PANMOL®
NADH

THE PATENTED ACTIVITY NUTRIENT COFACTOR
1. GENERAL BACKGROUND

While the caloric doctrine primarily deals with the caloric value of our nutrition, electron donors have rapidly become more important within modern nutritional medicine. Electron donors provide our bodies with electrons. They strengthen the immune system, supply energy to our bodies (brain, cardiovascular system, muscular system), support regeneration processes and delay aging. Vitamins and enzymes are to be seen as the most important electron donors. Vitamin deficient nutrition, through an increased intake of pleasure products (alcohol, tobacco), permanent stress and high performance sport activities also increase the demand and utilization of electron donors. Deficits of electron donors lead to mental and physical loss of activity and accelerate the aging process.

Biological electron donors can be described within this sphere of nutritional medicine as antioxidants (such as carotenoids, vitamin C and vitamin E). The strongest antioxidant in our body is NADH. NADH (short form for nicotinamide-adenine-dinucleotide) is a critical cofactor in hundreds of biochemical reactions associated with energy metabolism. NADH is the biologically active form of vitamin B3 (niacin). The extremely high activity of NADH however pays its price. NADH is very sensitive to light and oxygen and therefore very unstable. Consequently, until only a few years ago, it was possible to administer NADH only via infusions. PANMOL®-NADH is stabilized by a natural process and is therefore orally administrable and stable for years. Finally an organic form of NADH is now available.

1.1. ROLE AND FUNCTION OF NADH IN THE BODY

Central role in the cellular energy production
NADH stands at the first place of the respiratory chain and belongs to the most important and energy rich electron carriers of human metabolism. With oxidation of nutrients it is biologically irreplaceable for the production of cellular energy (ATP-production) and for cell regulation. *, **

Element of various regenerative enzymes
NADH is the coenzym of various hydrogen transferring enzymes (dehydrogenases, oxidoreductases) and supports the regeneration of the brain, heart, vascular and muscle cells including the functions of the reproductive organs.

Positively influences the synthesis of activating neurotransmitters
Studies confirm that NADH has a positive influence on the synthesis of neurotransmitters. In-vitro studies show that NADH, dosage dependantly, increases the production of the neurotransmitter dopamine up to 6 times.* Furthermore NADH stimulates tyrosine hydroxylase (TH), the key enzyme for the production of dopamine up to 70 %.***

The neurotransmitter dopamine deserves special consideration from a biopsychological standpoint, as it is apparently important for several basic behavioural functions such as motivation, learning, psychomotility and attentiveness.

Works as an antioxidant protecting against free radicals****
NADH is the most powerful antioxidant present in the human body and fulfils a major role in the neutralization of cytotoxic radicals and peroxides.
NADH-deficits demonstrate reduced:

- memory and concentration performance
- intellectual performance
- alertness and attentiveness
- heart function
- muscle performance
  and
- reduced cell- and tissue protection

REFERENCES:


### 2. PANMOL®-NADH

<table>
<thead>
<tr>
<th></th>
<th>PANMOL®-NADHMICRO</th>
</tr>
</thead>
<tbody>
<tr>
<td>NADH - content</td>
<td>10%</td>
</tr>
<tr>
<td>combinable</td>
<td>also with other non-active and active ingredients.</td>
</tr>
<tr>
<td>possible products</td>
<td>capsules</td>
</tr>
<tr>
<td>endproduct must be coated (acidic resistant)</td>
<td>no</td>
</tr>
</tbody>
</table>
4. DEVELOPMENT HISTORY

During the middle of the last century NADH was successfully used as an infusion to treat various neurological illnesses such as Parkinson’s disease, Alzheimer’s disease and also for late-onset dementia. Due to its chemical instability, infusions always had to be freshly prepared. Although the central activating and stimulating characteristics of this biomolecule were identified 5 decades ago, broad usage of NADH, because of its highly sensitive and instable molecular-characteristics, was not possible. An oral application of NADH was totally out of the question, because the substance would have been destroyed immediately after ingestion through contact with gastric acid.

4.1. PANMOL®-NADH

At the turn of this century a Research Team of the vis vitalis-group / Austria, began to study the biochemical and biological characteristics of NADH. Through various ongoing comparison investigations with plant cells it was possible to identify a plant molecule that had similar characteristics to NADH: chlorophyll. If NADH is the strongest electron donor in humans, then chlorophyll is the plant’s counterpart. After a series of research tests it was possible to combine NADH and chlorophyll in a manner that resulted in a natural stabilized NADH-complex. Therefore for the first time the highly active and highly sensitive biomolecule NADH was stabilized through combination with a natural element.
4.2. PANMOL®-NADH <sub>MICRO</sub>

After extensive tests at a second phase it became possible to coat the stable NADH/chlorophyll-complex with other natural substances – in particular plant fats and bees wax – to form microcapsules resistant to gastric acid. With this PANMOL®-NADH<sub>MICRO</sub> was born, a stabilized oral form of NADH that, for the first time, is also combinable with other biologically active ingredients.

5. PANMOL® NADH - SCIENTIFIC BACKGROUND

Being a heterotrophic creature, man needs macromolecules (protein, fats, carbohydrates) every day in order to meet his/her energetic and substantial demands. In the context of energy metabolism, human cells burn macronutrient cleavage products by transferring the electrons of these cleavage products to molecular respiratory oxygen. In doing so, thermal energy and biochemical storage energy are formed as depots in the form of ATP and other phosphates rich in energy. The major portion of these cleavage products is, however, also used for the biosynthesis and regeneration of new biological structures (e.g., enzymes, hormones, body cells, connective tissue). Those synthetic procedures too require both electrons and biochemical energy (e.g., ATP) released from combustion processes. Both oxidation (which releases energy by destroying chemical structures) and reduction (which builds up new biological structures by energy consumption) are metabolic processes by which electrons are transferred from macronutrient molecules. Thus, all metabolic processes occurring in the human body are oxidation and reduction processes, so-called redox processes, for which „health-promoting“ antioxidants are essential. Since electrons cannot be transferred in isolated states during biochemical processes, the cell requires transfer molecules, which are also referred to as electron carriers. In principle, every atom, every molecule, which transfers electrons to a reaction partner, is an electron carrier or—in relation to that partner onto which the electron is transferred—an electron donor, or a reductant, or an antioxidant, respectively. The potential of transferring electrons varies from one molecule to another. The lower the standard redox potential, the stronger the electron pressure exerted by that molecule, and vice versa. Molecules having low standard redox potentials carry electrons rich in energy, which flow either into biochemical energy or into the synthesis of new biological structures of high order, which means that the vitality of the human body depends, in the end, on the magnitude of the fraction of energy-rich electrons in food.

NADH, for instance, ranks among the most important and energy-richest electron carriers in human metabolism because of its extremely low standard redox potential of -320 mV. NADH transfers electrons (a bound „hydride“) to molecular respiratory oxygen primarily to recover energy (while simultaneously forming ATP). That reaction process - which is referred to as oxidative phosphorylation or respiratory chain-takes place in the mitochondria of the cells. Thus, NADH is, so to speak, symbolic of cellular energy. Supplementation studies with NADH have shown that NADH promotes cognitive, intellectual and motoric abilities in patients with Parkinson’s disease and Alzheimer’s disease as well as in those with fatigue, lethargy and chronic fatigue syndrome. NADH also enhances vitality and motivation, and occasionally has been reported to eliminate libido and potency disorders. Depression, learning difficulties and concentration can also be a consequence of limited endogenous NADH synthesis, as can reduced physical performance and stressful circumstances and during endurance sport. Last but not least, NADH promotes the synthetic and detoxicating functions of the liver.

Cognitive function: Based on its biochemical mode of action, NADH may promote cognitive
function by indirectly increasing dopamine synthesis, decreasing mitochondrial dysfunction by helping to restore function of certain NADH-dependent mitochondrial enzymes that are closely linked to energy metabolism of neurons and by increasing energy production of the cells in the brain and periphery, which may lead to a higher capacity in their metabolic performance.

The efficacy of a stabilized NADH as a countermeasure for jet lag was examined. Thirty-five healthy, employed subjects participated in a double-blind, placebo-controlled study. Training and baseline testing were conducted on the West Coast before subjects flew overnight to the East Coast, where they experienced a 3-hour time difference. Upon arrival, individuals were randomly assigned to receive either 20 mg NADH or identical placebo tablets. All participants completed computer-administered tests (including Cog Screen) to assess changes in cognitive functioning, mood, and sleepiness in the morning and afternoon. Jet lag resulted in increased sleepiness for over half the participants and deterioration of cognitive functioning for approximately one third. The morning following the flight, subjects experienced lapses of attention in addition to disruptions in working memory, divided attention, and visual perceptual speed. Individuals who received NADH performed significantly better on 4 cognitive test measures and reported less sleepiness compared with those who received placebo. NADH significantly reduced jet lag-induced negative cognitive effects and sleepiness, was easily administered, and was found to have no side effects.¹

Demarin et al. evaluated the effect of stabilized oral reduced nicotinamide adenine dinucleotide (NADH) on cognitive functioning in patients with Alzheimer’s disease (AD). The trial was a randomized, placebo-controlled, matched-pairs, double-blind, 6-months clinical study. Patients with probable AD (n = 26) were randomized to receive either stabilized oral NADH (10 mg/day) or placebo. Twelve pairs of subjects were matched for age and baseline total score on the Mattis Dementia Rating Scale (MDRS) and the Mini Mental State Examination. After 6 months of treatment, subjects treated with NADH showed no evidence of progressive cognitive deterioration and had significantly higher total scores on the MDRS compared with subjects treated with placebo (p < 0.05). Analysis of MDRS subscales revealed significantly better performance by NADH subjects on measures of verbal fluency (p = 0.019), visual-constructional ability (p = 0.038) and a trend (p = 0.08) to better performance on a measure of abstract verbal reasoning.²

In older learning-impaired rats cognitive-enhancing properties of NADH could also be observed. After receiving 10 – 100 mg NADH/kg for 10 days, the treated animals showed improved navigation performance, which was in some instances superior to the performance of their untreated, younger counterparts.³

**Physical endurance:** There have been several experimental and animal studies published, suggesting that strenuous exercise correlates with levels of NADH. In an open label trial the dispersion of reaction times and the ergometric performance of seventeen competition level athletes (cyclists and long distance runners) have been examined before and 4 weeks after a daily supplementation of 5 mg of NADH. The dispersion of reaction times (DRT) became better and so did the quality and the speed of recognizing symbols in a certain pattern. In 9 of the 17 athletes the continuous attention as well as the maximum performance did improve.⁴

**Cardiovascular health:** Dysfunction of the blood vessels, particularly the endothelium, may result from increased production of free radicals and decreased availability of the endothelial derived relaxing factor nitric oxide (NO) either so comprised synthesis, release or its activity. Lack of NO contributes to impaired blood vessel relaxation, platelet aggregation, enhanced leucocyte adhe-
sion to the endothelium, and increased blood pressure. A double-blind, placebo-controlled study with spontaneously hypertensive rats (SHR) investigated the effects of oral NADH on blood pressure. Ten rats received placebo; and ten, NADH for ten weeks. Systolic BP was measured by tail plethysmography. Blood was collected terminally, and chemistries were performed by routine methodologies. Thiobarbituric acid reactive species (TBARS) (an estimate of lipid peroxidation/free radical formation) was measured in renal and hepatic tissues. Although systolic BP did not differ between the two groups over the first month, it decreased and stayed markedly lower for the remainder of study in SHR receiving oral NADH. At the end of 60 days, SBP in NADH-treated SHR was 184 mm Hg +/- 2.8 (SEM) compared to 201 mm Hg +/- 2.1 (SEM) in control SHR (p < 0.001). NADH intake lowered total cholesterol (p < 0.002) and LDL (p < 0.02). Renal TBARS were also significantly lower in SHR receiving NADH (P < 0.001). Accordingly, supplementation with the natural coenzyme NADH theoretically could prove to be useful in preventing age-related increases in BP and, thus, various cardiovascular maladies.\(^{\text{V}}\)

**Liver metabolism and reproductive Organs:** NADH promotes the synthesis and detoxification functions of the liver. Alcoholic beverages primarily consist of water, pure alcohol (chemically known as ethanol), and variable amounts of sugars (i.e., carbohydrates); their content of other nutrients (e.g., proteins, vitamins, or minerals) is usually negligible. Alcohol is broken down (i.e., metabolized) in the liver primarily through two pathways: the alcohol dehydrogenase (ADH) pathway and the microsomal ethanol–oxidizing system (MEOS). In people who consume alcohol at moderate levels and/or only occasionally, most of the alcohol is broken down by ADH, an enzyme found in the fluid that fills the cell (i.e., the cytosol). ADH converts alcohol (chemically known as ethanol) to acetaldehyde, a toxic and highly reactive molecule. During this reaction, hydrogen is removed from the alcohol and transferred to nicotinamide adenine dinucleotide (NAD), converting it to reduced NAD (NADH). NADH participates in numerous other metabolic reactions, passing on the hydrogen to other compounds.\(^{\text{VI, VII}}\)

NADH is also reported to eliminate libido and potency disorders. Testosterone is thought to be a male hormone but it exists in both genders and fulfills many functions. Women produce up to 2% of what a man would produce. Testosterone is produced in the testes of men and in the ovaries of women. In both sexes, it aids in bone and muscle development and blood cell turnover among its many functions. It also influences libido in both men and women. Research has shown that alcohol suppresses testosterone production. Alcohol increases the breakdown and removal of testosterone from the blood and decreases testosterone production rate.\(^{\text{VIII}}\) NADH is able to decrease negative effects of alcohol on testosterone levels.\(^{\text{VII, IX}}\)

**References:**


6. DATA AND FACTS

6.1. STABILITY OF COMBINED PANMOL\textsuperscript{®}-NADH\textsubscript{MICRO} (storage conditions: 30°C, 60 % rel.h.)

<table>
<thead>
<tr>
<th>months</th>
<th>0</th>
<th>4</th>
<th>8</th>
<th>12</th>
<th>16</th>
<th>20</th>
<th>24</th>
<th>28</th>
</tr>
</thead>
<tbody>
<tr>
<td>content of NADH in %</td>
<td>100</td>
<td>50</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

PANMOL\textsuperscript{®}-NADH in combination with vitamins and trace elements

Common NADH in combination with vitamins and trace elements

6.2. ORAL NADH MICROENCAPSULATION OF PANMOL\textsuperscript{®}-NADH\textsubscript{MICRO}

Enteric-coated NADH microspheres

Enteric shell

Stabilized NAD sphere

Other active & inactive ingredients

100 µm
7. PATENT DOCUMENT

Short title: NADH/NADPH-Containing compound

European patent number: 15 62 613

US patent number: 72 55 813

Russian patent number: 23 38 540

Canadian patent number: 25 03 860

Patents pending.

Patents pending.

Japanese patent pending number: 2004-55 22 58

Patents pending.

Patents pending.

Patents pending.

Patents pending.

Serbian patent number: 51606

Croatic patent pending number: P200 50 573 A

United Arab Emirates patent pending number: 248/2005

Norwegian patent pending number: 200 52 839
8. SCIENTIFIC LITERATURE


Background / Objective: Depression and dementia represent two of the most important diseases in advanced age. The positive influence of particular nutrients such as NADH, B-vitamins and various trace elements is well documented in this connection. The aim of this study was to test the influence of a complex nutritional supplement on memory, concentration and mood.

Methods: The Hamilton Depression Scale (HAM-D-17) was used for investigating the parameter depression. In order to quantify dementia (divided into memory achievement and attention), the Syndrome Short Test (SKT) was used. The study was designed as a double-blind, placebo-controlled, prospective trial.

Results: Within three months 28 residents of nursing homes (24 women, 4 men) of the age range 49-94 years (78.39 ± 9.19 years) could be included in the study. The verum group showed a significant improvement of depressive symptomatology (p = 0.049) in the comparison to placebo group. With regard to the memory and attention parameters, a clear benefit for the supplemented group also resulted.

Conclusion: This study could document a beneficial effect of the nutritional supplement on the examined parameters dementia and depression.


BACKGROUND: Chronic fatigue syndrome (CFS) is a disorder of unknown etiology, consisting of prolonged, debilitating fatigue and a multitude of symptoms including neurocognitive dysfunction, flu-like symptoms, myalgia, weakness, arthralgia, low-grade fever, sore throat, headache, sleep disturbances and swelling and tenderness of lymph nodes. No effective treatment for CFS is known.

OBJECTIVE: The purpose of the study was to evaluate the efficacy of the reduced form of nicotinamide adenine dinucleotide (NADH) i.e., ENADA the stabilized oral absorbable form, in a randomized, double-blind, placebo-controlled crossover study in patients with CFS. Nicotinamide adenine dinucleotide is known to trigger energy production through ATP generation which may form the basis of its potential effects.

METHODS: Twenty-six eligible patients who fulfilled the Center for Disease Control and Prevention criteria for CFS completed the study. Medical history, physical examination, laboratory studies, and questionnaire were obtained at baseline, 4, 8, and 12 weeks. Subjects were randomly assigned to receive either 10 mg of NADH or placebo for a 4-week period. Following a 4-week washout period, subjects were crossed to the alternate regimen for a final 4-week period.
RESULTS: No severe adverse effects related to the study drug were observed. Within this cohort of 26 patients, 8 of 26 (31%) responded favorably to NADH in contrast to 2 of 26 (8%) to placebo. Based upon these encouraging results we have decided to conduct an open-label study in a larger cohort of patients.

CONCLUSION: Collectively, the results of this pilot study indicate that NADH may be a valuable adjunctive therapy in the management of the chronic fatigue syndrome and suggest that further clinical trials be performed to establish its efficacy in this clinically perplexing disorder.


Current remedies for jet lag (phototherapy, melatonin, stimulant, and sedative medications) are limited in efficacy and practicality. The efficacy of a stabilized, sublingual form of reduced nicotinamide adenine dinucleotide (NADH, ENADAlert, Menuco Corp.) as a counter-measure for jet lag was examined. Because NADH increases cellular production of ATP and facilitates dopamine synthesis, it may counteract the effects of jet lag on cognitive functioning and sleepiness. Thirty-five healthy, employed subjects participated in this double-blind, placebo-controlled study. Training and baseline testing were conducted on the West Coast before subjects flew overnight to the East Coast, where they would experience a 3-hour time difference. Upon arrival, individuals were randomly assigned to received either 20 mg of sublingual stabilized NADH (n = 18) or identical placebo tablets (n = 17). All participants completed computer-administered tests (including Cog Screen) to assess changes in cognitive functioning, mood, and sleepiness in the morning and afternoon. Jet lag resulted in increased sleepiness for over half the participants and deterioration of cognitive functioning for approximately one third. The morning following the flight, subjects experienced lapses of attention in addition to disruptions in working memory, divided attention, and visual perceptual speed. Individuals who received NADH performed significantly better on 4 cognitive test measures (P ≤ .05) and reported less sleepiness compared with those who received placebo. No adverse effects were observed with NADH treatment. Stabilized NADH significantly reduced jet lag-induced negative cognitive effects and sleepiness, was easily administered, and was found to have no side effects.


This study was designed to evaluate the effect of stabilized oral reduced nicotinamide adenine dinucleotide (NADH) on cognitive functioning in patients with Alzheimer’s disease (AD). NADH is a coenzyme that plays a key role in cellular energy production and stimulates dopamine production. In previous trials NADH has been shown to improve cognitive functioning in patients with Parkinson’s disease, depression and AD. The present trial was a randomized, placebo-controlled, matched-pairs, double-blind, 6-month clinical study. Patients with probable AD (n = 26) were randomized to receive either stabilized oral NADH (10 mg/day) or placebo. Twelve pairs of subjects were matched for age and baseline total score on the Mattis Dementia Rating Scale (MDRS) and the Mini Mental State Examination.
After 6 months of treatment, subjects treated with NADH showed no evidence of progressive cognitive deterioration and had significantly higher total scores on the MDRS compared with subjects treated with placebo \( (p < 0.05) \). Analysis of MDRS subscales revealed significantly better performance by NADH subjects on measures of verbal fluency \( (p = 0.019) \), visual-constructional ability \( (p = 0.038) \) and a trend \( (p = 0.08) \) to better performance on a measure of abstract verbal reasoning. There were no differences between groups in measures of attention, memory, or in clinician ratings of dementia severity (Clinical Dementia Rating). Consistent with earlier studies, the present findings support NADH as a treatment for AD.


The coenzyme Nicotinamide adenine dinucleotide (NADH) has been used as novel medication in 34 Parkinson patients in an open label trial. In all patients, a beneficial clinical effect was observed. Twenty-one patients (61.7 percent) showed a very good (better than 30 percent) improvement of disability and 13 patients (38.3 percent) a moderate (up to 30 percent) improvement. The effect of NADH was dependent on the dosage and the severity of the case. The best therapeutic dose was in the range of 25 to 50 mg per day. The clinical improvement was more pronounced after i.v. and less after i.m. administration. Concomitant with improvement of the disability, the urine level of homovanillinic acid (HVA) increased significantly in all patients (in some patients by more than a 100 percent), indicating a stimulation of the endogenous L-DOPA biosynthesis. The daily „on phases“ of the patients could be increased from two up to nine hours in the individual patients by NADH administration.


The reduced coenzyme nicotinamide adenine dinucleotide (NADH) was used as medication in 885 parkinsonian patients in an open label trial. About half of the patients received NADH by intravenous infusion, the other half received capsules, orally. In about 80% of the patients a beneficial clinical effect was observed: 19.3% of the patients showed a very good (30-50%) improvement of disability, 58.8% a moderate (10-30%) improvement. 21.8% did not respond to NADH. Statistical analysis of the improvement in correlation with the disability prior to treatment, the duration of the disease and the age of the patients revealed the following results: All these 3 parameters have a significant although weak influence on the improvement. The disability before the treatment has a positive regression coefficient \( (t \text{ value } < 0.01) \). The duration of the disease has a negative regression coefficient \( (t \text{ value } < 0.05) \). In other words younger patients and patients with a shorter duration of disease have a better chance of gaining a marked improvement than older patients and patients who have had the disease longer. The orally administered form of NADH was associated with an overall improvement in the disability which was comparable to that of the parenterally applied form.

Drugs intended to increase wellness or quality of life ("lifestyle drugs") have gained popularity and/or importance over recent years. Biogenic substances such as nicotinamide adenine dinucleotide (NADH) are credited with increasing physical and intellectual performance without side-effects. NADH is an energy-delivering co-substrate in the respiratory chain. Clinical studies showed positive effects of parenterally administered NADH in Parkinson’s disease and major depression. NADH can be measured by its fluorescence. In this study a pulsed N2-laser combined with a fibre-optic probe and photomultipliers were used to induce and measure NADH fluorescence in the rat cortex. The aims of the study were to assess the suitability of the laser-induced spectroscopy for in vivo and on-line measurement of NADH in neuroscience and the assessment of the central availability of NADH after peripheral administration. NADH (50 mg/kg), but not the precursor nicotinamide, caused a significant rise of the NADH fluorescence intensity indicating an increase of the NADH concentration in the rat cortex. In conclusion, the results suggest that NADH given orally or intraperitoneally increases the amount of NADH in the brain. The results may thus help to explain the clinical effects reported.


A gradual increase in blood pressure (BP), often attaining hypertensive levels, is common during aging—"age-related hypertension." Therefore, means to prevent or ameliorate this elevated BP safely are important. Although oral B-nicotinamide adenine dinucleotide (NADH), a natural coenzyme, is used principally to treat various neurologic disorders, we wished to investigate whether this agent had the same potential to lower BP and benefit the cardiovascular system as does coenzyme Q10, a similar-type agent. As a first approximation, spontaneously hypertensive rats (SHR) were used to determine effects of oral NADH. In a blinded, placebo-controlled study, ten rats received placebo; and ten, NADH for ten weeks.

Systolic BP was measured by tail plethysmography. Blood was collected terminally, and chemistries were performed by routine methodologies. Thiobarbituric acid reactive species (TBARS) (an estimate of lipid peroxidation/free radical formation) was measured in renal and hepatic tissues. The following was noted: water and food intake were comparable, and the steady weight gain of young SHR were similar in the placebo and NADH groups. Although systolic BP did not differ between the two groups over the first month, it decreased and stayed markedly lower for the remainder of study in SHR receiving oral NADH. At the end of 60 days, SBP in NADH-treated SHR was 184 mm Hg +/- 2.8 (SEM) compared to 201 mm Hg +/- 2.1 (SEM) in control SHR (p < 0.001). No significant differences were seen in blood levels of glucose, insulin, triglyceride, and HDL levels but NADH intake lowered total cholesterol (p < 0.002) and LDL (p < 0.02). Renal TBARS were also significantly lower in SHR receiving NADH (P < 0.001). Accordingly, supplementation with the natural coenzyme NADH theoretically could prove to be useful in preventing age-related increases in BP and, thus, various cardiovascular maladies.
9. TECHNICAL DATA

9.1. DAILY DOSAGES/CHARACTERISTICS

9.1.1. PANMOL®-NADH_{MICRO}

RECOMMENDED DAILY DOSAGES:
50 mg PANMOL®-NADH_{MICRO} (corresponding to 5 mg NADH) to 200 mg PANMOL®-NADH_{MICRO}
(corresponding to 20 mg NADH)

PHYSICAL-CHEMICAL CHARACTERISTICS:
Appearance: dark green granulate
Odour: slightly oily
Taste: oily
Solubility: pH-dependent
pH: neutral
Origin: plant fats, bees wax, chlorophyll, NADH
Applicability: in hard gelatin capsules and soft gelatin capsules
9.2. SPECIFICATIONS

9.2.1. PANMOL®-NADH$_{\text{MICRO}}$

| Raw material: | PANMOL®-NADH$_{\text{MICRO}}$ | Supplier: vis vitalis gmbh |
| Product code: | 910510  (R008.095) | Version: 9 |
| Batch Number: | |

**Specification released of QA / Date:** 05.07.2012  
**Name:** DI Guggemos  
**Sign:**  
**Certificate of analysis released of QA / Date:**  
**Name:** Mag. Hlebaina  
**Sign:**

**Definition**

PANMOL®-NADH$_{\text{MICRO}}$ (Plant fats, bees wax, chlorophyll, NADH)

<table>
<thead>
<tr>
<th>Characters</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
<td>SOP-02-026</td>
<td>Granulate</td>
<td></td>
</tr>
<tr>
<td>Colour</td>
<td>SOP-02-026</td>
<td>Dark green</td>
<td></td>
</tr>
</tbody>
</table>

**Assay**

<table>
<thead>
<tr>
<th>stabilized NADH</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>puroNADH*</td>
<td></td>
<td>9.5 – 10.5 g/100g</td>
<td></td>
</tr>
</tbody>
</table>

**Tests**

<table>
<thead>
<tr>
<th>Arsenic</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAM07 **</td>
<td></td>
<td>max. 1 ppm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cadmium</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAM07 **</td>
<td></td>
<td>max. 1 ppm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mercury</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAM07 **</td>
<td></td>
<td>max. 0.1 ppm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lead</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAM07 **</td>
<td></td>
<td>max. 1 ppm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total plate count</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOP-02-032</td>
<td></td>
<td>max. 100.000 cfu/g</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Enterobacteriaceae</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOP-02-032</td>
<td></td>
<td>max. 200 cfu/g</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mould</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOP-02-032</td>
<td></td>
<td>max. 200 cfu/g</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Loss on drying</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOP-02-001</td>
<td></td>
<td>max. 2 %</td>
<td></td>
</tr>
</tbody>
</table>

* HPLC-Method, based on the publications of Dr. Antal Bognár and SLMB 03:2000.
** ICP-MS standard addition method for analysis of heavy metals, with certified CertiPUR® Standards.

**Release data and Storage**

<table>
<thead>
<tr>
<th>Retest schedule</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>FB (1996)</td>
<td></td>
<td>2.5 years</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Containers</th>
<th>Reference</th>
<th>Requirements</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>FB (1996)</td>
<td></td>
<td>Tightly closed, cool, dark and dry</td>
<td></td>
</tr>
</tbody>
</table>

**Characteristic properties**

<table>
<thead>
<tr>
<th>Nutritional values (per 100g):</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat</td>
<td>~ 63.0 %</td>
</tr>
<tr>
<td>Energy</td>
<td>~ 2338kJ / 568 kcal</td>
</tr>
</tbody>
</table>

**Excipients:**

| Chlorophyll | 9.5 – 10.5 g/100g | calculated |
| Bees Wax | 15.0 – 17.0 g/100g | calculated |
| Vegetable fat | 62.0 – 64.0 g/100g | calculated |
9.3. FLOW CHARTS

9.3.1. PANMOL®-NADH micro

Flow Chart of PANMOL®-NADH micro

Vegetable fat (solid) △
Hot melt
Chlorophyllin
Suspension
NADH
Bees Wax
Sprinkling (Micro-encapsulation)
PANMOL®-NADH micro
packaging under nitrogen atmosphere
9.4. DATA OF FOOD IDENTIFICATION CODE

9.4.1. PANMOL®-NADH\textsubscript{MICRO}

TEXT OF INGREDIENTS:

PANMOL®-NADH (Plant fats, Bees Wax, Chlorophyll, NADH)

TEXT OF THE VALUE INDICATION INGREDIENTS PER 50 mg/100 mg/200 mg:

<table>
<thead>
<tr>
<th>Value requirements factors</th>
<th>per 50 mg</th>
<th>per 100 mg</th>
<th>per 200 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>NADH</td>
<td>5 mg</td>
<td>10 mg</td>
<td>20 mg</td>
</tr>
</tbody>
</table>

TEXT OF NUTRITIONAL VALUE:

<table>
<thead>
<tr>
<th></th>
<th>per 100 g</th>
<th>per 50 mg</th>
<th>per 100 mg</th>
<th>per 200 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caloric value</td>
<td>568 kcal</td>
<td>0.284 kcal</td>
<td>0.568 kcal</td>
<td>1.136 kcal</td>
</tr>
<tr>
<td></td>
<td>2338 kJ</td>
<td>1.169 kJ</td>
<td>2.338 kJ</td>
<td>4.676 kJ</td>
</tr>
<tr>
<td>Protein</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fats</td>
<td>63 g</td>
<td>31.5 mg</td>
<td>63 mg</td>
<td>126 mg</td>
</tr>
<tr>
<td>Bread units</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The values of the content description are based on an average analysis.

TEXT RECOMMENDATION FOR PACKAGES AND PACKAGE INSERTS:

...contains/contain patented PANMOL®-NADH. NADH is the biologically active form of niacin (vitamin B3) and has the function of a biological spark plug in the cellular energy metabolism. In a patented process, the highly reactive and oxygen-sensitive power vitamin NADH has been stabilised and made available without chemical additives.
FREE SALES CERTIFICATE Nr. 110160-2

In my profession as a certified Expert to the Court, Governmental Certified Food Chemist according to § 73 LMSVG (General Food Law) and Expert for food and cosmetics, officially appointed and sworn by the Austrian Federal Ministry of Health and Woman as well as a Full Professor of Food Chemistry and Head of the Institute of Food Chemistry and –technology, Graz University of Technology I do certify:

According to the data given to me in 2011/01/12 by vis vitalis gmbh, the product

- Aktives Vitamin B3 mit Pflanzenfett, Bienenwachs und Chlorophyll
  (active vitamin B3 with plant fats, bees wax and chlorophyll)
  described in appendix – page 1

manufactured and distributed by vis vitalis gmbh to

- all countries of the European Union

is duly safe and do not contain any prohibited substances, do not contain any controlled substances which are required to be registered, do not contain any dangerous substances which are not safe for use.

The products do not contain materials of bovine origin and so cannot represent any risk of BSE.

These products contain suitable substances not exceeding the specified amount. The products are also permitted to be exported to other countries outside the EU.

O.Univ.Prof. Dr. Werner Pfannhauser
Certified Expert to the Court, Governmental Certified Food Chemist according to § 73 LMSVG and Expert for food and cosmetics, Full Professor of Food Chemistry and –technology

Aktives Vitamin B3 mit Pflanzenfett, Bienenwachs und Chlorophyll
(active vitamin B3 with plant fats, bees wax and chlorophyll)

The active vitamin B3 with plant fats, bees wax and chlorophyll, accordant to EP process patent Nr. 1562813 ("ZUSAMMENSETZUNG UMFASSEND NADH/NADPH" / "NADH/NADPH-CONTAINING COMPOUND"), is marketable according to effective European food law (VO (EG) 178/2002) as a food or a food ingredient.

The declaration of this active vitamin B3 with plant fats, bees wax and chlorophyll in the list of ingredients of food, food supplements or dietary products as:

- "NADH, Pflanzenfett, Bienenwachs und Chlorophyll" (NADH, plant fats, bees wax and chlorophyll) and/or
- „Aktives Vitamin B3 mit Pflanzenfett, Bienenwachs und Chlorophyll" (active vitamin B3 with plant fats, bees wax and chlorophyll)

is legally allowed and corresponds to effective European food labelling directives (VO 2000/13/EG).
9.6. CERTIFICATE - FEDERAL MINISTRY OF HEALTH

9.6.1. PANMOL®-NADH<sub>MICRO</sub>

CERTIFICATE

The Federal Ministry of Health (Division II/B: Consumer Health) confirms that Univ.Prof. Dr. Werner Pfannhauser is – based on the Food Safety- and Consumer Protection Law (BGBl. I Nr. 13/2006 idgF) – competent and authorized to perform investigations and expertises.

Based on the expertise of Univ.Prof. Dr. W. Pfannhauser from January 20th, 2011, Nr. 110160-2, the product active vitamin B3 with plant fats, bees wax and chlorophyll manufactured and distributed by vis vitalis gmbh to

- all countries of the European Union

is duly safe and to not contain any prohibited substances, do not contain any controlled substances which are required to be registered, do not contain any dangerous substances which are not safe for use.

The product do not contain materials of bovine origin and so cannot represent any risk of BSE.

The product contain suitable substances not exceeding the specified amount.

The product is also permitted to be exported to other countries outside the EU.

The active vitamin B3 with plant fats, bees wax and chlorophyll, accordant to EP process patent Nr. 1562613 (NADH/NADPH-containing compound), is marketable according to effective European food law (VO (EG) 178/2002) as a food or a food ingredient.
The declaration of this active vitamin B3 with plant fats, bees wax and chlorophyll in the list of ingredients of food, food supplements or dietary products as:

- NADH, plant fats, bees wax and chlorophyll and/or
- active vitamin B3 with plant fats, bees wax and chlorophyll

is legally allowed and corresponds to effective European food labelling directives (VO 2000/13/EG).

For the Minister:

Dr. Karl Pisek

Die vorstehende umschriebene Unterschrift von Herrn Dr. K. Pisek
wird hiermit beigefügt.
Wien, am 3. FEB. 2011

Erwin Schuller
10. LOGISTIC DATA

10.1. PACKAGE PANMOL\textsuperscript{®}-NADH\textsubscript{MICRO}

- outside carton
- inside with aluminium bags
- packed under nitrogen atmosphere
- filled loosely
- 1 kg weight per unit

CUSTOMS NUMBER:
2106 9092
**General distribution Austria:**
vis vitalis gmbh  
Moosham 29  
5585 Unternberg  
AUSTRIA  
Phone: +43 (0) 6476 8052-00  
Fax: +43 (0) 6476 8052-22  
office@panmol.com  
www.panmol.com

**General distribution Europe:**  
K.-W. Pfannenschmidt GmbH  
Habichthorst 34-36  
22459 Hamburg  
GERMANY  
Phone: +49 (0) 40555-8660  
Fax: +49 (0) 40555-3898  
info@pfannenschmidt.de  
www.pfannenschmidt.de

**General distribution USA:**  
Stauber Performance Ingredients Inc.  
4120 N. Palm Street  
Fullerton, CA 92835-1026  
USA  
Phone: 714-441-3900 / 888-441-4233  
customerservice@stauberusa.com  
www.stauberusa.com