

Based on my review of the reliable and credible scientific literature regarding cognitive function, aging, cognitive decline, cognitive dysfunction and dementia, I conclude that there is significant scientific agreement in support of the following health claims:

- The consumption of phosphatidylserine may reduce the risk of cognitive dysfunction in the elderly.
- The consumption of phosphatidylserine may reduce the risk of dementia in the elderly.

Cognitive Functions, Aging and Cognitive Decline

Cognitive processes range from “automatic” (requiring little attention) to “effortful” (requiring considerable attentional capacity).^{1,2} The “capacity theory of attention” suggests that the ability to perform complex cognitive tasks will reflect the amount of processing resources available for the performance of such tasks.³ Consequently, any reduction in resource availability will affect automatic cognition processes more slowly while effortful cognition processes will decline more rapidly.³ Increasing task complexity will increase the rate of decline in ability to perform that task.³ For example, memory is impaired when the number of stimuli exceeds the remaining immediate memory capacity.⁴ In contrast, increasing task simplicity will be associated with a decrease in the rate of decline in ability to perform that task.

In some individuals, aging may be accompanied by a mild degree of decline in the ability to perform effortful cognitive processing.^{5,6} The early signs of declining ability to perform effortful cognitive processing typically include reduced ability to perform on tests of reaction time, language, declarative memory, working memory, free recall, visuospatial/reasoning skills, executive functions and problem solving.^{5,6,7-11,13} In contrast, performance deficits are least likely to occur in automatic processing tasks, such as location memory and recognition memory.⁷⁻¹¹

Several investigators have reported inability to document a relationship between age and ability to maintain cognitive function in individuals over 50 years of age who remained otherwise healthy.¹²⁻¹⁴ In contrast, functional decline in cognitive abilities was associated with failing health.¹²⁻¹⁴ It appears that even among the elderly, functional decline in cognitive abilities requires the presence of active disease processes.¹²⁻¹⁵

Pathophysiologic Processes that Contribute to Cognitive Decline

Decreased Cholinergic Function: Cholinergic neural functioning is required for learning, memory, and other attention-demanding processes in humans.¹⁶⁻²² Decreased

cholinergic function is associated with impairments in memory and other cognitive functions^{20,23-33} and the extent of memory loss is correlated with decreased cerebral glucose metabolism (as revealed by magnetic resonance imaging),³⁴ impaired synthesis of acetylcholine and sensitivity of postsynaptic membranes to acetylcholine³⁵⁻³⁷ and loss of cholinergic neurons.^{16,38} In humans with demonstrable impairments in cognitive function, the severity of cognitive deficit has been reported to be correlated with the degree of imbalance in the cholinergic system (relative excess of intrasynaptic AChE degradation activity in comparison to the rate of presynaptic *de novo* acetylcholine synthesis).^{39,40} In contrast, cholinesterase inhibitor drugs (e.g., tacrine; donepezil) and other agents that increase cholinergic activity have been found to enhance learning and memory and attenuate cognitive deficits.^{31,41-55}

Altered Neuronal Membrane Lipid Composition: Other structural and biochemical changes in the brain commonly associated with aging include derangement of neuronal membrane lipid composition and enzymatic activity (especially decreased endogenous synthesis of phosphatidylserine), decreased neurotransmitter synthesis and metabolism, and decreased synaptic density.⁵⁶⁻⁵⁹ Age-related alterations in neuronal membrane composition (particularly a decrease in the ratio of phosphatidylserine to cholesterol in the membrane) can cause neurochemical changes which can contribute to an increase in the viscosity of cellular membranes, thus reducing enzymatic activities that require optimum fluidity. These cell membrane changes can be indirectly responsible for alterations in enzymatic activities, receptor functions, membrane carriers, and neuronal electrical characteristics, resulting in functional impairments in behavior, memory and learning.^{23,59}

Cerebral Atrophy: During normal aging, shrinkage and loss of neurons in the cerebral cortex occurs, resulting in dendritic atrophy, decreased cortical synaptic density, decreased cortical synaptic connectivity and cerebral cortical atrophy.⁶⁰⁻⁶⁹ Neuronal shrinkage and loss are the most important determinants of cerebral neurodegenerative changes and cognitive decline during aging⁷⁰ and the presence of significant cerebral cortical atrophy increases the risk for cognitive decline.^{71-84,85} Similarly, magnetic resonance images of elderly humans have revealed a direct correlation between hippocampal volume and the retention of memory functions.⁸⁶⁻⁸⁸

White Matter Lesions (Leukoaraiosis): In addition to explicit volumetric change, lesions in the white matter of the brain ("leukoaraiosis") are associated with signs of declining cognitive function,^{85,89-91} especially decrements in short-term memory,^{89,92} attention⁹³ and other frontal lobe functions.⁹² Evidence of the presence of leukoaraiosis precedes evidence of cognitive decline.⁹⁴ The first clinical sign of the presence of leukoaraiosis is reduced ability to focus attention.^{93,95-97} Even among otherwise apparently healthy elderly subjects, the presence of leukoaraiosis is correlated with decreased ability to perform executive functions, reduced mental speed, delayed verbal recall and impaired ability to focus attention.⁹⁸⁻¹⁰⁰ However, this finding has not been universal.¹⁰¹ Nonetheless, the degree of overall progressive cognitive impairment among older adults

and the elderly is increased in the presence of extensive leukoaraiosis^{18,20,72,85,91,102-106} Significant leukoaraiosis increases the risk for development of clinical signs of dementia.^{102,107}

Hypoperfusion: Cerebral hypoperfusion is a self-sustaining process,¹⁰⁸ promoting collagen deposition into and thickening of the capillary basement membrane, endothelial cell collapse, pericyte degeneration and laminar flow-disrupting distortion of the capillary lumen.¹⁰⁹⁻¹¹³ These lesions accumulate during chronic cerebral hypoperfusion and culminate in degeneration of the capillary network within the brain.^{109-111,113-114} The degeneration of this network disrupts cerebral hemodynamics and impairs oxygenation and nutriture of the brain.¹¹⁵⁻¹¹⁷ The resulting malnourishment of affected areas of the brain impairs neuronal energy metabolism^{118,119} and glial cell uptake of potentially excitotoxic glutamate from the extracellular space.¹²⁰⁻¹²² Extracellular glutamate may reach neurotoxic concentrations.¹²⁰ Intracellular energy deficiency impairs neurotransmitter synthesis,^{118,119} prevents maintenance of cell membrane potential and the generation of action potentials,¹¹⁸ inhibits axoplasmic flow and axo-dendritic synaptic function^{115,119,121-123} and stimulates degeneration of the Golgi apparatus¹²⁴⁻¹²⁵ which eventually triggers apoptotic or necrotic neuronal cell death.¹²⁵⁻¹²⁶ Consequently, chronic cerebral hypoperfusion causes cerebral degeneration,^{94,127-133} with the severity of subsequent leukoaraiosis,^{62,94,108,130,134-136} cortical atrophy⁹⁴ and loss of cognitive function^{108,137,138,139} proportional to the extent of the decline in cerebral perfusion.

Cerebrovascular Disease: Cerebrovascular disease reduces cerebral blood flow⁶⁸⁻⁷⁵ and increases the risk for dementia.^{140,141} Prior to the appearance of signs of dementia, patients with cerebrovascular disease first experience increasing decline in memory performance.¹⁴²⁻¹⁵¹

Hypertension: Hypertension may contribute to the etiology of degenerative changes in the brain, particularly leukoaraiosis,^{72,152} and accelerates cerebral degeneration.^{70,94,153,154} Increased incidence of cerebrovascular disease secondary to hypertension¹⁵⁵⁻¹⁶⁰ likely participates in the pathogenesis of cerebral hypoperfusion,⁷² Hypertension-induced cerebral hypoperfusion may account for the increased incidence of cognitive decline that accompanies hypertension in middle-aged and older adults.¹⁶¹⁻¹⁶⁸

Oxidative Stress and Failure of Neuroprotection: "Neuroprotection" is the process of protecting the central nervous system from oxidative damage by reactive oxygen species.¹⁶⁹ Neuroprotective capacity anywhere within the brain may be compromised by increased local generation of reactive oxygen species or by exhaustion of local antioxidant capacity. A number of pathological processes characterized by impairments in learning, memory, attention and concentration are associated with loss of neuroprotection in the central nervous system.^{169,137}

In aging mice, the extent of oxidative damage to brain proteins increases with age¹⁷⁰⁻¹⁷³ and is inversely proportional to performance on tests of cognitive function and motor coordination.¹⁷⁰ These findings have led to the suggestion that the accumulation of oxidatively-modified proteins in the brain impairs cognitive function.¹⁷⁴

In humans, reduced production of the vasodilator, nitric oxide, within the cerebral endothelium is associated with impairment in endothelium-dependent vasodilation^{137,175-179} and may result in compromise of cerebral blood flow.¹³⁷ Chronic oxidative stress inhibits nitric oxide availability and contributes to compromise of cerebral blood flow.¹³⁷ Chronic cerebral hypoperfusion accelerates cognitive decline.^{108,137-139}

It has been suggested that cognitive or memory deficits are consequences of imbalances between local reactive oxygen species and antioxidant capacity within the brain.^{169,180} This balance is particularly critical in the locus coeruleus in the hippocampus, a region of the human central nervous system that is vital for the formation, integration and retrieval of relational memory and learning.^{4,181-186} On the other hand, the oxidative effects of reactive oxygen species can be mitigated by inhibition of the generation of reactive oxygen species or by acceleration of their reduction by detoxification reactions.^{169,187} In addition, neuropathologic changes induced by reactive oxygen species can be repaired.¹⁶⁹

Accelerated Cognitive Decline is a Required Precursor to Dementia Unrelated to Stroke

Only over the age of 70 years does cognitive performance decline with advancing age irrespective of health status.^{15,188-190} Nonetheless, the presence of mild cognitive decline is not predictive of later dementia.^{15,191} In one study of elderly individuals with mild degrees of cognitive impairment at the beginning of the study, after 2 to 3 years only 20% had progressed to clinically evident dementia while another 20% exhibited improved cognitive function and the remaining 60% were relatively unchanged.¹⁹² Similar rates of progression to dementia in cohorts of elderly individuals with initially mild cognitive impairment who have been followed for several years have been reported by several other groups of investigators.^{191,193-195} These findings have led to the suggestion that mild cognitive decline in the elderly does not always lead inevitably to dementia.¹⁵

Cognitive changes that differ from the changes seen in normal aging precede the appearance of signs of dementia.^{38,196-204} In particular, the accelerated cognitive decline preceding the clinical expression of dementia is characterized most typically by accelerated deficits in “effortful” (“controlled”) processing, with little or no decline in “automatic” processing.^{197,205-209} Controlled processing requires intact attentional resources and involves voluntary retrieval and integration of information and the performance of complex cognitive functions requiring planning, inductive thinking and flexible thought.^{205,206} Automatic processes involve well-learned, spontaneous responses

and require less cognitive effort.²⁰⁶ Examples of intellectual tasks that require the largest number of attentional resources include verbal learning,^{197,202,203,206,210-212} delayed short-term recall,^{197,202,206,210-212} visuospatial/reasoning skills,^{196,197,199,206,210} category verbal fluency,^{197,204,211,213,214} word finding^{197,199,203,204,210,213,214} and perceptual speed^{197,204,206,207,211,215} and ability to focus attention on attention-demanding tasks.^{203,204,206,212} The most sensitive indicators of initial acceleration of cognitive decline are reduced short-term memory,^{201,216-219} hippocampal atrophy^{220,221} and decreased hippocampal function.²²¹ Usually there is no loss in reading skills.^{210,214}

It has been suggested that cognitive decline in the absence of dementia typically accompanies aging and becomes "converted" to dementia only after a threshold of accumulation of small pathological changes within the brain is reached.²²² The "brain reserve" theory of the etiology of functional cognitive impairment suggests that during development the brain acquires a reserve capacity (volume, intensity of metabolism, connectivity in neural networks, dendritic branching, synapse density, efficiency of functioning, etc.).²²³ This capacity may become diminished later in life as a result of any of a number of insults or triggers of deterioration. Cognitive symptoms will appear only after this reserve capacity is either surpassed or eliminated. For example, patients with symptomatic dementia exhibit significantly accelerated loss of functional neurons from the posterior hippocampus.⁶⁴

Accelerating impairment in cerebral energy metabolism contributes to accelerating cognitive decline. In elderly individuals with or without mild decline in cognitive functioning abilities and with no symptoms of dementia, cerebral glucose metabolism (estimated using positron emission tomography; PET) declines about 2% per decade.²²⁴ This decline parallels insignificant abnormalities in cerebral perfusion that are unrelated to memory impairment or other cognitive decline.²²⁵ In contrast, older individuals without memory impairment but who later develop significant cognitive decline exhibit PET evidence of declining cerebral glucose utilization which accelerates during the progression to dementia.^{224,226}

The best predictors of future dementia or later development of Alzheimer's disease in asymptomatic individuals without signs of dementia are declining performance on tests of short-term memory,^{197,210,227-229} especially verbal memory,^{230,231} and decreasing ability to focus attention on attention-demanding tasks.^{12,197,199-201,203,204,206,210-212,227,229,231-238} Memory impairment and reduced ability to learn new information precede behavioral and emotional changes.²⁴⁰ In addition, individuals at the greatest risk of later developing dementia often also exhibit significantly impaired performance on tests of motor functions.^{240,241} Although mild cognitive deficits occur among the healthy elderly, the subsequent rates of decline in these cognitive functions are accelerated in individuals who progress to symptomatic dementia or Alzheimer's disease.^{140,242-244} The acceleration in functional decline is accompanied by extensive reduction in cholinergic innervation of the hippocampus and neocortex,²⁴⁵ widespread loss of cholinergic neurons

in the medial system²⁴⁵ and significantly decreased rates of cerebral energy extraction from glucose.²²⁴

Biochemical and Physiologic Roles of Phosphatidylserine in Cognitive Function

Phosphatidylserine is the major acidic phospholipid in the brain and is a basic structural component of all cell membranes. Within the cell membrane, small amounts of phosphatidylcholine, another cell membrane phospholipid, are produced from phosphatidylserine.⁵⁶

Within the brain, phosphatidylserine is incorporated into neuronal cell membranes, influencing the metabolism of the neurotransmitters acetylcholine, norepinephrine, serotonin and dopamine.^{56-58,246} Adequate intramembrane phosphatidylserine content is required for the fusion of intraneuronal secretory granules with the presynaptic membrane and the subsequent release of neurotransmitter molecules into the synaptic cleft.⁵⁹ In addition, adequate intramembrane phosphatidylserine content is required for proper postsynaptic neurotransmitter-receptor interactions. The activation of calcium/calmodulin-dependent protein kinase C requires an interaction between cytosolic protein kinase C and membrane-bound phosphatidylserine; subsequent phosphorylations of intracellular proteins by activated protein kinase C and the biochemical consequences of those phosphorylations “translates” the presynaptic message into specific responses within the postsynaptic cell.^{59,247}

Supplemental Phosphatidylserine and Cognitive Function

In intact aged rats, ingested phosphatidylserine increases intercellular communication by increasing the fluidity of cell membranes,^{56-58,246} eliminates the typical age-dependent decreases in stimulus-evoked acetylcholine release and cholinergic and cognitive problem-solving functions^{230,231,248} and stimulates enhanced performance on tasks that test learning ability and short-term memory.^{231,249-252} These beneficial outcomes have been associated with rapid incorporation of supplemental phosphatidylserine into neuronal cell membranes,²⁴⁸ increased cell membrane-associated ATPase activity and intraneuronal synthesis of acetylcholine and dopamine in the cerebral cortex,^{248,253-255} increased cholinergic neurotransmission and signal transduction,²⁵⁴⁻²⁵⁸ deceleration of the rate of loss of dendritic connections (maintenance of pyramidal dendritic spine density) in the hippocampus,²⁵⁹ attenuation of the rate of loss of receptors for nerve growth factor in the hippocampus²⁵⁹ (which might facilitate the ability of nerve growth factor to stimulate effective remodeling of interneuronal connections, possibly restoring dendritic spine density²⁵⁹), arrest of atrophy of cholinergic cells in the basal forebrain²⁶⁰ and reduced frequency of the normal rodent age-associated episodes of erratic electroencephalographic patterns.²⁴⁸ It has been suggested that phosphatidylserine enhances the neural events involved in the encoding and consolidation of new

information into memory, as well as facilitating the retrieval of that information when required.²⁴⁹

Oral phosphatidylserine has been reported to be highly bioavailable in humans and to cross the blood-brain barrier.^{248,261} Young adults given single intravenous doses of phosphatidylserine (75 mg) have exhibited significantly improved performance on tests of short-term memory and changes in electroencephalographic readings that were consistent with enhanced cognitive abilities.²⁶¹

These findings and the consistently positive responses of animals to dietary supplementation with phosphatidylserine have stimulated interest in phosphatidylserine supplementation in humans. In open-label trials, elderly subjects with mild degrees of decline in cognitive function have responded to oral phosphatidylserine (100 mg, t.i.d., for 60 days) with significantly improved performance on tests of verbal learning, verbal recall, verbal fluency, visual learning, attention, communication skills, initiative, socialization and self-sufficiency.^{262,263} Similar results were obtained following 90 days of the same level of daily supplementation, with significant improvements in the abilities to recall names and recognize faces also reported.²⁶⁴ Significant improvements in verbal learning, verbal recall, attention span and ability to concentrate, vigilance, initiation, socialization and self-sufficiency also were observed in elderly adults with more severe, moderate degrees of cognitive impairment following 2 months of oral supplementation with phosphatidylserine (100 mg, t.i.d.).^{265,266}

The effectiveness of oral phosphatidylserine supplementation also has been studied in double-blind placebo-controlled randomized clinical trials. Elderly men and women over 60 years of age exhibiting mild memory loss have been given placebo or oral phosphatidylserine (100 mg, t.i.d.) for 90 days. Compared to the effects of placebo, phosphatidylserine supplementation produced significantly greater improvements in short-term recall, immediate memory, vocabulary skills and ability to recall words, attention and vigilance.²⁶⁷ More severe deterioration of cognitive functions (such as attention, concentration, learning ability, and ability to perform daily activities) but without dementia or pseudodementia, also has responded to supplementation with oral phosphatidylserine (100 mg, t.i.d., for 2 months), with significantly greater improvements in verbal recall, initiation, withdrawal, apathy and overall cognitive functioning than those produced by placebo.²⁶⁸ Similar results were obtained when elderly adults with moderately severe cognitive impairment were supplemented with oral phosphatidylserine (100 mg, t.i.d.) for 6 months.⁵⁶ In addition, long-term memory and ability to perform the activities of daily living were improved significantly.

Patients also exhibiting symptoms of chronic depression also have responded to phosphatidylserine supplementation with decreased apathy, withdrawal and sleep disturbances and increases in motivation and interest in others.^{56,268,269} These beneficial effects have been accompanied by improved memory performance,²⁶⁹ increases in electroencephalographic alpha rhythm that are indicative of increased acetylcholinergic

activity²⁶¹ and positron emission tomography (PET) evidence of increased brain glucose utilization.^{270,271}

Supplemental Phosphatidylserine and Dementia

Supplementation with phosphatidylserine decreases the risk for dementia by interrupting accelerated cognitive deterioration. In a placebo-controlled randomized double-blind trial of nondemented elderly patients with mild degrees of accelerated cognitive deterioration, 8 weeks of supplemental phosphatidylserine (100 mg t.i.d.) was accompanied by improved ability to perform executive functions and electroencephalographic evidence of normalization of some brain functions; these improvements persisted for at least 16 weeks (the extent of follow-up) after discontinuation of supplementation.²⁷² However, in a placebo-controlled randomized double-blind trial of elderly patients with more severe memory loss and cognitive decline, although 6 weeks of daily supplemental phosphatidylserine (100 mg t.i.d.) stabilized cognitive function, with improvements in recall, long-term memory, pattern recognition and ability to perform the activities of daily living significantly greater than those produced by placebo, discontinuation of phosphatidylserine supplementation was followed by resumption of pre-supplementation rates of cognitive deterioration.²⁷³

Elderly patients diagnosed with Alzheimer's disease also have been given supplemental phosphatidylserine. For example, in one placebo-controlled randomized double-blind trial of elderly patients with severe cognitive impairments secondary to Alzheimer's disease who were given supplemental phosphatidylserine (200 mg daily for 3 months), the investigators reported significantly greater improvements in memory, information processing and the ability to perform activities of daily living than those produced by placebo.²⁷⁴ In another trial in which oral phosphatidylserine (400 mg daily) was administered to patients with Alzheimer's disease, the addition of phosphatidylserine supplementation to a cognitive training program for 16 weeks resulted in significantly greater improvements in performance on neuropsychological tests than did cognitive training alone.²⁷⁰ However, the progression of disease was not halted by phosphatidylserine, with deterioration of performance noted in most patients four months later despite continued phosphatidylserine supplementation. It is not known whether larger phosphatidylserine intakes may have attenuated disease progression in these patients. In other trials that have studied patients with confirmed Alzheimer's disease, improvements in cognitive function associated with phosphatidylserine supplementation (300 to 400 mg daily) generally have been greatest in the least severely impaired patients.^{224,246,271}

Human studies using positron emission tomography (PET) to investigate brain glucose utilization in patients with Alzheimer's disease have noted evidence of significantly increased glucose utilization in response to supplementation with phosphatidylserine (300 to 500 mg daily), especially in the temporo-parietal areas which are specifically affected by this disease.^{224,270,271,275}

Phosphatidylserine also may protect neuronal cell membranes from oxidative damage. In cell culture studies, phosphatidylserine supplementation has been reported to inhibit the oxidation of cell membrane phospholipids by reactive oxygen species generated by xanthine oxidase.²⁷⁶ Concurrent with inhibition of oxidation of cell membrane phospholipids was reduction in the rate of free radical-induced cell death.

Daily Intake of Supplemental Phosphatidylserine that is Effective in Reducing the Risk of Dementia

In the majority of studies, 100 mg of phosphatidylserine t.i.d. (i.e., 300 mg daily) have been effective in retarding, arresting or reversing cognitive deterioration and therefore in reducing the risk of later development of dementia. Most studies have employed phosphatidylserine that was extracted from bovine or porcine sources; however, phosphatidylserine of plant origin appears to be equally effective.²⁶⁴

Safety of Daily Intake of Supplemental Phosphatidylserine that is Effective in Reducing the Risk of Dementia

In addition to the absence of reports of adverse reactions in the published scientific literature concerning oral supplementation with phosphatidylserine, the safety of phosphatidylserine has been documented in detail by several investigators. A cohort of 130 elderly adults (65 men and 65 women) who received oral phosphatidylserine (100 mg, t.i.d.) for 60 days exhibited no changes in hematocrit, white blood cell count, blood urea nitrogen concentration, serum total cholesterol concentration, plasma SGOT, SGPT, alkaline phosphatase or CPK activities, or plasma fasting glucose, uric acid, creatinine, total bilirubin or total triglyceride concentrations.²⁷⁷ Similar groups of elderly individuals who received phosphatidylserine (100 mg, t.i.d.) for 60 days²⁶⁶ or 6 months⁵⁶ also exhibited no changes in blood chemistry analytes or in indicators of liver or kidney function and no adverse interactions between phosphatidylserine and concurrent diuretic, antithrombotic, hypoglycemic, antiarrhythmic, antihypertensive, anti-inflammatory, antacid, antiulcer, mucolytic, chemotherapeutic or calcium channel blocking medications. Young adults who received phosphatidylserine via intravenous injection exhibited no effects on blood pressure or heart rate.²⁶¹

The safety of the use of phosphatidylserine obtained from animal sources has come under criticism because of the possibility of enzootic transmission of viruses.²⁷⁸ A source of phosphatidylserine that is the result of extraction from plant sources exclusively has been reported to be without side effects and poses no risk as a vector of viral pathogens.²⁶⁴

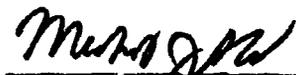
Conclusions

1. Reduced ability to perform complex executive functions through effortful or controlled mental processing ("cognitive decline") is not a mandatory component of human aging.
2. Evidence of cognitive decline reflects neuropathological changes in hippocampal and cerebral functioning.
3. In the absence of symptoms of dementia, the progression of cognitive decline to dementia is not universal.
4. An acceleration of cognitive decline is required in order for declining function to progress to dementia.
5. Cognitive decline in the absence of symptoms of dementia represents a modifiable risk factor for later development of dementia.
6. Oral phosphatidylserine supplementation is an effective modifier of cognitive decline and reduces the risk for dementia.

Summary Conclusion

In conclusion, I find that there is significant scientific agreement in support of the following health claims:

- The consumption of phosphatidylserine may reduce the risk of cognitive dysfunction in the elderly.
- The consumption of phosphatidylserine may reduce the risk of dementia in the elderly.



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A copy of my CV is attached.

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EDUCATION:

Ph.D., Animal Science - Nutrition 1979
Cornell University, Ithaca, New York

Bachelor of Science, Molecular Biology 1973
Massachusetts Institute of Technology, Cambridge, Massachusetts

PROFESSIONAL AND CAREER OBJECTIVES:

To contribute to the improvement of public health in the areas of nutrition and public health policy through an internationally recognized nutrition program

LICENSES, CERTIFICATIONS, HONORS:

Licensed Dietitian, State of Illinois 1995 to present
Certified Nutrition Specialist (C.N.S.) 1993 to present
Fellow, American College of Nutrition 1992 to present

EXPERIENCE:

Independent Consultant May 1998 to present
Senior Research Analyst, ECRI, Plymouth Meeting, PA 1997 to 1998
Senior Scientist, American Medical Association, Chicago, IL 1990 to 1997
Visiting Scientist/Research Assistant Professor
Northwestern University, Chicago, IL 1986 to present
Assistant Professor, University of Maryland, College Park, MD 1981 to 1986
Assistant Professor, Rutgers University, New Brunswick, NJ 1979 to 1981

Michael J. Glade, Ph.D.

Director and Nutritionist Adviser to the Board of Directors International College of Advanced Longevity Medicine	1998 to present
Member, Advisory Board Society for Integrative Medicine National Graves' Disease Foundation	1998 to present 1992 to 2001
Recorder Nutrition Sciences Education and Research Fund	1997 to present
Designated Representative of the C.B.N.S. Intersociety Physician Nutrition Education Consortium	1996 to present
Policy Paper Reviewer Council for Agricultural Science and Technology (CAST).	1996 to present
Lecturer Capital University of Integrative Medicine, Washington, DC New York Chiropractic College (Diplomate in Nutrition program) Northwestern University Medical School, Chicago, IL	1999 to present 1998 to present 1990 to present
Part-Time Faculty Biostatistics, University of Bridgeport, Bridgeport, Connecticut	1993 to present
Adjunct Faculty Union Institute, Cincinnati, Ohio	2000 to present
Book Review Editor <i>Nutrition: The International Journal of Applied and Basic Nutritional Sciences</i>	1992 to present
Manuscript Reviewer <i>The Journal of the American Medical Association, The Journal of the American College of Nutrition, Nutrition,</i> and other peer-reviewed journals	1980 to present
Council Coordinator American College of Nutrition	1994 to 1998
Certification Board for Nutrition Specialists Director Director of Educational Programs President Vice-President Editor, Certifying Examination, Certification Board for Nutrition Specialists Editor/Author <i>1996 Study Guide for the Certifying Examination for Certified Nutrition Specialists</i> <i>1996 Candidate's Guide for Licensure as a Nutrition Counselor, State of Illinois</i> <i>1999 Study Guide for the Certifying Examination for Certified Nutrition Specialists</i>	1999 to present 2001 to present 1996 to 1999 1992 to 1996 1992 to 2001 1996 1996 1999

Complete Nutrition Expertise

May 1998 to present

PO Box 6377
Evanston IL 60204-6377

- technical support
- educational/promotional materials
- seminars and symposia
- publications
- labeling
- regulatory affairs
- scientific product support
- policy development
- research protocol evaluation
- research design/implementation
- data analysis and interpretation
- product formulation

Product formulation and development projects have emphasized the rational combination of select vitamins, minerals, herbs, and phytonutrients and phytomedicines into formulas for individuals who are attempting to quit smoking or who are afflicted with alcoholism, caffeine dependency, colorectal cancer, breast cancer, cardiovascular disease, osteoporosis, arthritis or celiac disease. These projects have included the assembly of scientific substantiation for both product ingredients and product labeling.

Consulting Clinical Nutritionist
North Shore Wellness and Cosmetic Surgery
281 Waukegan Road, Northfield, IL 60093

September 1999 to present

Patient care in the areas of cancer management, restoration of intestinal function, diabetes, chronic fatigue, multiple sclerosis, mental illness, skeletal function, heart disease, chronic fatigue syndrome, fibromyalgia, morbid obesity, yeast infection and smoking cessation.

Nutritionist/Medical Advisor
Lake County Chapter, Celiac-Sprue Association

September 2000 to present

Other past consulting projects:

Identification and substantiation of structure/function statements for dietary supplements containing ginseng (prepared for a commercial client).

Substantiation of new health claims for dietary supplements and foods containing folic acid (prepared for a petition submitted to the FDA).

Substantiation of new health claims for dietary supplements and foods containing various nutrients (prepared for petitions submitted to the FDA).

Design of human trials to demonstrate the safety of a new dietary ingredient (prepared for a commercial client).

Preparation of a petition to FDA requesting approval to import a new dietary ingredient (prepared for a commercial client).

Comparison of scientific manuscripts in several copyright infringement cases.

Substantiation of structure/function statements made for several dietary supplements (prepared for commercial clients).

Data analysis for the development of normal reference intervals for a series of new diagnostic tests.

Scientific substantiation and validation of a survey instrument for the assessment of overall health.

Scientific substantiation of a dietary supplement formulation for the support of cognitive functions (prepared for a commercial client).

Evaluation of the safety and effectiveness of a dietary supplement formulation for the chelation of heavy metals (prepared for a commercial client).

Evaluation of the safety and effectiveness of a dietary supplement formulation for enlargement of the human female breast (prepared for a commercial client).

Evaluation of the safety and effectiveness of dietary supplement formulations for enhancement of weight loss (prepared for commercial clients).

Evaluation of the safety and effectiveness of dietary supplement formulations for enhancement of sexual function (prepared for a commercial client).

Evaluation of the safety and effectiveness of dietary supplement formulations for enhancement of immune function (prepared for a commercial client).

Evaluation of the safety and effectiveness of dietary supplement formulations for enhancement of sleep (prepared for a commercial client).

Presentations since May 1998:

Herbal management of diabetes. Natural Pharmacy East, Arlington, VA, October 1998.

Nutritional support for breaking nicotine addiction. International College for Advancement of Longevity Medicine Fall Symposium, Reno, NV, October, 1998.

Nutritional support for breaking nicotine addiction. Sixth International Congress of the American Academy of Anti-Aging Medicine, Las Vegas, NV, December, 1998.

Nutritional support for breaking nicotine addiction: A randomized, double-blind, placebo-controlled evaluation of a proprietary dietary supplement. American College of Nutrition Annual Symposium, Washington, DC, October, 1999

Efficacy of an enzyme product derived from *Aspergillus niger* and bromelain (AbsorbAid™) in improving protein absorption in nursing home patients on tube feeding. American College of Nutrition Annual Symposium, Las Vegas, NV, October, 2000.

Preventing cancer with nutrition. Prevention Plus Seminar Series, NSA, Inc., Oak Park, IL, October, 2000.

Celiac disease. Healthy Eating Seminar Series, Lake County Chapter, Celiac-Sprue Association, Waukegan, IL, October, 2000.

Gluten sensitivity and other digestive disorders. Healthy Eating Seminar Series, Lake County Chapter, Celiac-Sprue Association, Deerfield, IL, January, 2001.

Digestive disease; celiac disease; digestive ecology; using diagnostic technology to target trace elements and vitamin therapy. American Naprapathic Association, Countryside, IL, April 22, 2001.

Restoration of digestive ecology. Designs for Health – Advanced Training in Clinical Nutrition, Designs for Health Institute, Boulder, CO, June 30, 2001.

The relationship between digestive tract function and autism. In-service training, Pfeiffer Foundation, Naperville, IL, July 2001.

Michael J. Glade, Ph.D.

Upcoming Presentations:

Nutrition and brain function. American Naprapathic Association, Countryside, IL, April 7, 2002.

Applied genomics, Genovations, Oak Park, IL, April 27, 2002.

Review course in preparation for the certifying examination of the Certification Board for Nutrition Specialists, Ft. Lauderdale, FL, May 13-14, 2002.

Teaching Lecture Topics since May 1998:

Environmental medicine and detoxification therapy.

Carbohydrate nutrition and nutritional therapy.

Protein nutrition and nutritional therapy.

Nutritional and herbal management of diabetes.

Nutritional therapeutics in cancer.

Nutrition and cancer prevention for consumers.

Celiac disease and its prevention and treatment.

Free radical and antioxidant biology.

Biostatistics for nutritionists (I designed and am teaching course over the internet)

Michael J. Glade, Ph.D.

ECRI

5200 Butler Pike, Plymouth Meeting, PA 19462

August 1997 to May 1998

SENIOR RESEARCH ANALYST
Technology Assessment

Evaluation of medical, nutritional, and technological therapies and diagnostic techniques for human endocrine, metabolic, musculoskeletal, and nutritional diseases.

Quality Assurance Manager, National Guidelines Clearinghouse (with AHCPH)

Participant in database design, National Guidelines Clearinghouse (with AHCPH)

Statistical expert, diagnostic technologies and meta-analysis

Provide in-house expertise to ECRI Management on food, device, drug, agriculture and nutrition-related health, policy, legal, and regulatory matters.

SUPERVISOR: Charles Turkelson, Ph.D.
Chief Research Analyst
Technology Assessment
ECRI

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1993 to 1997

SENIOR SCIENTIST, Technology Assessment & Nutrition Department of Technology Assessment

Evaluation of medical, nutritional, and technological therapies and diagnostic techniques for human endocrine, metabolic, musculoskeletal, and nutritional diseases.

Development of Technology Assessments for the AMA *Diagnostic and Therapeutic Technology Assessment (DATTA)* project:

Diagnostic Value of Plasma Lp(a) Concentrations

Diagnostic Value of Plasma Apolipoproteins

Diagnostic Value of Serum Thyroid-Stimulating Hormone (TSH)

Diagnostic Value of Computerized Dynamic Posturography

Diagnostic Value of 24-hour Esophageal pH Monitoring

Therapeutic Value of Peripheral Parenteral Nutrition

Therapeutic Value of Intraoperative Radiotherapy

Therapeutic Value of Speech Therapy in Otitis Media

Therapeutic Value of Recombinant Human Growth Hormone (rhGH) in Children with Short Stature

Therapeutic Value of Mononuclear Leukocyte ("Buffy Coat") Infusions in Chronic Myelocytic Leukemia

Therapeutic Value of Medicinal Leeches

Therapeutic Value of Pedicle Screw Spinal Fixation Systems

Therapeutic Value of Recombinant Human Growth Hormone (rhGH) in Children with Gonadal Dysgenesis

Related Duties:

Statistician; perform statistical analyses for all physician surveys administered by the *DATTA* project.

Co-Editor of the monthly AMA newsletter, *Technology News*.

Provide in-house expertise to AMA Senior Management on food, device, drug, agriculture and nutrition-related health, policy, legal, and regulatory matters.

Secretary, AMA House of Delegates Reference Committee E (advise AMA policy committees on medicine, nutrition, and public health).

Michael J. Glade, Ph.D.

Publications:

Published In:	No. of Publications:
<i>DATTA</i> Assessments:	13
peer-reviewed journals:	4
Proceedings chapters:	4
book reviews:	11
general public press:	16
peer-reviewed journals (submitted):	5

Original articles published in the monthly AMA newsletter, *Technology News*:

Risk Assessment in the Establishment of Upper Safe Limits for Nutrient Intakes	12/96
Dietary Fat and Cancer: Molecular Mechanisms	10/96
Clinical Significance of Melatonin (with B. Kendler)	9/96
Designing, Testing, and Labeling Reusable Medical Devices for Reprocessing in Health Care Facilities	6/96
Dietary Phytochemicals in Cancer Prevention and Treatment	11/95
Electromagnetic Compatibility for Medical Devices: Issues and Solutions	9/95
FDA/NIH-Sponsored Conference: Comparing Treatments: Safety, Effectiveness, and Cost-Effectiveness	5/95
Clinical Significance of Oxidative Stress (with B. Kendler)	11/95
Diet and Cancer: Molecular Mechanisms of Interactions	1-2/95
Management of Disorders of Cholesterol, Triglyceride, and Lipoprotein Metabolism	11/94
AMA Annual Meeting Update (with S. Kalousdian)	7-8/94
Drug and Device-Induced Disease: Developing a Blueprint for the Future	/94
AMA Interim Meeting Update (with S. Kalousdian)	1-2/94
AMA Annual Meeting Update (with S. Kalousdian)	8/93
Breast Cancer Risk and Diet	1/93

Author of AMA policy statements on nutrition issues:

- food irradiation;
- lipoproteinemia;
- bacterial contamination of meat;
- dietary calcium requirements;
- folic acid supplementation to prevent neural tube defects;
- thiamin supplementation of alcoholic beverages to prevent polyneuropathy;
- neonatal hyponatremia from hypo-osmolar bottled water

Michael J. Glade, Ph.D.

Speaking Invitations:

The Dietary Supplement and Health Education Act of 1994. Annual Meeting of the American College of Nutrition, Washington, DC, October, 1995.

Innovation in clinical nutrition. Harvard University, May 6, 1995.

Environmental medicine. New York Chiropractic College, April 29, 1995.

Environmental medicine. New York Chiropractic College, September 11, 1994.

Additional Responsibilities:

Meeting with representatives of the Food and Drug Administration, the US Department of Agriculture, and other federal agencies concerning:

food, device and drug regulation;

food safety;

direct to consumer advertising of medical therapies.

Collaboration with other AMA staff in the development of scripts for television programs aired on American Medical Television.

Represented AMA on "National Educational Forum on Food Safety Issues."

Book Review Editor, *Nutrition: The International Journal of Applied and Basic Nutritional Sciences*.

Reviewed manuscripts submitted to *the Journal of the American Medical Association*, *the Journal of the American College of Nutrition*, and other peer-reviewed journals.

Reviewed advertisements intended for use in AMA publications.

Policy paper reviewer for the Council for Agricultural Science and Technology (CAST).

Invitations to Chair National Meetings:

Invited to chair and organize a session on "Nutritional Controversies" at the 1996 Annual Meeting of the American College of Nutrition, San Francisco.

Invited to serve as co-chairman of a session of the 1994 Malnutrition and AIDS Symposium, Los Angeles.

Invited to serve as co-chairman of a session of the 1994 Annual Meeting of the American College of Nutrition, Atlanta.

SUPERVISOR: Sona Kalousdian, MD, MPH
Department Director, Department of Technology Assessment
American Medical Association
(773) 384-4915

Michael J. Glade, Ph.D.

AMERICAN MEDICAL ASSOCIATION

515 N. State St. Chicago, IL 60610

1990 to 1993

SENIOR SCIENTIST, Endocrinology, Metabolism & Nutrition Department of Drugs

Evaluation of medical and nutritional therapies and diagnostic techniques for human endocrine, metabolic, musculoskeletal, and nutritional diseases.

Extensive revision of chapters in the Congressionally-recognized compendium of FDA-approved unlabeled drug use and nutritional therapy, *AMA Drug Evaluations*:

Fluid, Electrolyte, and Acid-Base Therapy (pp. 865-880*)

Drugs Used for Urolithiasis (pp. 907-924)

Drugs Used in Adrenocortical Dysfunction (pp. 1017-1036)

Drugs Used in Thyroid Disease (pp. 1037-1062)

Vitamins and Minerals (pp. 2283-2306)

Parenteral and Enteral Nutrition (pp. 2307-2362)

Drugs Used in Obesity (pp. 2439-2454)

Treatment of Disorders of Cholesterol and Lipoprotein Metabolism (pp. 2455-2500)

(* page numbers as in the 1995 edition)

Assistant Secretary, AMA House of Delegates Reference Committee E (advise AMA policy committees during development of policies concerning medicine, nutrition, and public health).

Collaboration with other AMA staff in the development of scripts for television programs aired on American Medical Television

Publications:

Published In:	No. of Publications:
<i>AMA Drug Evaluations</i> Chapters:	8
peer-reviewed journals:	12
Proceedings chapters:	6
book reviews:	1
general public press:	6

Speaking Invitations:

A review of hormonal regulation of cartilage growth in foals. Symposium on Equine Osteochondrosis, Cambridge University, United Kingdom, September, 1992.

Marginal copper deficiency as a cause of defective angiogenesis in chondrodysplasia. Symposium on Equine Osteochondrosis, Cambridge University, United Kingdom, September, 1992.

Michael J. Glade, Ph.D.

Endocrine regulation of equine growth plate chondrocytes. Symposium on Equine Osteochondrosis, Cambridge University, United Kingdom, September, 1992.

Equine osteochondrosis as a manifestation of induced episodic "pseudohypothyroidism." Symposium on Equine Osteochondrosis, Cambridge University, United Kingdom, September, 1992.

Insulin and thyroid hormones influence matrix production by chondrocytes. Seminars in Endocrinology, Northwestern University, Chicago, IL, April 2, 1991.

Additional Responsibilities:

Meetings with representatives of the Food and Drug Administration, the US Department of Agriculture, and other federal agencies concerning:

food, device and drug regulation;

food safety;

direct to consumer advertising of medical therapies

Collaboration with Centers for Disease Control in development of recommendations concerning folic acid and the prevention of neural tube defects (*Morbidity and Mortality Weekly*, August 2, 1991, and September 21, 1992).

Author of AMA policy statement on monosodium glutamate.

Provide in-house expertise to AMA Senior Management on food, device, drug, agriculture and nutrition-related health, policy, legal, and regulatory matters.

Represented AMA on "National Educational Forum on Food Safety Issues".

Book Review Editor, *Nutrition: The International Journal of Applied and Basic Nutritional Sciences*.

Review manuscripts submitted to *the Journal of the American Medical Association*, *the Journal of the American College of Nutrition*, and other peer-reviewed journals.

Review advertisements intended for use in AMA publications.

Coordinator, Council on Endocrinology, Bone, and Minerals; American College of Nutrition.

Advisory Board Member, National Graves' Disease Foundation

SUPERVISOR: Joseph Cranston, Ph.D.
Department Director
Department of Drugs
American Medical Association

Michael J. Glade, Ph.D.

NORTHWESTERN UNIVERSITY

303 E. Chicago Avenue, Chicago, IL 60610

1986 to 1990

RESEARCH ASSISTANT PROFESSOR Department of Pharmacology

Funded originally as an NIH Senior Fellowship, this position - including both research and teaching - has been continued on a part-time, unpaid basis through the present time as a Visiting Scientist, Department of Molecular Pharmacology and Biological Chemistry

Laboratory and field research; presentation and publication of research findings; fund raising; maintenance of laboratory; practice of safe and proper animal housing and handling; practice of safe handling of hazardous substances.

Concentration on the effects of nutrients, hormones and growth factors on skeletal development and disease.

Guest lectures on pancreatic and thyroid disease and their prevention and medical and nutritional management.

Publications:

Published In:	No. of Publications:
peer-reviewed journals:	11
Proceedings chapters:	8
abstracts:	4
general public press:	98

Speaking Invitations:

Response of arthritic chondrocytes to polysulfated glycosaminoglycans. Skeletal Biology Program, Case Western Reserve University, Cleveland OH, May 14, 1990.

Flora and fauna of Africa and Europe. Department of Pharmacology, Northwestern University, Chicago, IL, February 9, 1989.

Influences of diet and endocrinology on equine developmental orthopedic disease. Department of Animal Sciences, University of Guelph, Ontario, Canada, January 18, 1989.

Diet and growth quality. Equine management class, University of Guelph, Ontario, Canada, January 18, 1989.

Fermentation enhancers. Department of Animal Sciences, University of Guelph, Ontario, Canada, January 17, 1989.

Nitrogen metabolism in the equine. Equine management class, University of Guelph, Ontario, Canada, January 16, 1989.

Michael J. Glade, Ph.D.

Feeding and management of pleasure and show horses. Potomac Horse Club, Silver Spring, MD, October, 1988.

Feeding and management of pleasure and show horses. Potomac Horse Club, Silver Spring, MD, October, 1988.

Homeorrhexis and the growing animal. Biological Sciences Seminar, University College, Dublin, Ireland, October 17, 1988.

Nutrition and developmental disorders of equidae. Department of Zoology, University College, Dublin, Ireland, October 17, 1988.

Nitrogen metabolism in horses. Veterinary College of Ireland, Dublin, Ireland, October 14, 1988.

The role of yeast culture in the nutritional management of young horses. 100th Irish Veterinary Congress, Dublin, Ireland, September 23, 1988.

The role of endocrine factors in equine developmental orthopedic disease. Developmental Orthopedic Disease Panel, American Association of Equine Practitioners Annual Meeting, New Orleans, LA, November 29, 1987.

Diet, chondrodysplasias and animals. Oral Biology Seminar, Northwestern University, Chicago, IL, October 29, 1987.

Effects of yeast culture on nitrogen metabolism in young horses. Alltech Biotechnology Symposium, Lexington, KY, April, 1987.

Bibliometric analysis of research activity in Brazil. Central Intelligence Agency, MacClean, VA, March, 1987.

Bibliometric analysis of research activity in Spain. Ministry of Science and Education, Madrid, Spain, March, 1987.

Cartilage disorders associated with changes in thyroid hormone metabolism. The Chicago Endocrine Society, Chicago, IL, December, 1986.

Dietary causes of osteochondrosis. Pathology Seminar, Northwestern University, Chicago, IL, April, 1986.

Michael J. Glade, Ph.D.

UNIVERSITY OF MARYLAND

College Park, Maryland

1981 to 1986

**ASSISTANT PROFESSOR, Department of Animal Sciences
College of Agricultural Sciences**

Teaching: (Class, laboratory, barn; lecture, hands-on formats)

Animal Husbandry (nutrition, diet formulation, diseases, management, genetics, physiology, functional morphology)

Animal Training (including principles of animal behavior and their application to training)

Safe Animal Handling (including principles of animal behavior and their application to safe practices in handling animals)

Protein Nutrition (graduate course)

Training:

How to Teach and Supervise Animal Training (undergraduate and graduate students; written materials; videotapes)

Laboratory Techniques (undergraduate and graduate students)

Field Research Techniques (undergraduates and graduates)

Dissertation and Scientific Writing

Grant Proposal Preparation

Research:

Animal Nutrition and Physiology Projects, including several in collaboration with the National Zoo, Washington, DC

Publications:

Published In:	No. of Publications:
peer-reviewed journals:	17
Proceedings chapters:	8
abstracts:	8
general public press:	73

Other projects: (in addition to those documented in publications)

hormone secretion rates in pigs

skeletal growth in monkeys

pharmacokinetics of ivermectin in bullfrogs

growth hormone concentrations in horses and zebras

Michael J. Glade, Ph.D.

Invitation to Chair National Meeting:

Invited to serve as co-chairman of a Non-Ruminant Nutrition session at the 1982 meeting of the American Society of Animal Science, Guelph, Ontario, Canada.

Speaking Invitations:

Quality feed management: tips for proper production and storage. Baltimore Horse Seminar, March, 1985.

Dietary carbohydrate induction of the multiple-messenger, inositol-calmodulin pathway. Animal Sciences Seminar, University of Maryland, February, 1985.

The use of ultrasound to monitor neonatal bone development. Invited seminar, Walter Reed Medical Center, Washington, DC, December, 1984.

Mechanisms of dietary induction of osteochondrosis. Invited seminar, Department of Animal Science, University of Alberta, Edmonton, Canada, August, 1984.

The Use of Self-Supervised Activity to Acquaint College Students with the Teacher-Student Dynamic. 10th International Conference, Improving University Teaching, College Park, MD, July, 1984.

Diagnostic ultrasound - a non-invasive method for examining bone. Pediatric Research Conference, University of Maryland School of Medicine, May, 1984.

Electrical stimulation of bone healing. Alice Deal Science Day, May, 1984.

Non-Traditional feeding practices for the performance horse. Maryland Nutrition Conference, Baltimore, MD, March, 1984.

The use of ultrasound. Nutritional Sciences Colloquium, University of Maryland, February, 1984.

Nutrient-hormone interactions and their impact on growth. Nutritional Sciences Colloquium, University of Maryland, February, 1984.

Feeding horses for a lot less money. Eastern Amateur Arabian Horse Show Circuit Fall Meeting, December, 1983.

Equine nutritional requirements. Baltimore Horse Seminar, November, 1983.

The costs of owning a horse, Maryland Society for the Prevention of Cruelty to Animals Field Day, May, 1983.

Ultrasonic measurement of bone strength. Alice Deal Science Day, April, 1983.

Nutritional manipulation of bone and joint development in growing horses. Maryland Nutrition Conference, Washington, DC, March, 1982.

Developmental origins of growth abnormalities. Animal Sciences Seminar, University of Maryland, October, 1981.

Michael J. Glade, Ph.D.

Additional Responsibilities:

Design of Animal Habitats:

Personally redesigned three multi-acre animal housing facilities, and assisted in their physical renovation

Animal Care:

Collaboration with veterinarians in prophylactic and interventive medical care, including personally:

- administering medications by mouth
- injection (intramuscular; intravenous)
- nasogastric intubation; rectal gavage
- bandaging; suturing
- development of growth plate biopsy procedure for ungulates
- necropsy

Animal Management:

Directly responsible for the management, breeding, and training of up to 120 horses residing at multi-building and multi-site facilities whose activities encompassed teaching, research, breeding, continuing adult education, veterinary care, demonstrations

Supervision of Personnel:

Supervision of up to two dozen permanent and temporary full and part time employees and volunteers engaged in animal husbandry

Record Keeping; Budgets:

Directly responsible for planning, developing, administering, and adhering to expense and revenue budgets, and for extensive and comprehensive record-keeping concerning all facets of a major university equine program

Fund-Raising:

Obtaining funds to support all programs and activities

Sources included federal agencies, state agencies, private foundations, private individuals, corporate entities, animal sales, animal rental

Michael J. Glade, Ph.D.

RUTGERS UNIVERSITY

New Brunswick, NJ

1979 to 1981

ASSISTANT PROFESSOR, Department of Animal Sciences

Teaching: (Class, laboratory, barn; lecture, hands-on formats):

Animal Husbandry (nutrition, diet formulation, diseases, management, genetics, physiology, functional morphology)

Animal Training (including principles of animal behavior and their application to training)

Safe Animal Handling (including principles of animal behavior and their application to safe practices in handling animals)

Training:

Field Research Techniques (undergraduates and graduates)

Grant Proposal Preparation

Research:

Animal Nutrition and Physiology Projects

Publications:

Published In:	No. of Publications:
Proceedings chapters	1
abstracts	1

Speaking Invitations:

Digestive physiology of the horse. Animal Sciences Seminar, University of Maryland, September, 1980.

Similarities between effects of dexamethasone on growing cartilage and osteochondrosis dissecans. Animal Science Seminar, University of California at Davis, April, 1980.

Osteochondrosis dissecans and growth suppression in dexamethasone treated horse foals. American Association of Equine Practitioners Annual Meeting, Miami Beach, December, 1979.

Effects of dexamethasone on calcium metabolism of pony foals. Animal Sciences Seminar, Rutgers University, May, 1979.

Michael J. Glade, Ph.D.

Additional Responsibilities:

Design of Animal Habitats:

Personally redesigned a multi-acre animal housing facility, and assisted in its physical renovation

Animal Care:

Collaboration with veterinarians in prophylactic and interventive medical care, including personally:

- administering medications by mouth
- injection (intramuscular; intravenous)
- nasogastric intubation; rectal gavage
- bandaging; suturing; necropsy

Animal Management:

Directly responsible for the management, breeding, and training of up to 11 horses residing at multi-building and multi-site facilities whose activities encompassed teaching, research, continuing adult education, veterinary care, demonstrations

Supervision of Personnel:

Directly responsible for the supervision of two permanent part time employees and a dozen or so volunteers engaged in animal husbandry

Record Keeping; Budgets:

Directly responsible for planning, developing, administering, and adhering to expense and revenue budgets, and for extensive and comprehensive record-keeping concerning all facets of a major university equine program

Fund-Raising:

Obtaining funds to support all programs and activities

Sources included federal agencies, state agencies, private foundations, private individuals, corporate entities, animal sales, animal rental

Refereed Journal Articles:

1. Glade, M.J. Nutritional support for breaking nicotine addiction: A randomized, double-blind, placebo-controlled evaluation of a proprietary dietary supplement as an aid to smoking cessation. *Journal of Alternative and Complementary medicine*: submitted for publication.
2. Glade, M.J. Polysulfated glycosaminoglycan stimulation of macromolecule synthesis and inhibition of macromolecule degradation by primary cultures of rabbit articular chondrocytes. *Journal of Orthopedic Research*: submitted for publication.
3. Glade, M.J. The effects of gestation, lactation, yeast culture and maternal calcium intake on the mechanical strength of equine bone. *Journal of Equine Veterinary Science*: submitted for publication.
4. Heimbürger, D.C., and the Intersociety Professional Nutrition Education Consortium. 2000. Physician-nutrition-specialist track: If we build it, will they come? *American Journal of Clinical Nutrition* 71:1048-1053.
5. Glade, M.J. 1997. Intake of dietary calcium to reduce the incidence of osteoporosis. *Archives of Family Medicine* 6:491-494.
6. Glade, M.J. 1995. Management of disorders of cholesterol, triglyceride, and lipoprotein metabolism. *Archives of Family Medicine* 4:869-878.
7. Glade, M.J. 1995. Continuous ambulatory esophageal pH monitoring. *Journal of the American Medical Association* 274:662-668.
8. Glade, M.J., Y.S. Kanwar and P.H. Stern. 1994. Insulin and thyroid hormones alter chondrocyte metabolism in cell culture independently and in combination. *Connective Tissue Research* 31:37-44.
9. Glade, M.J. 1993. The effects of gestation, lactation, and maternal calcium intake on the mechanical strength of equine bone. *Journal of the American College of Nutrition* 12:372-377.
10. Glade, M.J. 1992. Effects of *Yucca shidigera* extract on feed utilization by equine weanlings. *Journal of Equine Veterinary Science* 12:93-98.
11. Letcher, J. and M.J. Glade. 1992. Efficacy of ivermectin as an anthelmintic in leopard frogs. *Journal of the American Veterinary Medical Association* 200:537-538.
12. Glade, M.J., Y.S. Kanwar and T.J. Hefley. 1991. Enzymatic isolation of chondrocytes from immature rabbit articular cartilage and their maintenance of phenotypic expression in culture. *Journal of Bone and Mineral Research* 6:217-226.
13. Glade, M.J. 1991. Timed administration of leucine, isoleucine, valine, glutamine, and carnitine to enhance athletic performance. *Equine Athlete* 4:1-10.
14. Glade, M.J. 1991. Effects of dietary yeast culture supplementation of lactating mares on the digestibility and retention of the nutrients delivered to nursing foals via milk. *Journal of Equine Veterinary Science* 11:323-329.

Michael J. Glade, Ph.D.

15. Glade, M.J. 1991. Dietary yeast culture supplementation of mares during late gestation and early lactation. 3. Effects on mare and foal plasma metabolite, amino acid and endocrine profiles. *Journal of Equine Veterinary Science* 11:167-175.
16. Glade, M.J. 1991. Dietary yeast culture supplementation of mares during late gestation and early lactation. 2. Effects on milk production, milk composition, weight gain and linear growth of nursing foals. *Journal of Equine Veterinary Science* 11:89-95.
17. Glade, M.J. 1991. Dietary yeast culture supplementation of mares during late gestation and early lactation. 1. Effects on dietary nutrient digestibilities and fecal nitrogen partitioning. *Journal of Equine Veterinary Science* 11:10-16.
18. Glade, M.J. and M.D. Sist. 1990. Supplemental yeast culture alters the plasma amino acid profiles of nursing and weanling horses. *Journal of Equine Veterinary Science* 10:369-379.
19. Glade, M.J. and N.K. Luba. 1990. Benefits to foals of feeding soybean meal to lactating broodmares. *Journal of Equine Veterinary Science* 10:422-428.
20. Glade, M.J. and M. Campbell-Taylor. 1990. Effects of dietary yeast culture supplementation during the conditioning period on equine exercise physiology. *Journal of Equine Veterinary Science* 10:434-443.
21. Glade, M.J. 1990. Polysulfated glycosaminoglycan (PSGAG) accelerates the synthesis of collagen and glycosaminoglycans by arthritic equine cartilage tissues and chondrocytes. *American Journal of Veterinary Research* 51:779-785.
22. Sist, M.D., Youngblood, M.A., Williams, J.F. and Glade, M.J. 1988. Salivary and serum estrone sulfate levels in pregnant mares. *Journal of Equine Veterinary Science* 8: 164-167.
23. Glade, M.J. and M.D. Sist. 1988. Dietary yeast culture supplementation enhances urea recycling in the equine large intestine. *Nutrition Reports International* 37: 11-19.
24. Wright, L.L., M.J. Glade and J. Gopal. 1987. The use of transmission ultrasonics to assess bone status in the human newborn. *Pediatrics Research* 22:541-544.
25. Glade, M.J. and N.K. Luba. 1987. Serum triiodothyronine and thyroxine concentrations in weanling horses fed carbohydrate by direct gastric infusion. *American Journal of Veterinary Research* 48:578-582.
26. Glade, M.J., N.K. Luba, and H.F. Schryver. 1986. Effects of age and diet on the development of mechanical strength by the cannon bones of young horses. *Journal of Animal Science* 63:1432-1444.
27. Glade, M.J. and L.M. Biesik. 1986. Changes in serum hormone concentrations in weanling horses following gastric infusion of sucrose or casein. *Nutrition Reports International* 33:651-659.
28. Glade, M.J. and L.M. Biesik. 1986. Enhanced nitrogen retention in yearling horses supplemented with yeast culture. *Journal of Animal Science* 62:1633-1640.

Michael J. Glade, Ph.D.

29. Glade, M.J. 1986. Estimation of urine flow rate in weanling and yearling horses. *American Journal of Veterinary Research* 47:2151-2156.
30. Glade, M.J. and T.H. Belling. 1986. A dietary etiology for osteochondrotic cartilage. *Journal of Equine Veterinary Science* 6:151-154.
31. Glade, M.J. 1986. The control of cartilage growth in osteochondrosis. *Journal of Equine Veterinary Science* 6:175-187.
32. Glade, M.J. 1986. "Social Sleeping" among confined horses. *Journal of Equine Veterinary Science* 6:155-157.
33. Glade, M.J. and R.A. Salzman. 1985. Effects of hoof angulation on hoof growth and contraction in the horse. *Journal of Equine Veterinary Science* 5:45-50.
34. Glade, M.J. and T.J. Reimers. 1985. Effects of dietary energy supply on serum thyroxine, tri-iodothyronine and insulin concentrations in young horses. *Journal of Endocrinology* 104:93-98.
35. Glade, M.J., D. Beller, J. Bergen, D. Berry, E. Blonder, J. Bradley, M. Cupelo and J. Dallas. 1985. Dietary protein in excess of requirements inhibits renal calcium and phosphorus reabsorption in young horses. *Nutrition Reports International* 31:649-659.
36. Glade, M.J. 1985. Stimulation of electromagnetic osteogenesis in healthy growing yearlings. *Journal of Equine Veterinary Science* 5:149-153.
37. Glade, M.J. 1985. Overfeeding energy to horses. *Journal of Equine Veterinary Science* 5:95.
38. Glade, M.J., S. Gupta and T.J. Reimers. 1984. Hormonal responses to high and low planes of nutrition in weanling Thoroughbreds. *Journal of Animal Science* 59:658-665.
39. Glade, M.J. and T.H. Belling. 1984. Growth plate cartilage metabolism, morphology and biochemical composition in over- and underfed horses. *Growth* 48:473-482.
40. Glade, M.J. 1984. Feeding innovations for the performance horse. *Journal of Equine Veterinary Science* 4:165-168.
41. Glade, M.J. 1984. "Social sleeping" behavior in young horses. *Equine Practice* 6:10-14.
42. Glade, M.J. 1984. The influence of dietary fiber digestibility on the nitrogen requirements of mature horses. *Journal of Animal Science* 58:638-646.
43. Belling, T.H. and M.J. Glade. 1984. A non-destructive biopsy method allowing the rapid removal of live growth plate cartilage. *Veterinary Medicine/Small Animal Clinician* 79:528-531.
44. Glade, M.J. 1983. Nitrogen partitioning along the equine digestive tract. *Journal of Animal Science* 57:943-953.
45. Glade, M.J. 1983. Nutrition and performance of racing Thoroughbreds. *Equine Veterinary Journal* 15:31-36.

Michael J. Glade, Ph.D.

46. Glade, M.J., L. Krook, H.F. Schryver and H.F. Hintz. 1982. Morphologic and biochemical changes in cartilage of foals treated with dexamethasone. *Cornell Veterinarian* 73:170-192.
47. Glade, M.J., L. Krook, H.F. Schryver and H.F. Hintz. 1982. Calcium metabolism in glucocorticoid-treated foals. *Journal of Nutrition* 112:67-76.
48. Glade, M.J. and L. Krook. 1982. Glucocorticoid-induced inhibition of osteolysis and the development of osteopetrosis, osteonecrosis and osteoporosis. *Cornell Veterinarian* 72:76-91.
49. Glade, M.J., L. Krook, H.F. Schryver and H.F. Hintz. 1981. Growth inhibition induced by chronic dexamethasone treatment of foals. *Journal of Equine Veterinary Science* 1:198-201.
50. Matteo, C.M., M.J. Glade, A. Tanaka, J. Piret and A.L. Demain. 1975. Microbiological studies on the formation of gramicidin S synthetases. *Biotechnology and Bioengineering* 17:129-142.

Abstracts and Proceedings:

1. Glade, M.J., Kendra, D., Kaminsky, M.V., Jr. 2000. Efficacy of an enzyme product derived from *Aspergillus niger* and bromelain (AbsorbAid™) in improving protein absorption in nursing home patients on tube feeding. *Proceedings, Annual Meeting of the American College of Nutrition*, Las Vegas, NV, October.
2. Heimburger, D., and IPNEC. 2000. Training the Physician Nutrition Specialist (PNS). *Proceedings, Annual Meeting of the American College of Nutrition*, Las Vegas, NV, October.
3. Glade, M.J. 1998. Nutritional support for breaking nicotine addiction. *Proceedings, Sixth International Congress on Anti-Aging and Biomedical Technologies* (American Academy of Anti-Aging Medicine), Las Vegas, NV, December, p. unpagged.
4. Glade, M.J. 1998. Nutritional support for breaking nicotine addiction. *Proceedings, International College for Advancement of Longevity Medicine Fall Symposium*, Reno, NV, October, unpagged.
5. Glade, M.J. 1998. Herbal management of diabetes. *Proceedings, Second Annual Natural Pharmacy East Conference*, Arlington, VA, October, unpagged.
6. Glade, M.J., and M.E. Allen. 1996. Assessment of skeletal development in leopard geckos. II. Long bone morphometry and breaking strength. *Proceedings, Ninth Dr. Scholl Nutrition Conference*, Chicago, IL, October, unpagged.
7. Glade, M.J. 1995. The Dietary Supplement and Health Education Act of 1994. *Proceedings, Annual Meeting of the American College of Nutrition*, Washington, DC, October, p. 557.
8. Glade, M.J. 1993. CuSO₄ and chelated copper are bioequivalent when added to the diets of nursing foals. *Proceedings, Annual Meeting of the American College of Nutrition*, Chicago, October, p. 589.
9. Glade, M.J. 1993. CuSO₄ and chelated copper are bioequivalent when added to the culture medium of cartilage tissue and cells. *Proceedings, Annual Meeting of the American College of Nutrition*, Chicago, October, p. 589.
10. Glade, M.J. 1992. Equine osteochondrosis as a manifestation of induced episodic "pseudohypothyroidism." *Proceedings, Symposium on Equine Osteochondrosis*, Cambridge University, United Kingdom, September, p. 44.
11. Glade, M.J. 1992. Endocrine regulation of equine growth plate chondrocytes. *Proceedings, Symposium on Equine Osteochondrosis*, Cambridge University, United Kingdom, September, pp. 42-43.
12. Glade, M.J. 1992. Marginal copper deficiency as a cause of defective angiogenesis in chondrodysplasia. *Proceedings, Symposium on Equine Osteochondrosis*, Cambridge University, United Kingdom, September, pp. 30-31.

Michael J. Glade, Ph.D.

13. Glade, M.J. 1992. A review of hormonal regulation of cartilage growth in foals. *Proceedings, Symposium on Equine Osteochondrosis*, Cambridge University, United Kingdom, September, pp. 19-20.
14. Glade, M.J. 1992. The effects of gestation, lactation, and maternal calcium intake on the mechanical strength of equine bone. *Proceedings, Annual Meeting of the American College of Nutrition*, San Diego, October, p. 600.
15. Glade, M.J. 1992. Marginal copper deficiency as a cause of defective angiogenesis in chondrodysplasia. *Proceedings, Annual Meeting of the American College of Nutrition*, San Diego, October, p. 600.
16. Glade, M.J., C. Cahill and M. Campbell. 1989. Effect of exercise on plasma growth hormone concentrations in foals. *Proceedings, Equine Nutrition and Physiology Society*, pp. 63-64.
17. Glade, M.J. 1989. Effects of specific amino acid supplementation on lactic acid production by horses exercised on a treadmill. *Proceedings, Equine Nutrition and Physiology Society*, pp. 244-251.
18. Glade, M.J. 1989. Undergraduates and publishable equine research. *Proceedings, Equine Nutrition and Physiology Society*, pp. 233-235.
19. Glade, M.J. 1989. Supplemental yeast culture alters the plasma amino acid profiles of weanling Quarter horses. *Proceedings, Equine Nutrition and Physiology Society*, pp. 119-123.
20. Campbell, M. and M.J. Glade. 1989. Effects of dietary yeast culture supplementation during the conditioning period on heart rates and lactic acid production by horses exercised on a treadmill. *Proceedings, Equine Nutrition and Physiology Society*, pp. 72-78.
21. Glade, M.J. and P.H. Stern. 1988. Effect of polysulfated glycosaminoglycan (PSGAG) on monolayer cultures of articular chondrocytes. *Journal of Bone and Mineral Research*: 3: Suppl. 1:465.
22. Glade, M.J. 1988. The role of endocrine factors in equine developmental orthopedic disease. *American Association of Equine Practitioners* 33:171-189.
23. Wright, L.L., M.J. Glade and J. Gopal. 1987. Transmission ultrasonics to assess bone status in the human newborn. *Pediatrics Research*: 21:440A.
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