

# *Parkinson's Patients Support Groups, Inc.*

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## **UCLA Researchers Discover Link Between Parkinson's and Narcolepsy**

Parkinson's disease is well-known for its progression of motor disorders: stiffness, slowness, tremors, difficulties walking and talking. Less well known is that Parkinson's shares other symptoms with narcolepsy, a sleep disorder characterized by sudden and uncontrollable episodes of deep sleep, severe fatigue and general sleep disorder.

Now a team of UCLA and Veterans Affairs researchers think they know why — the two disorders share something in common: Parkinson's disease patients have severe damage to the same small group of neurons whose loss causes narcolepsy. The findings suggest a different clinical course of treatment for people suffering with Parkinson's that may ameliorate their sleep symptoms.

In their report (currently online) in the June issue of the journal *Brain*, Jerry Siegel, professor of psychiatry and biobehavioral sciences at the Semel Institute for Neuroscience and Human Behavior at UCLA, assistant resident neurobiologist Thomas C. Thannickal and associate research physiologist Yuan-Yang Lai have determined that Parkinson's disease patients have a loss of up to 60 percent of brain cells containing the peptide hypocretin. In 2000, this same group of UCLA researchers first identified the cause of narcolepsy as a loss of hypocretin, thought to be important in regulating the sleep cycle. This latest research points to a common cause for the sleep disorders associated with these two diseases and suggests that treatment of Parkinson's disease patients with hypocretin or hypocretin analogs may reverse these symptoms. More than 1 million people in the U.S. have been diagnosed with Parkinson's disease, and approximately

20 million worldwide. (The percentage of those afflicted increases with age.) Narcolepsy affects approximately one in 2,000 individuals — about 150,000 in the United States and 3 million worldwide. Its main symptoms are sleep attacks, nighttime sleeplessness and cataplexy, the sudden loss of skeletal muscle tone without loss of consciousness; that is, although the person cannot talk or move, they are otherwise in a state of high alertness, feeling, hearing and remembering everything that is going on around them.

"When we think of Parkinson's, the first thing that comes to mind are the motor disorders associated with it," said Siegel, who is also chief of neurobiology research at the Sepulveda Veterans Affairs Medical Center in Mission Hills, Calif. "But sleep disruption is a major problem in Parkinson's, often more disturbing than its motor symptoms. And most Parkinson's patients have daytime sleep attacks that resemble narcoleptic sleep attacks."

In fact, said Siegel, Parkinson's disease is often preceded and accompanied by daytime sleep attacks, nocturnal insomnia, REM sleep disorder, hallucinations and depression. All of these symptoms are also present in narcolepsy.

In the study, the researchers examined 16 human brains from cadavers — five from normal adults and 11 in various stages of Parkinson's — and found an increasing loss of hypocretin cells (Hcrt) with disease progression. In fact, said Siegel, the later stages of Parkinson's were "characterized by a massive loss of the Hcrt neurons. That leads us to believe the loss of Hcrt cells may be a cause of the narcolepsy-like symptoms of [Parkinson's] and may be ameliorated by treatments aimed at reversing the Hcrt deficit."

Funding for the study was provided by the National Institutes of Health and the Medical Research Service of the U.S. Department of Veterans Affairs.

The Semel Institute for Neuroscience and Human Behavior at UCLA is an interdisciplinary research and education institute devoted to the understanding of complex human behavior, including the genetic, biological, behavioral and sociocultural underpinnings of normal behavior and the causes and consequences of neuropsychiatric disorders. In addition to conducting fundamental research, the institute faculty seeks to develop effective treatments for neurological and psychiatric disorders, improve access to mental health services, and shape national health policy regarding neuropsychiatric disorders.

[www.newsroom.ucla.edu/page.asp?relnum=7905](http://www.newsroom.ucla.edu/page.asp?relnum=7905)

*This article was forwarded by Robin Riddle, the support group leader of Atypical Parkinson's Support Group in San Mateo. Thanks, Robin!*

## **Vibrational spectroscopy reveals Parkinson's biomarkers**

Dr Matt Wilkinson

09/05/2007(DrugResearcher.com) - US-based Molecular Biometrics has used vibrational spectroscopy techniques to identify biomarkers for Parkinson's disease that, if validated, could speed up the drug discovery process.

Researchers have used the vibrational spectroscopy techniques of Raman spectroscopy (RS) and near infra-red (NIR) spectroscopy to study the metabolic changes that occur with the onset of Parkinson's.

These biomarkers could not only be used as a tool in the early diagnosis of sufferers of Parkinson's but also as a tool to monitor the progression of the disease during clinical trials.

Trials currently use neuroimaging analysis to study disease progression but visually noticeable changes in the brain are slow leading to long trials that hold up the development of important new drugs.

Parkinson's is a neurological degenerative disorder of the central nervous system that impairs

the motor skills and speech of over 1.5m people in the US.

This is caused by certain neurons dying or failing to work properly which leads to the reduced production of the neurotransmitter dopamine that sends information to those parts of the brain controlling movement and coordination.

"Currently there are no proven biomarkers to aid in the diagnosis of Parkinson's. Advanced neuroimaging analyses such as positron emission tomography (PET) are too complex and expensive to be used on a routine basis," said Dr James Posillico, CEO of Molecular Biometrics.

Oxidative metabolism (OM) has been implicated in the onset of disease, and the use of the Molecular Biometrics biospectroscopy-based metabolomics (BSM) platform showed that both RS and NIR could quantify biomarkers of OM with a sensitivity of over 75 per cent.

This latest study involved examining the blood plasma of 52 patients, with 20 of those probably suffering from early stage Parkinson's and found that the techniques detected unique metabolic profiles or fingerprints for the two groups.

"We expect that metabolomic signatures like those identified in this pilot study will eventually provide clinicians with a rapid, cost-effective tool for diagnosing and monitoring patients with Parkinson's," continued Posillico.

"Biospectroscopic tests may also be useful in clinical trials to determine if new drug candidates are having the desired effect of modifying disease progression."

Oxidative stress and free radical-mediated damage to proteins, lipids and nucleic acids has also been demonstrated in the brains and blood of Alzheimer's disease patients.

Scientists at Molecular Biometrics have shown that this oxidative modification of blood proteins can be readily detected using biospectroscopy of small volumes of human plasma to reveal metabolomic signatures unique to patients with early sporadic Alzheimer's, Parkinson's and normal elderly controls (NEC). [www.npf.org](http://www.npf.org)

## **FDA Approves 1st Parkinson's Patch**

Once-Daily Skin Patch, Called Neupro, Treats Early Symptoms of Parkinson's Disease

By [Miranda Hitti](#)

WebMD Medical News

Reviewed by [Louise Chang, MD](#)

May 9, 2007 -- The FDA today announced the approval of Neupro, the first skin patch designed to treat symptoms of early Parkinson's disease. In a healthy brain, certain brain cells produce a chemical called dopamine, which helps the brain coordinate the body's movements. In Parkinson's disease, dopamine-producing brain cells falter and die. Parkinson's disease progresses gradually. Its four main symptoms are trembling in the hands, arms, leg, jaw, and face; stiffness of the limbs and trunk; slowness of movement; and impaired balance and coordination. As these symptoms become more pronounced, patients may have difficulty walking, talking, or completing simple tasks.

Neupro patches, which are changed daily, deliver a new drug called rotigotine through the skin. Rotigotine is a member of a class of drugs called dopamine agonists, which mimic dopamine's effects. Other Parkinson's disease drugs are given orally. Those drugs are effective, but some patients experience a wearing-off effect at the end of each dose. The Parkinson's patch is designed to solve that problem.

The FDA says Neupro's effectiveness was demonstrated in three studies that included 1,154 patients with early Parkinson's disease who were not taking other Parkinson's medications.

### **Side Effects**

The most common side effects in clinical trials included skin reactions at the patch site, dizziness, nausea, vomiting, drowsiness, and insomnia. Most of those side effects are typical for this class of drug, states the FDA in a news release.

Other potential safety concerns with Neupro include sudden onset of sleep while engaged in routine activities such as driving or operating machinery (sleep attacks), hallucinations, and decreased blood pressure on standing up (postural hypotension), according to the FDA.

Neupro Patch is made by Schwarz Bioscience of Research Triangle Park, N.C.

*Source: Web MD*

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## **Parkinson's cure possible in next decade**

Charles Enman, Ottawa Citizen

Published: Monday, April 23, 2007

Given enough funding, a cure for Parkinson's disease could be in the hands of physicians within 10 years, a noted researcher says.

"It would take money and researchers are in desperate times financially — but I'm fairly certain we could have a cure within a decade," said Jackalina Van Kampen, who will deliver a lecture on her research tomorrow at the Civic campus of The Ottawa Hospital.

How much money is needed? Ms. Van Kampen doesn't have a precise figure, but says "likely not \$100 million, but a few tens of millions of dollars."

That would be good news for the 100,000 Canadians diagnosed with Parkinson's, the degenerative disorder that causes tremors, rigidity and the gradual loss of mobility. The average age of onset is 60, but many are diagnosed earlier, including Canadian-born actor Michael J. Fox, who was only 30 when told he had the disease.

Ms. Van Kampen, a native of Charlottetown, is an assistant professor of neuroscience at the Mayo Clinic in Jacksonville, Florida. Her findings were published in July in a much-noticed article in *The Journal of Neuroscience*.

Parkinson's is caused by the degeneration of neurons in a structure of the midbrain called the substantia nigra. Those neurons produce dopamine, a neurotransmitter that helps the brain control movement.

By the time Parkinson's is diagnosed, there is already extensive damage of those neurons, damage that physicians and researchers always assumed was irreversible.

Working with rats, Ms. Van Kampen has found ways of coaxing dormant neurons to take on the dopamine-producing role of the damaged neurons and to restore the brain's control of movement.

Scientists have gradually been accepting that many structures in the brain can regenerate themselves. In the hippocampus, which helps create memories, generation of new neurons is part and parcel of the process of memory creation.

"But no one thought regeneration occurred in the substantia nigra," Ms. Van Kampen says. "Five years

ago, they called my idea ‘completely crazy.’”

Throughout the brain, there are undifferentiated cells called progenitor cells that, with the right stimulation, can transform themselves into more specific types of cells. Some of them are in the substantia nigra, and Ms. Van Kampen hoped to find a way to convince those cells to become dopamine neurons. Working with Parkinsonian rats, she found a drug that increased the number of dopamine neurons by 180 per cent.

Sophisticated brain scanning showed that those new dopamine neurons were working well. But the proof of the pudding was the behaviour of the rats: Their movements, which previously showed problems typical of Parkinson’s, were now almost fully restored to normal — what Ms. Van Kampen calls “a functional recovery.”

Rats aren’t people, and finding the equivalent way of coaxing progenitor cells in human brains to appropriately mutate is not a slam-dunk exercise, but Ms. Van Kampen feels sure the task is doable, given enough funding and perhaps a decade of hard research.

Equally important as generating cells to replace damaged neurons is the protection of neurons that are still intact. Ms. Van Kampen has found that ginseng, that most ancient of healing herbs, is very effective. When she treated rats with ginseng and then administered a toxin that would destroy cells in the substantia nigra, she found on post mortem examination that those cells were “almost completely protected.”

Finally, in a finding that will gratify mothers and gym teachers, she’s found that exercise reduces the severity of Parkinson’s.

She put some Parkinsonian rats, normally housed in shoe box-sized cages, into a much larger, three-level ferret cage that amounted to “a giant rat condo,” complete with running wheels and chewing blocks.

“We found that rats in this enriched environment recovered some neurons in the substantia nigra,” she says. “And I guess that speaks to the importance of keeping active, for everyone.”

She also believes that the work she’s doing will have application in treatment of other neurodegenerative disorders, such as Alzheimer’s disease.

Her main interest, though, is Parkinson’s. Her father developed the disease when she was in her early teens. When she was 14, the leader of a church group for

girls asked her to write down her dream of her future. “And I wrote that I would cure Parkinson’s,” she says. “Family history surely set me on a path.”

*Source: attawacitizen.com. This information was forwarded by Robin Riddle. Thanks, Robin.*

## **High-Frequency Stimulation Provides Long-Term Efficacy for Parkinson’s Symptoms: Presented at AANS**

Mashawnda Dowell

May 4, 2007(Drs. Guide) - High-frequency stimulation (HFS) of the subthalamic nucleus (STN) is a safe, effective, and stable treatment for patients with advanced Parkinson’s disease, according to a retrospective evaluation of this technique.

Lead author Alim L. Benabid, MD, PhD, director, department of neurosurgery, Centre Hospitalier Universitaire A. Michallon, Grenoble, France, discussed the study’s results here at the American Association of Neurological Surgeons (AANS) annual meeting.

Currently, STN-HFS is the treatment of choice for treating patients with advanced Parkinson’s disease. Dr. Benabid and colleagues studied the evolution of the cardinal features of this technique in a 14-year series that included 304 patients.

Enrolled patients underwent surgery in the STN since 1993 (296 bilaterally, 600 electrodes). The researchers retrospectively analyzed the duration of benefit, active contact coordinates, complications, and long-term changes in the nature of the disease.

Improvement in major symptoms was an average of 65% for the major Parkinson’s symptoms as evaluated using the Unified Parkinson’s Disease Rating Scale, Hoehn and Yahr Staging of Parkinson’s Disease, and quality-of-life scales. Improvement in speech was an average of 35%.

Drug dosages, and subsequently the levodopa-induced dyskinesias, were decreased by an average of 65%. This was linearly correlated to the preoperative levodopa-induced improvement and was strictly dependent on the accurate placement of the leads, according to the researchers.

The benefit of STN-HFS persisted in levodopa-sensitive symptoms such as tremor, akinesia, and rigidity, but not for midline symptoms such as balance and gait. Patients’ baseline condition (off medication, off stimulation) worsened in 25% of patients, was

stable in 36% of patients, and improved in 38%. Improvements continued over 5 years in 19% of patients.

The researchers determined that mild and transient complications were due to pretargeting (5.7%), implantation (30%), stimulation (19.6%), and hardware failure (16.7%). Complications decreased by a ratio of 2.3 with the learning curve of operators who performed the procedure.

There were psychic disorders in 8.3%, one procedure-related death, and one suicide.

The researchers concluded that STN-HFS is safe, effective, and stable over time. "Careful surgical practice and patient selection improve the outcome," the noted.

[www.nwppf.org/articles.asp?id=2175](http://www.nwppf.org/articles.asp?id=2175)

## Magnesium in diet

### Definition

Magnesium is an essential mineral for human nutrition.

### Function

Magnesium in the body serves several important metabolic functions. It plays a role in the production and transport of energy. It is also important for the contraction and relaxation of muscles. Magnesium is involved in the synthesis of protein, and it assists in the functioning of certain enzymes in the body.

### Food Sources

Most dietary magnesium comes from vegetables, particularly dark green, leafy vegetables. Other foods that are good sources of magnesium are:

Soy products, such as soy flour and tofu

Legumes and seeds

Nuts (such as almonds and cashews)

Whole grains (such as brown rice and millet)

Fruits or vegetables (such as bananas, dried apricots, and avocados)

## Side Effects

Toxic symptoms from increased magnesium intake are not common because the body eliminates excess amounts. Magnesium excess almost always occurs only when magnesium is supplemented as a medication.

Magnesium deficiency is rare. The symptoms include muscle weakness, fatigue, hyperexcitability, and sleepiness. Deficiency of magnesium can occur in alcoholics or people whose magnesium absorption is decreased due to surgery, burns, or problems with [malabsorption](#) (inadequate absorption of nutrients from the intestinal tract). Certain medications or low blood levels of calcium may be associated with magnesium deficiency.

### Deficiency symptoms have three categories:

Early symptoms include irritability, anorexia, fatigue, insomnia, and muscle twitching. Other symptoms include poor memory, apathy, confusion, and reduced ability to learn.

Moderate deficiency symptoms consist of rapid heartbeat and other cardiovascular changes.

Severe deficiency of magnesium could lead to tingling, numbness, sustained contraction of the muscles, and hallucinations and delirium.

[www.nlm.nih.gov/medlineplus/ency/article/002423.htm](http://www.nlm.nih.gov/medlineplus/ency/article/002423.htm)

## Dementia: It's not always Alzheimer's

**Many disorders, some curable, can cause dementia. Don't assume it's always Alzheimer's.**

Dementia is a neurological disorder that affects your ability to think, speak, reason, remember and move. While Alzheimer's disease is the most common cause of dementia, many other conditions also can cause similar symptoms. Some of these disorders get worse with time and cannot be cured. Other types respond so well to treatment, their symptoms may even be reversed.

### Types of dementia

After Alzheimer's disease, the most common forms of dementia are vascular dementia and Lewy body dementia. Sometimes, a person can have more than one of these problems at the same time. Frontotemporal dementia is less common, but may be mistaken for Alzheimer's.

## **Vascular dementia**

In vascular dementia, arteries feeding the brain become narrowed or blocked. The onset of symptoms usually is abrupt, frequently occurring after a stroke. However, some forms of vascular dementia progress so slowly that they are difficult to distinguish from Alzheimer's disease. Some people have both Alzheimer's and vascular dementia. Vascular dementia often causes problems with thinking, language, walking, bladder control and vision. Preventing additional strokes by treating underlying diseases, such as high blood pressure, may halt the progression of vascular dementia.

## **Lewy body dementia**

Lewy bodies are abnormal clumps of protein that have been found in the brains of people with Lewy body dementia, Alzheimer's disease and Parkinson's disease. This suggests that the three ailments are related, or that Lewy body dementia and Alzheimer's disease or Parkinson's disease sometimes coexist in the same person. Some people with Lewy body dementia have experienced improvements in symptoms when treated with Alzheimer's or Parkinson's medications.

## **Frontotemporal dementia**

Because it affects the areas of the brain that are responsible for judgment and social behavior, frontotemporal dementia can result in socially inappropriate behavior. Symptoms of this form of dementia, which runs in families, usually appear between the ages of 40 and 65.

## **Other disorders linked to dementia**

Several less common brain disorders also can result in dementia.

**Huntington's disease.** Symptoms of this hereditary disorder typically begin between the ages of 30 and 50, starting with mild personality changes. As the disorder progresses, a person with Huntington's develops involuntary jerky movements, muscle weakness and clumsiness. Dementia commonly develops in the later stages of the disease.

**Parkinson's disease.** People with Parkinson's disease may experience stiffness of limbs, shaking at rest (tremor), speech impairment and a shuffling gait. Some people with Parkinson's develop dementia late in the disease.

**Creutzfeldt-Jakob disease.** This extremely rare and fatal brain disorder belongs to a family of

human and animal diseases known as the transmissible spongiform encephalopathies. A new variety of Creutzfeldt-Jakob disease has emerged — particularly in Great Britain. It's believed to be linked to the human consumption of beef from cattle with mad cow disease (bovine spongiform encephalopathy).

**AIDS.** People in the advanced stages of AIDS also may develop a form of dementia.

## **Some causes are treatable**

Many other conditions, some reversible, can cause dementia or dementia-like symptoms.

**Reactions to medications.** Some medications have side effects that mimic the symptoms of dementia. A single medicine may trigger such a reaction in an older person or in someone whose liver fails to eliminate the drug normally. Interactions among two or more drugs may lead to reversible symptoms of dementia as well.

**Metabolic abnormalities.** Decreased thyroid function (hypothyroidism) can result in apathy or depression that mimics dementia. Hypoglycemia, a condition in which there isn't enough sugar in the bloodstream, can cause confusion or personality changes. Pernicious anemia caused by an inability to absorb vitamin B-12 also can cause cognitive changes.

**Nutritional deficiencies.** Chronic alcoholism can result in deficiencies of thiamin (vitamin B-1), which can seriously impair mental abilities. Severe deficiency of niacin (vitamin B-3) may cause pellagra, a neurological illness with features of dementia. Dehydration also can cause confusion that may resemble dementia.

**Emotional problems.** The confusion, apathy and forgetfulness associated with depression are sometimes mistaken for dementia, particularly in older individuals.

**Infections.** Meningitis and encephalitis, which are infections of the brain or the membrane that covers it, can cause confusion, memory loss or sudden dementia. Untreated syphilis can damage the brain and cause dementia.

**Normal-pressure hydrocephalus.** If cerebrospinal fluid builds up in the ventricles of the brain, the brain tissue is compressed

even though the fluid pressure remains normal. This may cause dementia. If this condition is identified in time, it may be treated by draining the excess fluid via a tube (shunt) leading into the abdomen.

### **Don't jump to conclusions**

Dementia isn't always due to Alzheimer's. Before you conclude that a loved one's memory loss and confusion stem from an irreversible disease process, get a thorough medical evaluation. Even if the evaluation uncovers no underlying condition that, with treatment, can reverse dementia, options may be available for easing its symptoms. Knowing the likely cause of dementia, however, is the essential first step toward managing it appropriately.

[www.mayoclinic.com](http://www.mayoclinic.com)

## **Malnutrition and seniors: When a relative doesn't eat enough**

**Malnutrition doesn't affect only frail or impoverished older adults. Active, independent seniors also are at risk. Learn to spot the signs of malnutrition and what you can do to help.**

At 82, your aunt is still beautiful, with a slender build and great charm. Active and outgoing, she recently moved to a retirement community where she hikes, plays bridge, and entertains her fiancé and large extended family. By all objective measures, your aunt is thriving, but in fact, like many older Americans, she's malnourished.

According to some estimates, as many as 85 percent of adults in long term care facilities and more than half of all seniors cared for at home suffer from under-nutrition or malnutrition — low or dangerously low levels of protein and other nutrients. But a growing number of active, independent older adults also are malnourished, especially women who live alone, minorities and low-income seniors. Experts say that spotting people who are clearly in trouble — those who are losing weight and muscle tone or becoming weak and confused — isn't difficult. The real challenge is identifying people who appear well nourished but aren't.

### **Tracking the causes of malnutrition**

At first glance, the causes of malnutrition seem straightforward: too little food, a diet lacking in nutrients or absorption problems. But malnutrition is much more complex, a web of physical, emotional and

social problems that traps vulnerable people in a self-perpetuating cycle of dependence and declining health.

For instance, older single adults, even energetic and self-sufficient ones like your aunt, often don't cook for themselves; unless invited out, their typical dinner may be nothing more than a handful of popcorn or a cup of tea. Carried on for long, a nutrient-poor diet accelerates the loss of muscle mass and strength that normally comes with aging. Shopping and preparing food become more difficult, which reinforces the tendency to subsist on easy but empty fare — toast, cold cereal, saltines. Eventually, the chronic lack of nourishment leads to increased frailty and dependence, which in turn can trigger depression — itself a major drain on appetite.

Malnutrition weakens the immune system, increasing the risk of pneumonia and other serious infections and exacerbating existing health conditions. And it can also contribute to mental confusion. Very ill or disorientated people are unlikely to eat well, if at all, and they're more likely to end up in a hospital or long-term care facility, where they're vulnerable to pressure sores, infections, post-surgical complications and further malnutrition.

### **When does malnutrition begin?**

If the scenarios are complicated, so are the factors that lead people to eat poorly in the first place. Often, the trigger isn't a single event, but a cascade of difficulties, many of which are common among people who are aging:

**Chronic illness.** Older adults may have debilitating, long-term conditions that affect their ability to shop, prepare meals or feed themselves. What's more, both chronic and acute ailments often suppress the appetite even as they increase the body's need for nutrients. Dementia, Alzheimer's disease, alcoholism and other illnesses that affect mental functioning also have a profound influence on appetite and on the capacity to prepare and eat healthy meals. Caregivers also are often at risk, neglecting themselves while they tend an ailing partner.

**A recent hospitalization or nursing home stay.** Acute illness or surgery can take a tremendous toll on the health of seniors, often resulting in loss of appetite, weakness, weight loss and debilitation.

**Depression.** Although frequently unrecognized and underreported in older adults, depression

affects as many as 6 million Americans over 65, including more than a third of those in nursing homes. As with other aspects of aging, the reasons for depression are complicated and interrelated: grief, loneliness, isolation, failing health, retirement, lack of mobility, concurrent illnesses such as Parkinson's disease, cancer, or diabetes, medications and malnutrition itself, which makes depression worse.

**Alcoholism.** It's not clear how many older adults have alcohol-related problems, but experts say the number is far larger than suspected even a decade ago. And alcoholism is a leading contributor to malnutrition — decreasing appetite, destroying vital nutrients and frequently serving as a substitute for meals.

**Malabsorption.** Some of the physiological changes that occur with aging affect the way the body absorbs and uses nutrients. In many older adults, the production of certain digestive enzymes and acids diminishes, interfering with protein breakdown and with the absorption of vitamin B12, folate, and possibly calcium and iron. Lack of vitamin B12 can have a devastating effect on the nervous system, leading to an unsteady gait, muscle weakness, slurred speech and psychosis — signs and symptoms similar to those of such age-related diseases as Parkinson's and Alzheimer's. Other illnesses — cancers of the gastrointestinal tract, inflammatory bowel disease and even diarrhea — can interfere with absorption.

**Medications.** Many drugs commonly prescribed for older adults can contribute to malnutrition by suppressing appetite, altering the way food tastes, causing nausea and vomiting, or interfering with absorption. Offending drugs include some antidepressants, certain blood pressure and osteoporosis medications, and even common analgesics such as aspirin. The problem is often compounded because many older people take several medications, all of which may affect the ability to eat and digest nourishing foods.

**Difficulty chewing and swallowing.** Dental problems, including gum disease, cavities and poorly fitting dentures can affect the taste of food and make chewing nearly impossible. When people with chewing problems do eat nutritious foods, they may have trouble digesting them. A dry mouth — a side effect

of many drugs — and diseases such as Parkinson's that affect the nervous system can interfere with swallowing.

**Diminished taste and smell.** For most people, food is as much about comfort and enjoyment as it is about survival, and this may be especially true as people age. Yet taste and smell often diminish later in life, robbing food of much of its flavor. Although some sensory loss seems to be a normal part of aging, certain medications and diseases can make matters worse, especially Alzheimer's, which affects the pathways in the brain related to the sense of smell.

**Restricted diets.** Seniors are more likely than any other group to have dietary restrictions, including limits on salt, fat, protein and sugars. Although such diets play a central role in managing many medical conditions, they can be so bland and unappealing that older adults simply stop eating. For that reason, some nutrition experts recommend rethinking restrictive diets, noting that malnutrition is more detrimental to overall health than rich or salty foods are.

**Limited income.** Some seniors with limited incomes go hungry, especially if they're taking expensive medications. It's not uncommon for older adults to have to choose between drugs and groceries at the end of the month when Social Security checks are stretched thin.

**Loneliness and isolation.** One of the biggest contributors to malnutrition is a solitary life and the loneliness, depression, grief, boredom and fear that often go along with it.

## What to look for

The signs of malnutrition are often hidden, especially in people who don't seem at risk. To help uncover problems before they become more serious:

**Quiz older people about what they eat**, but don't rely on self-reports alone. Try to spend time with them during normal meals at home, not just in restaurants or on special occasions. If you have an older friend or family member in a hospital or long term care facility, make a point of visiting during mealtimes. When you're concerned about weight loss, request a calorie count from the hospital or nursing home dietitian.

**Look for physical problems** such as poor wound healing, easy bruising and dental difficulties.

**Know what drugs older family members take** and how they affect appetite and digestion. Many commonly prescribed medications can reduce hunger and prevent nutrient absorption.

Ask an older person's doctor to check certain protein levels (serum albumin, prealbumin or retinol binding protein levels). Despite some drawbacks, these tests can often help identify chronic malnutrition.

### What you can do

Malnutrition is a complex problem, but the solutions can be surprisingly simple. Even small changes make a big difference in an older person's health and well-being:

**Enrich spartan diets.** To boost nutrition, encourage seniors to spread peanut or other nut butters on toast and crackers, on fresh fruits such as apples and bananas, and on raw vegetables. Other suggestions include sprinkling nuts or wheat germ on yogurt, fruit and cereal; adding extra egg whites to scrambled eggs and omelets; and melting cheese on sandwiches, vegetables, soups, rice and noodles.

**Restore life to bland food.** Try to make special diets more appealing by using lemon juice, herbs and spices and by varying the texture, color and temperature of foods. If loss of taste and smell is a problem, experiment with strong seasonings and flavors. Careful chewing can sometimes increase enjoyment because more flavor molecules come into contact with taste receptors. A dietitian can also suggest ways to perk up dull meals.

**Plan between-meal snacks.** This can be particularly helpful for people who get full quickly. A piece of fruit or cheese, a spoonful of peanut butter, even a milkshake for people who aren't lactose intolerant can add nutrients and calories.

**Use nutritional supplements.** Undernourished seniors are especially likely to be deficient in protein, vitamins B-6 and B-12, folate, niacin, vitamin D, calcium and zinc. Supplements are important because they can help supply

missing nutrients, but they can't provide protein or needed calories and shouldn't become a substitute for meals.

**Consider outside help.** If an older adult is very frail, consider hiring a home health aide to help prepare meals or investigate Meals-On-Wheels and other community services, including home visits from registered dietitians. Local churches and civic groups often have volunteers willing to shop and cook for seniors who live alone. Your department of health or community hospital can usually provide information on programs in your area.

**Engage doctors.** Talk to doctors about changing medications that affect appetite and nutritional status and about screening for nutrition problems during routine office visits. Be sure to inform doctors if you notice that an older adult is losing weight. And consult a dentist about oral pain or chewing problems.

**Make meals social events.** This may be the most important step in curbing malnutrition; older people clearly do better when they have company. Drop by during mealtime or invite seniors to your house. Or try eating out on a regular basis, which can boost spirits along with appetite. Take advantage of discount meals at earlier hours, and ask for small portions of food. If you can't share meals with the older adults in your life, encourage them to join programs where they can eat with others or arrange for them to have meals with friends. In one study, older adults who attended nutrition classes improved their nutritional status, but the change seemed to result from the social interaction the classes provided, not from the information they received.

**Encourage regular exercise.** Many seniors, even those with serious health problems, can benefit from daily exercise — it stimulates appetite, helps depression, and strengthens bones and muscles. Exercising with others also provides motivation and social interaction.

[www.mayoclinic.com](http://www.mayoclinic.com)

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PPSG recently received gift donations honoring the following individuals: Eric and Polly Bergstrom, Evelyn Fox, and Bernice Sarina.

## In Memory

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If you would like to be removed from our mailing list or know someone who would like to be included, please take a minute, call us at **408.734.1593**, or e-mail [ppsginfo@yahoo.com](mailto:ppsginfo@yahoo.com), and let us know.

## PPSG SUPPORT GROUPS

**Berkeley** 3<sup>rd</sup> Mon 10-12 North Berkeley Senior Center, 1901 Hearst Av, Roddy Raikow 510-231-1998 or Mitzi Cahn 510-527-9075 **Fremont** 4<sup>th</sup> Mon 7:00 pm Fremont Senior Center 40086 Paseo Padre Parkway, Lettie Webb 510-656-6393 **Fremont Caregivers** Contact Nancy Rothschild, Caregiver Project Coordinator, 510-574-2035 **Marin County** 4<sup>th</sup> Tue most mo., 2-4 Redwoods Auditorium 40 Camino Alto, Mill Valley, Gloria Rashti 415-381-6680. Redwoods 415-383-2741 **Mt. Diablo Parkinson's Network General Meetings** 2<sup>nd</sup> Sat 10-12, Grace Presbyterian Church, 2100 Tice Valley Blvd, Walnut Creek, Nancy Walls, 510-236-7065, Philip Wheeler, 510-527-3588, Margy Hansell, 925-939-4210, or Ronalee Spear, 925-284-2189 **Oakland** 1<sup>st</sup> Thur 1:30-3:30 Easter Seals Bay Area, 180 Grand Av, Suite 300, Karen & Jim Eagan, 510-763-4492 **Petaluma** Last Sat 1:30-3:30 Sunrise of Petaluma, 815 Wood Sorrel Dr, John & Mamie Strong 707.763.3522 **Pleasanton Tri-Valley** 2<sup>nd</sup> Sat 10-12, Senior Center, 5353 Sunol Blvd, Norm & Jackie Bardsley 925-244-1231 or 925-831-9940 **San Leandro** 1st Thur (except Jul & Aug) 10-11:30, (NEW LOCATION) San Lorenzo Community Church, 945 Paseo Grande, Norma Zeff, 510-663-6435 **Sonoma County** 1<sup>st</sup> Sat (not Jan, Jul, Sep) 1-3, First Congregational Ch, 2000 Humboldt St, Santa Rosa, Ron & Colleen Trowse 707-526-4373 **Vallejo** 3<sup>rd</sup> Mon (except 2nd Mon, Jan & Feb) 2:00 Kaiser Medical Center, 975 Sereno Drive, Evelyn Fox 707-644-3390

## ---PENINSULA REGION---

**Daly City** 1<sup>st</sup> Tue 3-4 Doelger Senior Center, 101 Lake Merced Blvd, Leonard Ke 415-587-1285 **Los Altos Young Parkinson's Support Group** 2<sup>nd</sup> Sat 10-12, United Methodist Ch/Los Altos, Foothill at Magdalena, Dean Prescott 408-738-2505 or dean53@yahoo.com **Magnolia Peninsula** 2<sup>nd</sup> Thur 1:30 main conference room Magnolia Apart, 201 Chadbourne Av, Millbrae, Leon Rosenthal, 650-348-3480 **Palo Alto** 2<sup>nd</sup> Wed 2:00-3:30 Avenidas Senior Center dining room, 450 Bryant St, 650-289-5400 **Redwood City Positive People Against Parkinson's** 3<sup>rd</sup> Fri 1-2:30, (No meetings Aug, Nov, Dec) Sequoia Hospital, Health & Wellness Ctr, 749 Brewster Ave, Tom Constantino 650-366-7166, or David Shein, 650-367-5998 **(NEW) San Francisco Caregivers** Thur (varies) 12-12:50 Veterans Affairs Med Ctr, Parkinson's Ctr conf room, Bldg 203 Room 1B26A, Susan Heath 415-379-5530 or Aliza Benditsky 415-221-4810 X 4741 **San Mateo Atypical Parkinsonism (PSP, LBD, MSA, CBD) Bay Area Caregivers** Sundays 5-7 about every 6 weeks, Mimi's Café 2208 Bridgepointe Parkway, San Mateo, Robin Riddle 650-233-9277 or riddle@stanfordalumni.org **San Mateo Caregivers** 1<sup>st</sup> Wed 2:30-4:30 Ellsworth Room 100 San Mateo Dr., Call Ann Sasaki, Mills Health Center 650-696-4741 **Sunnyvale** 2<sup>nd</sup> Wed 1-3 First United Methodist Ch, 535 Old San Francisco Rd, Phyllis & Henry Ng 408-733-5648 **YOPD** (Young Onset Parkinson's Disease) 2<sup>nd</sup> Tue 6:30-8:00, Board Room, Lucile Packard Child Hosp, 725 Welch Road, Palo Alto, Martha Gardner, 866-250-2414.

## ---SOUTHERN REGION---

**Hollister** 1<sup>st</sup> Tue 1:30-3:30 First Presbyterian Ch, 2066 Cienega Road, Shirley Kennedy 831-637-3839 or John Skinner 831-637-6755 **Monterey** 3<sup>rd</sup> Mon 2:30-4:00 SHARE Room, Monterey Adult School, 200 Coe Av, Seaside. Helen Garrett 831-657-4241 or Kathy Warthan 831-372-7510 **Salinas** 4<sup>th</sup> Wed 2:00-3:30 Salinas Adult School, 20 Sherwood Place, Sherry Whitcomb, 831-796-6920 **San Jose-Berryessa** 1<sup>st</sup> Wed 1:00-2:30 Berryessa Community Center, 3050 Berryessa Rd, Bob & Jane Pomeroy 408-263-8485 **San Jose Caregivers** usually 4<sup>th</sup> Wed 1:30-3:30 St Francis Episcopal Church, 1205 Pine Ave, Charmaine Eng 408-723-8116 **San Jose-Willow Glen** 1<sup>st</sup> Fri 10-12 St Francis Episcopal Church, 1205 Pine Ave, Betty Havens 408-227-8593, or Joan Lorentson, 408-997-7009 **Santa Cruz** 1<sup>st</sup> Wed 12:30-2:00 St. Stephen's Lutheran Church, 2500 Soquel Ave, David Donahoe 831-479-4485 **Saratoga** 3<sup>rd</sup> Tue 2-4 19449 Via Real, Lois McPherson 408-867-1807

## ---CENTRAL VALLEY REGION---

**Fresno, Greater** 2<sup>nd</sup> Sat 10 at San Joaquin Valley Rehab Hosp 7173 N. Sharon Ave, Max Robinson, 559-226-2673 **Merced** 4<sup>th</sup> Thur 10AM (Nov 17, Dec no meeting) Mission Gardens 1450 E. 27<sup>th</sup> St, Amie Marchini 209-384-3300 **Modesto** 3<sup>rd</sup> Wed 1:30-3:00 Centenary United Methodist Ch, Fireside Room 1911 Toyon Av, JoAnn & David Ryan 209-529-5643 or [davejoann@sbcglobal.net](mailto:davejoann@sbcglobal.net) **(NEW) Pine Grove** 1<sup>st</sup> & 3<sup>rd</sup> Thur 2-4 Calvary Chapel Patio Bldg 18400

Ridge Road, Sarah Johnson 209-296-2575 **Roseville** 1<sup>st</sup>  
Tues 1:30-3:00 Roseville Maidu Comm Ctr, 1550 Maidu  
Drive, Linda Krisa 916-261-1321 **Tulare-Kings** 1<sup>st</sup> Fri  
10:30 Visalia United Methodist Church, 5200 W. Caldwell  
Av, Mary Dickerson 559-622-9044, Church Office 559-  
627-1660 (**FORMING**) **Turlock** Donald Jackson 209-606-  
9127 November 22, 2006

## EXERCISE CLASSES

**Berkeley:** Vista College, Joan Nielsen, 510-981-2800

**Berkeley:** Mon. 10:30-11:30 & 1-2:30, John Argue 510-  
985-2645

**Daly City:** Tue./Thur. 1-2, Doelger Sr. Ctr. Pat Armstrong  
650-991-8012

**Gilroy:** Gavilan College, Dave Ellis, 408-848-4878

**Hayward:** Kaiser Permanente, Wed. 10-11:30, John Argue  
510-985-2645

**Kensington:** Tue. 1:30-3:00, John Argue 510-985-2645

**Marin Cty:** Tue. 10-11:30; 12-1:30. Osher Marin JCC, San  
Rafael. 415-479-2000

**Monterey:** Monterey Peninsula College, Mark Clements,  
831-646-4231

**Orinda:** Tue. & Fri. 1:00-2:30, In Forma Gym. Dean  
Dallman 925-283-5019

**Palo Alto:** CAR, Aquatic Therapy, 650-494-1480

**Palo Alto:** Mon. & Fri., 9:15-10:15, Sr. Ctr. 450 Bryant St.  
650-289-5400

**Redwood City:** Canada College, 4200 Farm Hill Blvd.  
Barbara McCarthy 650-306-3473

**Salinas:** Hartnell College, Melissa Stave, 831-755-6876

**Saratoga:** Mon. – Fri. 9-12; 1:30-3, W. Valley Comm. Coll.  
Joan 408-741-2420

**San Bruno:** Mon. & Wed. 1:10-2:30, Tue. & Thur. 12:35-  
1:50, Skyline Coll. Bess 650-738-4286

**San Francisco:** Fri. 11-12, SFSU, Marsha Melnick 415-  
338-1360.

**San Jose:** Mon. & Wed. 10:30-11:45, Houge Ctr. Tue. &  
Thur. 10-11:45, Evergreen Ctr. Deanna, 408-369-6435

**San Jose:** Easter Seals Comm Ctr. Aquatic Exercise  
programs, 408-295-0228

**San Jose:** Evergreen Valley College, Rich Wagner, 408-  
274-7900 X 6447

**San Mateo:** College of San Mateo, 1700 W. Hillsdale  
Blvd., John Hogan, 650-574-6469

**Sunnyvale:** Tue. & Thur. 9-10, Sr. Ctr. 550 Remington Dr.  
Ruth Hanes 408-864-8873

**Sunnyvale:** Wed. 10-12 noon, The Parkinson's Institute,  
1170 Morse Ave., Marilyn Basham:

408-734-2800.

## Exercise Classes – New Addition

### Palo Alto

Tai Chi/Chi Kung for Parkinson's in the Atrium at Stanford  
Medical Center

Every Saturday 10:00 am – 11:30 am (short break in  
between).

Mwezo & Jane

Kujiweza Healing Arts Institute

Call Jane: (408) 315-1179

Email: [Kujiweza@sjyogataichi.com](mailto:Kujiweza@sjyogataichi.com)

### San Jose

The Villages Golf & Country Club

Parkinson's Exercise Program (PEP)

Balance, gait, posture, Tai Chi/Chi Kung

Mondays 11:30 – 12:30 pm

Optional 3-day program

Mwezo & Jane

Kujiweza Healing Arts Institute

Call Jane: (408) 315-1179

Email: [Kujiweza@sjyogataichi.com](mailto:Kujiweza@sjyogataichi.com)

### Los Gatos

The Terraces of Los Gatos

Parkinson's Learning Lifelong Useful Skills (PLLUS)

Balance, gait, posture & Tai Chi/Chi Kung

Mon. 2:00 pm & Wed. 10:30 am

Mwezo & Jane

Kujiweza Healing Arts Institute

Call Jane: (408) 315-1179

Email: [Kujiweza@sjyogataichi.com](mailto:Kujiweza@sjyogataichi.com)

## June 29, Caregivers Appreciation Luncheon

Mark your calendar! PPSG is having its 4th Appreciation Luncheon on June 29 for caregivers who have devoted their ongoing efforts to caring for people with Parkinson's. The event is an opportunity for caregivers to meet with each other, to share their concerns and experiences and to take a break. The program will include speakers (TBA). As in the past, there will be a separate program for patients who can not be left alone.

Location: Basque Cultural Center, 599 Railroad Ave.(Off Grand Ave and Hwy 10), South. San Francisco, 94080.

Phone: 650.583.8091.

Date: Friday, June 29, 2007.

Time: 10:00 am to 1:30 pm.

If you would like to attend, and you do not have the sign-up form that is on the May support group newsletter, please contact Charmaine Eng, at 408.723.8116 or Viola Mays, at 408.225.7465. Space is limited, please RSVP by June 13. Thank you!

**If you would like to receive our newly-assembled caregivers' packet, please call or write to PPSG.**

*This newsletter was assembled by  
The Morgan Center. Thank You!*



Parkinson's Patients Support Groups, Inc.  
1170 Morse Avenue  
Sunnyvale, CA 94089-1605  
408.734.1593

**ADDRESS SERVICE REQUESTED**

**Summer Quarterly 2007**

## PPSG Board Meetings

You are welcome to drop by our board meetings and share ideas with us! We meet on the **3rd Monday** of the month between **1:30 and 3:30 PM** at the Parkinson's Institute. To confirm meeting dates and time, please call us at 408.734.1593. If you are planning to attend, please call Charmaine Eng at 408.723.8116 (dial \*82 before the number).

### Board Members

#### Chair:

Charmaine Eng

#### Vice Chair:

Dean Prescott

#### Secretary:

Carla Gwosden

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If you would like to be removed from our mailing list or know someone who would like to be included, please take a minute, call us at **408.734.1593**, or e-mail [ppsginfo@yahoo.com](mailto:ppsginfo@yahoo.com), and let us know. Thank you.

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