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A B O U T U S

Parkinson's Post is published by the Northwest Parkinson's Foundation, a 501(c)(3) charitable organization.

Our mission is to establish optimal quality of life for the Northwest Parkinson's community.

We welcome comments and inquiries regarding this newsletter and all our activities. Our contact information can be found on the last page of this newsletter. We look forward to hearing from you!



NORTHWEST
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FOUNDATION

A lesson in slowing down

By Elisa Murray

My mother went to the Booth Gardner Parkinson's Care Center because of a glass of wine—or, to be truthful, because of several glasses of wine.

My younger sister and I, both Seattle residents, held our annual “lowbrow” wine-tasting in March this year. A high school friend of mine came with her sister and her sister's friend, Craig Howard. Craig—one of those people who is completely at ease in situations where he doesn't know a soul—told us about his life and work. Central to his story was the Northwest Parkinson's Foundation, which he co-founded in 1999. We were immediately interested: Our mother had been diagnosed with Parkinson's in 2002, and she'd been having little luck finding a therapeutic program that worked for her.

He gave us contact information for the Foundation. We passed it on to our mother. She called the Foundation the next day, spoke to co-founder Bill Bell at length—“We talked for an hour,” she said, disbelieving, “and this Center is one of only two in the country!”—and by the end of the week she had set up a series of appointments. My mother has always moved quickly.

For much of her life, in fact, the best word to describe her was “movement.” As a child, she was a tomboy with braids who excelled at basketball, swimming, tennis and skiing. As an adult, she raised three daughters as a single mother, worked full time and was an active



Photo courtesy of Elisa Murray

The author, center, flanked by sister Vicki Kurzban, left, and mother Anya Murray, on a trip to the Equadorian jungle in 1993.

church member. And she managed to have a social life, keep her tennis game sharp and her ski turns graceful. She also kept the entire family on the move; we moved six times over the course of about 10 years, finally ending up near her hometown in Massachusetts.

She was dedicated to health and physical fitness long before it became fashionable. The year she turned 40, 1978, she gave up smoking, started jogging and filled our cupboards with natural foods—despite the objections of her three Snickers-bar-addicted daughters. At age 50, she became a massage therapist. At 60, she moved to Boulder, near my older sister and her family, and immediately took up sufi and contra dancing, hiking and camping. When I lived in Ecuador for several years in the early

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1990s, she visited me twice, cheerfully hitching rides on the most rickety of transportation options. My friends still talk about it: While their parents demanded taxis, my mother hopped on decrepit buses and pickups.

So it was a shock to all of us when several years ago—at 62—something finally slowed Anya Murray down: Parkinson's disease.

None of us realized this for a long time. Her symptoms accumulated bit by bit: a slower gait, a slight shuffle, a curve to her usually erect back, a softer voice. Finally, a friend mentioned differences he'd noticed, and she began to take note. She told us what she suspected months before a neurologist confirmed the diagnosis in early 2002.

The problem with Parkinson's is that even a clear diagnosis does not lead to a clear anything else. My mother, who believes in helping the body heal itself, decided to postpone going on medications until she had tried other routes. She did an impressive amount of research and eventually put together a program whose foundation was a certain kind of Chinese medicine—called *yin tui na*, or forceless spontaneous release—as well as massage, exercises, vitamins and other therapies. With her usual discipline, she drafted a schedule that she stuck to religiously. When we became concerned the program wasn't helping her, she told us she was going to try it for a certain amount of time. Then she would consider alternatives.

This year, after deciding her Chinese medicine program was not providing enough relief, she decided to try conventional drugs. Her neurologist thought she should start with Sinemet. More frustration: Two weeks went by with no apparent effects. Understandably, after waiting so long she wanted results. At about that time, she spoke to Bill Bell and scheduled a visit with the Booth Gardner Parkinson's Care Center.

By the time she arrived for her evaluation at the Center, the Sinemet

had finally kicked in. She reported higher energy levels and better coordination than she'd had in a long time. And her spirits had lifted. Though she used a wheelchair in the airport during her trip to Seattle, she hadn't really needed it.

Visits to the Center didn't change her life—does anything ever really change anyone's life?—but it was very valuable. The staff was extremely helpful in assessing her and laying out an array of treatment possibilities. She saw a physical therapist, neurologist, occupational therapist, speech therapist and psychologist—all with deep knowledge and experience in Parkinson's. The appointments were thorough and centered on her and her experience, not just on symptoms.

And the visits were covered by Medicare.

"I felt a lot better and more confident about what I was doing after I went there," Mom says. "They had more resources than I had had at my disposal, and it was really helpful to get an integrated approach." She was impressed with the Center's thorough follow-up as well. Interestingly, her Center assessment also helped her with her relationship with her Colorado doctor because she felt more confident and proactive as a patient, which made her more adept at assessing and choosing options.

The visits were equally valuable for me and my sister. We attended visits with her, and we learned much more not just about Parkinson's, but about *her* Parkinson's. We learned she has low blood pressure, which could be a dangerous condition. We learned she has good walking coordination, her speech volume is high and her cognitive ability is sharp. And we learned she has a lot of anxiety, has difficulty expressing herself and keeps quieter than she used to. My mom, never one to burden

others with her problems, had not told us this in phone conversations: It took an appointment with a psychologist for us to hear that.

Things have improved for her since the visits to the Center. She's slowly phased off Sinemet and onto Requip, with fairly good results. And Selegiline is helping control her tremors. But it's been a difficult road. Since her diagnosis, she's had to give up many beloved activities, including skiing and driving, because of a growing lack of coordination and low energy. But she continues to hike; she still goes to her weekly sufi dances—the cornerstone of her social life—and in recent months she started jogging again. "It's almost easier to jog than to walk," she says.

'The problem with Parkinson's is that even a clear diagnosis does not lead to a clear anything else.'

Perhaps the most difficult challenge has been adjusting to her new self. She's no longer the Anya who ran from one activity to the other. "I'm now the slowest person at the checkout line," she told me once, ruefully. "It takes me forever to count out change. I'm the slowest at everything."

This, I realize, isn't necessarily because Parkinson's folks are so much slower, it's because our society seems to be getting measurably faster. I'm used to the fast pace: My job is a flurry of emails, quick conversations and meetings ruled by the relentless hand of efficiency. It's almost a relief to get on the phone with Mom—former queen of multi-tasking—and consciously slow myself down to hear how she's *really* doing.

She, meanwhile, has become religious about what she calls her "sleep therapy"—taking a nap every day. Perhaps we could all use some of that.

—Elisa Murray is communications director with Northwest Environment Watch, a nonprofit organization in Seattle.

Researcher expands on family Parkinson's link

By Ian Corks

At its 2003 Annual General Meeting in Montreal, Parkinson Society Canada was proud to sponsor the inaugural Donald Calne Lecture, which was delivered by one of the world's foremost Parkinson's researchers, Dr. Yoshikuni Mizuno, chairman of the Department of Neurology at Tokyo's Juntendo University. With 16 staff neurologists working primarily on the diagnosis and management of Parkinson's patients and more scientists dedicated to basic research, the team is probably the largest, and certainly one of the most influential, academic groups in the world devoted to Parkinson's disease. Dr. Mizuno is also president of the 8,000-member Japanese Neurological Association.

Dr. Mizuno is dedicated to finding the cause of Parkinson's and has led pioneering research in many aspects of the disease. One of his greatest accomplishments came in the late 1990s with his identification of a new gene implicated in familial forms of Parkinson's. This discovery has shed light on how dopamine cells die prematurely and has implications not only for familial Parkinson's but for the more common sporadic (i.e. non-hereditary) form of the disease.

Much of Dr. Mizuno's work throughout the years has been directed at the role of genetics in Parkinson's disease. Introduced by Dr. Donald Calne himself, Dr. Mizuno directed much of his fascinating lecture to the significant progress being made in this area. What follows is an overview of Dr. Mizuno's presentation on genetics.

The genetic link

Although it is not a true hereditary condition, as many as one in 20 people with Parkinson's have an inherited form of the disease. This is often referred to as familial Parkinson's disease. Parkinson's can be "inherited" through two pathways. In so-called autosomal dominant Parkinson's, the disease is passed down from grandparents to parents and then to children and grandchildren. In autosomal recessive Parkinson's, the parents remain apparently unaffected, yet can pass on the condition to several of their children.

Researchers have identified 11 distinct forms of familial Parkinson's disease involving both autosomal dominant and autosomal recessive inheritance pathways. These are named Park 1 through 11, according to the order of their discovery.

The first of these, Park 1, was identified among families living in the Contursi region of southern Italy. Park 1 is an autosomal dominant form and the age of onset is slightly younger than normal. One of the great advantages for researchers studying familial Parkinson's is the ability to identify and study the causes of the disease at the molecular level, something that can't currently be done in other types of the disease. This kind of molecular study has enabled re-

searchers to determine that Park 1 occurs in the long arm of chromosome 4 and is caused by mutations of the alpha-



Dr. Yoshikuni Mizuno

synuclein gene and protein. Alpha-synuclein is an important protein that also plays a role in the causes of cell death in the substantia nigra in non-familial Parkinson's.

In the case of Park 1, it was found that altering a single amino acid within alpha-synuclein—specifically changing alanine to threonine—could trigger the complex condition that is Parkinson's.

Discovering a new gene

Park 2 is an autosomal recessive form of the disease and was detected in a small population in Japan. Park 2 usually occurs around the age of 40, and while people with the condition respond well to levodopa, they tend to develop dyskinesias and motor fluctuations from the treatment more easily than other groups. Dr. Mizuno's team located Park 2 on the long arm of chromosome 6 and, perhaps more importantly, has identified a new gene—which they named the parkin gene—as the causative factor.

This identification of the parkin gene occurred in a relatively short period of time by normal research standards. While investigating the manganese superoxide dismutase gene as a potential target gene for a genetic risk factor for Parkinson's, Dr. Mizuno's team came across a family in which four out of six siblings had young-onset Parkinson's. All affected individuals had two alanine molecules in their chromosomes, compared to one in the general population. Using a process called linkage analysis, 12 similar families were identified and analyzed. This provided data that eventually allowed the team to locate the disease gene on the long arm of chromosome 6.

Next, the team encountered a patient who seemed to be lacking one of the DNA markers being used in their linkage analysis. They theorized that this marker might be within the disease gene itself. They then employed an experimental technique called molecular cloning—something Dr. Mizuno compares to fishing—trying to find one molecule (in this case one that absorbs a substance known as D6S395) in thousands.

Dr. Mizuno was able to catch that "fish" and the parkin

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New drug shows promise for advanced Parkinson's

By **Ember Sturgis**
and **Brandi Kimball, PharmD**

Levodopa, a precursor of dopamine, has been used for the treatment of Parkinson's disease since it received FDA approval in 1970. It is usually combined with carbidopa and/or entacapone (levodopa + carbidopa = Sinemet® and levodopa + carbidopa + entacapone = Stalevo®).

Many patients experience a marked improvement in symptoms after starting levodopa therapy. Unfortunately, as the disease progresses the response to levodopa often fluctuates and diminishes. Diminished effects from the drug are referred to as "wearing off."

Variations in motor function are often correlated with levels of levodopa in the body and may include sudden, frequent "off times" that impair a person's ability to perform activities of daily living and have a negative impact on quality of life. Increases in dose and frequency can be helpful to a point, but often the side

effects limit this approach to therapy.

Research has been ongoing by Neopharma, a drug company based in Sweden, to find a solution to manage motor fluctuations and "off" times for patients on levodopa therapy. In January, the Swedish Medical Products Agency approved the manufacture and marketing of a gel form of carbidopa/levodopa—called Duodopa®—delivered via pump.

Duodopa is indicated for patients with advanced levodopa-responsive Parkinson's who are experiencing severe motor fluctuations. This gel suspension is formulated to allow continuous intestinal administration via a portable pump. Duodopa is pumped directly into the duodenum through a surgically inserted tube. The tube can remain in place for several years for long-term administration. It is designed to hold enough medication for one day of normal therapy, usually 16 hours.

The dosage comprises three individually adjusted doses: the morning bolus dose, the continuous maintenance

dose and extra bolus doses. This unique method of delivery results in a constant level of levodopa in the body, which results in fewer motor fluctuations and "off" times. The side effects reported were similar in frequency and severity when compared to oral therapy.

A majority of patients in two studies reported improvement of motor fluctuations and quality of life. Despite the inconvenience and expense of the delivery system, some patients chose to remain on Duodopa after the study ended.

The goal of any pharmacological treatment of Parkinson's is to help patients maintain mobility for as long as possible. For patients living with advanced Parkinson's who are suffering from disabling motor fluctuations, Duodopa may be beneficial. Additional research needs to be completed before this drug and delivery device become commercially available.

Ember Sturgis is a PharmD candidate at Washington State University. Brandi Kimball is a geriatric resident at WSU.

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gene was discovered. When the blood from patients within the families was analyzed, many different kinds of mutations in the parkin gene were found.

It has since been discovered that mutations in the parkin gene usually result in the absence of the parkin protein. In patients with Park 2, parkin protein is usually low or absent, while in people with regular sporadic Parkinson's there is abundant parkin protein.

In Park 2, therefore, the researchers determined that the lack of parkin protein is the cause of nigral neuronal cell death. They discovered that parkin protein is an enzyme called ubiquitin ligase, which plays a role in a cell "cleaning" system (called the ubiquitin proteasome system) that removes unnecessary proteins or proteins that are no longer needed from the cells.

Without the existence of this ubiquitin ligase, the system does not function properly. Therefore, without the parkin protein, the proteins that should be removed instead accumulate in the cells. Dr. Mizuno compared it to dust and dirt accumulating in a carpet if you don't vacuum regularly because you don't have a vacuum cleaner.

This accumulation of abnormal protein eventually kills the cells. By putting together this process, researchers have found a significant piece of the puzzle—the molecular mechanism by which the lack of parkin protein kills nigral neuron cells.

The discovery of the parkin gene and how it is linked to cell death represents a significant advance. It has helped shed light on how dopamine cells die prematurely in familial Parkinson's, which, in turn, has important implications in all forms of the disease. Similarly, the work done on other types of familial Parkinson's have also provided valuable data that is being used by researchers around the world.

Closer to the cause

Dr. Mizuno emphasized the importance of familial Parkinson's research. He believes that in the near future, researchers will find the cause of regular sporadic Parkinson's using the information obtained from the studies on familial Parkinson's. Once that is achieved, we will be that much closer to finding ways to stop the disease process.

—*Reprinted with permission from Parkinson Society Canada (Spring 2004, Volume 4, Issue 1, Parkinson Post), www.parkinson.ca.*

Life lesson comes in small (furry) package

By Carey Christensen

My dog died not long ago. I was away from home, visiting friends in California, driving an unfamiliar freeway, when the call came. It was Elizabeth, my soon to be 16-year-old daughter. "Mia died. Mia died at the vet's, Mom," her words barely audible, choked by tears and nearly drowned by the roar of traffic. "What?" I replied, hoping I'd heard wrong, knowing I hadn't.

It took less than 10 seconds for grief to overcome shock. I navigated the Friday afternoon California traffic with one hand; the other held the phone that connected me to my daughter, our disbelief and terrible sadness shared wirelessly and wordlessly as we cried together for mile upon mile.

The next few days of my trip were thankfully packed with activity. I greeted each new person with, "I'm so happy to meet you. My dog died yesterday," hoping that saying it out loud would somehow make it normal. The joy I felt being among friends was shadowed by the sadness I was able to keep at bay—for awhile. I didn't want to go home.

The last leg of my journey was blurred by tears. I drove from Seattle to Stanwood dreading the thought of being without Mia, of being home alone. I thought of her perched on the sofa pillows, surveying the neighborhood; of her resting beneath my chair as I worked at the computer; the way she looked after me like the true Aussie herder she was—ever alert, ever concerned, ever completely engaged and engaging. Lacking a tail, she wiggled her whole body in delight—and everything and everyone delighted her.



Top: The author with new family member Truly. Above: The incomparable Mia.

My thoughts were intense and contradictory. I wondered who I would talk to in the middle of the night as I coped with typical Parkinsonian wakefulness; I hoped my unexpected newfound freedom from the responsibilities of dog ownership would feel liberating; I was angry with the unfairness of losing my canine companion while dealing with divorce and disease; I felt guilty for allowing her teeth to be cleaned—the routine procedure that claimed her life; I felt consoled at the thought she was probably sweetly dreaming at the end; I was overwhelmed with the pain of never seeing her again.

I also remembered the shedding; the accidents on the carpet; the difficulty of willing my slow-moving self off the couch to take her outside because our new condo lacked our old backyard. I thought of the expense and trouble of boarding her when I traveled; the vet bills; the grooming. But, surprisingly, no matter how hard I willed it to come, the feeling of liberation never arrived. I knew I would gladly take back the burden of responsibility for the love of Mia and her companionship.

That's how I know I'm alive and that life is worth living. That despite the debilitating nature of my current situation and the depths of despair I have known, I can still open my heart to love—and the potential of heartbreak. Life is not rich if lived safely.

So instead of doing laundry upon my return, I found "Truly"—a new pup to share my life. She will not replace Mia—no dog could. Instead she'll join Mia, whose joyful existence continues to fill my heart, ensuring it will always remain open, alive and ready to love.

—Carey Christensen lives in Stanwood, WA, with her 16-year-old daughter Elizabeth, Harvey the cat, and Truly. She was diagnosed with Parkinson's in 1999 at age 41.

Community mourns loss of Parkinson's advocate

Millicent Martinez Kondracke, a psychotherapist and advocate whose battle with Parkinson's was documented in a book by her journalist husband, died July 22 at her home in Washington, D.C., of complications of her illness. She was 64.

The wife of TV commentator and writer Mort Kondracke, Milly

Kondracke had suffered from Parkinson's disease for 17 years. She was the subject of his 2001 book, *Saving Milly: Love, Politics and Parkinson's Disease*, about the couple's relationship and her battle with Parkinson's.

Milly Kondracke took up the fight for a Parkinson's cure through advocacy work on Capitol Hill. She pushed for increased funding for the National Institutes of Health and for federal support for stem cell research.

In 2002, the Parkinson's Action Network established the Milly Kondracke Award for Advocacy in her honor, and she was also the recipient of PAN's Morris K. Udall Award for Public Service.

Mort Kondracke wrote in *Saving Milly*, "I want a medical miracle to save her. But even if there is none, our story will not end. ... I will keep working to end Parkinson's disease on her behalf, and I will hug her in my heart forever."

The Northwest Parkinson's Foundation would not exist without your support. We rely on donations to carry out our work.

Donations support a wealth of programs and services to help Parkinson's people live well, including the high-quality specialty care of the Booth Gardner Parkinson's Care Center; a growing Parkinson's Telehealth Program that reaches out to people in rural communities; educational and support materials, such as the *Parkinson's Post*, a weekly email update, our website, the Parkinson's Educator, and the book, *H.O.P.E.*; and advocacy efforts toward funding for Parkinson's research.

If Parkinson's has touched you or someone you care for, please consider supporting this work through a gift today. Those listed here gave between Aug. 1 and Sept. 19, 2004. Thank you, friends!

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In honor of Booth Gardner

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CYCLING FOR THE CAUSE



Eight Oregonians got together to ride the 100-mile "Rim to Roseburg" bicycling event on Aug. 21. The team collected pledges for the Northwest Parkinson's Foundation as part of Team Parkinson's 2004. The are pictured at the rim of Crater Lake before their long descent into Roseburg, OR. The riders are (front row, from left) Greg Olsen, Jacqui Snyder, Randy Rogers, Steve Ogle and Robert Ogle, and (back row, from left) Duane Parazoo, Linda Parazoo and Lynette Brown.



Paul Menard and Wendy Holman rode together in the Seattle "Spawning Cycle" bike ride on Sept. 19, gathering pledges for the Northwest Parkinson's Foundation as members of Team Parkinson's. Team members taking part in various rides this summer have collectively raised more than \$95,000 for the Northwest Parkinson's Foundation.

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Mercer Island, WA 98040

PHONE

1.877.980.7500 (toll-free)

EMAIL

nwpf@nwpf.org

WEBSITE

www.nwpf.org

B O A R D

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“Counting on Kindness: The Dilemmas of Dependency” / Speaker and author Wendy Lustbader, MSW, will lead a conference for Parkinson’s caregivers (family and professional) / 8:30 a.m. to 4 p.m. Oct. 7 / Sunset Presbyterian Church, Portland, OR. To learn more or register, call Parkinson’s Resources of Oregon at 800.426.6806 or visit www.parkinsonsresources.org.

Booth Gardner Parkinson’s Care Center Quarterly Meeting / Featuring Alida Griffith, M.D., on “Cutting Edge Research in Parkinson’s Disease” / Anthony Mosley, M.D., on “Clinical Research at the Booth Gardner Parkinson’s Care Center” / and Shirley Glazer, SLP, on “Management of Speech and Voice Disorders in Parkinson’s Disease—Revisited” / 3:30 p.m. to 6 p.m. Oct. 7 / Evergreen Hospital and Medical Center, Kirkland, WA / Call 425-899-3000 for registration and directions.

Parkinson’s Telehealth Series / John Roberts, M.D., Parkinson’s specialist with Virginia Mason Medical Center in Seattle, WA, will discuss treatment options for Parkinson’s. The presentation will be aired live to communities connected to the telehealth network. Viewers in connected communities will be able to ask questions. 2 p.m. (PST) Oct. 18. Call 1.877.980.7500 to learn more.

Annual Washington APDA Symposium / Nov. 6 / West Seattle campus, South Seattle Community College, Seattle, WA / Find program details in the fall *APDA Parkinson Pathfinder*, online at www.waparkinsons.org or by calling 206.543.5369.

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