Optimizing cognitive function

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Greetings from the Bay area/San Francisco

- Birdman at US Open
San Rafael

- Is the 2nd most northerly mission on the El Camino Real
- to Northern most mission from Sonoma, California to Guatemala

-in honor of Arcangel Raphael
San Rafael

Arcangel Raphael is the arcangel that supervises healers and healing for all Earth’s population. Raphael coaches and motivates healers whispering instructions into the ears of doctors, surgeons, psychologists, nurses and other caregivers including scientists. His intervention helps scientists in making breakthroughs in medical cures. When asked for healing assistance, Raphael surrounds and nurtures people with the emerald green light of his halo. Green is the color of healing; clairvoyant people may see emerald green sparkles when Raphael is around.

“It is God Who heals… in Judaism, Christianity, Catholicism and Islam…”

Reverend Daniel Kudra
What do these people have in common?
Optimizing cognitive function

For the Brain:
Feed
Protect
Stimulate

For Children And Adults
Optimizing cognitive function

1. Optimal
   A. 3D models of brain - in childhood and adulthood do not exist!
   B. General concepts of brain development and aging
      i. baseline, normal neuroimaging over the lifespan
         (prenatal, natal, toddler, adult, elderly)
      ii. Prevent decline -- Normal aging of brain
         Decline due presumably to genetics, loss of telomerase, hormonal

2. Feed the brain (chemical)
   A. Diet
   B. Nutrients
   C. Medications i.e. theoretically, telomerase
   D. Hormones (primary, secondary and trophic effects)

3. Mechanical
   A. Exercise
   B. Osteopathy
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4. Energetic (affecting brain coherence, electricity, & magnetism)
   A. acupuncture
   B. aromatherapy
   C. crystals and coherence
   D. electricity
   E. flower essences
   F. fusion energy - scalar
   G. grounding (earthing)
   H. hands on healing (Therapeutic Touch)
   I. homeopathy (vibrational)
   J. Intuitive Medical consulting
   K. light therapy (i.e. LED and infrared light emitting diodes)
   L. magnetism (i.e. TMS)
   O. music therapy
   P. quantum healing from a distance
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5. Brain stimulation programs
   A. Neurofeedback
      sound entrainment for postTBI (visual entrainment may be too strong)
      a. LENS (Low Energy Neurofeedback Stimulation)
   B. Children: Brain Spark
   C. Adults: Brain Fitness program
   D. TMS to lessen depression
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6. Psychologic therapies/ self-concept/ internal work
   A. self-esteem
   B. work: meaningful, moderate amount of good stress, maximize employee control
   C. resolving depression
   D. for anxiety, mood lability, rage attacks- organic disease evaluation
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7. Mental/ Mind Therapies
   A. Cognitive strategies
      i. children who had multiple strategies, both involving verbal and imagery were better able to solve problems (Charness and Schaie, 2003)
      ii. Knowing how one’s brain works accelerates cognitive abilities (math research)
      iii. Use it or lose it (Diamond et al, 1966)
      iv. Dedifferentiation of learning systems in older adults
   B. Lucid dreaming (Waggoner, 2009)
   C. Meditation
      i. gratitude (other sources of human strength: hope, humility, love, spirituality, wisdom)
   D. Visual imagery (Gawain, 2008)
   E. Do what makes you happy (no evidence for this)
   F. Become a Renaissance man/woman- use more of brain when Participating in different activities
   G. Balance of activities (meditation daily, exercise 2x/day, meaningful work, social, relationship, nature, pets, friends and family, community, ongoing spiritual practice, not overschedule)
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8. Social
   a. Benefits of marriage to the individual
   b. Benefits of community involvement
   c. Benefits of holding precious all living beings
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9. Protect
   A. Remove toxins and toxicants
   B. Remove EMF stress
   C. Remove emotional stressors/emotional abuse
   D. Remove negative psychological factors
      i. low self esteem
      ii. lack of hope
      iii. lack of motivation
   E. Remove preventable mechanical trauma
      i. physical abuse, violence involving head trauma
      ii. Seat belt
      iii. Sports: Stop concussions (football, soccer (headers), boxing)
      iv. use helmets (bicycling, sports, atonic epilepsy)
      v. no helmet use in plagiocephaly
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9. E. Preventable mechanical trauma
   vi. Violence prevention
      A. remove guns
      B. create world of focusing on people’s needs, instead of fear based
      C. Stop violence in the media (movies, TV), which is modeled by a subset of the population with poor social skills
      D. Create and maintain active peace
         i. United Nations
         ii. World Child Peace Council
1. A. Optimal Cognitive Function
Child brain and neurodevelopment

<table>
<thead>
<tr>
<th>Fetus</th>
<th>18 days</th>
<th>neurulation (plate, groove to tube). Genome controlled neuroblast differentiation, migration, and neuronal multiplication (250,000 neurons/min (Cowan, 1979)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>56 days</td>
<td>cerebral cortex differentiation</td>
</tr>
<tr>
<td></td>
<td>5 months</td>
<td>migration complete, primary cerebral fissures, 50-100 billion cerebral cortex cells</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>secondary cerebral fissures and start of myelination</td>
</tr>
<tr>
<td></td>
<td>6-9 months</td>
<td>myelination, growth brain, synaptogenesis, pruning, cortical thinning</td>
</tr>
<tr>
<td>Birth</td>
<td>375-400 gm brain, start of myelination visual system &amp; cerebrum (post front and pariet lobes)</td>
<td></td>
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<tr>
<td></td>
<td>2 months</td>
<td>myelination visual system complete</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>glial cells divide</td>
</tr>
<tr>
<td></td>
<td>1yr</td>
<td>1 kg, myelination frontal and temporal lobes</td>
</tr>
<tr>
<td></td>
<td>18 months</td>
<td>cortical spinal and other principal tracts myelinated</td>
</tr>
<tr>
<td></td>
<td>2 yr</td>
<td>cerebrum myelinated</td>
</tr>
<tr>
<td></td>
<td>4-20 yrs</td>
<td>steady increase cortical white matter (WM) 12%</td>
</tr>
<tr>
<td></td>
<td>6 yrs</td>
<td>decreases cort GM (preadolescent increase, then post-adolescent decrease front GM peak age 12; temp GM peak age 16, occip lobe GM peak age 20 (Giedd et al., 1999)</td>
</tr>
<tr>
<td></td>
<td>puberty</td>
<td>2000 words, spatial and temporal relationships, causality, read, write, and later calculate</td>
</tr>
<tr>
<td></td>
<td>puberty</td>
<td>1250 gm girls, 1375 gm boys, slower brain growth for both</td>
</tr>
<tr>
<td></td>
<td>18-21 yrs</td>
<td>motor skills maximal precision athletes, artists, and musicians</td>
</tr>
<tr>
<td></td>
<td>late childhood-adulthood</td>
<td>increased complexity fiber systems (dendritic arborizations, cortical interneuronal connections, complexity reduced number of neurons per volume of tissue (Conel, 1939-1967)</td>
</tr>
<tr>
<td></td>
<td>35 yrs</td>
<td>myelin frontal lobes complete, judgment, inhibition of impulses (Adams and Victor, 1993)</td>
</tr>
</tbody>
</table>
1.A. Brain development, Child

31 wk (7 ¾ mo), 40 wk, 15 month, (Huppi et al, 1998)

Shatz (UCBerkeley) in 1997 discussed the phases of brain development.
First phase - before birth, when genetics directs the “hardwiring”.
Second phase, before birth-- spontaneous brain activity.
After birth, third phase sensory experience completes the wiring process.
Third phase relies on relevant environmental stimuli to appropriately, hard wire the brain with fine tuning.
1.A. Child Brain development

Fagiolini et al in 2009 state that brain development is more complicated than Shatz had proposed in 1997, with the concept of epigenetics- many environmental factors influence the expression of the genome pre and postnatally, including

a. Brain Stimulation (i.e. playing music after 8\textsuperscript{th} month)

b. Emotions (Church, 2012)

c. Hormones (Hertoghe, 2006)

d. Nutrition

d. Toxins

Therefore, theoretically one might optimize/enhance brain development, lessen the decline in brain functioning, and lessen the brain atrophy of aging, if one modifies the epigenetic or environmental factors.
A fine interplay exists between sensory experience and innate genetic programs leading to the sculpting of neuronal circuits during early brain development. Recent evidence suggests that the dynamic regulation of gene expression through epigenetic mechanisms is at the interface between environmental stimuli and long lasting molecular, cellular and complex behavioral phenotypes acquired during periods of developmental plasticity. Understanding these mechanisms may give insight into the formation of critical periods and provide new strategies for increasing plasticity and adaptive change in adulthood. (Fagiolini et al, 2009)
1.A. Brain development

“In the cerebral cortex, functional domains such as visual processing, attention, memory, and [cognition] rely on the development of distinct yet interconnected sets of anatomically distributed cortical and subcortical regions. The developmental organization of these circuits is a remarkably complex process that is influenced by genetic predispositions, environmental events, and neuroplastic responses to experiential demand that modulates connectivity and communication among neurons, within individual brain regions and circuits, and across neural pathways” (Tau and Peterson, 2009)
FIG. 2.16. Normal corpus callosum development. A: Normal 1-month-old. On this SE 500/20 image, the corpus callosum is isointense with the rest of the brain. The corpus callosum is uniformly thin at this age without the normal bulbar enlargement of the genu and splenium: the genu, body, and splenium are all of the same thickness. B: Normal 4-month-old. By 3-4 months of age, the splenium of the corpus callosum increases in size and begins to show an increased signal intensity as compared to the rest of the brain on SE 600/20 images. These changes probably result from the process of myelination in the visual association fibers. C: Normal 7-month-old. By 6-7 months of age, the corpus callosum is of a uniformly high signal intensity as compared with surrounding brain. The genu and splenium of the corpus are now large, compared to the body. The corpus is still relatively thin. D: Normal 10-month-old. By 8-9 months, the corpus callosum begins to thicken in the genu and splenium, taking on more of an adult appearance. The thinning of the corpus at the junction of the posterior body and splenium (arrow) is a normal variant and does not denote pathology. E: Normal mature corpus callosum in a 15-year-old.
1.A. Child brain development

Myelination

myelin content

- 40% water; dry weight: 70-85% lipids, 15-30% protein
- Cholesterol
- phospholipids
  - phosphatidylcholine
  - phosphatidylethanolamine
  - phosphatidylserine

myelination period (6 mos gestation to 35 yrs)

myelination as vulnerable period in brain development

maintenance of myelin/turnover/oxidative stress
1. Child optimal brain functioning

- Nutrition (aa, vitamins, minerals, iron, iodine, antioxidants, probiotics, inositol, choline, EFA, DHA)
The combined visual resolution acuity difference measured with behaviorally based methods between human milk fed groups and DHA-free formula fed groups is 0.49±0.09 octaves (P≤0.000001) at 2 months of age and 0.18±0.08 octaves (P=0.04) at 4 months of age. Acuity differences for electrophysiologic-based measures are also greater than zero at 4 months (0.37±0.16 octaves, P=0.02). Conclusion: Some aspect of dietary n-3 intake is associated with performance on visual resolution acuity tasks at 2, and possibly, 4 months of age in healthy full term infants. Whether n-3 intake confers lasting advantage in the development of visually based processes is still in question.

- Exercise
  - Sign language in infancy (lessens crying, increases ability of child and parent to communicate, greater percentage needs met and met more rapidly, infant self-directive)

- Exposure in first 5 years to
  - Language
  - Sports such as golfing
  - Music

- Computer programs-Brain Spark, Fast Forward, math.com

- Prevention –
  - Heavy metal poisoning (distilled or reverse osmosis water filtration)
  - Diabetes mellitus
  - Tick born disease (condoms, screen in blood supply Babesia), outdoor exposure and from pets

- Not overschedule
- Stress management
- Meditation
1. Child optimal cognitive functioning

- Hair testing (lithium deficiency and “cheap man’s screen” for breadth of possible heavy metals)
- Urine fractionated porphyrins (heavy metal poisoning), screen for first and check nutrients needed for detoxification (concern of chelating agent challenge without repletion of nutrients of detox)
- Daily am/noon mvi/mmi with antioxidants, inositol, choline/methylB12 and methylated folate
- Ensure excellent thyroid function
1. Child optimal brain health

- Autism(s), global epidemic, 1 in 88 children in the US (1 million children) (Bacon et al., 2012)
- Complex genetic predisposition of weaknesses in detoxification and/or the immune system with multiple environmental factors
- 2 dominant environmental factors from my clinical experience are Toxins/Toxicants (heavy metal poisoning being most common) and Tick born disease

Heavy metal poisoning - if prevent heavy metal exposure, could positively affect/prevent disease in up to 80% of children with autism (Rimland survey of 24,578 parents of autistic children)

- Tick born disease contributes to 22-30% of Autism
- Tick born disease, global epidemic, (CDC states only 10% diagnosed). Cases more than doubled 1992-2006 with a total of 248,074. Lyme disease is highest in children 5-14 yrs. Lyme d is grossly underestimated given arcane criteria for diagnosis with lab testing and in arduous reporting form to be completed by MD to Public Health Depts.
- Thus, if we prevent Tick born disease, we may be able to prevent Lyme-induced autism (in 22-30% of cases).
1. Child optimal brain health

- Computer exposure early
- Limit TV time (Rod Ingram), MD resource
- Howard- Multiple Intelligences
- Teach intuition to 4-18 yr olds, psychic abilities and meditation (Academy of Intuitive Medicine, since 1998, McCartney)
1.A. Optimal Cognitive Function Adult

Healthy Adult

81 y/o healthy woman (R)
(Whole Brain Atlas, Johnson and Becker)
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Adult brain, “Normal” aging

<table>
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<tr>
<th>Percent decrease</th>
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<tbody>
<tr>
<td>Brain weight</td>
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<tr>
<td>Blood flow to brain</td>
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- Steady decline in cognitive function starting at 30 yrs of age, and progressing into senium (WAIS, 1955)
- Performance functions greater decline (block design, reversal of digits, picture arrangement, object assembly, and digit symbol task) than verbal functions.
- Performance on tests of abstraction (digit symbol substitution) and tests measuring reaction time or speed in processing information, declines slowly throughout life. (Adams and Victor, 1993)
- Aging is associated with structural and functional decline in frontal lobes, striatum and medial temporal lobes (Dennis and Cabeza, 2008; Raz, 2005)

- But hormonal levels also decline with age in all but 10% of the population (Hertoghe, 2012). Therefore, maintenance/loss of decline in cognitive function can be mitigated, at least in part, by bioidentical hormone replacement therapy to the level of healthy 21-30 yr olds.
1.A. Adult “healthy” brain aging

- Lifespan average 85 yrs
- Maximal lifespan, 120 years
- Possible limiting factor: length of telomeres, since piece of telomere is lost with each chromosome replication. When neurons can no longer replicate, neurogenesis cannot occur, and there is no replacement of dying neurons. Thus, with age, there is loss of neuronal number, and ultimately loss of brain function. Treatment theoretically, would be to administer telomerase.
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Optimal Adult

- Insight into critical periods, the mechanisms by which they influence circuit development, and the forces that cause them to end may be fruitful for maximizing neuroplasticity-based treatments in adults.

(Cramer et al, 2011)
Optimizing Cognitive Function

- Cognitive processes depend on brain anatomy and physiology.
- (Dennis and Cabeza, 2008)
1.A. Association Between Decline in Brain Dopamine Activity With Age and Cognitive and Motor Impairment in Healthy Individuals

- American Journal of Psychiatry, VOL. 155, No. 3

OBJECTIVE:
Although it is documented that brain dopamine activity declines with age, the functional significance of this is not known. This study assessed the relation between measures of brain dopamine activity and indexes of motor and cognitive function in healthy individuals.

METHOD: Thirty healthy volunteers aged 24–86 years were studied with positron emission tomography and [11C]raclopride to assess dopamine D2 receptors. All subjects underwent a neuropsychological test battery that included tasks found to be sensitive to dopamine alterations in patients with neurodegenerative disease and control tasks.

RESULTS: Transfer of [11C]~raclopride from plasma to brain in the striatum and cerebellum was not affected by age. In contrast, D2 receptor availability in the caudate and putamen declined with age. Correlations between D2 receptors and neuropsychological test performance were strongest for the motor task (Finger Tapping Test) and were also significant for most tasks involving frontal brain regions, including measures of abstraction and mental flexibility (Wisconsin Card Sorting Test) and attention and response inhibition (Stroop Color-Word Test, interference score). These relationships remained significant after control for age effects. CONCLUSIONS: Age-related decreases in brain dopamine activity are associated with a decline in motor function and may also contribute to impaired performance on tasks that involve frontal brain regions. Interventions that enhance dopamine activity may improve performance and quality of life for the elderly. The fact that correlations remained significant after age effects were partialed out suggests that dopamine activity may influence motor and cognitive performance irrespective of age.
2.A. Diet

- Paleolithic (high protein, 5 different colored vegetables/day, handful of nuts, 1-2 high antioxidant fruits, oils (olive))
- Organic
- Free range, grass fed animals
- Wild fish, minimize mercury exposure
- Antifungal
- Antiinflammatory (production PGE3 which inh TNFalpha and IL1beta, inh COX1 and COX2 which reduces inflammatory prostaglandins. (Lorenz, 2006)
- Antioxidants
2.B. Brain nutrients- Amino Acids

- Most neurotransmitters are made from amino acids
- Need all 20 (like slats of a barrel, to hold water/to make proteins)
- **Glycine** - inhibits in the brainstem
- **Tryptophan** - promotes contentment and sleep
- **Phenylalanine, Tyrosine** - for alertness and thyroid hormone synthesis

(Bourre, 2006)
2.B. Brain nutrients

Cholesterol

Brain content and neurodevelopmental windows

- **Abstract**
  - One hundred and thirty-nine complete human brains ranging in age from 10 weeks' gestation to 7 postnatal years, together with 9 adult brains, have been analysed in order to describe the human brain growth spurt quantitatively. The three major regions were examined for weight, DNA, cholesterol, and water content. The growth spurt period is much more postnatal than has formerly been supposed. The cerebellum has special growth characteristics; and there is a separate period from 10 to 18 weeks' gestation when adult neuronal cell number may largely be achieved. The implications of these findings for the vulnerability of developing brain are discussed.

- Original article
- Quantitative growth and development of human brain
2.B. Essential Fatty-Acids

- Are part of all tissues and crucial for cell membrane synthesis. The brain is full with long-chain polyunsaturated fatty acids.

- **Docosahexaenoic acid (DHA):** essential nutrient for neurodevelopment and cognitive functions. (SanGiovanni et al, 2000).
  - neurotransmitters involved in the signal transduction process,
  - dendrite connectivity
  - functional maturation of the central nervous system. (Bourre, 2006)
Prenatal ingestion of DHA and omega 9

- Abstract
  - Fatty acid components of infant brain were determined to assess fatty acid requirements for synthesis of structural lipids in brain tissue during the last trimester of development in the fetus. Quantitative fatty acid analysis of cerebellum, frontal and occipital brain lobes indicated rapid accretion of chain elongation and desaturation products during the last trimester of brain growth. Frontal and occipital brain lobes were similar in fatty acid content. Fatty acid accretion rates were determined by regression analysis of tissue fat components at varying gestational ages. Tissue accretion of saturated and ω-9 fatty acids, as well as total fatty acid content, paralleled increases in whole brain weight. Levels of linoleic (C\textsubscript{18:2} ω-6) and linolenic (C\textsubscript{18:2} ω-3) acids were consistently low in brain during the last trimester of development, while marked substantial accretion of long chain desaturation products, arachidonic (C\textsubscript{20:4} ω-6) and docosahexaenoic (C\textsubscript{22:6} ω-3) acids occurred. Accretion of individual fatty acids of cerebellum also reflected changes in tissue total fatty acid content, with exception of the levels of C\textsubscript{18:3} ω-3 and its chain elongation products present in cerebellum during the last trimester. These developmental changes and estimates of fatty acid incorporation into whole brain and cerebellum are quantitatively relevant to estimation of fatty acid requirements of the low birth weight neonate.

- Intrauterine fatty acid accretion rates in human brain: implications for fatty acid requirements
  - Department of Nutrition and Food Science and Department of Pediatrics, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada
2.B. Essential fatty acids and cholesterol

- Prenatal-DHA and omega 9
- Myelin deposition

Rat brain 70% of brain cholesterol is in myelin (Davison and Dobbing, 1966)
(keep total cholesterol 160-180 (Dr. Walsh, Great Plains labs)
If cholesterol <160, need to increase cholesterol in either in diet or administer supplement.
2. B. Essential fatty acids

- John Paul SanGiovanni Catherine S Berkey Johanna T Dwyer Graham A Colditz

Background: Biologically active neural tissue is rich in docosahexaenoic acid (DHA), an omega-3 long-chain polyunsaturated fatty acid (LCPUFA). We conducted a systematic review to examine the nature of discordant results from studies designed to test the hypothesis that dietary DHA leads to better performance on visually-based tasks in healthy, fullterm infants. We also conducted a meta-analysis to derive combined estimates of behavioral- and electrophysiologic-based visual resolution acuity differences and sample sizes that would be useful in planning future research. Study design and methods: Twelve empirical studies on LCPUFA intake during infancy and visual resolution acuity were identified through bibliographic searches, examination of monograph and review article reference lists, and written requests to researchers in the field. Works were reviewed for quality and completeness of information. Study design and conduct information was extracted with a standardized protocol. Acuity differences between groups consuming a source of DHA and groups consuming DHA-free diets were calculated as a common outcome from individual studies; this difference score was evaluated against a null value of zero and then used, with the method of DerSimonian and Laird (Meta-analysis in clinical trials. Control Clin Trials 1986;7:177–188), to derive combined estimates of visual resolution acuity differences within seven age categories. Results of randomized comparisons: The combined visual resolution acuity difference measured with behaviorally based methods between DHA-supplemented formula fed groups and DHA-free formula fed groups is 0.32±0.09 octaves (combined difference±S.E.M., P=0.0003) at 2 months of age. The direction of this value indicates higher acuity in DHA-fed groups. Results of non-randomized study designs: The combined visual resolution acuity difference measured with behaviorally based methods between human milk fed groups and DHA-free formula fed groups is 0.49±0.09 octaves (P≤0.000001) at 2 months of age and 0.18±0.08 octaves (P=0.04) at 4 months of age. Acuity differences for electrophysiologic-based measures are also greater than zero at 4 months (0.37±0.16 octaves, P=0.02). Conclusion: Some aspect of dietary n-3 intake is associated with performance on visual resolution acuity tasks at 2, and possibly, 4 months of age in healthy fullterm infants. Whether n-3 intake confers lasting advantage in the development of visually based processes is still in question.
2.B. Antioxidants

- Alpha lipoic acid
- Glutathione-tripeptide thiol
  - Different types of brain cells prefer different glutathione precursors (Dringen, 1999)
- Vitamin C
- Vitamin E (alpha, beta and gamma tocopherol)
- Melatonin
Abstract
Cerebral metabolite concentrations and water content were measured by means of localized proton magnetic resonance spectroscopy in 50 children, while metabolite peak ratios in short echo time spectra were evaluated in 173 examinations. Normative curves for normal development were established for two cerebral locations. The current report presents the first study of absolute metabolite concentrations and $T_1$- and $T_2$-relaxation as a function of age. Myo-inositol was found dominating the spectra at birth (12 mmoles/kg), while choline is responsible for the strongest peak in older infants (2.5 mmoles/kg). Creatine and N-acetyl groups are at significantly lower concentrations in the neonate than in the adult (Cr: 6, NA: 5 mmoles/kg). NA and Cr are determined by gestational age, whereas the concentration of ml correlates best with postnatal age. Quantitative $^1$H MRS is expected to be of particular value in diagnosis and monitoring of pathology in infants, since metabolite ratios are often misleading.


2.B Choline as brain nutrient.
2.B. Brain nutrients

Vitamins

**Vitamin A**
- Improves visuospatial performance (along with B12, B6, and E)
- Control of differentiation and proliferation of cells
- Antiviral (herpes has affinity for nervous system, temporal lobes)
- Assists liver, indirectly protecting the brain

**Vitamin B1**
- Facilitates use of glucose (as do all B vitamins)
- Modulates cogn perform (especially in elderly)
- Lowers/normal homocysteine level, preventing CVA
- Improves levels of abstract thought (with B2 and B3) (Bourre, 2006)
2.B. Brain nutrients

Vitamin B2
- Improves levels of abstract thought (B1, B3)
- After brain injury, improves outcome and reduces edema formation and glial fibrillary acid protein expression.
- Enhances utilization of B1 and B3

Vitamin B3
- Improves levels of abstract thought

Vitamin B4
- Adenine- chemical component of DNA

Vitamin B5
- (found in blood plasma) Essential to maintain the body's balance of sodium and potassium

Vitamin B7
- Coenzyme in metabolism of fatty acids (Bourre, 2006)
2.B. Brain nutrients

- **Vitamin B6**
  - Helps in the production of neurotransmitters
  - Important for immune system function (especially in elderly)
  - Lowers irritability and depression
  - High levels associated with high performance in memorization tests
  - Improves visuospatial performance (A, B12, and E)
  - Lowers levels of homocysteine, lowering risk of hyperhomocystenemia and stroke (B9,12) (Bourre, 2006)
2.B. Brain nutrients

- **Vitamin B9**
  - Preserves brain during development and memory during aging
  - Mental and emotional health
  - B9 and B12 are utilized to synthesize red blood cells and help iron work properly in the body

- **Vitamin B12**
  - Improves functions in frontal lobe and language
  - If deficient, memory loss, dementia, and pain can occur
  - Involved in synthesis of some neurotransmitters (with B6)
  - Improves visuospatial performance (with A, B6, and E) (Bourre, 2006)
2.B. Brain nutrients

- Vitamin C
- Nerve endings contain the highest concentrations
- Transformation of dopamine into noradrenaline
- Anti-stress effects (in rats)
- Lessens oxidative stress to the brain and nervous system
- Vitamin C with beta-carotene helps long term memory
- Improves visuospatial performance (along with A, B6, 12, and E)

(Bourre, 2006)
2.B. Brain nutrients

- **Vitamin D is a hormone**
- Involved in normal structural brain development
- Protects neurons in hippocampus
- Modulates transport of glucose to the brain
- Helps prevent neurodegenerative/ neuroimmune diseases
- Low levels appear to raise risk for fatal stroke, dementia and multiple sclerosis

(Bourre, 2006)
2.B. Brain nutrients

- **Vitamin E** (alpha, beta, and gamma tocopherols)
  - Helps cognitive performance (along with C)
  - Helps hippocampal memory cells resist oxidative stress

- **Alpha tocopherol**: actively taken up by the brain.
  - Directly involved in membrane protection
    - plays a role in cognition

- **Tocopherol**: protects against aging

- **Gamma**: still being investigated

- Only Alpha-tocopherol, not gamma tocopherol are integrated into biological membranes
  (Bourre, 2006)
2.B. Brain nutrients

- **Minerals**
  - **Calcium**
    - Vital for communication between neurons (neurotransmitter release)
    - Helps lower blood pressure
      - (benefits found in dairy products, not non-dairy sources of calcium)
  - **Lithium**
    - Mood stabilizing (lithium orotate)
    - Low but long exposure can enhance neurotropic and neuroprotective factors, and neurogenesis (Bourre, 2006)
    - If deficient, causes global neurodevelopmental delay (Adams et al, 2011)
2.B. Brain nutrients

- **Sodium**
  - Maintains blood pressure
  - Nerve cells sensitive to amount of sodium in blood
    - Dangerous to have too much or too little
  - Used in signal transduction (action potential)

- **Potassium**
  - Builds electrical potential in neurons
  - Sodium and potassium work together to regulate water balance in cells
(Bourre, 2006)
2.C. Adult Optimizing Brain function Medication

- To lengthen telomeres, possibly extending the period of neurogenesis via Telomerase (FDA approved Feb. 2012 in US, product is TA65.) Can measure telomere length for range of lengths of telomeres to determine chromosomal age of person, compared to their chronological age.

Can Telomerase really extend the period of neurogenesis? Neurogenesis occurs into our 80’s at baseline.

Concern: Telomerase may be tumorigenic (Magalhães and Toussaint, 2004)
2.D. Hormone management for optimal cognitive functioning

18 hormones—perhaps in order of importance for brain function
17/18 hormones affect brain function
thyroid
Melatonin (importance of sleep, antioxidant)
Cortisol
Pregnenolone in position of steroid hormonal pathway
Estradiol
Testosterone
DHEA
androstenedione
Oxytocin
Growth hormone
Progesterone
ADH
Aldosterone
Parathyroid
Insulin
Glucagon
Calcitonin
Prolactin
and Gut hormones, vasoactive intestinal peptide, motilin, gastrin...
2.D. Hormone management

Methodology Dr. Thierry Hertoghe developed through review of the literature and extensive clinical experience.

Goal: hormone levels replace with push to midway or 2/3 up the normal range of a 21-30 y/o, replacing with bioidentical hormones and maintaining at physiological levels, by blood, saliva and urine testing.
2.D. Hormone management

Hormones are trophic to organs and restore health.
Hormones prevent cancer and disease. As hormone levels decline with aging at menopause, this is coincident with the rise of incidence of cancer.

(Hertoghe, 2012)
2.D. Thyroid Management

Lab blood testing
- 3rd Generation TSH
- Free T3
- Free T4
- Total T3
- Reverse T3
- Fasting amino acids, iodine
- Thyroid antibodies

Treatment
- TSH 0.5-1.1
- Free T3 2/3 up normal range
- Total T3/ Reverse T3 ratio greater than 10 (McDaniel, 2011)
2.D. Hormone Management

Melatonin
- Salivary testing at bedtime (goal 20 pg/mL)
- 1-20mg ER at bedtime (or booster regular if middle of night)
- Grade A recommendation of supplement for ASD (Rossignol, 2009)

Growth Hormone Testing
- IGF 1
- IGF-BP 3
- Fasting amino acids

(Hertoghe, 2006)
## Growth Hormone Treatment

### DEFICIENT or Moderately weak ADRENALS
(Patient with average to low cortisol & 17-OH-steroid levels, with or without signs and complaints of cortisol deficiency)

<table>
<thead>
<tr>
<th>PRINCIPLES</th>
<th>TIMING</th>
<th>VIAL:</th>
<th>VIAL:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start GH at a lower dose and gradually increase</td>
<td>Before bedtime</td>
<td>GH DOSE</td>
<td>mg</td>
</tr>
<tr>
<td>Inject every day before bedtime (eventually 113” of dose in the morning, 2/3” in the evening)</td>
<td>1st 10 days</td>
<td>0.02 ml/day</td>
<td>0.027</td>
</tr>
<tr>
<td></td>
<td>2nd 10 days</td>
<td>0.04 ml/day</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>3rd 10 days</td>
<td>0.06 ml/day</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>4th month</td>
<td>0.08 ml/day</td>
<td>0.11</td>
</tr>
<tr>
<td></td>
<td>5th month</td>
<td>Follow-up consultation: check and adjust doses</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6th month</td>
<td>0.10 ml/day</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>7th month</td>
<td>0.12 ml/day</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>8th month</td>
<td>0.14 ml/day</td>
<td>0.19</td>
</tr>
<tr>
<td></td>
<td>9th month</td>
<td>0.16 ml/day</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td>4th, 8th &amp; ... 1st month</td>
<td>Follow-up consults: patient is examined and GH dose adjusted, either increased or decreased depending on needs; Increase the dose by 0.02 ml every 10-30 days; Optimal dose (average): 0.06 ml - 0.30 ml/day</td>
<td></td>
</tr>
<tr>
<td>Correct any cortisol deficit</td>
<td>Before bedtime</td>
<td>INJECTION PEN:</td>
<td>Norditropin Simplex (Nordi-pen)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GH DOSE</td>
<td>mg</td>
</tr>
<tr>
<td></td>
<td>1st 15 days</td>
<td>1 click/day</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>2nd 15 days</td>
<td>2 clicks/day</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>2nd to 4th mo.</td>
<td>2 or 3 clicks/day</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>4th month</td>
<td>Follow-up consultation: check and adjust doses</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5th month</td>
<td>4 clicks/day</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td>6th month</td>
<td>5 clicks/day</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td>7 &amp; 8th months</td>
<td>6 clicks/day</td>
<td>0.30</td>
</tr>
<tr>
<td></td>
<td>4th, 8th &amp; 16th month</td>
<td>Follow-up consultations: the GH dose is adjusted, Increase the dose with 0.10 mg every month; Optimal Dose (on average): 0.1 - 0.4 mg/day</td>
<td></td>
</tr>
<tr>
<td>Severe adrenal deficiency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increase two times slower and remain at lower doses even if patient is treated for the adrenal deficiency: Increase with 0.01 ml/day every 10 days or 0.05 mg per day every 30 days go up to maximum of 0.06 ml - 0.10 mg/day before the next follow-up</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hertoghe, 2006
Suggested order of priority in learning hormone therapies for neophyte physicians and patients

1° Thyroid hormone therapy
2° Testosterone therapy in men
3° Safe female hormone therapy in women
4° Safe cortisol replacement
5° DHEA therapy
6° Fludrocortisone (aldosterone) therapy
7° Growth hormone
8° Others: pregnenolone
9 Androstenedione
(Hertoghe, 2006)
2.D. Hormone Management

The following table shows the hormone tests useful for an initial assessment. The essential tests are highlighted in yellow colour, whereas tests to do occasionally, only when a person’s signs and complaints require further investigation, are not highlighted in color.

<table>
<thead>
<tr>
<th>HORMONES</th>
<th>BLOOD TESTS</th>
<th>24-hour URINE TESTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pituitary hormones</td>
<td>GH, LH, FSH, ACTH, Prolactin</td>
<td>GH</td>
</tr>
<tr>
<td>Pineal h.</td>
<td>6-sulfatoxy-melatonin</td>
<td></td>
</tr>
<tr>
<td>Thyroid hormones</td>
<td>TSH, Free T3, Free T4, thyroid antibodies, Anti-thyroglobulin (ATG), Anti-thyroid peroxidase (ATPO)</td>
<td></td>
</tr>
<tr>
<td>Liver GH axis hormone</td>
<td>IGF-1 (somatomedin C), IGF-BP-3, Calcitonin</td>
<td></td>
</tr>
<tr>
<td>Adrenal hormones</td>
<td>Cortisol (total), Transcortin (CBG), Free cortisol¹, DHEA, sulfate</td>
<td>Free Cortisol, 17-hydroxy-steroids, Free DHEA</td>
</tr>
<tr>
<td>Female hormones</td>
<td>Estradiol, Progesterone (premenopausal 21st day of the menstrual cycle)</td>
<td>Free Estradiol, estrone, estradiol, 16-alpha-hydroxy-estrene, 2-hydroxy-estrene, 4-hydroxy-estrene, 4-methoxy-estrene, Pregnandiol</td>
</tr>
<tr>
<td>Male hormones</td>
<td>Testosterone, SHBG, Androstanediol glucuronide (or if not available: dihydrotestosterone)</td>
<td>Free Testosterone</td>
</tr>
</tbody>
</table>

Note: ¹ The free cortisol is calculated from a formula based on the ratio total cortisol – CBG.

(Hertoghe, 2006)
1. Overview of Mental and Emotional Complaints of Hormone Deficiencies

The table below is a quick overview of the main complaints a hormone deficient patient can have:

<table>
<thead>
<tr>
<th>MENTAL and EMOTIONAL COMPLAINTS of HORMONE DEFICIENCIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult Growth Hormone Deficiency</td>
</tr>
<tr>
<td>Quality of life  • Poor quality of life, feels unwell</td>
</tr>
<tr>
<td>• Lack of inner peace</td>
</tr>
<tr>
<td>• Chronic anxiety, without any reason</td>
</tr>
<tr>
<td>• Tendency to be depressed</td>
</tr>
<tr>
<td>Mood</td>
</tr>
<tr>
<td>• Lack of self-control; Excessive emotional reactions</td>
</tr>
<tr>
<td>• Melodramatic, histrionic</td>
</tr>
<tr>
<td>• Outbursts of panic and anxiety</td>
</tr>
<tr>
<td>• Sharp verbal retorts</td>
</tr>
<tr>
<td>• Tendency to social isolation</td>
</tr>
<tr>
<td>• Impaired social status (lower professional position, lower income, generally living without partners, alone or still at parents' home, poor social integration)</td>
</tr>
<tr>
<td>Social behavior</td>
</tr>
<tr>
<td>• Poor sleep:</td>
</tr>
<tr>
<td>• A superficial, anxious, agitated sleep</td>
</tr>
<tr>
<td>• With a lot of anxious thinking</td>
</tr>
<tr>
<td>• Easily waking up during the night</td>
</tr>
<tr>
<td>• Difficulties to fall asleep and fall back asleep</td>
</tr>
<tr>
<td>• Poor dreaming</td>
</tr>
<tr>
<td>Adult Melatonin Deficiency</td>
</tr>
<tr>
<td>Sleep</td>
</tr>
<tr>
<td>• Slowness</td>
</tr>
<tr>
<td>• Apathy (lack of interest, initiative)</td>
</tr>
<tr>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Behavior</td>
</tr>
<tr>
<td>• Morning depression</td>
</tr>
<tr>
<td>Mood</td>
</tr>
<tr>
<td>• Slowness thinking and reaction</td>
</tr>
<tr>
<td>• Easily distracted</td>
</tr>
<tr>
<td>Memory</td>
</tr>
<tr>
<td>• Poor concentration, poor attention</td>
</tr>
<tr>
<td>• Poor memory</td>
</tr>
<tr>
<td>• Poor school performance</td>
</tr>
<tr>
<td>Pregnenolone Deficiency</td>
</tr>
<tr>
<td>Possible DIRECT complaints</td>
</tr>
<tr>
<td>Memory</td>
</tr>
<tr>
<td>• Poor memory</td>
</tr>
<tr>
<td>Vision</td>
</tr>
<tr>
<td>• Poor color vision</td>
</tr>
<tr>
<td>INDIRECT complaints: resulting from deficiencies in hormones derived from pregnenolone (mainly adrenal cortex &amp; sex hormones)</td>
</tr>
</tbody>
</table>
### 2.D. Hormone Management

#### Mental and Emotional Complaints of Hormone Deficiencies

**Women**

<table>
<thead>
<tr>
<th>Estrogen Deficiency</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vitality</strong></td>
<td>Fatigue (persistent)</td>
</tr>
<tr>
<td><strong>Mood</strong></td>
<td>Depression (persistent)</td>
</tr>
</tbody>
</table>

**Progesterone Deficiency (or Estrogen excess)**

| **Vitality**   | Muscle and nervous tension |
| **Mood**       | Irritability, aggressiveness |
|                | (especially during premenstrual syndrome) |
|                | Anxiety and anger, outbursts of panic or rage |

**Testosterone Deficiency**

| **Character** | Lack of mental firmness: |
|               | Undecided, hesitating |
|               | Loss of self-confidence, lack of assertiveness |
|               | Lack of authority, submissiveness |

| **Mood** | Chronic depression |
|          | Excessive anxiety, fears |
|          | Nervous |
|          | Irritable |
|          | Ill at ease |

| **Stress** | Excessive emotions |
|            | Excessive sensitivity to difficulties |
|            | Low resistance to stress |
|            | Unnecessary worry |
|            | Hysterical reactions |

**Men**

<table>
<thead>
<tr>
<th>Testosterone Deficiency</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Character</strong></td>
<td>Lack of mental firmness:</td>
</tr>
<tr>
<td></td>
<td>Indecisive, hesitating</td>
</tr>
<tr>
<td></td>
<td>Loss of self-confidence</td>
</tr>
<tr>
<td></td>
<td>Lack of authority, submissiveness</td>
</tr>
</tbody>
</table>

| **Mood** | Chronic depression |
|          | (may include crying spells, suicidal tendencies) |
|          | Excessive anxiety, fear |
|          | Excessive emotions |

| **Stress** | Excessive sensitivity to difficulties |
|            | Unnecessary worry |

**Progesterone deficiency**

| **Emotional** | Anxiety, lack of inner peace |
| **Sleep**     | Superficial nervous sleep |
### 2.D. Hormone Management

<table>
<thead>
<tr>
<th>MENTAL and EMOTIONAL COMPLAINTS of HORMONE DEFICIENCIES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DHEA (&amp; Androstenedione) Deficiency</strong></td>
</tr>
<tr>
<td><strong>Men</strong></td>
</tr>
<tr>
<td>- Low sexual desire</td>
</tr>
<tr>
<td>- Erectile dysfunction</td>
</tr>
<tr>
<td><strong>Women</strong></td>
</tr>
<tr>
<td>- Low sexual desire</td>
</tr>
<tr>
<td>- Low sexual satisfaction</td>
</tr>
<tr>
<td><strong>Cortisol Deficiency</strong></td>
</tr>
<tr>
<td>- Poor resistance to stress, great difficulty to function well in stressful situations or even react to them, paralyzed in stressful situations, experiencing stress as being too much, as an unfair event</td>
</tr>
<tr>
<td>- Excessive sensitivity to human suffering</td>
</tr>
<tr>
<td>- Excessive compassion for the pain of others</td>
</tr>
<tr>
<td><strong>Stress</strong></td>
</tr>
<tr>
<td>- Irritability</td>
</tr>
<tr>
<td>- Negativism (experiencing reality as being more negative than it really is for others)</td>
</tr>
<tr>
<td>- Feeling of being of a victim</td>
</tr>
<tr>
<td>- Paranoid-like reactions: accusatory behavior, quarrelsome</td>
</tr>
<tr>
<td><strong>Character</strong></td>
</tr>
<tr>
<td>- Excessive emotions: outbursts of anger or anxiety, panic attacks</td>
</tr>
<tr>
<td>- Easy screaming or yelling</td>
</tr>
<tr>
<td>- Sharp verbal retorts, use of strong, dramatized words</td>
</tr>
<tr>
<td><strong>Behavior</strong></td>
</tr>
<tr>
<td>- Drowsiness, zombie-like feeling</td>
</tr>
<tr>
<td>- Easily distracted, absent-minded</td>
</tr>
<tr>
<td>- Day dreaming</td>
</tr>
<tr>
<td>- Difficulty focusing on tasks</td>
</tr>
<tr>
<td>- Feels better in head when laying flat on a bed</td>
</tr>
<tr>
<td>- or moving all the time</td>
</tr>
<tr>
<td><strong>Aldosterone Deficiency</strong></td>
</tr>
<tr>
<td><strong>When STANDING UP OR SITTING</strong></td>
</tr>
<tr>
<td>- Troubled vision with difficulty to focus on objects and tasks when standing</td>
</tr>
</tbody>
</table>
2.D. Hormone Management

The most important effects a hormone treatment can have on other hormone activities are summarized in the following table.

<table>
<thead>
<tr>
<th>Corrective Hormone therapy</th>
<th>Endocrine deficiency that makes the hormone therapy not well-tolerated(^1)</th>
<th>Intolerance to the hormone therapy</th>
<th>Degree</th>
<th>Hormone imbalance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melatonin therapy</td>
<td>Cortisol deficiency</td>
<td></td>
<td>++</td>
<td>Melatonin and growth hormone (GH) treatment aggravate the cortisol deficiency by reducing cortisol production and levels.</td>
</tr>
<tr>
<td>GH therapy</td>
<td></td>
<td></td>
<td>+++</td>
<td>The thyroid treatment aggravates the cortisol deficiency by increasing cortisol catabolism; Even small doses of thyroid treatment result in hyperthyroid episodes of excess T3 because in cortisol deficiency the conversion of thyroid hormone T4 to the very active T3 is accelerated. Hyperthyroid episodes more frequently occur in stressful conditions where more cortisol is needed.</td>
</tr>
<tr>
<td>Thyroid therapy</td>
<td>Cortisol deficiency</td>
<td>+++</td>
<td></td>
<td>Hyperthyroid episodes, because a low estradiol level increases the conversion of T4 into the active T3</td>
</tr>
<tr>
<td></td>
<td>Estradiol deficiency</td>
<td>+</td>
<td></td>
<td>When doses of calcitonin are too high, patients report suffering from a state that resembles overt cortisol deficiency with nausea</td>
</tr>
<tr>
<td></td>
<td>Calcitonin therapy</td>
<td>+ ±</td>
<td></td>
<td>Note: (^1) A borderline or severe hormone deficiency that causes intolerance if it remains untreated.</td>
</tr>
</tbody>
</table>

(Hertoghe, 2006)
4. Overview of Disease Susceptibility

The following table is an overview of the most important age-related diseases each hormone deficiency could promote as suggested by animal and human studies:

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Neuropsy.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Alzheimer's</td>
</tr>
<tr>
<td></td>
<td>Depression</td>
</tr>
<tr>
<td></td>
<td>Anxiety,</td>
</tr>
<tr>
<td></td>
<td>stressable.</td>
</tr>
<tr>
<td>GH Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>Melatonin Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>Thyroid Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>Pregnenolone Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>Cortisol Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>DHEA Deficiency</td>
<td>?</td>
</tr>
<tr>
<td>Aldosterone Deficiency</td>
<td>?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Estrogen Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>Progesterone Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>Testosterone Deficiency</td>
<td>+</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Men</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Testosterone Deficiency</td>
<td>+</td>
</tr>
<tr>
<td>Progesterone Deficiency</td>
<td>+</td>
</tr>
</tbody>
</table>

Code:
"+" signifies that there is data suggesting that a deficiency or lower levels of the hormone are associated with an increased risk of the disease.
"-" signifies that scientific researchers generally report that a deficiency or lower levels of the hormone reduces the risk of disease.
"?" or brackets "( )" signify that there is doubt or controversy about the information.
7.B. Lucid dreaming

- The conscious awareness of being in a dream while you’re dreaming.
- You can act deliberately (flying, walking through walls, create a desired object, or make objects disappear)
- Conduct experiments in the unconscious mind or seek information from the apparently conscious unconscious (Waggoner, 2009)
7.B. Lucid dreaming, naturally

- Repetitive nightmares, must change outcome
- Obsessives who continually ask question, “What was I just doing?”
“Lucid dreamers must learn to focus simultaneously on both their conscious awareness and the apparent dreaming activities. Lucid dreamers who become overly focused on the dreaming activities [as an outsider looking in, are not active in the dream]. So too, lucid dreamers who become inattentive to the fact of their conscious awareness risk becoming lost to the dreaming.” (Waggoner, 2009)
7.B. Lucid dreaming

To maintain lucidity, we must develop a proper balance of mindful, aware interacting to engage the dream consciously. Waggoner, 2009
7.B. Lucid dreaming, benefits

- Creating the dream in reality
- Finding information
- Healing yourself and others
- Telepathy
- Precognitive
- Mutual lucid dreaming
- Interacting with the deceased
- Unified self in a connected universe (Waggoner, 2009)
7. Mind Therapies C. Meditation, i. Gratitude

- Recognize have received gift
- Recognize value of gift
- Appreciate intentions of the giver
- ----acknowledgement of goodness in one’s life
- Re-cognized the event (lemon into lemonade)
- Receiving what we do not expect to receive or have earned or receiving more than we have deserved or earned
7. Mind therapies, C. meditation, i. gratitude

- Gratitude (Emmons and McCoullough) - how gratitude can make you happier
Giving thanks for abundance is sweeter than the abundance itself

~ Rumi
7. E. Happiness

- Happiness can add as much as 9 years to life expectancy (Emmons, 2007)
- Cheerful students earned $25,000 more than their more dour classmates, 16 years later (Emmons, 2007)
- Happy people are successful and flourishing.
- Happy people are more charitable, creative, generous, more helpful, self-confident, have better self-control, self-regulation, better coping skills (Emmons, 2007)
7.E. Optimal job

- “just right” mentally stimulating career
- Directing own course to not be overstressed– decision to leave job if necessary
- Sense of purpose
- Feeling of mastery
Abstract

Purpose of review: Recent research has revealed that the population of older adults is composed not only of individuals who are either healthy or have an age-related disease, most commonly Alzheimer's disease, but also individuals with mild cognitive impairment who are at-risk for or already in the prodromal stage of Alzheimer's disease. These variations in cognitive aging can be related to their neural bases via structural and functional neuroimaging methods.

Recent findings: Healthy aging appears to primarily affect a frontal-striatal system that undergirds executive control of cognition, while minimally affecting medial temporal lobe structures. Functional imaging studies suggest that enhanced prefrontal engagement may offer compensatory plasticity that minimizes age-related cognitive losses. Mild cognitive impairment appears to affect the entorhinal cortex in particular, with functional consequences in other brain regions. Alzheimer's disease is characterized by severe hippocampal injury, although early-stage Alzheimer's disease may relatively spare some cortical regions.

Summary: Advances in in-vivo imaging methods are providing the tools for identifying different trajectories of neurocognitive aging, and knowledge about these brain changes may promote opportunities for treatment.

7.A. iv. Mind Therapies

Dedefferentiation in aging across memory systems.

Young adults use medial temporal lobes for explicit learning and striatum for implicit learning; older adults show no preferential recruit for either task. Thus in aging, the two systems become less specialized. Older adults choose neural dedifferentiation to compensate for loss of medial temporal lobe/explicit learning function. (Dennis and Cabeza, 2011)
8. Brain stimulation

- Neurofeedback
  - LENS (Len Ochs, founder) assists with
    - assimilating, processing, organizing and retrieving information
- Brain Spark
- Brain Fitness program
9. Neurotoxicity

- Common symptoms can include problems with memory, concentration, reaction time, sleep, thinking, language, as well as depression, confusion, personality changes, fatigue, and numbness of the hands and feet. Many types of nervous system disorders could be caused by neurotoxicity, including numerous neurologic and psychiatric disorders.

- Interpersonal and legal difficulties may result from mental health problems stemming from neurotoxicity, including job loss, family difficulties, and irrational, unusual, criminal or violent behavior. Dr. Raymond Singer, Ph.D., Neuropsychologist, Member of Society of Toxicology, written 125 books
9.A. Neurotoxins are biologic partial list- prevent exposure or treat

- **Borrelia burgdorferi**
- **BOTULINUM TOXIN** A toxin made by the microorganism *Clostridium botulinum*; found in improperly canned food; affects the nerve-muscle junction, causing severe weakness and death in most cases; is used clinically to reduce muscle spasms and even reduce facial lines
- **Mycotoxin – aflatoxin (aspergillus, ochratoxin and Tricothecene)**
- **PFIESTERIA PISCIDIA** A microorganism found in fish off the East Coast; has caused memory loss among fishermen in Chesapeake Bay
- **TETRODOTOXIN** A toxin absorbed via consumption of improperly prepared puffer fish, called fugu in Japan; blocks motor and sensory nerve impulses, causing respiratory paralysis and death in most cases
- **TUBOCURARINE** Also called *curare*, a toxin from the plant *Chondodendron tomentosum*; blocks motor neurons and causes paralysis; is used as an arrow poison, and to achieve immobility in general anesthesia
9.A. Neurotoxins

Mycotoxins

- Aflatoxin - is the most carcinogenic naturally occurring substance ever encountered.
- Fumonisin B – is toxic to brain and is carcinogenic. Produced by Fusarium growing on corn.
- Ochratoxin - carcinogenic kidney toxin (generated by Aspergillus ochraceus (from nuts, corn and peanuts) and Penicillium verrucosum)). (Penicillium verrucosum causes mycotoxicosis and is isolated from wheat and barley).
- Trichothecenes - potent inhibitors of protein synthesis with immunosuppressive and dermotoxic effects, are toxins produced by Stachybotrys
9.A. Neurotoxins

Mycotoxin

prior to Operation Desert Storm, Sadam Hussein had his Scud missiles loaded with a number of deadly chemicals and biologic agents. One of these was aflatoxin. This was verified after the war by a special United Nations inspection team.

(Davis, 1999)
## 9.A. Neurotoxicants are man made
### Top 100 most common

<table>
<thead>
<tr>
<th>2-Ethoxyethyl Acetate</th>
<th>Bisphenol A Halothane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acibenzolar-S-methyl</td>
<td>Bromodeoxyuridine(5-)</td>
</tr>
<tr>
<td>Acrylamide</td>
<td>Butylated Hydroxy Anisol</td>
</tr>
<tr>
<td>Aldicarb</td>
<td>Butylated hydroxytoluene</td>
</tr>
<tr>
<td>Allethrin</td>
<td>Cadmium</td>
</tr>
<tr>
<td>Aluminum (cl or lactate)</td>
<td>Caffeine</td>
</tr>
<tr>
<td>Amino-nicotinamide(6-)</td>
<td>Carbamazepine</td>
</tr>
<tr>
<td>Aminopterin</td>
<td>Carbaryl</td>
</tr>
<tr>
<td>Amphetamine(d-</td>
<td>Carbon monoxide</td>
</tr>
<tr>
<td>Arsenic</td>
<td>Chlordecone</td>
</tr>
<tr>
<td>Aspartame</td>
<td>Chlorodiazepoxide</td>
</tr>
<tr>
<td>Azacytidine(5-)</td>
<td>Chlorine dioxide</td>
</tr>
<tr>
<td>Benomyl</td>
<td>Chlorpromazine</td>
</tr>
<tr>
<td>Benzene</td>
<td>Chlorpyrifos</td>
</tr>
<tr>
<td>Bioallethrin</td>
<td>Cocaine</td>
</tr>
<tr>
<td>Bis(tri-n-butyltin)oxide</td>
<td></td>
</tr>
</tbody>
</table>

**Building a Database of Developmental Neurotoxicants: Evidence from Human and Animal Studies**


9.A. Neurotoxicants

Top 100 most common

- Colchicine
- Cypermethrin
- Cytosine Arabinoside
- Dexamethasone
- Diamorphine hydrochloride
- Diazepam
- DEET
- Deltamethrin
- Diazinon
- Diethylstilbestrol
- Diphenylhydantoin
- Epidermal Growth Factor
- Ethanol
- Ethylene thiourea
- Flourouracil(5-)
- Fluazinam
- Fluoride
- Griseofulvin
- Haloperidol
- Heptachlor
- Hexachlorobenzene
- Hexachlorophene
- Hydroxyurea
- Imminodipropionitrile
- Ketamine
- Naltrexone
- Nicotine
- Methoxyethanol, 2-
- Methylazoxymethanol
- Methylmercury
- Lead

Building a Database of Developmental Neurotoxicants: Evidence from Human and Animal Studies

9.A. Neurotoxicants

Top 100 most common

- Lindane
- LSD
- Maneb
- Medroxyprogesterone
- Mepivacaine
- Methadone
- Methanol
- Methimazole
- Methylparathion
- Monosodium Glutamate Valproate
- MPTP Vincristine
- Naloxone
- Ozone
- Paraquat
- Parathion (ethyl)
- PBDEs
- PCBs (generic)
- Penicillamine
- Permethrin
- Phenylacetate
- Phenylalanine (d,l)
- Phthalate, di-(2-ethylhexyl)
- Propylthiouracil
- Retinoids/vit.A/isotretinoin
- Salicylate
- Tebuconazole
- Tellurium (salts)
- Terbutaline
- (IDPN) Thalidomide
- THC
- Toluene
- Triamcinolone
- Tributyltin chloride
- Trichlorfon
- Trichloroethylene
- Triethyllead
- Triethyltin
- Trimethyltin
- Trypan blue
- Urethane

Building a Database of Developmental Neurotoxicants: Evidence from Human and Animal Studies
Neurotoxicology Div, U.S. EPA, RTP, NC 27711; 2Curriculum in Toxicology, Univ. of N.C. at Chapel Hill, Chapel Hill, NC, 27514; NCEA/ORD, U.S. EPA,
9.a. Sick building syndrome
9.a. Indoor chemical toxicant emissions

Objectives for classroom workplaces include:

- A source of fresh air is essential.
- Fresh air is easily provided by opening windows or doors.
- Maintain a supply of fresh air in the classroom.
- Keep the windows and doors opened when the classroom is not occupied.
- Use fans to circulate fresh air in the classroom.

Sources of chemical emissions

<table>
<thead>
<tr>
<th>Source</th>
<th>Formaldehyde</th>
<th>Hexane</th>
<th>Benzene</th>
<th>Trichloroethylene</th>
<th>Chloroform</th>
<th>Ammonia</th>
<th>Alcohol</th>
<th>Isopropyl Alcohol</th>
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</thead>
<tbody>
<tr>
<td>Adhesives</td>
<td>✓</td>
<td></td>
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<tr>
<td>Bioeffluents</td>
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<td>✓</td>
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<tr>
<td>Blueprint machines</td>
<td></td>
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<td></td>
<td></td>
<td>✓</td>
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<tr>
<td>Carpeting</td>
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<tr>
<td>Caulking compounds</td>
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<tr>
<td>Ceiling tiles</td>
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<td></td>
<td>✓</td>
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<td></td>
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<tr>
<td>Chlorinated tap water</td>
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<td>Cleaning products</td>
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<td>Computer VDU screens</td>
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<td>Cosmetics</td>
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<td>Duplicating machines</td>
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<tr>
<td>Electrophotographic printers</td>
<td>✓</td>
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<td>✓</td>
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<tr>
<td>Draperies</td>
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<td>✓</td>
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<td>Fabrics</td>
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<tr>
<td>Facial tissues</td>
<td></td>
<td>✓</td>
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<tr>
<td>Floor coverings</td>
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<td>Gas stoves</td>
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<td>Grocery bags</td>
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<td>Microfiche developers</td>
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<td>Office correction fluid</td>
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<td>Paints</td>
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<td>Paper towels</td>
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<tr>
<td>Particleboard</td>
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<tr>
<td>Permanent-press clothing</td>
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<td>Photocopiers</td>
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<td>Plywood</td>
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<tr>
<td>Pre-printed paper forms</td>
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<tr>
<td>Stains and varnishes</td>
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<td></td>
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<tr>
<td>Tobacco smoke</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
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<td></td>
<td></td>
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<tr>
<td>Upholstery</td>
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<td></td>
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<tr>
<td>Wall coverings</td>
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</tr>
</tbody>
</table>

Some common emissions and their sources are listed in the table above.

A thorough understanding of fresh air sources is essential for a healthy classroom environment.
9.A. Toxicants

- **Formaldehyde**
  - The aim of this study was to determine whether the inhalation of formaldehyde has a neurotoxicological impact.
  - Forty Wistar rats (Lew.1/K) were trained to find food in a maze within a particular time. When all animals were at an equal level, 13 rats inhaled 2.6 ppm and 13 others inhaled 4.6 ppm formaldehyde 10 min/d, 7 d/week for 90 d. The control group comprised 14 animals inhaling water steam according to the same exposure pattern. During the exposure period and the post-trial observation stage (30 d), the time required to find the food and the number of mistakes made on the way were recorded.
  - Between the animals exposed to formaldehyde and the control group a statistically significant difference for both parameters was observed (p<0.05). The animals exposed to formaldehyde needed more time and made more mistakes than the animals of the control group while going through the maze.
  - The results underline the necessity for a systematic observance of precautions in case of occupational or dwelling-related formaldehyde exposure, and allow us to classify formaldehyde as “probably neurotoxic”. Further investigations are required to assess the neurotoxicologic impact of subchronic formaldehyde exposure.

- **Formaldehyde Neurotoxicity in Animal Experiments**
- Pitten and Koch, Pathology-Research and Practice, 2000, 196 (3):193-8
## 9.A. Removal of formaldehyde by houseplants

Wolverton, D.C., 1996 How to Grow Fresh Air  P. 23

### Table 1: Removal of the toxic gas formaldehyde by houseplants

<table>
<thead>
<tr>
<th>Plant</th>
<th>L/H per hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boston fern</td>
<td></td>
</tr>
<tr>
<td>Florist's mum</td>
<td></td>
</tr>
<tr>
<td>Gerbera daisy</td>
<td></td>
</tr>
<tr>
<td>Dwarf date palm</td>
<td></td>
</tr>
<tr>
<td>Janet Craig</td>
<td></td>
</tr>
<tr>
<td>Bamboo palm</td>
<td></td>
</tr>
<tr>
<td>Kimberley queen fern</td>
<td></td>
</tr>
<tr>
<td>Rubber plant</td>
<td></td>
</tr>
<tr>
<td>English ivy</td>
<td></td>
</tr>
<tr>
<td>Weeping fig</td>
<td></td>
</tr>
<tr>
<td>Peace lily</td>
<td></td>
</tr>
<tr>
<td>Areca palm</td>
<td></td>
</tr>
<tr>
<td>Corn plant</td>
<td></td>
</tr>
<tr>
<td>Lady palm</td>
<td></td>
</tr>
<tr>
<td>Schefflera</td>
<td></td>
</tr>
<tr>
<td>Dragon tree</td>
<td></td>
</tr>
<tr>
<td>Warneckei</td>
<td></td>
</tr>
<tr>
<td>Lily turf</td>
<td></td>
</tr>
<tr>
<td>Dendrobium orchid</td>
<td></td>
</tr>
<tr>
<td>Dumb cane (Exotica)</td>
<td></td>
</tr>
<tr>
<td>Tulip</td>
<td></td>
</tr>
<tr>
<td>Ficus alii</td>
<td></td>
</tr>
<tr>
<td>King of hearts</td>
<td></td>
</tr>
<tr>
<td>Parlor palm</td>
<td></td>
</tr>
<tr>
<td>Azalea</td>
<td></td>
</tr>
<tr>
<td>Chinese evergreen</td>
<td></td>
</tr>
<tr>
<td>Spider plant</td>
<td></td>
</tr>
<tr>
<td>Banana</td>
<td></td>
</tr>
<tr>
<td>Red emerald philodendron</td>
<td></td>
</tr>
<tr>
<td>Dumb cane (Camilla)</td>
<td></td>
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<tr>
<td>Elephant ear philodendron</td>
<td></td>
</tr>
<tr>
<td>Golden pothos</td>
<td></td>
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<tr>
<td>Norfolk island pine</td>
<td></td>
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<tr>
<td>Wax begonia</td>
<td></td>
</tr>
<tr>
<td>Prayer plant</td>
<td></td>
</tr>
<tr>
<td>Oak leaf ivy</td>
<td></td>
</tr>
<tr>
<td>Christmas cactus</td>
<td></td>
</tr>
<tr>
<td>Lacy tree philodendron</td>
<td></td>
</tr>
<tr>
<td>Arrowhead vine</td>
<td></td>
</tr>
<tr>
<td>Heart-leaf philodendron</td>
<td></td>
</tr>
<tr>
<td>Lady Jane</td>
<td></td>
</tr>
<tr>
<td>Peacock plant</td>
<td></td>
</tr>
<tr>
<td>Poinsettia</td>
<td></td>
</tr>
<tr>
<td>Cyclamen</td>
<td></td>
</tr>
<tr>
<td>Moth orchid</td>
<td></td>
</tr>
<tr>
<td>Urn plant</td>
<td></td>
</tr>
<tr>
<td>Croton</td>
<td></td>
</tr>
<tr>
<td>Snake plant</td>
<td></td>
</tr>
<tr>
<td>Aloe vera</td>
<td></td>
</tr>
<tr>
<td>Kalanchoe</td>
<td></td>
</tr>
</tbody>
</table>
9.A. MAN MADE NEUROTOXICANTS (PARTIAL LIST)

- **PESTICIDES**  Many types; exposure is common in farming, spraying of fruit trees, houseplants, pests like ants, bees, and roaches; these agents often overexcite the cholinergic system, producing neuropathies, weakness, and mental status changes.

- **NERVE GASES**  Many types; the most common work just like pesticides, overexciting the cholinergic system, often to the point of death.

- **SOLVENTS**  Many types are used in different industries with different clinical effects, including:
  - **ALIPHATIC HYDROCARBONS**: gasoline, kerosene, propane, butane, pentane, hexane, heptane, and octane.
  - **ALIPHATIC HALOGENATED HYDROCARBONS**: chloroform, carbon tetrachloride, methylene chloride, methyl chloride, trichloroethylene, tetrachlorethylene, vinyl chloride.
  - **ALCOHOLS**: methanol, isopropyl alcohol.
  - **GLYCOLS**: ethylene glycol.
  - **AROMATIC HYDROCARBONS**: benzene, toluene, polycyclic aromatic hydrocarbons (PAHs).
9.A. Solvent-induced Alzheimer's

- Solvent Exposure as a Risk Factor for Alzheimer's Disease: A Case-Control Study

- Abstract
  
  This case-control study investigates whether history of organic solvent exposure is associated with increased risk of Alzheimer's disease. The study base includes about 23,000 persons aged 60 years or more from the local membership of a health maintenance organization in Seattle, Washington, who entered the study between 1987 and 1992. Probable Alzheimer's disease cases (n = 193) who had presented with new dementia symptoms were identified, enrolled, and diagnosed by our Alzheimer's Disease Patient Registry following standardized criteria. Control subjects (n = 243), free of dementia and neurologic disease causing dementia, were selected randomly from the study base and frequency matched to cases for age and sex. Proxy informants provided specific solvent exposure history as well as job descriptions likely to involve solvent use as part of a comprehensive risk factor interview. Kappa statistics indicated substantial agreement for control-control proxy solvent responses. History of exposure to one or more solvent groups (benzene and toluene; phenols and alcohols; ketones; other solvents) yielded an adjusted Alzheimer's disease odds ratio of 2.3 (95 percent confidence interval 1.1–4.7); among males only, it increased to 6.0 (95% confidence interval 2.1–17.2). Thus, past exposure to organic solvents may be associated with onset of Alzheimer's disease. Am J Epidemiol 1995;141:1059–71.

- (Kukull et al, 1993)
9.A. Pesticide-induced Parkinson's
9.A. Heavy metals

- **Arsenic**  Used in copper smelting and semiconductor manufacture; found in some herbal remedies; interferes with oxidative metabolism; causes muscle spasms, seizures, agitation, memory loss
- **Lead**  Used in battery production, foundries; jewelry production; found in old painted surfaces; interferes with many enzymes; causes fatigue, headaches, anxiety, memory loss
- **Manganese**  Used in battery production, fertilizers, gasoline additives; causes free radical damage, especially to cells in the substantia nigra and basal ganglia; causes a parkinsonian disorder
- **Mercury**  Used in the production of chlorine, paints, paper; also found in thermometers; inhibits many enzymes and harms membrane function; causes emotional instability, memory loss, spasticity
- **Thallium**  Used in rat and ant poisoning, infrared detectors, and photocells; causes numbness, weakness, respiratory paralysis, emotional instability, and delirium
9.A. Heavy metal detoxification

- Consider ACAM heavy metal detoxification/chelation module
  - Remove exposure (distilled water or reverse osmosis water filtration for cooking and drinking water)
  - Stooling 2-3 x/day
  - Nutrients: aa, Mg, Mn, Mo, Se, Zn, cysteine, glutathione, sulfate, Vit C, E
9.C. HIGH STRESS JOBS

- Soldier in battle
- Disaster relief worker (especially those who recover bodies) Military trainee in basic training
- Public schoolteacher
- Astronaut
- Nurse (especially in intensive care, cancer, burn, HIV, and some pediatric wards)
- Air traffic controller
- Professional working with the mentally disabled, mentally ill, or victims of head trauma
- Sex worker (male or female prostitute)
- Short-haul bus or taxi driver
- Social worker
- Airline pilot
- Deep-sea fisherman
- Hard-hat diver
- Factory worker doing a monotonous, rapid, repetitive task Farmer on a small farm
- Policeman or policewoman
- Firefighter
- Emergency medical technician
- Attorney (especially a new associate in a law firm)
- Physician (especially one in training, or transitioning to managed care)
- Flight attendant (especially on long-haul routes)
Introduction
The term *plagiocephaly*, from the Greek *plagios* (oblique) and *kephalê* (head), means distortion of the head, and refers clinically to cranial asymmetry. Cranial Osteopathy, since it was first proposed, has focussed upon the diagnosis and treatment of birth trauma and cranial asymmetries, and consequently specific therapy for plagiocephalic deformities has been described. Osteopathic manipulation also has been proposed as a treatment for torticollis, a condition associated with plagiocephaly. For these reasons, we decided to look at the mechanics of the occipital bone and the adjacent atlas and bones of the cranial base, in relation to functional plagiocephaly.

Methods
The records of 649 children seen in an osteopathic practice in Lyon, France, were reviewed retrospectively, in compliance with the legal requirements of the (CRIL) and the Helsinki accord, for gender, age at presentation, birth history, obstetrical data (breech presentation, vacuum extraction, forceps delivery or Caesarean section), presenting complaint, side of posterior plagiocephaly, side of frontal plagiocephaly, torticollis, motion pattern of the occipital bone upon the atlas, and motion pattern of the spheno-occipital synchondrosis.

Results
We found significant correlations between plagiocephaly (right/left) and primipara (), use of forceps () and extractor suction (). Correlations were also found between flattening of the occiput (right/left) and lateral strain of the spheno-occipital synchondrosis () and between plagiocephaly (right/left) and occipito-atlantal motion ().

Conclusion
We found a significant correlation between the lateral strain pattern of the spheno-occipital synchondrosis and plagiocephaly and between rotational dysfunction of the occiput upon the atlas and the side of posterior plagiocephaly. We suggest that thorough neonatal osteopathic examination can identify individuals predisposed to develop posterior plagiocephaly. (Seraqueet et al, 2006)

The authors propose that cranial osteopathy to children could prevent development of plagiocephaly when the lateral strain pattern of the spheno-occipital synchondrosis and the rotational dysfunction of the occiput upon the atlas were identified, and treated.
9.E.V. Protect - Remove preventable mechanical trauma

In The Netherlands helmet therapy is a commonly used treatment in infants with skull deformation (deformational plagiocephaly or deformational brachycephaly). However, evidence of the effectiveness of this treatment remains lacking. The HEADS study (Helmet therapy Assessment in Deformed Skulls) aims to determine the effects and costs of helmet therapy compared to no helmet therapy in infants with moderate to severe skull deformation.

HElmet therapy assessment in infants with deformed skulls (HEADS): protocol for a randomised controlled trial
Optimizing cognitive function
Feed
Protect
Stimulate
Squash blossoming
Dennis NA and R Cabeza, 2008. Neuroimaging of Healthy Aging. 1-57
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- Thich Nhat Hanh, *Living Buddha, Living Christ*

**BREATH-WORK BOOKS**
- Farhi, Donna, *The Breathing Book: Vitality and Good Health through Essential Breath Work*
- Lewis, Dennis, *The Tao of Natural Breathing: For Health, Well-Being and Inner Growth*
Resources

- RELAXTV - beautiful scenery and music