A Literature Review on the Effect of Changing Blood Tryptophan Concentration on Mood, in Particular Depression and How Concentration of Tryptophan can be Altered Through Diet and Supplements

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A Literature Review on the Effect of Changing Blood Tryptophan Concentration on Mood, in Particular Depression and How Concentration of Tryptophan can be Altered Through Diet and Supplements

Author(s): Mohammad M, Siddiqui M R

Abstract

Background: Tryptophan is an essential amino acid which acts as the precursor for Serotonin. Concentration of Tryptophan in the blood plasma has a direct effect on the concentration of Serotonin. A decrease in Serotonin in the brain may cause depressive symptoms. Studies have investigated the effect of changing Tryptophan intake and its effect on mood and psychological behaviour. The aim of this review is to investigate the effect of changing Tryptophan concentration in the blood through diet, and how this affects mood, in particularly depression.

Method: Electronic databases of Medline were searched. Inclusion criteria were on changing blood Tryptophan concentration through diet. Four studies were critically appraised for their validity and reliability.

Results: The literature supports the concept that tryptophan depletion caused low mood in individuals with a history of depression. One study carried out psychological tests and found that tryptophan depletion affected the recognition of facial expressions in healthy individuals and those who had recovered from depression. Increased intake of the amino acid showed a positive effect on mood in all participants.

Conclusion: The 4 studies suggest that Tryptophan decreases positive mood in individuals, even though it is more apparent in individuals with a history of depression. The amount of Tryptophan in blood may be manipulated through diet, especially through controlling the intake of LNAAs. The effect of changing the concentration of free plasma Tryptophan need to be further investigated.

Background

Tryptophan is an amino acid that needs to be ingested as it cannot be synthesised in the human body, therefore it is grouped as being one of the ten essential amino acids. There are two versions of each amino acid called stereoisomers. The two versions of Tryptophan are named L-Tryptophan and D-Tryptophan. The human body only has receptors to recognise the L-Tryptophan, therefore that is the isomer which will be discussed.

One of the many functions of Tryptophan in the body is to act as a precursor for neurotransmitters; these are molecules that relay a message between two adjacent neurons through a gap called a synapse. Serotonin (5-HT) is such a neurotransmitter that is derived from Tryptophan. Serotonin is found all around the body especially in the gastrointestinal tract, platelets and the central nervous system. This article will focus on the latter location of Serotonin. Serotonin is known for its function to elevate mood, a low concentration of Serotonin due to Acute Tryptophan Depletion (ATD) has been associated with lowering of mood. The concentration of it in the body can be altered through diet and supplementation. Therefore diet may be a factor for the onset of depressive symptoms. Depression as a disorder is the fourth highest cause of disability worldwide, affecting about two thirds of adults sometime during their life. Depression is also very common in long term hospital patients, and this may be linked to the level of malnutrition in hospitals, as it has risen by 85% over the past 10 years.

The concentrations of Tryptophan vary amongst different type of food. The average daily requirement for an adult is 4mg/kg body weight. Therefore for an average adult weighing 70kg, 280mg of Tryptophan would be required daily. For example this can be achieved by taking 100g of sunflower seeds, but a more reasonable diet would be to take 1 large egg, which on average has 30 grams of egg white, providing 300mg of Tryptophan. Some foods are a poor source of this amino acid, such as bananas, which have low overall protein content, so approximately 28 bananas are required to meet the recommended daily intake of tryptophan.

Recently, supplements for Tryptophan have been available in health stores, as no serious side effects have been associated with them. Some supplements are 500mg Tryptophan, which is almost double the required intake. This increases the Tryptophan
concentrations greatly in the blood and therefore also in the brain. This increases Serotonin production in the brain and has shown to benefit depressed individuals11.

Most of the conversion of Tryptophan to Serotonin takes place in the gut, but some does occur in the brain. As Serotonin cannot go past the blood-brain barrier (BBB), therefore only the amount of Tryptophan that crosses the BBB can be used to create Serotonin. Tryptophan is transported past the BBB via capillaries alongside all the other large neutral amino acids (LNAAs: tryptophan, phenylalanine, tyrosine, leucine, isoleucine, valine, methionine, threonine, serine and cysteine). As all the LNAAs use the same transport system there is competition between them, so that a higher concentration of one amino acid will lower the affinity for the other ones. Therefore the amount of Tryptophan in the brain doesn’t only depend on the amount of Tryptophan in the blood plasma, but also on the concentrations of other LNAAs in the plasma12, 13.

The concentration of Tryptophan in the plasma varies with the type of food ingested. As the Tryptophan plasma concentration rises, assuming other LNAAs concentrations remain constant, there will be more Tryptophan crossing the BBB into the brain, where it will be converted into Serotonin; hence the amount of Serotonin will increase in the brain. In contrast if the concentration is low, due to inadequate intake through diet or physiological problems, the Serotonin concentration in the brain will be lower than the norm.

Albumin is a protein found in the plasma, and its main function is to transport various molecules including Tryptophan. The bound amino acids cannot pass the BBB, therefore only the free plasma Tryptophan is available to the brain. Fatty acids also have an affinity for albumin, therefore an intake of fatty acids, increases plasma fatty acids which bind to albumin instead of Tryptophan. Therefore the amount of free plasma tryptophan increases, which makes more tryptophan available to the brain, and as a consequence Serotonin concentration increases as well14. Also, the ingestion of a high carbohydrate meal triggers the release of Insulin from the beta cells in the islets of Langerhans in the pancreas15. Insulin stimulates the uptake of LNAAs, but not Tryptophan, therefore the ratio of Tryptophan to other competing LNAAs increases, hence a higher proportion of Tryptophan crosses the BBB into the Central Nervous System (CNS), where it is converted into Serotonin16, 17. Therefore the concentration of Serotonin in the brain doesn’t just increase by taking larger amounts of Tryptophan through diet and supplementation, but also through the proportion of fatty acids and carbohydrates ingested.

The free plasma Tryptophan enters the CNS through the capillary transport system and past the BBB. The Conversion of Tryptophan into Serotonin only occurs in the serotonergic neurons in the brain. The conversion involves Tryptophan Hydroxylase which adds an alcohol group to the aromatic ring of Tryptophan and creates 5-hydroxytryptophan (5-HTP), which in turn is converted to Serotonin (5-HT) by the enzyme aromatic-L-amino-acid decarboxylase 19. The Serotonin that is produced is then released by the raphe nuclei in the brain, which are 9 pairs of nuclei distributed along the brain stem18.

Primarily the action of serotonergic neurons is stopped by the reuptake of serotonin at the synapses; this is also where the effect of Selective Serotonin Reuptake Inhibitors (SSRIs) takes action, which inhibit the reuptake of serotonin and therefore preventing the concentration of it going low. The other pathway involves the breakdown of Serotonin; this is initiated by monoamine oxidase, which converts Serotonin into 5-hydroxyindoleactic acid (5-HIAA). The 5-HIAA is excreted in urine and is a main way to indirectly evaluate the amount of serotonin in the body20.

The aim of this review is to investigate the effects of Tryptophan concentration though diet.

Methods

Electronic medical databases such as Medline, Scopus, Compendex, JSTOR and ScienceDirect were searched. Search engines such as Google Scholar and EBSCO Host Discovery service were also used. To make sure that almost all the relevant articles were being searched for, it was important to use medical subject headings (MeSH). In order to obtain more appropriate articles, the results were filtered by applying limits, such as to only include articles from 1995 onwards. This was to ensure that the material used in this review was up-to-date. The search strategy used in this review is summarised in a Table 1 below.

The strategic use of correct MeSH terms alongside limits, narrowed down the results as well as helping to find more relevant articles about Tryptophan concentrations relating to depression. This procedure was repeated on all the databases and search engines. Table 2 below demonstrates how the searches were carried out on Medline via PubMed using MeSH terms, and how these narrowed down the results.

Results
Study 1
Effect of Acute Tryptophan Depletion on Emotions in Individuals with Personal and Family History of Depression following a Mood Induction21.
As there haven’t been many studies testing the effect of ATD on emotions apart from depression, this study also aimed to measure the positive affect in anxiety and anger following a negative mood induction.
At the beginning participants were interviewed about stressful experiences within the past year, and this information was used to generate events that would induce negative moods. These events were then rated on a 10-point scale to which degree they felt a negative emotion.
The study was double-blind and counterbalanced, which included an ATD and a placebo challenge drink. The ATD drink contained 15 amino acids that have been known to decrease Tryptophan in the body, most likely LNAAs. The placebo drink included sweeteners in similar taste to the ATD drink. A blood test confirmed the ATD drink lowered the ratio of tryptophan to the LNAAs much more compared to the placebo.
Baseline measures of mood were taken, before the intervention was used. After 5 hours, which is the estimated onset of maximal Tryptophan depletion22, the participants underwent a negative mood induction. This involved listening to a 1-3 minute recording of a negative event, and after this they had to complete mood measures. For depressive symptoms, the Beck Depression Inventory (BDI)23 was used along with the Hamilton Depression Rating Scale (HDRS)24. The second set of test was taken after 1 week of the first test.
The results showed that controls and the vulnerable to depression had different peak positions on the positive affect graph, and overall it can be concluded that individuals who currently do not have depression but are susceptible to it by having a personal and family history, exhibit a greater reduction in serotonergic activity, causing depressive symptoms.
Study 2
Response to tryptophan depletion in major depression treated with either cognitive therapy or selective serotonin reuptake inhibitor antidepressants25.
This study compared the effect of Rapid Tryptophan Depletion (RTD) on patients remitted with depression treated with SSRIs or treated with Cognitive Therapy (CT). This study chose a group of 20 Participants who met the DSM-IV criteria for a major depressive episode of at least a moderate level. 10 of these participants had been treated with SSRIs (Fluoxetine and Paroxetine), the other 10 were treated with weekly session of CT.
The study included an active experiment were the Tryptophan was depleted and a “sham” experiment was the control. A 24 hour period of a low protein diet (160mg/day) was followed by supplements 3 times a day. The active group received a placebo whereas the control group received 500mg of l-Tryptophan.
The blood was collected at set times to assess if the intervention did cause Tryptophan depletion, and was compared with the behavioural tests including the BDI and HDRS taken at the same time. Physicians observed the behaviour of the participants throughout the study. The analysis consisted of comparing the differential change between the control and active groups.
The age of the 2 groups was significantly different, and analysis was carried out with and without age as a covariate.
Table 7 below shows the Tryptophan blood concentrations of the 2 groups. Statistical analysis showed it wasn’t statistically significant. The BDI and the HDRS in the other hand showed that the SSRI group showed a much more worsening of mood with the Tryptophan depletion. There were greater changes in the BDI score than the HDRS. Therefore the study concluded that the CT group was resistant to the effect of RTD on mood.
Study 3
Mood and cortisol responses following tryptophan-rich hydrolysed protein and acute stress in healthy subjects with high and low cognitive reactivity to depression26.
This study aimed to investigate the effects of high blood Tryptophan concentration in individuals with a high or low Cognitive Reactivity (CR). This involved 150 university students filling in a questionnaire about their history, and to test for CR, the Leiden Index of Depression Sensitivity27 was used. This left with 18 participants with low CR and 20 with high CR.
The participant’s mood and cortisol concentration in saliva was assessed before and after the sessions involving stress exposure. A variety of mood tests were given along with saliva being taken. One such time was 2 hours after ingestion of the Drink. Salivary cortisol post stress test was measured. The results showed that the cortisol concentration increased after the stress test. Higher in the Casein Protein (CP) group than in the Hydrolysed Protein (HP) group.
The mood test showed a decrease in anger, depression and tension following the HP drink. Likely due to the increased Tryptophan concentration in the brain. The CP group didn’t show a significant change. There was no relation between CR and the beneficial effects of HP in this study, even though other studies have shown the relationship in remitted patients28.
However this Tryptophan augmentation was sufficient to show a positive effect on behaviour in healthy individuals.

**Study 4**

Low-dose tryptophan depletion in recovered depressed patients induces changes in cognitive processing without depressive symptoms. This study explored the effects of a low-dose Tryptophan depletion on the cognition and emotions of individuals who are medication free recovered from depression as well as healthy individuals. It used a Tryptophan-Depleting (TD) mixture alongside controls. The mixture used in this study is shown to cause a 66% decrease in total tryptophan. Before and after ingesting the mixture depression tests such as BDI, HDRS were taken. Five hours after the ingestion, series of Psychological tests were also carried out to measure any aspect of change in behaviour and thinking due to the TD. One example of the results is that TD specifically decreased the recognition of happy faces in the recovered patients, which was opposite for the healthy individuals. This effect has been shown in another study, which highlights that the emotional dysfunction caused by depression gives rise to misperception of emotional displays.

**Discussion**

A variety of studies, all involving the manipulation of Tryptophan concentration in the body, have been analysed. In all the studies the intake of special amino acid mixtures, significantly reduced the concentration of total blood Tryptophan. This mixture included LNAAs that compete with the Tryptophan and hence reduce the entry of Tryptophan into the brain. Studies 1, 2 and 4 included participants with a history of depression. These included individuals treated with SSRIs and CTs as well as recovered patients free of medication. These individuals have consistently showed a more significant change in mood after TD. This could be due to the drugs such as the SSRIs making their serotoninergic system more sensitive to changes in Tryptophan concentration. Although the specific inclusion criteria, of only using individuals treated with a specific antidepressant had its limitations.

To detect any changes in mood, BDI and HDRS were used in study 1, 2 and 4. They all indicated a similar outcome, TD caused a reduction in mood whereas an increase in Tryptophan through dietary means (study 3) increased positive mood. Study 4 on the other hand used a variety of psychological tests, and found out that TD also affects aspects of facial recognition and response times.

Even though many studies have been carried out about TD’s effect on the brain, the studies often include a small number of people, for example study 2 only had 19 participants that completed the experiment. This becomes a factor affecting reliability. The effect of TD on participants, who had a history of depression or were being treated for it, was consistent throughout the 4 studies as it significantly lowered the moods of the individuals. The effect on healthy individuals was less consistent as shown by study 1 and 4. Study 1 found no significant change in positive affect in individuals without a history of depression, but study 4 highlighted that some cognitive changes did occur in healthy individuals although to a lesser extend compared to the recovered patients. This difference could have come from the methods used, study 4 tested for a greater range of symptoms but study 1 only focused on the mood states using BDI, HDRS, POMS. Study 4 also used these and showed that a low-dose TD did not affect the BDI and HDRS scores in both healthy and recovered individuals, but the study lasted less than 1 day, compared to approximately 1 week for the others, therefore it excluded the delayed effect of low-dose TD. Study 3 also included healthy individuals, albeit they were split into groups of high CR and low CR and it tested the opposite effect of TD, the effect of a HP. It demonstrated that that the increase in Tryptophan intake caused by the HP was sufficient to affect behaviour in healthy individuals. This effect was only shown by one of their tests and not the other one, which questions the validity of this study.

The use of different mood tests with specific scoring systems also affected the validity, as the studies reviewed had different scores that were counted as a high or low. For example in study 2 a score of 14 in the BDI showed a re-emergence of mood symptoms whereas study 2 looked more at significant changes in scores before and after the stress test. The difference in scales to detect depressive symptoms has made it difficult to compare the outcomes of each study. To eliminate bias, all the studies used a double-blind design, but randomisation only occurred in study 4, which makes it a more robust than the others. To minimise the confounding factors, all the studies had an exclusion criteria, such as not to include individuals who had done substance abuse in the past year, as this can affect their mood and may cause error in the results.

For future consideration, studies investigating the effect of blood Tryptophan concentration on mood,
should include a larger sample size, because the largest investigated in this review was 70, which is not good enough for a fully robust study. Also a more appropriate test should be used to detect mood changes, which takes into account the subjectivity of feelings. A range of participants should be used, to provide a broad understanding of the effect of Tryptophan depletion. To provide stronger evidence that Tryptophan also affects non-clinical populations; further studies should consider using more healthy participants. To eliminate biases, randomisation should be performed in each study, because it was not done in 3 of the 4 studies reviewed and this weakens the validity of the outcome.

Table 10 outlines some of the limitations of this review.

**Conclusion**

The 4 studies suggest that Tryptophan decreases positive mood in individuals, even though it is more apparent in individuals with a history of depression. The amount of Tryptophan in blood may be manipulated through diet, especially through controlling the intake of LNAAs. The effect of changing the concentration of free plasma Tryptophan need to be further investigated.

**References**

18. Frazer A HJ. Understanding the neuroanatomical organization of serotonergic cells in the brain provides insight into the functions of this neurotransmitter. Basic Neurochem 1999.
22. Delgado PL CD, Price LH, Aghajanian GK, Landis H, Heninger GR. Serotonin function and the mechanism of antidepressant action. Arch Gen
Illustrations

Illustration 1

Tables.

Table 1 – Search Strategy, Summary

<table>
<thead>
<tr>
<th>Database Used</th>
<th>MeSH Terms</th>
<th>Limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medline</td>
<td>Tryptophan</td>
<td>Published: 1995-2011</td>
</tr>
<tr>
<td>Scopus</td>
<td>Depletion</td>
<td>Language- English</td>
</tr>
<tr>
<td>Compendex</td>
<td>Serotonin</td>
<td>Humans</td>
</tr>
<tr>
<td>JSTOR</td>
<td>Depression</td>
<td></td>
</tr>
<tr>
<td>PubMed</td>
<td>Diet/Supplements</td>
<td></td>
</tr>
<tr>
<td>ScienceDirect</td>
<td>Remitted</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 - Search Strategy, narrowing down results on PubMed

<table>
<thead>
<tr>
<th>Attempts</th>
<th>MeSH Terms</th>
<th>Results</th>
<th>Limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Tryptophan</td>
<td>1076</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Tryptophan</td>
<td>514</td>
<td>English Only 1995-2011</td>
</tr>
<tr>
<td></td>
<td>Depletion</td>
<td></td>
<td>Human</td>
</tr>
<tr>
<td>3</td>
<td>2 + depression</td>
<td>187</td>
<td>English Only 1995-2011</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Humans</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
After all the databases mentioned earlier were searched using this method, about 10 appropriate articles were left. To narrow down the number even further, the abstract for each of them was read and the articles that did not particularly study the topic in question were taken out of the count. At the end 6 articles remained which were read and from these 4 articles were chosen using our inclusion and exclusion criterion (Table 3 and 4).

Table 3 - Selection Criteria for Studies

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Inclusion</th>
<th>Exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nature of Participants</td>
<td>Human</td>
<td>Non-human</td>
</tr>
<tr>
<td>Age of Participants</td>
<td>Over the age of 18</td>
<td>Under the age of 18</td>
</tr>
<tr>
<td>Sex of Participants</td>
<td>Men and Women</td>
<td>Single sex</td>
</tr>
<tr>
<td>Number of Participants</td>
<td>Over 20</td>
<td>Less than 20</td>
</tr>
<tr>
<td>History of Participants</td>
<td>Depression in Past, and now being treated with Antidepressants or family history of depression. Healthy.</td>
<td>Any other mental disorder apart from depression e.g. Axis I Disorders. Taken Illicit Drugs.</td>
</tr>
<tr>
<td>Language of Article</td>
<td>English</td>
<td>Non-English</td>
</tr>
<tr>
<td>Publishing date</td>
<td>From 1995 onwards</td>
<td>Older than 1995</td>
</tr>
</tbody>
</table>
Table 4 – Studies to be included in this review.

<table>
<thead>
<tr>
<th>Study</th>
<th>Author</th>
<th>Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Altman et al.</td>
<td>Effect of ATD on emotions in individuals with personal and family history of depression following a mood induction.</td>
</tr>
<tr>
<td>2</td>
<td>O’Reardon JP, et al.</td>
<td>Response to tryptophan depletion in major depression treated with either cognitive therapy or selective serotonin reuptake inhibitor antidepressants</td>
</tr>
<tr>
<td>3</td>
<td>Firk C, Markus CR,</td>
<td>Mood and cortisol responses following tryptophan-rich hydrolysed protein and acute stress in healthy subjects with high and low cognitive reactivity to depression</td>
</tr>
<tr>
<td>4</td>
<td>Hayward G, et al.</td>
<td>Low-dose tryptophan depletion in recovered depressed patients induces changes in cognitive processing without depressive symptoms</td>
</tr>
</tbody>
</table>
### Table 5 – Summary of Study 1.

<table>
<thead>
<tr>
<th>Aim</th>
<th>To assess if individuals with a personal and family history of depression are more susceptible to a positive effect on feeling Anxiety, Anger and Sadness after ATD</th>
</tr>
</thead>
</table>
| Method | Double-Blind Study  
Intervention was either an ATD drink containing amino acids to lower Tryptophan concentration in blood or a Placebo.  
Performing BDI, HDRS, POMS, and MAACL test as baseline, 5h and 7h of taking the intervention. |
| No. of Participants | Total 70  
30 with History of Depression  
40 Without History of Depression |
| Duration | Approximately 1 week. |
| Data Assessed | Tryptophan blood concentration after taking drinks.  
BDI  
HDRS  
POMS & MAACL |

**Conclusion**  
Individuals who currently do not have depression but are susceptible to it by having a personal and family history, exhibit a greater reduction in serotonergic activity.
### Table 7 – Summary of Study 2

<table>
<thead>
<tr>
<th><strong>Aim</strong></th>
<th>Comparing the effects of Rapid Tryptophan Depletion in remitted patients treated with SSRIs or Cognitive Therapy.</th>
</tr>
</thead>
</table>
| **Method** | Double-blinded, Placebo controlled, balanced crossover design  
Each participant received active experimental or control condition in a randomised manner. 24h Low protein diet – 160mg Tryptophan a day. Controls received 500mg L-Tryptophan 3 times a day were as the depletion group received placebos. Day 2, 100g Amino acid drink and capsules. + 2.2g L-Tryptophan supplements for controls. Bloods taken at 0, 2, 4, 6 hours after ingestion of drink. BDI and HDRS taken at each point of blood collection |
| **No. of Participants** | Total 20, 19 completed. 10 –remitted, treated with SSRI. 10- weekly session of CT |
| **Duration** | Approximately 1 week |
| **Data Assessed** | Depressive Symptoms assessed using BDI and Modified HDSR tests.  
Calculating changes in serum Tryptophan concentration. |
| **Results** | The group that was treated with SSRIs showed a significant worsening on mood especially on the BDI. |
Table 8 – Summary of Study 3

<table>
<thead>
<tr>
<th>Aim</th>
<th>To investigate the beneficial effect on mood by changing Tryptophan concentration in the blood, using Tryptophan-rich Hydrolysed Protein (HP).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Method</td>
<td>Double-Blind, crossover study. 312 ml Protein drink either containing HP or CP was given. CP drink consisted of standard Casein protein with 0.4g Tryptophan and 10g of other LNAAs whereas the HP contained 0.8g Tryptophan and 0.9g LNAAs. Stress tests and Mood tests along with obtaining Saliva samples. Variety of Mood tests at beginning and 2h ingestion of drink.</td>
</tr>
<tr>
<td>No. of Participants</td>
<td>Total 38. 20 with High Cognitive Reactivity. 18 with Low Cognitive Reactivity</td>
</tr>
<tr>
<td>Duration</td>
<td>Approximately 1 week</td>
</tr>
</tbody>
</table>
| Data Assessed | Profile of Mood states (POMS)\(^29\)  
Positive and Negative Affect Scale (PANAS)\(^30\)  
Trier Social Stress test (TSST)\(^31\)  
Level of cortisol in saliva before and after the stress test. |
| Results | An intake of Tryptophan-rich hydrolysed protein significantly increased positive mood in all subjects. Slower cortisol increase in participants ingesting HP. |
| Conclusion | Tryptophan-rich hydrolysed protein may be a good dietary way to change Tryptophan concentration in the brain, which induces a significant positive mood. |
### Table 9 – Summary of Study 4

<table>
<thead>
<tr>
<th><strong>Aim</strong></th>
<th>How a low-dose Tryptophan affects the cognition and emotions on individuals recovered from depression and healthy individuals.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Method</strong></td>
<td>Randomized, double-blind design. Either treated with Tryptophan-Depleting (TD) or control mixture. State of participant recorded at start and 5h after ingestion using BDI, POMS, and HDRS, all designed to detect short term changes. Blood samples taken at same time. Psychological tests after 5h ingestion of mixture.</td>
</tr>
<tr>
<td><strong>No. of Participants</strong></td>
<td>Total of 58. 24 healthy individuals. 24 individuals recovered from depression, free of antidepressant for at least 3 months.</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>Less than 1 day</td>
</tr>
<tr>
<td><strong>Data Assessed</strong></td>
<td>Blood samples measure amino acid levels. BDI, POMS and HDRS to measure state of individual. Psychological tests: Counting Stroop Task; Emotional Counting Stroop Task; Emotional memory Task; Auditory Verbal Learning Task; Emotion Potentiated Startle; Facial Expression Recognition</td>
</tr>
<tr>
<td><strong>Results</strong></td>
<td>TD mixture significantly lowered the ratio of Tryptophan to the other LNAAs in comparison to the control. TD did not show a significant difference in the BDI and HDRS in either the recovered and healthy individuals. TD caused a Decrease in recognition of happy expression in recovered individuals. TD enhanced the recognition of Disgust, more in recovered individuals. TD caused an increased startle response. Recovered individuals more sensitive to negative stimuli as they were more easily distracted</td>
</tr>
<tr>
<td><strong>Conclusion</strong></td>
<td>Low-dose TD changed perceptual biases in individuals, which was more apparent in individuals with a history of depression. The changes seen with the low-dose may be early indicators of depression.</td>
</tr>
</tbody>
</table>
Illustration 3

Tables

**Table 10 – Limitation of this review.**

<table>
<thead>
<tr>
<th>Limitations</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Access</strong></td>
<td>Access to full articles was often a limitation, due to the university not having a subscription to a specific journal. Therefore, a chance is that some important studies have been missed out from this review.</td>
</tr>
<tr>
<td><strong>Language</strong></td>
<td>The use of only English articles, excluded articles of other languages, which might have had important information regarding Tryptophan and depression.</td>
</tr>
<tr>
<td><strong>Number of Studies</strong></td>
<td>There weren’t a large number of studies done on this topic and some might have been missed out by the databases.</td>
</tr>
</tbody>
</table>
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