of randomized controlled trials of antidepressants for major depressive disorder in children and adolescents found small improvements in quality of life. Quality of life as an outcome measure is often selectively reported in trials of antidepressants.

1.1.2. Anxiety disorders:

For children and adolescents, fluvoxamine is effective in treating a range of anxiety disorders. Fluoxetine, sertraline, and paroxetine can also help with managing various forms of anxiety in children and adolescents.

Meta-analyses of published and unpublished trials have found that antidepressants have a placebo-subtracted effect size (standardized mean difference or SMD) in the treatment of anxiety disorders of around 0.3, which equates to a small improvement and is roughly the same magnitude of benefit as their effectiveness in the treatment of depression. The effect size (SMD) for improvement with placebo in trials of antidepressants for anxiety disorders is approximately 1.0, which is a large improvement in terms of effect size definitions. In relation to this, most of the benefit of antidepressants for anxiety disorders is attributable to placebo responses rather than to the effects of the antidepressants themselves.

1.1.3. Generalized anxiety disorder:

Antidepressants are recommended by the National Institute for Health and Care Excellence (NICE) for the treatment of generalized anxiety disorder (GAD) that has failed to respond to conservative measures such as education and self-help activities. GAD is a common disorder in which the central feature is excessively worrying about numerous events. Key symptoms include excessive anxiety about events and issues going on around them and difficulty controlling worrisome thoughts that persist for at least 6 months.

Antidepressants provide a modest to moderate reduction in anxiety in GAD. The efficacy of different antidepressants is similar. Social anxiety disorderedit. Some antidepressants are used as a treatment for social anxiety disorder, but their efficacy is not entirely convincing, as only a small proportion of antidepressants showed some effectiveness for this condition. Paroxetine was the first drug to be FDA-approved for this disorder. Its efficacy is considered beneficial, although not everyone responds favorably to the drug. Sertraline and fluvoxamine extended-release were later approved for it as well, while escitalopram is used off-label with acceptable efficiency. However, there is not enough evidence to support Citalopram for treating social anxiety disorder, and fluoxetine was no better than a placebo in clinical trials. SSRIs are used as a first-line treatment for social anxiety, but they do not work for everyone.

One alternative would be venlafaxine, an SNRI, which has shown benefits for social phobia in five clinical trials against a placebo, while the other SNRIs are not considered particularly useful for this disorder as many of them did not undergo testing for it. As of 2008, it is unclear if duloxetine and desvenlafaxine can provide benefits for people with social anxiety. However, another class of antidepressants called MAOIs are considered effective for social anxiety, but they come with many unwanted side effects and are rarely used. Phenelzine was shown to be a good treatment option, but its use is limited by dietary restrictions. Moclobemide is a RIMA and showed mixed results, but still received approval in some European countries for social anxiety disorder. TCA antidepressants, such as clomipramine and imipramine, are not considered effective for this anxiety disorder in particular. This leaves out SSRIs such as paroxetine, sertraline, and fluvoxamine CR as acceptable and tolerated treatment options for this disorder.

1.1.4. Obsessive–compulsive disorder:

SSRIs are a second-line treatment for adult obsessive–compulsive disorder (OCD) with mild functional impairment, and a first-line treatment for those with moderate or severe impairment.

In children, SSRIs are considered as a second-line therapy in those with moderate-to-severe impairment, with close monitoring for psychiatric adverse effects. Sertraline and fluoxetine are effective in treating OCD for children and adolescents.

Clomipramine, a TCA drug, is considered effective and useful for OCD. However, it is used as a second-line treatment because it is less well-tolerated than SSRIs. Despite this, it has not shown superiority to fluvoxamine in trials. All SSRIs can be used effectively for OCD. SNRI use may also be attempted, though no SNRIs have been approved for the treatment of OCD. Despite these treatment options, many patients remain symptomatic after initiating the medication, and less than half achieve remission.

Placebo responses are a large component of the benefit of antidepressants in the treatment of depression and anxiety. However, placebo responses with antidepressants are lower in magnitude in the treatment of OCD compared to depression and anxiety. A 2019 meta-analysis found placebo improvement effect sizes (SMD) of about 1.2 for depression, 1.0 for anxiety disorders, and 0.6 for OCD with antidepressants.

1.1.5. Post–traumatic stress disorder:

Antidepressants are one of the treatment options for PTSD. However, their efficacy is not well established. Paroxetine and sertraline have been FDA approved for the treatment of PTSD. Paroxetine has slightly higher response and remission rates than sertraline for this condition. However, neither drug is considered very helpful for a broad patient demographic. Fluoxetine and venlafaxine are used off-label. Fluoxetine has produced unsatisfactory mixed results. Venlafaxine showed response rates of 78%, which is significantly higher than what paroxetine and sertraline achieved. However, it did not address as many symptoms of PTSD as paroxetine and sertraline, in part due to the fact that venlafaxine is an SNRI. This class of drugs inhibits the reuptake of norepinephrine, which may cause anxiety in some patients. Fluvoxamine, escitalopram, and citalopram were not well-tested for this disorder. MAOIs, while some of them may be helpful, are not used much because of their unwanted side effects. This leaves paroxetine and sertraline as acceptable treatment options for some people, although more effective antidepressants are needed.

1.1.6. Panic disorder:

Panic disorder is treated relatively well with medications compared to other disorders. Several classes of antidepressants have shown efficacy for this disorder, with SSRIs and SNRIs used first-line. Paroxetine, sertraline, and fluoxetine are FDA-approved for panic disorders, while fluvoxamine, escitalopram, and citalopram are also considered effective for them. SNRI venlafaxine is also approved for this condition. Unlike social anxiety and PTSD, some TCAs antidepressants, like clomipramine and imipramine, have shown efficacy for panic disorder. Moreover, the MAOI phenelzine is also considered useful. Panic disorder has many drugs for its treatment. However, the starting dose must be lower than the one used for major depressive disorder because people have reported an increase in anxiety because of starting the medication. In conclusion, while panic disorder treatment options seem acceptable and useful for this condition, many people are still symptomatic after treatment with residual symptoms.

1.2. CLASSIFACTION

1.2.1. Selective serotonin reuptake inhibitors.

Blister pack of Prozac (fluoxetine) a inhibitor. Selective (SSRIs) are believed to increase the extracellular level of the neurotransmitter serotonin by limiting its reabsorption into the presynaptic cell, increasing the level of serotonin in the synaptic cleft available to bind to the postsynaptic receptor. They have varying degrees of selectivity for the other monoamine transporters, with pure SSRIs having only weak affinity for the norepinephrine and dopamine transporters.
SSRIs are the most widely prescribed antidepressants in many countries. The efficacy of SSRIs in mild or moderate cases of depression has been disputed.

1.2.2. Serotonin–norepinephrine reuptake inhibitors:
The chemical structure of venlafaxine (Effexor),an SNRI. Serotonin–norepinephrine reuptake inhibitors (SNRIs) are potent inhibitors of the reuptake of serotonin and norepinephrine. These neurotransmitters are known to play an important role in mood. SNRIs can be contrasted with the more widely used selective serotonin reuptake inhibitors (SSRIs), which act mostly upon serotonin alone.
The human serotonin transporter (SERT) and norepinephrine transporter (NET) are membrane proteins that are responsible for the reuptake of serotonin and norepinephrine. Balanced dual inhibition of monoamine reuptake may offer advantages over other antidepressants drugs by treating a wider range of symptoms. SNRIs are sometimes also used to treat anxiety disorders, obsessive–compulsive disorder (OCD), attention deficit hyperactivity disorder (ADHD), chronic neuropathic pain, and fibromyalgia syndrome (FMS), and for the relief of menopausal symptoms.

1.2.3. Serotonin Modulators and Stimulators:
Serotonin modulator and stimulators (SMSs), sometimes referred to more simply as "serotonin modulators", are a type of drug with a multimodal action specific to the serotonin neurotransmitter system. To be precise, SMSs simultaneously modulate one or more serotonin receptors and inhibit the reuptake of serotonin. The term was coined in reference to the mechanism of action of the serotonergic antidepressant vortioxetine, which acts as a serotonin reuptake inhibitor (SRI), a partial agonist of the 5-HT1A receptor, and antagonist of the 5-HT3 and 5-HT7 receptors. However, it can also technically be applied to vilazodone, which is an antidepressant as well and acts as an SRI and 5-HT1A receptor partial agonist.
An alternative term is serotonin partial agonist/reuptake inhibitor (SPARI), which can be applied only to vilazodone.

1.2.4. Serotonin antagonists and reuptake inhibitors.
Serotonin antagonist and reuptake inhibitors (SARIs) while mainly used as antidepressants are also anxiolytics and hypnotics. They act by antagonizing serotonin receptors such as 5-HT2A and inhibiting the reuptake of serotonin, norepinephrine, and/or dopamine. Additionally, most also act as α1-adrenergic receptor antagonists. The majority of the currently marketed SARIs belong to the phenylpiperazine class of compounds. They include trazodone and nefazodone.

1.2.5. Tricyclic antidepressants:
The majority of the tricyclic antidepressants (TCAs) act primarily as serotonin–norepinephrine reuptake inhibitors (SNRIs) by blocking the serotonin transporter (SERT) and the norepinephrine transporter (NET), respectively, which results in an elevation of the synaptic concentrations of these neurotransmitters, and therefore an enhancement of neurotransmission. Notably, with the sole exception of amine tine, the TCAs have weak affinity for the dopamine transporter (DAT), and therefore have low efficacy as dopamine reuptake inhibitors (DRIs).
Although TCAs are sometimes prescribed for depressive disorders, they have been largely replaced in clinical use in most parts of the world by newer antidepressants such as selective serotonin reuptake inhibitors (SSRIs), serotonin–norepinephrine reuptake inhibitors (SNRIs), and norepinephrine reuptake inhibitors (NRIs). Adverse effects have been found to be of a similar level between TCAs and SSRIs.

1.2.6. Tetra cyclic antidepressants:
Tetra cyclic antidepressants (TeCAs) are a class of antidepressants that were first introduced in the 1970s. They are named after their chemical structure, which contains four rings of atoms, and are closely related to tricyclic antidepressants (TCAs), which contain three rings of atoms.

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1.2.7. Monoamine oxidase inhibitors:

Monoamine oxidase inhibitors (MAOIs) are chemicals that inhibit the activity of the monoamine oxidase enzyme family. They have a long history of use as medications prescribed for the treatment of depression. They are particularly effective in treating atypical depression. They are also used in the treatment of Parkinson's disease and several other disorders.

Because of potentially lethal dietary and drug interactions, MAOIs have historically been reserved as a last line of treatment, used only when other classes of antidepressant drugs (for example selective serotonin reuptake inhibitors and tricyclic antidepressants) have failed.

MAOI have been found to be effective in the treatment of panic disorder with agoraphobia, social phobia, atypical depression or mixed anxiety and depression, bulimia, and post-traumatic stress disorder, as well as borderline personality disorder. MAOIs appear to be particularly effective in the management of bipolar depression according to a retrospective-analysis. There are reports of MAO efficacy in obsessive–compulsive disorder (OCD), trichotillomania, dysmorphophobia, and avoidant personality disorder, but these reports are from uncontrolled case reports.

MAOIs can also be used in the treatment of Parkinson's disease by targeting MAO-B in particular (therefore affecting dopaminergic neurons), as well as providing an alternative for migraine prophylaxis. Inhibition of both MAO-A and MAO-B is used in the treatment of clinical depression and anxiety disorders.

1.2.8. NMDA receptor antagonists.

NMDA receptor antagonists like ketamine and esketamine are rapid-acting antidepressants and seem to work via blockade of the ionotropic glutamate receptor. Other NMDA antagonists may also play a role in treating depression. The combination medication dextromethorphan/bupropion (Auvelity), which contains the NMDA receptor antagonist dextromethorphan, was approved in the United States in 2022 for treating major depressive disorder.

1.2.9. PHARMACOLOGY:

Antidepressants act via a large number of different mechanisms of action. This includes serotonin reuptake inhibition (SSRIs, SNRIs, TCAs, vilazodone, vortioxetine), norepinephrine reuptake inhibition (NRIs, SNRIs, TCAs), dopamine reuptake inhibition (bupropion, amineptine, nomifensine), direct modulation of monoamine receptors (vilazodone, vortioxetine, SARIs, agomelatine, TCAs, TeCAs, antipsychotics), monoamine oxidase inhibition (MAOIs), and NMDA receptor antagonism (ketamine, esketamine, dextromethorphan), among others (e.g., brexanolone, tianeptine).Some antidepressants also have additional actions, like sigma receptor modulation (certain SSRIs, TCAs, dextromethorphan) and antagonism of histamine H1 and muscarinic acetylcholine receptors (TCAs, TeCAs).

The earliest and most widely known scientific theory of antidepressant action is the monoamine hypothesis, which can be traced back to the 1950s and 1960s. This theory states that depression is due to an imbalance, most often a deficiency, of the monoamine neurotransmitters, namely serotonin, norepinephrine, and/or However, serotonin in particular has been implicated, as in the serotonin hypothesis of depression. The monoamine hypothesis was originally proposed based on observations that reserpine, a drug which depletes the monoamine neurotransmitters, produced depressive effects in people, and that certain hydrazine antituberculosis agents like iproniazid, which prevent the breakdown of monoamine neurotransmitters, produced apparent antidepressant effects. Most currently marketed antidepressants, which are monoaminergic in their actions, are theoretically consistent with the monoamine hypothesis. Despite the widespread nature of the monoamine hypothesis, it has several limitations: for one, all monoaminergic antidepressants have a delayed onset of action of at least a week; and secondly, many people with depression do not respond to monoaminergic antidepressants. Several alternative hypotheses have been proposed, including hypotheses.

Involving glutamate, neurogenesis, epigenetics, cortisol hypersecretion, and inflammation, among others.

In 2022, a major systematic umbrella review by Joanna Moncrieff and colleagues showed that the serotonin theory of depression was not supported by evidence from a wide variety of areas. The authors concluded that there is no association between serotonin and depression, and that there is no evidence that strongly supports the theory that depression is caused by low serotonin activity or concentrations. Other literature had described the lack of support for the theory previously in many of the expert responses to the review, it was stated that the monoamine hypothesis had already long been abandoned by psychiatry. This is in spite of about 90% of the general public in Western

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countries believing the theory to be true and many in the field of psychiatry continuing to promote the theory up to recent times. In addition to the serotonin umbrella review, reviews have found that reserpine, a drug that depletes the monoamine neurotransmitters including serotonin, norepinephrine, and dopamine shows no consistent evidence of producing depressive effects. Instead, findings of reserpine and mood are highly mixed, with similar proportions of studies finding that it has no influence on mood, produces depressive effects, or has antidepressant effects. In relation to this, the general monoamine hypothesis, as opposed to only the serotonin theory of depression, likewise does not appear to be well-supported by evidence.

The serotonin and monoamine hypotheses of depression have been heavily promoted by the pharmaceutical industry (e.g., in advertisements) and by the psychiatric profession at large despite the lack of evidence in support of them. In the case of the pharmaceutical industry, this can be attributed to obvious financial incentives, with the theory creating a bias against non-pharmacological treatments for depression.

An alternative theory for antidepressant action proposed by certain academics such as Irving Kirsch and Joanna Moncrieff is that they work largely or entirely via placebo mechanisms. This is supported by meta-analyses of randomized controlled trials of antidepressants for depression, which consistently show that placebo groups in trials improve about 80 to 90% as much as antidepressant groups on average and that antidepressants are only marginally more effective for depression than placebos. The difference between antidepressants and placebo corresponds to an effect size (SMD) of about 0.3, which in turn equates to about a 2- to 3-point additional improvement on the 0–52-point (HRSD) and 0–60-point (MADRS) depression rating scales used in trials. Differences in effectiveness between different antidepressants are small and not clinically meaningful. The small advantage of antidepressants over placebo is often statistically significant and is the basis for their regulatory approval, but is sufficiently modest that its clinical significance is doubtful. Moreover, the small advantage of antidepressants over placebo may simply be a methodological artifact caused by unblinding due to the psychoactive effects and side effects of antidepressants, in turn resulting in enhanced placebo effects and apparent antidepressant efficacy. Placebos have been found to modify the activity of several brain regions and to increase levels of dopamine and endogenous opioids in the reward pathways.

1.3. CASSYTHA FILIFORMIS:

Cassythafiliformis or love-vine is an orangish, wiry, parasitic vine in the family Lauraceae. It is found In warm tropical regions worldwide including the Americas, Indomalaya, Australasia, Polynesia and tropical Africa.

Vines.

- Cassythafiliformis is a twining vine with yellow or orange to pale green hollow stems with a length between 3–8 metres long. The stems attach to host plants by growing shoots from the base of its root, they have haustoria that fold inside the hosts' phloem and xylem membranes to absorb water a

Clump of C.filiformis on Florida Rosemary, Southwest florida.

Leaves are reduced to scales about 1 mm long and can be seen near stem ends.

1.3.1. SCIENTIFIC CLASSIFICATION:

KINGDOM: Plantae.
CLADE: Trachophytes,Angiosperms, magnoliids.
ORDER: Laurales.
FAMILY: lauraceae.
GENUS: Cassytha.
SPECIES: C.filiformis.
BINOMINALNAME: Cassythafiliformis.
SYNONYMS: Cassythaamericananees.

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TELUGUNAME: Passiteega.

1.03.2. Flowers and fruit:

Flowers are borne in spikes 1–2 cm long from short stalks or sometimes solitary. There are six curved inward tepals made of 3 outer oval ones 1 mm long and three inner ones 2.5 mm long. Each one has smooth (glabrous) and broad stamens with short pointy ends forming into a beak shape.


Fruit is a round and green or whitish drupe about 7 mm in diameter. Its juicy flesh is eaten and dispersed by birds.
Cassytha is a genus of some two dozen species of obligately parasitic vines in the family Lauraceae. Superficially, and in some aspects of their ecology, they closely resemble plants in the unrelated genus Cuscuta, the dodders. When fruit and flowers are absent in the field, the physical resemblance is so close that few people without technical training can discern the difference. In this respect and in their ecology the two genera present a spectacular example of convergent evolution.Nonetheless, Nickrent comments that "Cassytha is uneqivocally assigned to Lauraceae based on (both) morphological and molecular data." In its divergence from habits typical of the Lauraceae, Cassytha also presents examples of mosaic evolution.
Several species of Cassytha are regarded as pests in various regions, though as a rule they are not as serious a problem as the true dodders. Some even yield a welcome harvest of fruit or are valued for their perceived medicinal or aphrodisiac properties, partly because, like many members of the Lauraceae, some are fragrant when bruised. Their stems make useful strings for construction of thatched roofs and certain styles of lei and the like.
Probably the most useful common names for Cassytha species are laurel dodder or dodder laurel, because they look like dodder and are fragrant members of the laurel family, Lauraceae. The name love vine has merit because some species, in particular C. filiformis, are regarded as aphrodisiacs in the Caribbean region. In practice, the confusion between the various species of Cassytha and Cuscuta is so unavoidable that their common names are more or less interchangeable. Practically all the common names for dodder accordingly are widely applied in error to Cassytha as well, but as a matter of convenience in Florida at least, where members of both groups of plants are present as agricultural pests, a publication Of the deparrtment of the agriculture adopts the names woevine for cassythasnd dodder for cascuta.

1.3.3. Context and distribution:

Though the Lauraceae constitutes a large family, with thousands of species in tens of genera, Cassytha is its only known parasitic genus, and its climbing habit also is atypical of the family; most Lauraceae are woody shrubs or trees. The genus at one time was assigned its own family, Cassythaceae, but currently agreement on its inclusion into the Lauraceae is general.
As currently defined, Cassytha has a wide distribution for a genus of so few species. Most are native to Australia (including temperate regions, where they are the only native members of the family), but a few are indigenous to Africa, southern Asia, various islands, and regions in the Americas. Some species seem to have been spread inadvertently by humans and probably by birds as well, and now occur on several continents. C. filiformis, for example, grows in Hawaii (where it is said to be indigenous), the Australasian realm, northern South America, Central America, southern Florida, Japan, and South Africa. It also appears to have been transported to many major islands, and now is effectively pantropical.

1.3.4 Botanical details :

The genus is cited as Cassytha L., Sp. Pl. 35 (1753), which means that Carl Linnaeus formally described it in 1753 in his monumental work, Species Plantarum. Otto Stapf updated the work in Flora Capensis in 1912.

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Cassytha is unusual among Lauraceae in at least two respects: they are scandent herbaceous plants, and they are obligate parasites. Their stems are thread-like or wiry, and like most twining species, they twine round the host clockwise as seen from the source of growth. The vines generally turn yellowish once they have established themselves on a productive host because they then reduce or stop their production of chlorophyll. Cassytha species are stem parasites, adhering to their hosts by uniseriate haustoria that generally are small and oblong. Their leaves are without stipules, alternate, simple, and easily overlooked, being minute and scale-like.

Various species of Cassytha bear flowers in racemes, spikes, or heads. Depending on the species, the flowers are sessile or pedicellate. The individual flowers are hermaphroditic and bracteolate, each being attended by a bract and two smaller bracteoles. In general, the flowers are small, so much so that in many species they are inconspicuous. The perianth has six tepals, the three outer tepals smaller than the inner. The 12 stamens are in four whorls. The receptacle of the fertilised fruit gradually envelops the ovary, becoming the fleshy part of the ripe fruit, which often retains the dried remnants of the perianth at its tip. In effect, the resulting fruit structure is a tiny drupe. The endocarp is bony and plays an important part in the reproductive process, both in protecting the seed while the fruit is eaten and in inhibiting germination until the endocarp decays, thereby permitting long-lived soil seed banks to accumulate.

Some species of the cactus genus Rhipsalis once were assigned to Cassytha in an error arising from the resemblance in habitus. An unfortunate consequence has been that the homonym Cassytha Mill. (1768) is often mentioned as a synonym of the genus Rhipsalis, although this perception is incorrect, since the generic name Cassytha had already been applied to a completely different genus in a different plant family.

The morphology and ecology of Cassytha are so atypical of the family Lauraceae, they have been the subject of molecular genetic research to confirm their taxonomic relationships. Though special aspects to their phylogeny certainly are under debate, their assignment to the Lauraceae generally is regarded as undoubtedly correct.

1.3.5 Reproduction and ecology.

Cassytha fruits are ecologically valuable to some fruit-eating birds. The birds either regurgitate the seeds or pass them through their gut. Mammals, for example Australian macropods, also transport the seeds in their gut.The bony endocarp that protects the seed in its passage through animal gut also prevents the seed from immediate germination even if conditions are favourable. Instead, the seeds survive on or in the ground till decay weakens the endocarp sufficiently to permit moisture to enter and germination to begin. This process is not deterministic, so some of the seeds might remain inactive in the soil seed bank for many years before they germinate at unpredictable intervals. Accordingly, once soil is infested with large numbers of seeds, eradication of the population generally requires considerable time. On germination, the seedlings behave as aggressive parasites; they twist about till they find a host, and those that fail to locate hosts soon die, typically in months.

Seedlings and actively growing shoots are green at first. Once their haustoria are fully established on a suitable host, the plants lose most of their chlorophyll and generally become yellowish or orange, and the Cassytha plant abandons its connections with its root, which soon dies.

Cassytha species are perennials; although they attack practically whatever host plants they encounter, including suitable annuals. They seem to show some preference for woody perennial hosts. Consequently, they often find themselves on seasonally dormant host species. When that happens and the supplies from the host largely dry up, the stems of most species of Cassytha turn green until the host once again becomes productive. This suggests that such species are at least marginally photosynthetic, but do not invest resources unnecessarily when photosynthesis is not required. Cassytha species do produce some of their own nutrients while green, so their chlorophyll production is both actual and functional.

Technically, Cassytha could be regarded as hemiparasitic rather than holoparasitic, but their own autotrophic contributions are plainly limited to what it takes to tide over temporary shortages. When all the hosts of a plant die, so does the parasite, so whatever the details of their biology, Cassytha species certainly are unconditionally obligately parasitic. No doubt their lack of a persistent root system dooms any Cassytha plants whose hosts supply insufficient water and mineral nutrients.

2. REVIEW OF LITERATURE.

Masakenda et al reported by Medicinal plants used for anxiety, depression, or stress treatment: An update published online 2022 sep 15, molecules, 2022 sep; 27(18).;6021.

Depression, anxiety, stress, and other mental disorders, which are on the rise worldwide, are indications that pharmacological therapy can have serious adverse effects, Here, we reviewed plants and products derived from them that are commonly used for the above indications, focusing on clinical data and safety profiles. While lavender, hops, maypop, lemon balm, and valerian have consistently been shown in clinical trials to relieve mild forms of neurological disorders, particularly depression, anxiety, and stress, currently available data do not fully support the use of peppermint for anxiety disorders and depression. Recent studies support the use of saffron for depression; however, its toxicological profile raises safety concerns. St. John's wort is effective in alleviating mild to moderate depression, in conclusion, more studies are needed to validate the mechanism of action so that these plants can be used successfully and safely to alleviate or eliminate various mental disorders.
TalhaJawaid*, Roli Gupta and Zohaib Ahmed Siddiqui et al reported by a review on medicinal plants showing anti-depressant activity published on 30 nov 2011.
Depression is a heterogenous mood disorder that has been classified and treated in a variety of ways. Although several synthetic drugs are being used as standard treatment for clinically depressed patient, they have adverse effects that can compromise the therapeutic treatment. Thus, it is worthwhile to look for antidepressants from plants with proven advantage and favorable benefit to risk ratio. Several medicinal plants and medicine derived from these plants have shown antidepressant properties by virtue of combined effect of their medicinal constituents. The causes of depression are decreased brain levels of monoamines like noradrenaline, dopamine and serotonin. Therefore, drugs restoring the reduced levels of these monoamines in the brain either by inhibiting monoamine oxidase or by inhibiting reuptake of these neurotransmitters might be fruitful in the treatment of depression. The present review is focused on the medicinal plants and plants-based formulations having antidepressant activity in animal studies and in humans.
TahianaRamaholimihaso et al reported by curcumin in depression of potential mechanism of action published online 2020 nov 27.

AIM AND OBJECTIVE

Aim: Extraction & evaluation of biological activity of cassythafiliformis.
Objectives:

- Identification & authentication of plant material.
- Collection and shade range of plant material.
- Soxhlet extraction of powder plant material with suitable solvents methanol, chloroform, benzene ,N- hexane.
- Preliminary phytochemical of Phyto constituents.
- Screening of the extract for anti-depressant activity.

METHODOLOGY

Collection of plant material & extraction of fresh plantcassythafiliformis was collected in nalgonda& were authenticated by Dr. KNVenkateshwararao sir department of pharmacognosy. A vocher specimen was deposited in the college herbarium.The whole plant was shade dried and coarsely powdered. The coarse powder was subjected to extaction with solvents of methanol,chloroform, n-hexane ,benzene by soxhlet apparatus and extract was concentrated dryness in a vaccum .

4.1 Experimental design for anti-depressant activity.

- The rats were divided into 4 groups (N=6).
- Drugs/vehicle for administered to animals prior 60 min to study.
- Group-1: control administered saline 2mg/kg body weight orally.
- Group-2: Receive standard drug amitriptyline 10mg/kg for body weight orally.
- Group-3: Receive cassythafiliformis 100mg/kg body weight orally.
- Group-4: Receive cassythafiliformis 200mg/kg body weight orally.

4.2 Extraction procedure: collect the fresh aerial part of Cassythafillformis and shade dried the plant for 10 days.
*After dried the plant material and make into coarsely powder form sieved the powder.
*Take approximately 20g of casythafiliformis and extract with different solvents individually like methanol, n-hexane, chloroform, benzene by using Soxhlet apparatus.
*After collecting the individual filtrates of extraction.

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*Resulting extract were concentrated and evaporated to dryness using rotary evaporator at optimum temperature between 40&45degree celsis to avoid denaturation of active constituents.
*Then concentrated extracts are stored.

Fig1: Soxhlet extraction unit.

4.3.1. Preliminary phytochemical tests: A preliminary phytochemical investigation was carried out for all extracts by following the test methods.

1. Test for alkaloids:1.2mlof extract was taken in a test tube 0.2 ml of dilute hydrochloric acid and 0.1ml of Mayers reagent were added. Formation of yellowish buff colored precipitate gives positive test alkaloid.
2. 1.0ML of dilute hydrochloric acid and 0.1ml of dragendroffs reagent were added in 2ml solution of extract in test tube. The development of orange, brown coloured precipitate suggested the presence of alkaloid.
3. 2ml of extract solution was treated with dilute hydrochloric acid and 0.1 ml of wagers reagent. Formation of reddish-brown precipitate indicated the positive response for alkaloid. 2ml of extract was allowed to react with 0.2 ml of dilute hydrochloric acid and 0.1ml hangers reagent. A yellowish precipitate suggested the presence of alkaloid.

Test for amino acids:

- A small portion of all the extracts was individually dissolved in distilled water and treated with Ninhydrine at the pH range of 4-8.

4.3.2. Test for flavonoids & glycosides:

A small portion of all the extract was individually dissolved in ethanol and hydrolyzed with 10% sulphuric acid, cooled and extracted with diethyl ether and divided into three portions and treated as follows.

- 1ml of diluted sodium carbonate was added to the first test tube.
- 1ml of 0.1m sodium hydroxide was added to the second test tube.
- 1ml of diluted ammonia solution was added to the third test tube.

4.3.3. Test for anthroquinine glycosides:

A small portion of all extract was individually extracted with water immiscible organic solvent, filtered and made alkaline with caustic soda.

4.3.4. Test for steroids and triterpenoids:

a) **Libermann- Burchard test;** Asmall portion of all extracts was individually dissolved in chloroform and 1ml of acetic anhydrate was added to it. 2ml of concentration of sulphuric acid was added along the sides of the test tube.
b) **Nollers test:** A small portion of all the extract was individually dissloved in 2ml of 0.01% anhydrous stannic chloride in purely thionyl chloride.
c) **Salkowski test:** A small portion of all the extract was individually dissolved in choloform and few ml of conc. sulphuric acid was added to it.

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4.3.5 Test for reducing sugar:
A small portion of all the extract was individually dissolved in distilled water and filtered, to the filtrate add equal portions of Fehlings A &B solution and heated for few minutes.

4.3.6 Test for gums:
A small portion of all the extract was individually dissolved in a distilled water, filtered and added equal volume of concentrated sulphuric acid. 15% of alcoholic solution of alpha- naphthol (molishs reagent) was added to it.

4.3.7. Test for tannins:
A small portion of all the extracts was individually dissolved in distilled water and filtered. The filtrate was divided into three portions and treated as follows.

- To the first test tube 10% aqueous potassium dichromate solution was added- formation of yellow brown precipitate.
- To the second test tube 10% aqueous lead acetate solution was added formation of yellow precipitate.
- To the third test tube 1ml of 5% ferric chloride solution was added- formation of greenish black or bluish colour.

4.3.8. Test for saponins:
A small portion of all the extract was individually dissolved in small quantity of distilled water and shaken in a graduated cylinder for 5min.

4.4. Acute toxicity.
A number of 90 mice were divided into 4 groups, each group treated with ethanolic defatted extract of C. filiformis at doses of 25, 50, 100, 200 mg/kg. The number of animal death at 1, 2, 3, 24 and 48 hours were recorded to calculate LD50 on each time. Animal behavioral changes such as motoric activity, respiratory rate, and diarrhea were monitored.

Phytoconstituents	*Phytoconstituents extract of cassyta filiformis.*
GLYCOSIDES	+
SAPONINS	–
ALKALOIDS	+
TANNINS	+
FLAVANOIDS	+

4.5. Delayed Toxicity Study.
The survived animals from death on the acute toxicity study were monitored for the symptom(s) associated with the delayed toxicity, their body weight, 24 hour food intake and water intake for 14 days. At the end of experiment, the animals were sacrificed and their liver, heart and kidney were taken to measure their ratios to the body weight.

4.6. Experimental Animals.
Male wister rats, weighing 200-250gm were used in the study and fed with standard laboratory pellet to diet; provimi limited (india), provided water ad libitum and were maintained at 23-25C, 35-60% humidity, and 12h light /dark cycle. The rats were acclimatized to the laboratory conditions for a period of 7 days prior to the experiment. The experimental protocol (NCOP/VK/318/PO/Re/S/2001/CCSEA/01/2024) was duly approved by institutional animal ethics committee (IAEC).

4.7. ACUTE TOXICITY STUDIES.

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Acute toxicity studies are done by using OECD 423 annexure(D) acute toxic class method (OECD Guideline). This method is a step wise procedure with 3 animals of a single sex per step. The starting dose of Cassytha filiformis was 1000 mg/kg body weight p.o. using water as vehicle. Drug was administered to overnight fasted female rats. Food was withheld for 4hr after administration of Cassytha filiformis and observed for signs of toxicity.

4.8. INDUCTION OF DEPRESSION.

The rats were divided into 4 groups(n=4). The CF extract was dissolved in distilled water and administered orally at doses of 200&400mg/kg, 60min before conduction of the behavioral tests and the study was carried out for 14 days.

GROUP1; control, administered saline 2ml/kg orally.
GROUP2; FST+ Amitriptyline (10mg/kg orally).
GROUP3; FST+CF 100mg/kg orally.
GROUP4; FST+ CF 200mg/kg orally.

5.8 FORCED SWIMMING TEST(FST)

Rats were forced to swim individually in an open cylindrical tank (diameter 10cm,height 25cm), filled with 19cm of water at 25±10C. The total time that each rat remained immobile during a 6 min section was recorded as immobility time.

Immobility is judged when the rat ceases struggling and remains floating motionless in the water , only making necessary movements to remain its head above water. A decrease in the immobility duration is an indicator for anti-depressant effect.

5. RESULTS

Acute toxicity studies

The drug CF was found to be non-toxic, and the LD50 of 1000mgh/kg and above is said to be unclassified according to OECD 423.Hence, (100mg/kg) and (200mg/kg) of the dose were selected for further investigation.

Percentage of immobility time in forced swim test.

Sl.No.	Treatment.	Percentage immobility.			
		30min	60min	120min	240min
1	Control	50.5	52.4	51.3	50.2
2	Standard(10mg/kg)	29.6	31.3	30.4	29.5
3	Low dose Cassythafiliformis (100mg/kg)	15.5	16.2	16.5	17.2
4	High dose cassythafiliformis (200mg/kg)	10.2	9.3	7.6	5.9

6. DISCUSSION.

The present study states that administration of cassytha filiformis produced anti- depressant effect and prevented oxidative damage caused by forced swim test. Forced swimmed test predicted that efficacy of various anti-depressant and immobility behaviour of rats in forced swim test exactly resembles the state of depression in humans. The treatment with casytha filiformis significantly reduced immobility time in that dependent manner indicating anti-depressant activity. Further to validate anti- depressant activity it needed to be extended with other laboratory test oxidative stress involved in pathogenesis of various neuropsychiatric disorders including depression. A study also reported a involvement of radicals and pathogenesis of chronic depression. If oxidative stress generates reactive oxygen species and those in exit effect on signal transduction & neuroplasticity. Reactive oxygen species induce lipid peroxidation and causes damage to DNA& proteins. In order to confirm anti-depression activity of Cassytha filiformis forced swim test carried out which induce lipid peroxidation which was indicated by increasing immobility rate.

Anti-depressant activity access precipitated behaviour to most widely used method anti-depressant activity is forced swim test. It is quite sensitive and relatively a specific to all major classes of drugs.

In forced swim test immobility reflects a state of despair which can be reduced by several agents which are therapeutically effective in human depression.

Rats are forcefully swimmed in restricted space from which they cannot escape.

Results showed that the administration of Cassytha filiformis produced a diminution of the duration of immobility time of rats exposed to the forced swim test.

In present study Cassytha filiformis (200mg/kg p.o) close administered to the rats produced significant anti-depressant activity than (100mg/kg p.0) and their efficacies were found to be comparable to standard drug (10mg/kg p.o)

Flavonoid components are responsible for inhibiting the neurotransmitter& serotonin reuptake mechanism which mediate anti-depressant effect of Cassytha filiformis.

7. CONCLUSION

Cassytha filiformis, Amitriptyline, flowers and fruits of extract possess anti-depressant effect in animal models of depression which was comparable to that of standard drug as demonstrated in the study. The phytochemical screening reveals that presence of more number active constituents among which flavonoids are responsible anti-depressant activity. In presence study 200mg/kg body weight of Cassytha filiformis shows significantly decreasing immobility time 200mg/kg of the selected drug shows decreases immobility time. When compared with the control group of animals. It is needed to further evaluate with testing of biochemical parameters and histopathology studies for complete understanding of anti-depressant activity.

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