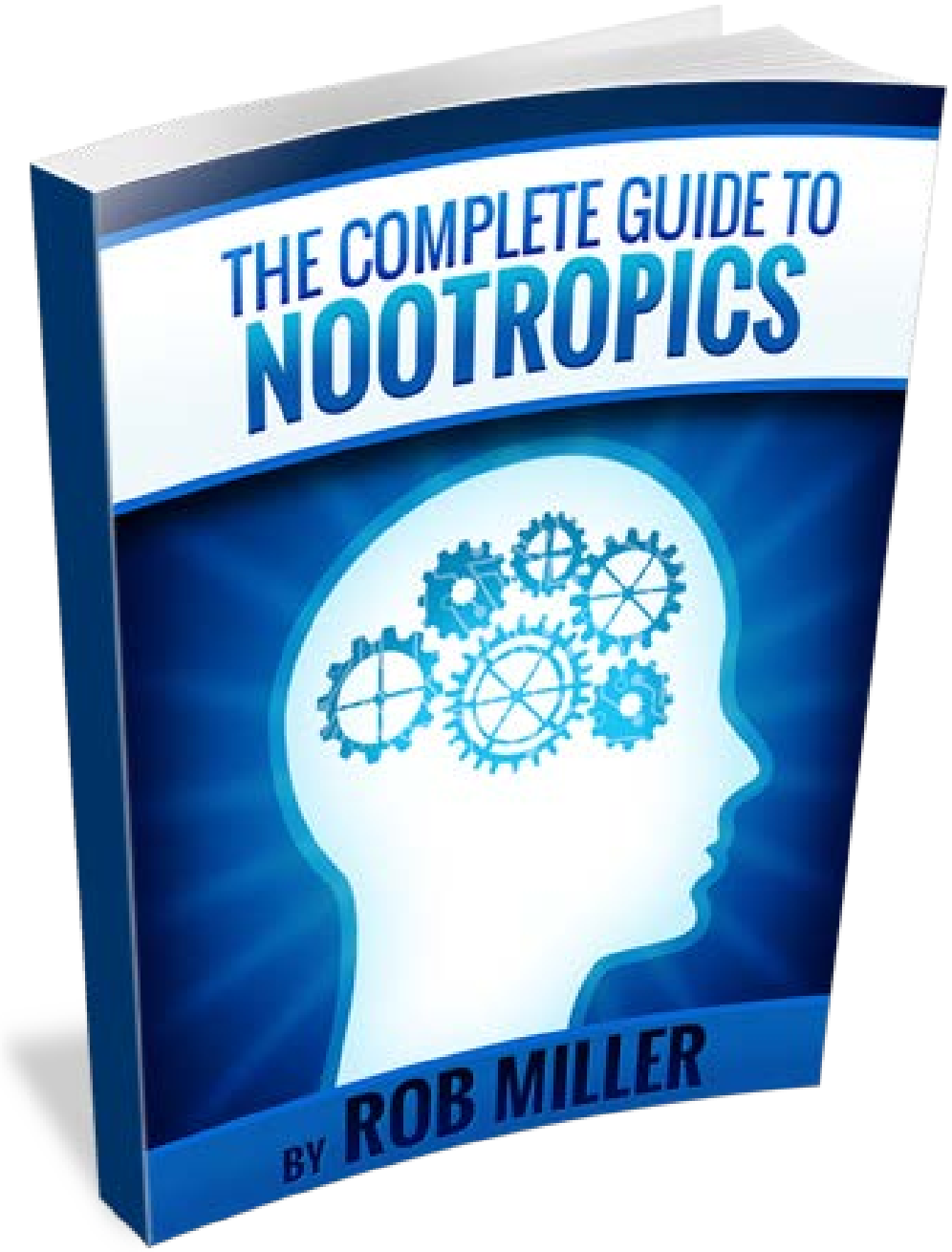

Note: The information provided in this guide was thoroughly researched and referenced. That being said, we are NOT doctors and you should not take any of this advice or anything we say in the ebook as medical advice. It is best to consult with your physician prior to taking any supplement.

My Top Choice For Nootropic Supplements

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Chapter I: Introduction to Nootropics

Living in the age of electronically distributed mass media definitely has both its perks and its downfalls. An obvious perk is that we are constantly deluged in easily accessible information about a wide variety of topics. On the other hand, this is also one of the chief downfalls, as it all too often results in a persistent, widespread sense of information overload. However, there is hope! Thanks to the Internet, the public has become more aware of the existence and benefits of a possible remedy to some of these informational ills of modern life: nootropics.

What are nootropics?

Also known as “smart drugs,” these substances are claimed to improve attention, motivation, intelligence, memory, and mood. Equally importantly, these substances are reportedly very safe. Within the community of nootropics researchers and enthusiasts, there is considerable debate over just which drugs, supplements, and nutraceuticals fall into this coveted category.

Remember the drugs depicted in the 2011 thriller “Limitless?” The main character Eddie is given a mysterious nootropic drug. In some ways, the comparison is fair; after all, nootropics have been shown to enhance attention, concentration, memory, and more – Eddie experienced quite a bit of financial success as a result of this in the film. At the same time, the mental and physical effects the main character undergoes are over-exaggerated if the directors has piracetam or aniracetam in mind. It's not likely that the average individual taking a nootropic will find him- or herself suddenly involved in disturbing situation after the next. Take note: “going off the deep end” is not a common consequence of taking nootropics as far as the information shows (and we encourage you to check out the studies referenced for yourself).

The term “nootropic” was coined by the Romanian chemist and psychologist Dr. Corneliu Giurgea in 1972 to describe the effects of piracetam, a compound that

he discovered in 1964 while working for the Belgian pharmaceutical company UCB. The term itself means “mind-turning” due to the observed positive effects of piracetam and related compounds on the brain and on mental function in both non-human animals and humans. Piracetam is, thus, the prototypical nootropic compound.

In addition to having discovered and developed piracetam, Dr. Giurgea is among those responsible for the dissemination of information on it. In turn, this has led to the research, discovery, and popularization of many other –racetams and other kinds of nootropics. In order to clarify research on and discussion of the –racetam compounds, Dr. Giurgea came up with a set of criteria that any given substance must meet in order for it to be classified as a nootropic.

Giurgea's Nootropic Criteria

According to Giurgea, a nootropic substance:

1. should enhance memory and learning
2. should enhance resistance of learned behaviors/memories to conditions which tend to disrupt them (e.g. electroconvulsive shock, hypoxia)
3. should protect the brain against various physical/chemical injuries (e.g. barbiturates, scopolamine)
4. should increase the efficacy of tonic cortical/subcortical control mechanisms
5. should lack the usual pharmacology of other psychotropic drugs (e.g. sedation, motor stimulation), and possess very few side effects and extremely low toxicity (Giurgea C., 1972, p. 108).

The issue of what qualifies as a nootropic, however, wasn’t so simply settled. As is usually the case in science, once the field was established other learned commentators weighed in with their own observations and proposals. In addition to Giurgea's efforts to sum up his thoughts on the matter, the pharmacologist V. Skondia was responsible for coming up with an even more detailed set of criteria,

which he published in 1979. Skondia chose to look in more detail at the metabolic effects of these compounds.

Skondia's Criteria

According to Skondia's criteria for nootropics, a nootropic should display:

1. No direct vasoactivity
    (a) No vasodilation
    (b) No vasoconstriction
2. No change in basic EEG rhythm
    (a) Quantitative EEG: increased power spectrum (beta 2 and alpha)
    (b) Qualitative EEG: decreased delta waves and cerebral suffering
3. Must pass blood-brain barrier
    (a) Under normal conditions
    (b) Under pathological conditions
4. Must show metabolic activity in:
    (a) Animal brain metabolism
        i. Molecular
        ii. Physiopathological
    (b) Human brain metabolism
        i. A-V differences
            A. Increased extraction quotients of O2
            B. Increased extraction quotients of glucose
            C. Reduced lactate pyruvate ratio
        ii. Regional cerebral metabolic rates (rCMR)

A. Increased ICMR of O2

B. Increased rCMR of glucose

iii. Regional cerebral blood flow: normalization

5. Minimal side effects

6. Clinical trials must be conducted with several rating scales designed to objectify metabolic cerebral improvement. (Skondia V., 1979)

As you can readily see, Skondia's requirements are more extensive and go much more in-depth than Giurgea's. One point that was agreed upon by both researchers was that nootropics should have a well-established safety profile and display little to no toxicity. Putting this information together, we can see that some of the essential aspects of the definition of a nootropic are: that (a) a given chemical is (b) a drug with (c) little to no side effects that (d) enhances cognition.

It is apt that nootropics are often referred to in the media as “smart drugs.” They are “smart drugs” not only in the sense of enhancing various aspects of intellectual performance (*a la*, for example, stimulant medications such as the amphetamines, which are commonly used in the treatment of ADD/ADHD), they are also “smart drugs” in the sense of being healthy choices for the enhancement of intellectual performance (quite unlike the amphetamines).

Who takes nootropics?

Not only do nootropics help the elderly and those suffering from brain trauma, but they also provide beneficial results to the young and healthy. Nootropics enthusiasts love to use them for studying and learning. Russian cosmonauts use them to sharpen their physical and mental skills in space (Malykh & Sadaie, 2010, p. 290).

Chapter II: Types of Nootropics

The -racetam Family (piracetam, aniracetam, nefiracetam, levetiracetam, pramiracetam, oxiracetam, phenylpiracetam, and noopept)

As was mentioned above, piracetam (trade name Nootropil, among others) was the first nootropic, in that its discovery led to the term being coined in the first place (Giurgea 1972). Since piracetam was discovered, various modifications of its chemical structure have yielded numerous other –racetams; among other distinguishing features, these have the advantage over piracetam of being more potent by weight—piracetam doses often go into the tens of grams.

The Russian-developed (and approved) nootropic Noopept (itself a trade name; in chemical nomenclature, Noopept is referred to as GVS-111), for example, is active in doses of 10-20 milligrams orally. Quite an improvement! These compounds have found a variety of medical uses in different countries. In Europe, for instance, piracetam has a long history of use as a cognition-enhancing agent in the treatment of impaired intellectual function due to a variety of causes.

The only -racetam that is approved for medical use in the United States is levetiracetam (trade name Keppra), which is an anticonvulsant used in the control of epileptic seizures. In contrast to the majority of other -racetams, levetiracetam has some serious safety issues (Malykh & Sadaie 2010).

“Eugeroic” Stimulants (modafinil, adrafinil, and armodafinil)

In contrast to traditional/conventional stimulant medications such as Adderall (mixed amphetamine salts), Dexedrine (dextroamphetamine) and Ritalin (methylphenidate, a compound structurally related to amphetamine), eugeroic (from Greek roots for “good arousal”) agents such as modafinil (trade name Provigil) do not tend to produce significant elevations in heart rate or blood pressure, do not tend to produce euphoria, and do not seem to display a high abuse liability.

It is extremely unlikely that people will end up on street corners selling their parents' TVs for their next hit of modafinil. Adrafinil (trade name Olmifon) is the parent compound of the group, and was discovered/developed in France in the 1970s. It is somewhat less potent than its derivatives, and also has unfortunate liver side-effects in some users that appear to have been eliminated in its successors. These are fascinating compounds, but an in-depth discussion of them is, unfortunately, not within the scope of this book.

Vitamins and Supplements (B and D vitamins and Omega-3 fatty acids)

While a great deal of quackery abounds in the alternative health field, there is solid evidence for the beneficial psychological effects (in terms of mood, motivation, alertness, and etc.) of some vitamins and supplements. Vitamins, minerals and other nutrients are often overlooked as possible pathways to personal enhancement, but the debilitating effects of bad diet should not be underestimated. On the other hand, some people seem to assume that because many supplements are "all natural," the substances contained in these supplements are not harmful. This line of thinking is both erroneous and extremely dangerous. Some of these supplements, including members of the B vitamin family as well as vitamin D, can cause physiological toxicity (including but not limited to neurotoxicity).

If you are a regular consumer of fish or of extracted fish oil as a source of Omega-3 fatty acids, it is important that you verify that the material you are consuming is not contaminated with mercury. Due to the detrimental effects on cognitive functioning that result from mercury poisoning, it would be accurate to think of mercury as an anti-nootropic.

Cholinergics (Lecithin, Choline, DMAE, Alpha-GPC, CDP-Choline, Galantamine, Donepezil, and Huperzine-A)

Cholinergic substances can be defined in multiple ways. In one sense, a cholinergic is a precursor to **acetylcholine**, one of the most important

neurotransmitters for, among other things, memory function. **Choline**, **Alpha-GPC**, and **CDP choline** (also known as citicoline) are cholinergics in this sense. Lecithin is not technically a cholinergic precursor itself, but instead contains phosphatidylcholine, a cholinergic precursor. DMAE's role as a choline precursor is debatable, and is consequently debated.

In a second sense, a cholinergic is a substance which affects acetylcholine receptors in a similar manner to acetylcholine itself. (Acetylcholine is an agonist at its receptors, which means that it "activates" them.) While DMAE's mode of action remains unclear, it may well be that DMAE is a cholinergic agonist in its own right. As an aside, nicotine is itself a cholinergic agonist, and, specifically, agonizes *nicotinic* cholinergic receptors. Nicotine displays various cognition enhancing effects, but because of its detrimental effects on cardiovascular function, its dependence potential and its relatively low margin of safety, nicotine cannot be considered a nootropic.

In a third sense, a cholinergic is a substance that inhibits acetylcholinesterase, the enzyme that breaks down acetylcholine. Galantamine, huperzine A and donepezil are all examples of cholinergics in this sense, and there is some reason to think that DMAE may be a cholinergic in this sense as well. Incidentally, some of the more potent members of this last class of cholinergics have seen battlefield use as chemical warfare agents!

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Chapter III: The Benefits of Nootropics for Users

The body of evidence for the benefits of using nootropics is rather substantial – there have been a wide variety of scientific studies conducted on multiple continents over the past half century. There are numerous animal studies that have established how nootropics positively affect the functioning of rats, guinea pigs, and monkeys. What you're likely more to be interested in, however, are the studies that employ humans as test subjects. Fortunately, there are a significant number of these as well.

-racetams:

Piracetam

Piracetam, the prototype, is one of the most commonly studied of all the nootropics. Structurally, piracetam is related to the inhibitory neurotransmitter GABA, though it has no appreciable affinity for GABA receptors. Originally approved for use in humans in Europe during the 1970s, piracetam has been used in the treatment of conditions such as vertigo and age-related disorders, among others. The adverse effects of piracetam are mild and short-lasting and consist mainly of anxiety, drowsiness, insomnia, and agitation (Malykh & Sadaie, 2010, p. 94). As is the case with all supplements that fit Dr. Corneliu E. Giurgea's criteria for being nootropics, piracetam has few side effects and a very low toxicity (Giurgea C., 1972, p.108).

Effects on senile dementia AD patients

Piracetam appears to have an overall positive effect on patients in patients with senile dementia. Alzheimer's disease (AD) patients, compared to those elderly who do not have it, frequently display a lower hydrocarbon core fluidity. *In vitro* administration of piracetam brought the AD patients' aforementioned fluidity up

to the same level as the non-demented elderly. On top of that, it enhanced the fluidity of the latter groups' hydrocarbon core.

For both groups, piracetam was shown to decrease age-related alterations of membrane fluidity (this result has also been demonstrated in animals). This could result in increased energy metabolism, as well as enhanced signal transduction – disruptions of each of which are common problems among AD patients (Eckert et al., 1999, pp. 757-758).

This single study on piracetam is reflective of 19 different double-blind studies done on the effectiveness of the drug in age-related cognitive impairment. In an analysis of this research, individuals treated with piracetam showed a 60.9% improvement vs. a 32.5% improvement for those treated with placebo (Malykh & Sadaie, 2010, p. 294).

Results in sufferers of chronic schizophrenia

A 1979 controlled double-blind crossover study set out to establish whether or not piracetam could improve processes of transmission between the left and right hemispheres of the brains in chronic schizophrenics. Traditionally, interhemispheric transmission is exceptionally difficult for these individuals.

The Facts

- The patients selected for this research were all inpatients, ranging from ages 38 to 63 years old.
- Of the patients, 13 were male and 11 were female.
- The subjects of this study were those who had achieved few positive results with conventional medication or social rehabilitation.
- Two 800 mg doses of piracetam were given three times daily for a total of 4.8 g daily.
- The placebo, which looked identical to the piracetam, was given just as often to the other half of the test subjects (the control group).

- Testing in this study consisted of four separate sessions:
    - pre-trial (baseline, the day before the piracetam or placebo was taken)
    - during the first four week period
    - during 2-week cross-over gap (the break period)
    - during the last four week period (when the placebo and piracetam group had switched)

Disappointingly, in this study piracetam did not appear to facilitate the transfer condition; on the positive side, however, the researchers found that the drug did reduce the amount of incorrect responses in tasks that measured the intrahemispheric (that is, “between hemispheres”) process of transmission. This is not to say that piracetam increased the number of correct answers from the subjects, but rather that they responded less over time. Piracetam was also shown to be more effective than placebo in facilitating the performance of specific tasks that resulted in a heavier mental workload.

The study brought up the question of whether piracetam could be especially effective at higher levels of cognitive function. The mental improvement in certain areas helped to highlight similarities between patients of chronic schizophrenia and senile dementia (Dimond et al., 1979, pp. 342-343).

Recovering from aphasic stroke

Piracetam was chosen for a double-blind placebo-controlled study of stroke patients with aphasia. Aphasics are usually deficient in memory, alertness, peripheral vasculature, and microcirculation in the central nervous system (CNS). Because piracetam has been shown to help reduce these symptoms in patients with other conditions, researchers gave 4.8 g daily doses of it to 24 aphasic patients. Twenty-six patients were given placebo.

- There were a total of 50 experimental subjects, all of whom were patients who had been experiencing aphasia for periods ranging between four weeks and 36 months.
- In both the placebo and piracetam groups, the type and severity of symptoms were similar.
- No patients suffering from mild aphasia were included in this study; further, all of the included patients had been healthy prior to experiencing ischemic stroke, hemorrhagic stroke, or brain trauma/surgery.
- In addition to treatment with piracetam or placebo, the subjects underwent intensive language therapy in the form of ten 60 minute sessions, half of which were conducted individually and the other half of which were conducted in a group setting.
- Piracetam had a significant positive effect on the Written Language subtest.
- On the Communicative Ability scale, the piracetam group's scores in spontaneous speech improved.
- Furthermore, there were no adverse effects reported in the piracetam group.

While the above results are interesting, this was only one trial and can hardly be considered definitive. However, this was not the only study performed on the subject. Another study confirmed piracetam's ability to facilitate improvement of written language skills, as well as a reduction of the overall severity of the subject's aphasia (measured by the Token Test) (Poeck K).

Treatment of alcohol organic mental disorder

In 1990, a group of Austrian researchers decided to test piracetam for possible therapeutic utility in the treatment of alcohol organic mental disorder. The best known example of an alcoholic organic mental disorder is Wernicke-Korsakoff’s syndrome, but alcohol can negatively affect cognitive ability in multiple ways.

Deficient cognitive functioning subsequent to alcohol abuse is a serious public health problem.

Published in *Psychopharmacology* with C. Barnas listed as the lead author, the resulting paper was titled “High versus low-dose piracetam in alcoholic organic mental disorder: a placebo controlled study.” Barnas and his co-authors compared the effectiveness of the daily administration for six weeks of placebo, 6 grams of piracetam, or 24 grams of piracetam in alleviating alcohol-induced cognitive deficits.

The Facts

- There were a total of 60 (52 male, 8 female) experimental subjects, all of whom were DSM III-R confirmed alcoholics who had cognitive impairment that was verified on day 0 by psychological tests. Subjects were again evaluated for cognitive impairment on days 7, 14, 28, and 42.
- Subjects’ mean duration of alcohol consumption was 17.5 years.
- The study was conducted in double-blind fashion, with subjects randomly assigned to the three groups.
- Psychological tests administered to assess cognition included a test to measure attention and concentration over a period of 4.5 minutes (the d-2 test), a test to do the same over a period of 15 minutes (the Pauli test), a test to measure short-term memory (the Syndrom Kurz Test, or SKT), and a vocabulary test to correct the SKT score (the Verbal Comprehension Test).
- D-2 test results for all groups improved after 14 days relative to baseline (day 0), which were below normal. The Pauli test results were below normal on day 0, but group 3 (the high dose group) experienced significant improvement by day 14, whereas similar improvement was not observed in the other two groups until day 28.
- SKT testing on day 0 showed more severe cognitive impairment in group 3

than in the placebo group at baseline. By the measures of the SKT, group one (the placebo group) improved 5%, while groups 2 and 3 only improved by 1%.

- The SKT power factor of group 3 showed what the authors referred to as "significant amelioration" after 42 days, while the SKT power factor for groups 1 and 2 had barely changed. Using d-2 test results as matching variables, it was found that the SKT scores for group 3 were the only ones that displayed significant amelioration of the pre-existing deficit.
- The SKT speed factor improved by 5% for group 1, and by 1% for groups 2 and 3.
- Based on the degree of improvement seen in all of the experimental groups, the authors noted that organic mental disorder appears to display a marked tendency towards spontaneous remission over short periods of time.
- The SKT results displayed improvement for all groups in speed, but only the high-dose piracetam group showed a significant increase in power-related variables.

This fact lead the authors to wonder if therapeutic use of high dose piracetam could result in "better insight and … a higher motivation for alcohol abstinence." Twenty-three years later, this question has still not been properly addressed. According to the SKT results, duration of alcohol use influenced the degree of improvement (as evaluated by power factor), while the quantity of alcohol consumed in the three months prior to the trial showed no relevance. The authors concluded that "chronic, long-term ingestions of CNS-toxic substances implicates a worse prognosis than a subchronic intoxication with high doses."

Enhancement of normal mental functioning

In 1976, two researchers at University College in Cardiff, Wales performed a small study on the effects of piracetam in normal subjects. The results of this study

were summarized in the seminal (and tantalizingly titled) paper “Increase in the Power of Human Memory in Normal Man through the Use of Drugs. As shown below, the results were promising.

The Facts

- There were 16 subjects (12 male, 4 female), all healthy 2nd or 3rd year students in the Psychology Course.
- The study was double-blind. Placebo capsules were of the same appearance as those containing the active drug.
- Subjects were tasked with learning series of words presented as stimuli on a memory drum.
- No effects were observed after 7 days, but verbal learning had increased after 14 days.
- Three tests were administered: a pre-test before drug administration began, a test one week after drug administration was initiated, and a test two weeks after drug administratration was initiated.
- The initial procedure included: a verbal memory task, a dichotic listening task, a pursuit rotor task, an intermanual transfer task, and a level of handedness performance evaluation measured by the Handedness Questionnaire, with the subjects’ age and sex taken into account.
- After the completion of the initial procedure, subjects were then matched with the closest scoring other subject. One of each pair was given piracetam, while the other was given placebo. Because the study was double-blind, neither the researchers nor the subjects knew which subject in each pair was receiving the active drug.
- The authors found clear evidence for enhancement in verbal learning due to piracetam administration, but the evidence was less positive for enhancement on the pursuit rotor non-verbal learning test.

- In this study, piracetam was found to act specifically and selectively, which led the authors to ponder whether human brain power can be selectively increased (Dimond & Brouwers, 1976).

We could literally go on for days discussing the extensive scientific data collected from piracetam trials, but then we'd miss out on all the other great nootropics out there. Piracetam, being the supplement responsible for the term “nootropic” itself, has been researched extensively; however, it is not the only substance of its type that enhances memory, attention, and learning. Let's get right to the point, and discuss the potential of other nootropics.

Aniracetam

Aniracetam, just like its sister compound piracetam, shows itself to be effective in selective and specific areas. Although **aniracetam** has fallen out of favor in clinical studies in the US, perhaps due to its short half-life, scientists have been looking into developing new compounds based on its structure. In three different animal studies, researchers found aniracetam to have anxiolytic properties.

Effects on senile dementia of AD patients

A double-blind multi-center study was conducted throughout western Europe to establish whether **aniracetam** would prove beneficial to people aged 68 to 80 who displayed mild to moderate cognitive impairment and fulfilled the criteria for likely having senile dementia of Alzheimer's type (also called probable SDAT).

The Facts

- This study gave one group of patients 1500 mg of **aniracetam**, another group 2400 mg of piracetam, and the last group placebo.
- Each group took their doses daily for a six month period of time.
- Throughout the trial, the placebo group's mental health continued to deteriorate.
- The **aniracetam** group experienced few side effects, and the ones that

were present were so mild and transitory that no subject had to cease treatment as a result.

- **Aniracetam** proved to be even more effective than piracetam in this trial, as established by a battery of psycho-behavioral tests (Lee & Benfield, 1994, pp. 96-97).

In other words, for patients who likely have senile dementia of Alzheimer's type, **aniracetam** could prove to be a useful adjuvant therapy (as in, therapy that strengthens your primary form of therapy or medication that works with your primary form of medication to give the most effective results).

Cognitive improvement in healthy volunteers

In a study aimed at healthy volunteers, single oral doses of 500-2000 mg of **aniracetam** were given to subjects experiencing hypoxic hypoxidosis. The **aniracetam** was able to reduce the electroencephalogram changes (EEC) that resulted from the induced hypoxidosis. In fact, in this trial, 1000 mg of **aniracetam** was found to be more active than 2000 mg of piracetam. A similar study was performed using 1500 mg of **aniracetam** on young, healthy volunteers. This 1500 mg was given after the subjects were exposed to scopolamine. **Aniracetam** reduced these individuals' cognitive impairment from scopolamine, and this dose was more active than 2400 mg of piracetam (Senin et al., pp.96-97).

Phenylpiracetam

Phenylpiracetam, marketed under the brand name “Phenotropil,” is a nootropic developed in Russia. It has been approved there for correcting cerebrovascular deficiency, focusing attention, ameliorating apathy, and slowing memory decline since 2003. Cosmonauts are even prescribe **phenylpiracetam** to sharpen both their physical and mental abilities while they're in space (Malykh & Sadaie, 2010, p. 290). As you can see, Russia has been giving this nootropic to people with jobs that require astute mental focus and high intelligence.

Improving mood and cognition after brain trauma

Phenylpiracetam is especially worth noting because it is fast-acting and absorbs well orally. For individuals with cognitive impairment and/or depression following encephalopathy and brain injuries (acute lesions, gliomas surgery, and brain traumas), **phenylpiracetam** improved cognition scores for every group. **Phenylpiracetam** did even better than piracetam because it worked much more quickly to ameliorate the impairment, as well as ridding the patients of headaches. Scores of those given **phenylpiracetam** improved in all cognition tests.

Levetiracetam

Levetiracetam, trade name Keppra, is a nootropic that has been approved for use in the United Status since 1999. It is the only -racetam that is registered as a medication there.(290) It is used as an adjunctive (additional) therapy for those four years and older who experience partial onset seizures. In a controlled study of patients with refractory partial seizures, **levetiracetam** improved memory and cognition. In an open-label study, **levetiracetam** helped with language dysfunction present in children who suffered from benign sporadic seizures (Malykh & Sadaie, 2010, p. 299).

Pramiracetam

Pramiracetam has been shown to improve cognitive deficits that are usually associated with or result from brain trauma (Malykh & Sadaie, 2010, p. 288). When compared to placebo, Italian researchers found that **pramiracetam** was 50% more effective than placebo in reducing amnesia-related effects caused by scopolamine intoxication (at hours 1 and 3 following injection). In addition, pramiracetam is even more potent than piracetam and Ukrainian studies found it better at restoring memory loss in patients suffering from mild craniocerebral trauma (298-299).

Noopept

Noopept is a -racetam derivative that is 1000 times more potent than piracetam and is prescribed to patients in Russia. Animal studies have shown this nootropic to be easily absorbed, neuroprotective, and memory enhancing. Moreover, **noopept** penetrates the blood-brain barrier efficiently. **Noopept's** animal studies look promising, similar to piracetam's animal studies before it was tested extensively in humans.

Cholinergics:

Choline

Choline and its derivatives are not vitamins *per se*, but in 1998 the Institute of Medicine (IOM) recognized them as essential nutrients. The reasoning underlying this decision was that **choline** is necessary in a variety of ways for the maintenance of human health. According to Oregon State University's Linus Pauling Institute, **choline** is: used in the synthesis of structural components needed to maintain cell membrane integrity, a precursor to the neurotransmitter acetylcholine (which is involved in muscle control and memory), and able to keep both fat and cholesterol from collecting in the liver.

Choline deficiency

The Linus Pauling Institute also warns individuals to make sure they are not deficient in choline. Among other problems it can cause, **choline deficiency** can lead to a condition called “fatty liver.” In a study of 57 individuals given a **choline** deficient diet, 80% of the postmenopausal women, 44% of the premenopausal women, and 77% of the men had liver damage, “fatty liver,” or muscle damage of some sort. Estrogen levels in the premenopausal women may be responsible for their resistance since estrogen endogenously induces choline synthesis through a couple of different enzymes. Adding choline to the subjects' diets fixed the problem rather quickly.

Benefits of choline

Jane Higdon, PhD. at the Linus Pauling Institute, has wondered whether choline can be used in the prevention of cardiovascular disease and cancer in humans since it has shown promise in this capacity in rats. Another tantalizing line of speculation is based on the fact that higher intake of **choline** on a daily basis by young rats has been shown to lessen later memory deficits due to age. Scientists are currently looking into whether choline or related substances could prove useful in the treatment of Alzheimer's disease.

Huperzine A

Huperzia serrata, a plant which contains the alkaloid **Huperzine A**, is another medicament that, for whatever reason(s), all too often gets swept under the rug and is not given nearly enough of the attention it deserves. WebMd has a great section on Huperzine A's uses in the treatment of age-related memory problems and Alzheimer's disease. This supplement shows promise in improving memory, learning, and energy levels.

As we discussed in the introductory section on cholinergics, huperzine A belongs to a group of cholinergics known as acetylcholinesterase inhibitors (AChEIs). By inhibiting the activity of the enzyme responsible for metabolizing acetylcholine (acetylcholinesterase), AChEIs increase both the amount of acetylcholine present in the synaptic cleft and its duration of action. **Huperzine A** also has other neuropharmacological effects; for instance, it is an NMDA antagonist like the prescription drugs memantine and ketamine.

Vitamins and Supplements:

B-Complex Vitamins

It's especially important to consider supplementation with B-complex vitamins because many of us don't get nearly enough of them in our diets. The highest amounts of B vitamins are found in whole unprocessed foods, which can often

prove difficult to find in local grocery stores. Pay special attention to your diet to make sure you're regularly getting enough B vitamins. **B vitamins** include: B_1, B_2, B_3, B_5, B_6, B_7, B_9, and B_{12}.

Vitamin B deficiency

A dietary deficiency in the consumption of any one of the B vitamins will result in negative consequences; for example, a severe, persistent thiamine (B_1) deficiency can cause limb pain, irregular heartbeat, edema, and even death. In addition, **thiamine deficiency** can result in various sorts of neurological dysfunctions that affect memory, coordination, and vision. Smokers, alcoholics, coffee drinkers, and people who eat large amounts of certain kinds of fish (among others) are all at risk for the development of thiamine deficiency.

As another example, **folic acid (B_9) deficiency** is worrisome in pregnant women because it can result in birth defects for their children. In addition, folic acid deficiency can also result in a number of other disorders, including impaired mental functioning. Some studies suggest that folic acid supplementation may be effective in reducing symptoms of depression, and this in turn suggests that low folic acid intake may be a contributing factor to depression.

Unfortunately, space limitations prevent us from going into every one of the possible adverse effects of each and every member of the B vitamin complex. It is our hope that the two examples given were sufficient to illustrate the importance of these vitamins in regulating both physiological and psychological health. We also hope that the all too brief discussion here will inspire you to do further research of your own, not just in relation to the B vitamins, but to many or most desirably all of the integral components of a healthy diet.

Using B vitamins

Because the B-complex represents an entire group of vitamins, there are quite a few purposes for which one may find it suitable to supplement with individual members of it, either alone or in combination. **Pyridoxine (B_6)**, for instance,

plays an important role in the metabolism of various amino acids. For instance, it is crucial in the creation of histamine, serotonin, and dopamine from their amino acid precursors. **Pyridoxine** supplementation relieves depression, memory problems and peripheral neuropathies that have been caused by dietary deficiencies. It has also been shown to reduce women's chance of developing macular degeneration, and is being examined for possible utility in ameliorating PMS symptoms in premenopausal women (Ehrlich 2011).

It should be noted that some anti-depressants like the irreversible monoamine oxidase inhibitors (or “MAOIs”) reduce pyridoxine levels. Recreational abusers of nitrous oxide and medical professionals who are frequently exposed to it are also both at risk for the development of pyridoxine deficiency, as are tobacco smokers and alcoholics. Adequate intake of pyridoxine at therapeutic levels is vital for all of these groups.

Biotin (B_7) is another vitamin that plays a significant role in metabolism, particularly in relation to carbohydrates, fats, and proteins. **Biotin** is necessary for the production of glucose and amino acids. Biotin deficiency is relatively uncommon in the modern world, but if you regularly eat large quantities of egg whites, you may be at risk of it due to the presence in egg whites of a protein called avidin.

Biotin deficiency can lead to worrisome problems such as: vomiting, anorexia, dermatitis and neurological symptoms including lethargy, hallucinations and peripheral neuropathy. Good dietary sources of biotin include: liver, egg yolks, kidney, and nuts. If for whatever reason you don't have access to these food items, try cereal or biotin supplements (Schnepp 2002).

While we don’t have space to go into much more detail in this section, one B vitamin that is especially relevant to mention in relation to nootropics is **pantothenic acid** (B_5). Among other of its myriad biological functions, pantothenic acid is a co-factor in the conversion of choline to acetylcholine.

Supplementation with **lecithin**, **choline**, **alpha-GPC**, **CDP choline**, and possibly **DMAE** should be enhanced by concurrent supplementation with **pantothenic acid**.

Finally, a notable B vitamin analogue with apparent nootropic properties is known as **sulbutiamine** (trade name Arcalon, among others). Sulbutiamine is related to thiamine (B_1), but has a greater ability to cross the blood-brain barrier than its parent compound. Sulbutiamine has shown promise in the treatment of asthenia as well as the treatment of memory impairment, and we anxiously await the results of future trials evaluating its effectiveness in treating cognitive and other forms of mental dysfunction in both active and abstinent alcoholics.

Bacopa (Brahmi)

Bacopa monierri, also known as **Brahmi**, is a plant native to India that has been traditionally used in Ayurvedic medicine for a whole host of problems, but especially those of anxiety and intellect. In rat studies, **Brahmi** improved memory retention and reduced amnesia caused by electroconvulsive shock, scopolamine intoxication, and/or immobilization (Roodenrys et al., 2002, p. 279).

Results for older healthy volunteers

Bacopa's effect on humans was put to the test in a double-blind, randomized study with a placebo control group. All of the volunteers were healthy and between the ages 40 and 65.

The Facts

- Each group was given a capsule (whether **Bacopa** or placebo) of either 300 mg if the individual weighed less than 90 kg or 450 mg if he or she weighed more than 90 kg.
- Seventy-six subjects completed the trial – four from each group left, only one of whom left due to gastrointestinal difficulties with **Bacopa**.
- Three tests (lasting approximately an hour) were given to each patient,

including:

- baseline (first) test (before medication is administered)
- six to eight weeks into the trial to check-in (no test)
- second test approximately three months later, then patient ceases taking capsules
- post-trial (third) test approximately six weeks later

- A testing session consists of completing various tasks that measure a multitude of memory-related abilities, as well as psychological state (Roodenrys et al., 2002, p. 280).

Now, in this particular study, **Brahmi** did not have a significant effect on subjects' short term memory. It did, however, improve their scores on recalling unrelated word pairs. It is possible that the Bacopa slowed down memory loss (Roodenrys et al., 2002, p. 281).

Brahmi's anxiolytic effects

Another double-blind, placebo-controlled study found that *B. monniera* extract improved processing speed of visual information in healthy volunteers (Stough et al., 2001, p. 481). Test scores showed significant improvement in the **Bacopa** group for learning rate and memory consolidation, along with a decrease in overall state of anxiety and rate of forgetting when compared to the placebo group. Researchers are positing that **Brahmi's** demonstrated modulation of brain serotonin levels in animals may be related to its anxiolytic effect in humans (Stough et al., 2001, p. 483). More in-depth research on humans is necessary to determine whether this is the case for us as well.

Supplements Worth Noting (not nootropics):

Yerba Mate

Yerba Mate is both a plant (*Ilex paraguariensis*) and a traditional South American

drink prepared from that plant that is typically served out of a gourd. The dried leaves are mixed with hot, but not boiling, water. The result is a tea-like beverage that contains three xanthine alkaloids. These are: caffeine, theobromine, and theophylline. **Yerba Mate** is highest in caffeine, as are its extracts. In the United States, Mate is also made using tea bags.

In the *Journal of Food Science's* November 2007 issue, there was an article by two researchers (Heck and Mejia) from the University of Illinois on the research to date about Mate. "Yerba Mate Tea (Ilex paraguariensis): a comprehensive review on chemistry, health implications, and technological considerations" came to a few conclusions about Mate's efficacy. Let's look at those now.

In the review, the authors discuss (among other benefits) Mate's abilities to protect the liver from damage, lower cholesterol levels, and reduce cardiovascular problems. Primarily due to the presence of the aforementioned xanthine alkaloids (particularly caffeine), Mate consumption results in central nervous system **stimulation**; this means that Mate has potential in combating obesity as well as in facilitating general performance enhancement. Due in part to its caffeine and theobromine content, Mate also displays powerful **antioxidant properties**.

However, it's not all good news. There are also some grounds for concern in relation to regular use of Mate. Caffeine and theobromine have both been reported to act as pro-oxidants in certain conditions. Beyond this, there is evidence that long-term antioxidant supplementation may lead to serious health complications. Finally, there is a positive correlation between Mate use and the development of some kinds of cancer.

Chapter IV: How Nootropics are Thought to Work

In this section, we will be going over what is known about the mechanisms of action of some of the best known members of the quintessential nootropic family. We are speaking, of course, of the "stars" among the –racetams and –racetam derived compounds. As an initial caveat, we should note that while there has been some research into the mechanisms by which the –racetams exert their effects, our understanding of how they work is still woefully incomplete.

That said, piracetam and its derivatives have been reported to affect the functioning and density of multiple neurotransmitter receptor types and subtypes. They also have a broad range of other known and suspected beneficial actions on membrane fluidity, cerebral blood circulation and other parameters implicated in the development and maintenance of cognitive functionality. Let's take a closer look at some of the more important members of this group.

Glutamate receptors

Piracetam and aniracetam have both been reported to activate the AMPA sub-type of glutamate receptor[1,2]; this effect was first noticed with aniracetam, and its observation and exploration has led to the development of a new series of smart drugs known as ampakines. In at least two rodent studies, chronic piracetam administration has been reported to significantly increase the density of the NMDA sub-type of glutamate receptors in different areas of the brains of aged

[1] http://www.ncbi.nlm.nih.gov/pubmed/22940587

[2] http://www.ncbi.nlm.nih.gov/pubmed/1975272

rats.[3,4] (A similar result has been reported for phenylpiracetam.) There is also evidence that suggests that piracetam works, in part, by activating NMDA receptors.[5] To our knowledge, there is no evidence that piracetam interacts in any way with the kainate subtype of glutamate receptor.

Cholinergic receptors

In addition, there are by now several decades worth of converging lines of data suggestive of a significant involvement of piracetam and its derivatives with cholinergic functioning in the CNS. Cholinergic functioning is the result of the activity of acetylcholine and its associated receptors, which fall into two sub-types: nicotinic receptors, which are also bound to by nicotine, and muscarinic receptors, which are also bound to by muscarine. Why is it thought that –racetams exert their effects via cholinergic mechanisms?

To begin with, it has long been known that piracetam and other –racetam compounds afford significant protection against the detrimental mental effects of scopolamine,[6] an anticholinergic drug – that is, a drug which antagonizes the action of acetylcholine, in this case at muscarinic receptors. Research and anecdotal reports also both indicate that the desired central effects of piracetam and its derivatives are enhanced by the use of cholinergic agents such as choline

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[3] http://www.ncbi.nlm.nih.gov/pubmed/8234409

[4] http://www.ncbi.nlm.nih.gov/pubmed/10338103

[5] http://www.ncbi.nlm.nih.gov/pubmed/21414388

[6] http://www.ncbi.nlm.nih.gov/pubmed/3137602

and DMAE.[7] This evidence, while suggestive, is indirect.

More directly, one study of piracetam administration in aged rats reported increases in muscarinic cholinergic receptor density in the hippocampi, striata and frontal cortices of the treated animals.[8] In vitro data on pramiracetam indicate that it increases the rate of sodium-sensitive choline uptake in rat synaptosomes (Malykh & Sadaie, 2010, p. 291). Finally, there is also evidence for interactions of both phenylpiracetam and nefiracetam with nicotinic acetylcholine receptors (ibid).

Summary

There are a number of other possible general modes of action of piracetam and its derivatives, some of which we have already touched on in previous sections, including alterations of membrane fluidity in cells, and others that we can only mention in passing, such as evidence for enhanced oxygen utilization in piracetam-treated animals. While researchers are still getting to the bottom of just how –racetam supplements work in the brain and body, what is known to date can help in the formulation of effective nootropic regimens. For instance, combining the known neuropharmacological features of –racetams with the extant data and user anecdotes on nootropic "stacking" makes a clear case for the combination of –racetams with cholinergics such as choline and DMAE.

In 2010, co-authors Malykh and Sadaie neatly summarized the broadly known facts regarding –racetam modes of action as follows: "[t]hese compounds interact with target receptors in brain and modulate excitatory and/or inhibitory processes of neurotransmitters, neurohormones, and/or post-synaptic signals." (291). To

[7] http://www.ncbi.nlm.nih.gov/pubmed/7301036

[8] http://www.ncbi.nlm.nih.gov/pubmed/10338103

narrow our focus a bit, let us now consider aniracetam (trade name Draganon, among others).

Brief discussion of aniracetam

As we previously noted, aniracetam facilitates activity at AMPA receptors. However, it would be a mistake to think that the AMPA activity neatly explains the effects profile of aniracetam. In addition, there is rodent evidence that aniracetam's anxiolytic or anxiety-relieving effects are mediated via dopaminergic, cholinergic and serotonergic neuronal mechanisms. [9]

This seems like a useful juncture to discuss some of the more interesting properties of aniracetam. In addition to its nootropic activity and greater potency by weight than piracetam, aniracetam has been reported to be an effective anxiolytic in both humans and non-human animals. This means that there is evidence for aniracetam being performance-enhancing in at least two different ways: first, via a direct nootropic effect promoting more efficient data processing, and, second, via the reduction of potentially performance-impairing anxiety.

Aniracetam has a fairly short half-life (~2 hours), which can be a detriment or a benefit depending on your purposes. If you are looking for a long-acting smart drug, aniracetam will not be the choice for you. On the other hand, if you want to enhance your intellectual performance over shorter spans of time (for instance, because you do not want to disrupt your sleep schedule), aniracetam will be of great utility.

At this point, we will have to bring this section to a close. We went over some basic points of the nootropic actions of other supplements in previous sections, and the main focus of this e-book is the –racetam nootropic compounds. We hope

[9] http://www.ncbi.nlm.nih.gov/pubmed/11412837

that this information has been of use to you.

Chapter V: Nootropics Compared to Prescription Medications

In Chapter III, we discussed the benefits of various nootropics when compared to placebo and against one another. What happens when we match them up against prescription medications?

-Racetams vs. Anti-depressants (MAOIs, SSRIs)

In Russia, phenylpiracetam is prescribed in the treatment of depression and apathy (Malaykh & Sadaie, 2010, p. 290). Piracetam has shown promise in treating the depression of those suffering from chronic cerebrovascular disorders (p. 297). Unlike MAOIs and SSRIs for depression, the -racetams can be stopped without fear of discontinuation syndrome (withdrawal). Many users of SSRIs remark on feeling “zombified” while under the influence of these substances, which only slightly out perform placebo. While on MAOIs, users must maintain a strict diet as foods high in tyramine (such as cured/dried meats, aged cheese, spoiled foods, soy sauce, and more) cause a dangerous spike in blood pressure (Hall-Flavin, Daniel, 2013). These dietary restrictions are not necessary with the -racetams.

Aniracetam vs. Benzodiazepines (alprazolam, diazepam, lorazepam, clonazepam)

In a study of mice in which one group was given aniracetam and another diazepam, the former showed anxiolytic effects in four different anxiety models compared to the latter's two (Nakamura & Kurasawa, 2001). Benzodiazepines are great as quick treatments for panic attacks, but frequent and extended use of them is not necessarily a good idea. Individuals habituated to benzodiazepines may experience negative withdrawal symptoms upon cessation; if serious enough, it can lead to seizures and even death. Aniracetam has no such withdrawal symptoms.

Modafinil vs. Amphetamine Salts

Modafinil is a “smart drug” that when taken allows the individual to sit down, study for hours, and easily recall the information he or she has memorized. Mind you, Modafinil is considered a nootropic by many and amphetamine is considered one by a few, but both are actually prescription medications in the United States at least. Adderall users with severe ADD and ADHD sometimes experiencing difficulty using modafinil in place of their usual medication, but they report positive results from lowering the former's dosage and adding the latter to their regimen. Both of these drugs require prescriptions in the United States; however, Modafinil is worth mentioning because it falls under the term “nootropic.”

As information currently stands, modafinil does not produce euphoria comparable to traditional stimulants. This information, along with trials conducted, have led scientists to conclude that modafinil may have abuse potential, but that it is much lower than with amphetamine or methamphetamine.

Modafinil has been promoted as a “wakefulness agent,” at first being used to treat narcolepsy in patients. Now, it has grown in popularity as a nootropic because it's effective in increasing memory and focus. Peter Borden, a fan of acupuncture and alternative health in general, had this to say about modafinil: “My senses sort of shifted to the visual, and my auditory sense went down. Sounds didn’t even register. It was like walking around on a winter day when it just snowed. It was very easy to stay visually focused” (Kolker 2013). On the other hand, if you're an insomniac, neither of these medications is an especially good idea.

Chapter VI: Stacking Nootropics

Now, it is usually not the best idea to take everything you hear/read on face value. Don't get too excited to run out and purchase a cart full of supplements until you've done further research. A few important questions to keep in mind are:

- What exactly do I want to take a supplement for? Do I want to increase my focus or ability to concentrate for extended periods of time? Do I simply want to relax?
- What medications or supplements am I currently on? Are any of them contraindicated to (as in, should not be used with) the nootropics I'm interested in?
- Is it necessary for me to take more than one nootropic at a time?

If you're new to the world of nootropics, don't be in a rush to take multiple types at once. You'll want to find out how each affects you when used on its own before you mix it with any other substances. Makes sense, right? This chapter is for those well-versed in the uses of nootropics who want to find the perfect combinations.

Caffeine + L-Theanine

In the strict sense of the term “nootropic,” caffeine doesn't really fit the criteria. At the same time, caffeine's stimulant properties have been noted to be helpful in increasing focus and energy. L-Theanine is used for treating anxiety, preventing Alzheimer's disease, and potentiating cancer drugs (making them more effective) according to its entry on WebMD. It may seem pointless to take a stimulant with a supplement originally found in green tea that relaxes you; however, users on the Smarter Nootropics website report that the two mix well. The normally unpleasant side effects of caffeine such as anxiety and jitteriness are countered by the l-theanine. You may experience alertness and lucidity without any

unnecessary nervousness.

-Racetam + Choline Source

Many nootropics enthusiasts would likely not even call this kind of combination a "stack" because they would consider it necessary. Various animal studies are out concluding that the -racetam family paired with a source of choline produces optimal results. One trial found that piracetam + choline led to enhanced memory retention more than piracetam alone (Bartus et al., 1981). Choline has also been noted to get the piracetam working more quickly. The cholinergic Huperzine A functions similarly when combined with piracetam.

Aniracetam + CDP choline (citicoline) is a favored combination according to the Best Nootropic review website due to aniracetam's anxiolytic effects and citicoline's effectiveness. One such combined stack on the market today is Nootrobrain, which uses the two aforementioned supplements along with Bacopa leaf and vitamin B6 – both of which we discuss in Chapter III.

Piracetam + Vasodilator Drug (in this case, cinnarizine)

In a study of multiple sclerosis patients, piracetam paired with cinnarizine, which acts as a vasoldiator, improved mental abilities even more than piracetam alone. Activity levels and mood increased in these patients who were assumed to still be suffering from encephalopathy (Malykh & Sadaie, 2010, p. 297).

-Racetams (and/or Modafinil and similar drugs) + Essential Vitamins

If you've noticed that you're not getting enough of certain vitamins in your diet, try supplementing with them in addition to piracetam, aniracetam, etc. and/or modafinil, adrafinil, etc. In Chapter III, we discuss the signs of certain B vitamin deficiencies. Check with your doctor, and have him or her run tests to determine how healthy your diet is. Since nootropics are rarely toxic, as long as you get your dosage of vitamins correct and don't overdo it, this type of combination will be beneficial. Nootrobrain, for example, combines vitamin B_6 with aniracetam

(plus citicoline and Bacopa leaf).

Choline Source + Huperzine A

In this combination, the choline (or choline derivative) boosts the effects of the Huperzine, even further enhancing concentraction. Choline itself shows evidence of being effective at slowing down age-related memory loss. One such product that combines these, along yerba mate, caffeine, guarana, and ginkgo biloba is AddieUp used for the purpose of promoting learning while increasing energy (hence the yerba mate, caffeine, and guarana).

My Top Choice For Nootropic Supplements

I have personally tested dozens of nootropic supplements, and I think that Mind Boost is the most effective in terms of results. It takes a few days to kick in, but when it does the effect is huge.

Check Out My Official **Mind Boost Review Here**

Chapter IX: Common Questions about Nootropics

“Are nootropics safe?”

Generally, yes, but it also depends on which nootropic you have in mind. Overall, the -racetams (piracetam, aniracetam, phenylpiracetam, levetiracetam, and noopept) have low toxicity, and side effects 1) are uncommon and 2) do not last long. According to Dr. Giurgea who coined the term “nootropic,” low toxicity is a requirement for supplements that fall into the aforementioned category. In addition, nootropics are not addictive and do not cause sickness upon cessation of treatment. In fact, the -racetams work better the more they build up in your system.

“There can be side effects though?”

Yes, you would be hard-pressed to find a medicine that does not have at least one possible side effect. It is possible to experience negative side effects from nootropics, although as previously discussed, they are rare and transitory. They include: anxiety, headache, confusion, and gastrointestinal upset. In the majority of -racetam studies on humans, patients who experienced these side effects were able to adjust without leaving the trials. Gastrointestinal problems led to the withdrawal of one patient using Bacopa from a trial of 84 healthy subjects.

“Is it safe to use a nootropic along with my prescribed medication?”

Most of the time, yes, although you will want to check with your doctor to be certain. In a study to test piracetam's effects on chronic schizophrenia patients, the piracetam was safely given to the subjects while they continued to take their psychotropic medications (Dimond et al., 1979, p. 343). Levetiracetam is approved in the United States as an additional treatment to antiepileptic drugs (Malykh & Sadaie, 2010, p. 302). If you have a serious condition, do not cease

treatment with your prescribed medication without talking to your doctor first.

“I'm happy and healthy. Do I really need nootropics?”

The answer to that question is up to you. Throughout this book, we've mentioned quite a few studies that were aimed at seeing how young, healthy people reacted to nootropics – remember Chapter III? From what research has established so far, the -racetams appear to act selectively and perhaps even more specifically at higher levels of cognition. They have demonstrated positive effects in different groups of people, young and old alike. This, along with their generally low toxicity, make for great supplements especially when compared to many prescription medications.

“If I stop taking nootropics, will I feel stupid?”

If you decide to quit supplementing with nootropics, you may notice your cognitive abilities returning to baseline. This does not mean that you will suddenly forget everything you studied while on nootropics, just that different aspects of your memory may be affected. For example, you could experience enhanced memory recall while taking aniracetam and then notice it's returning back to normal once you stop.

“How long should I take the -racetams for?”

Most of the human trials with piracetam noted significant effects at 12 or more weeks in. Set yourself a trial period of three to four months with regularly piracetam dosing, and make note of your results. Taking choline (or a choline derivative) can speed up how quickly you see effects. Remember, the -racetams are drugs that build up in your system, but they do not show any substantial tolerance increase. The more you take them, the better they work. As discussed in the aniracetam, phenylpiracetam, and Noopept sections in Chapter III, these three supplements have demonstrated significant effects in areas of memory and cognition much more quickly than piracetam by itself has.

“How much should I start with?”

This depends on various factors. Your best bet is to peruse the Internet for studies relevant to you – are you healthy, young, old, suffering from brain trauma, or what? Many of the studies we discuss in Chapter III state who the patients in each trial were, so that should provide you with a jumping off point. The Internet is particularly great in this instance too because it's incredibly easy to read experience reports posted by people who have and are experimenting with nootropics. A quick search of keywords (such as the nootropic you're interested plus the term “experiences” or “reports”) will bring a wealth of information directly to your screen (kind of like how this eBook is functioning as we speak).

“I suffer from narcolepsy. Would Modafinil be helpful?”

Modafinil is prescribed for exactly this purpose in the United States and has been approved by the FDA for it, along with sleep apnea. If you are experiencing these problems, it would be worthwhile to talk to your doctor about getting a prescription. The National Multiple Sclerosis Society warns users not take Modafinil if they experience insomnia frequently as it is. Due to Modafinil's long half-life (15 hours), you should be taking yours early in the morning.

Chapter VIII: How to Choose the Right Nootropic

Let's be honest, we've covered a significant amount of information in this book so it's understandable to feel a bit frazzled by it all. Because the world of nootropics is vast and continually expanding, it is particularly difficult to keep up with all of the latest novel supplements. This is certainly possible when you have spent years interested in the topic, but it is unrealistic when you are first learning about enhancing your cognition. For this reason, we have compiled a list of questions to ask yourself so that you can make a step in the right direction. Enjoy!

1. **Are you having difficulty staying awake even during your favorite activities, but especially during school or work?** Ask your doctor about Modafinil if you live in the United States. This medication is actually sold for the specific purpose of treating narcolepsy.
2. **Do you need a pick-me-up in the morning?** Try yerba mate, guarana, and/or caffeine (as found in the product AddieUp).
3. **Are you looking to enhance your cognitive abilities, particularly those of memory and concentration over an extended period of time?** Try one of the -racetams or Bacopa (check out Chapter III to note the differences between all of them). Use choline or a choline derivative with the -racetams to get significant effects more rapidly.
4. **Have you experienced brain trauma of some sort and want to improve learning scores as you recover?** Piracetam and aniracetam in particular are beneficial in this area, especially when it comes to encephalopathy and brain surgeries.
5. **Is your diet rich in vitamins and minerals? Have you seen your doctor recently about your overall health?** If you find that you are

deficient in specific vitamins, supplementing with them can easily fix the problem. Be sure to check whether the vitamins are fat or water soluble. If they are the former, take them with a source of fat (like whole milk). Vitamin deficiencies can cause all sorts of nasty problems, such as nausea, weakness, lethargy, and dermatitis (and these are not even the serious issues either).

6. My Top Choice For Nootropic Supplements

I have personally tested dozens of nootropic supplements, and I think that Mind Boost is the most effective in terms of results. It takes a few days to kick in, but when it does the effect is huge.

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References

Bartus R.T., Dean R.L. 3Rd, Sherman K.A., Friedman E., Beer B. (1981). Profound effects of combining choline and piracetam on memory enhancement and cholinergic function in aged rats. *Neurobiology of Aging, 2(2),* 105.

Dimond S.J. & Brouwers E.M. (1976). Increase in the power of human memory in normal man through the use of drugs. *Psychopharmacology, 49(3),* 307-309.

Dimond S.J., Scammel R.E., Price I.G., Huws D., Gray C. (1979). Some Effects of Piracetam (UCB 6215 Nootropyl) on Chronic Schizophrenia. *Psychopharmacology, 64,* 341-348.

Eckert G.P., Cairns N.J., Mueller W.E. 1999. Piracetam reverses hippocampal membrane alterations in Alzheimer's disease. *Journal of Neural Transmission, 106,* 757-761.

Ehrlich, S. (2011). Vitamin B6 (Pyridoxine). University of Maryland Medical Center. Retrieved from http://umm.edu/health/medical/altmed/supplement/vitamin-b6-pyridoxine

Hall-Flavin, D. M.D. (2013). I just started taking MAOIs for depression. Do I really need to follow a low-tyramine diet? Mayo Clinic. Retrieved from http://www.mayoclinic.com/health/maois/HQ01575

Kolker, R. (31 March 2013). The Real *Limitless* Drug Isn't Just for Lifehackers Anymore. New York Magazine. Retrieved from http://nymag.com/news/intelligencer/modafinil-2013-4/

Lee C.R., & Benfield, P. (1994). Aniracetam. An overview of its pharmacodynamic and pharmacokinetic properties, and a review of its therapeutic potential in senile cognitive disorders. *Drugs & Aging, 4(3)*, 257-273.

Malykh A.G. & Sadaie M.R. (2010). Piracetam and piracetam-like drugs: from basic science to novel clinical applications to CNS disorders. *Drugs, 70(3),* 287-312.

Nakamura K. & Kurasawa M. (2001). Anxiolytic effects of aniracetam in three different mouse models of anxiety and the underlying mechanism. *European Journal of Pharmacology, 420(1),* 34.

Ostrovskaia R.U., Gudasheva T.A., Voronina T.A., Seredenin S.B. (2002). The original novel nootropic and neuroprotective agent noopept. *Eksp Klin Farmakol, 65(5)*, 66-67.

Schnepp, Z. (2002). Biotin. School of Chemistry, University of Bristol. Retrieved from http://www.chm.bris.ac.uk/webprojects2002/schnepp/biotin.html

Senin U., Parnetti L., Cucinotta D., Criscuolo D., Longo A., Marini G. (1993). Clinical Experience with Aniracetam in the Treatment of Senile Dementia of the Alzheimer's Type and Related Disorders.” *Drug Investigation, 5(Suppl. 1),* 96-97.

Singh H.K. & Dhawan B.N. (1997). Neuropsychopharmacological effects of the Ayurvedic nootropic Bacopa monniera Linn. (Brahmi). *Symposium, 29(5),* 59-365.

Stough C., Lloyd J., Clarke J., Downey L.A., Hutchison C.W., Rodgers, T., Nathan P.J. (2001). *Psychopharmacology (Berlin), 156(4),* 481-484.

Poeck K. (1998). Piracetam Treatment in Post-Stroke Aphasia. *CNS Drugs, 9(Suppl. 1),* 51-56.