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Maine PD News

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What about medical marijuana and PD?

by Bill Stamey, M.D.

I am asked almost daily about medical marijuana for PD. For the purposes of this essay, I will call marijuana "MJ." I am not alone in being asked this question; my movement disorder colleagues around the state have had similar experiences. The National Parkinson Foundation (NPF) states that, among physicians surveyed at their 40 NPF Centers of Excellence, 95% of neurologists reported PD patients had asked for a medical MJ prescription, and 80% of patients with PD had used MJ, whereas only 10% of physicians had recommended it, and 75% of physicians felt that MJ would have a negative effect on short term memory (1).



MJ in PD is a complex subject, and to be clear, under Federal law, is not legal, though a couple of drugs derived from MJ are: dronabinol (Marinol) and nabilone (Cesamet).

Since President Nixon signed the Controlled Substances Act in 1970, MJ has been classified by the FDA as a Schedule I drug, defined as a drug with a "high potential for abuse ... no currently accepted medical use." Schedule I designation is an impedance to scientific study for potential medical use. In other words, it is hard to conduct trials with this designation. However, trials or not, in Maine medical MJ has been approved since 1999. Under the Rules Governing the Maine Medical Use of Marijuana Program (MMMP), there are certain indications for the use of medical MJ, but PD is not one of them (2). Regardless of medical law, the recent citizen initiative to legalize recreational MJ passed. Access is legal (within certain limits) for adults under State

law. Many people with PD tell me they will be, or have already been, trying MJ. I have heard from people who report improved tremors and dyskinesia, some who have better sleep, some who say it does nothing, and some who do not tolerate it due to side effects. This is all anecdotal. What does science show?

The pharmacology of MJ is complicated. Most MJ strains come from two species of plant: *Cannabis sativa*, and *Cannabis indica*. Over 60 neuroactive compounds have been identified in MJ. Many of these work on the brain's endocannabinoid system, which is known to affect neurotransmission in the motor system of the brain, and there are many receptors in the basal ganglia – the main region dopamine is used. Endocannabinoids also are implicated in the control of mood, cognition, and pain. THC is found in a higher concentration in *sativa* plants, and is thought to be the primary psychotropic compound in MJ, the cause of paranoia, and other psychotic features. Cannabidiol (CBD) is a non-psychoactive substance, and is in higher concentration in *indica* strains. CBD has sedating, anti-emetic (helps with nausea and vomiting), and analgesic (pain) properties.

There is a lot of data about MJ in the basic science, and some in the medical literature about people taking MJ as an intervention. The type of study is important. Case reports may describe the experience of only one, or of very few patients with an intervention. An open label study is one in which there is no placebo, and the concern is that there may be bias, whether conscious or unconscious, on the part of the participant or the evaluator. In other words, if you know you are taking an intervention, you may believe you are changed by that intervention. Belief is important in the mind-body connection, and is the basis of the “placebo effect.” This is not to say that open label trials are useless, just that they must be interpreted with caution.

In one open label observational study at an academic movement disorder center, 22 PD patients were tested at baseline and at 30 min

after smoking MJ (3). The group showed improvements of tremor, rigidity, and bradykinesia in the UPDRSIII motor score used to evaluate PD patients, improving on average from 33.1 to 23.2 after consumption. If the score means nothing to you, know that the UPDRSIII score ranges from 0, with no PD, to 108, the worst PD imaginable. Therefore, the lower the UPDRSIII score, the better, sort of like golf.

The scientific approach to prove the effectiveness of any drug or intervention would be to progress through trials which demonstrate safety with a few patients (phase I), a larger group (phase II), and efficacy (phase III). In all phases, side effects are noted. In the phase III trial, the gold standard is the well-designed, randomized, double-blinded, controlled trial (RDBCT). In this trial participants must meet certain criteria for entry, and take either the trial intervention (for example, MJ), or a placebo. When people participate in studies, they are evaluated periodically to measure effect. A study is double-blinded when neither the study participant nor the evaluator know who is taking the intervention, and who is taking placebo. All data is eventually collected, interpreted, described and discussed in peer-reviewed medical journals. Doctors then read these articles and use their own skills of critical analysis to interpret the study. There are many variables at play, and a study may raise many questions. A single study may need additional support. A study that shows unique results should ideally be repeated by a separate group of researchers and study participants.

Larger study groups tend to provide more valid information about how people may respond generally because researchers are able to average a lot of data. Unfortunately, there is very little study data with MJ in large groups of PD patients or the RDBCT. Some authors have tried to go through the available data and produce a conclusion from several case reports and studies. For example, researchers looked at a collection of papers and showed

that oral cannabis extract (OCE) is probably ineffective for treating levodopa-induced dyskinesias (4).

Papers such as this led a subcommittee of the American Academy of Neurology to review multiple studies involving the use of MJ in the treatment of neurologic diseases (5). These studies showed that, in PD, treatment with the compound 9-tetrahydrocannabinol (THC) or with OCE are probably ineffective in treating tremor. OCE is probably ineffective for treating levodopa-induced dyskinesias in PD. They noted that among 34 studies the risk of serious adverse psychopathologic effects was about 1%. The authors reported that comparative effectiveness of medical MJ vs. other therapies is unknown for these indications.

In 2015, researchers published in the journal of the Movement Disorders Society another summary of study data, which concluded that MJ is probably not helpful for the treatment of tremors or dyskinesias (6).

There are concerns about MJ in PD beyond paranoia and psychotic features. While MJ might help with pain, insomnia, nausea, and weight loss, it might cause side effects. MJ

use decreases reaction time, has negative effects on cognitive and executive function, may lead to risky behaviors, create apathy or a lack of motivation, cause dizziness and blurred vision, cause mood or behavioral changes, affect balance, or just make a person sleepy. All of these are already potential problems in PD, which may be exacerbated. Chronic use of MJ has been shown to unmask underlying psychiatric disorders. Finally, smoking MJ is associated with increased risk of lung cancer and stroke. Vaporizing MJ is also probably not a healthy option.

In conclusion, there is not much data to support the use of MJ in PD, but there is a huge political will to legalize it, and a growing feeling among patients that it is safe to try. This may not be true. As with any neuroactive substance, large RDBCTs are needed to demonstrate benefit and safety. In addition, if effective, MJ, which is a very potent compound, would ideally go through approval process with the FDA. MJ has not, and physicians do not have label or dose recommendations, timing instructions, or adequate description of all potential side effects.

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Maine PD News is a free online quarterly newsletter for Parkinson disease patients and their caregivers in Maine, though we welcome members of the PD community around the globe.

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PD Pearls



If levodopa is any higher than a low starting dose, it should never be stopped abruptly, but tapered slowly over at least 3 days to minimize risk of very rare, though severe, and possibly life-threatening side effects of rapid withdrawal.

All PD meds should ideally be tapered off, not stopped abruptly, if it is time to stop.

Sinemet is a portmanteau word built from Latin roots which mean "without vomiting." Levodopa given without carbidopa may cause nausea and vomiting.

Carbidopa, can be prescribed alone if needed to decrease nausea when the amount in Sinemet is not strong enough. It is known by the brand name Lodosyn.

High school senior helps the Maine Parkinson Society

by Bill Stamey, M.D.

We recently ran a notice in the Upcoming Events section of Maine PD News about a fundraiser to support the Maine Parkinson Society (MEPS). The event raised money by sale of movie tickets at Smitty's Cinema in Topsham on the 27th of October.

Cameron Loeschner, a senior at Mount Ararat High School, organized the event. He tells me this was a part of his capstone project. Thinking of his grandfather, Cameron noted, "I wanted to do something that would benefit Parkinson's disease." Seniors work with a mentor on the capstone, and Cameron reports his mentor was Amy Berube, LMSW at Mid Coast-Parkview Health. "We worked together, looked up different ideas, and came up with a movie night." They raised over \$620 and donated it MEPS. "I didn't know much about Maine Parkinson's at first, but I wanted to have more of a direct impact on my community. Amy helped me with selecting them, and I contacted Morgan Knox. Morgan was a big part of it."

The event apparently went well, and Cameron says, "I'm, glad I did it. To be the one that set that up was really cool. It made me see what I want to do in life, and it felt really good to do something that may help my grandfather.

James Pope, Cameron's grandfather, tells me "I am very proud of Cameron for spotlighting something this close to home. It means a lot that he thought of me, and that he kept the money right here in Maine. There are all kinds of programs around the country, but very few right here in our state."

Cameron has been accepted to Thomas College, in Waterville, Maine, and plans to major in communications with a concentration in public relations and marketing. He's off to a good start. Thank you, Cameron!



In Sickness and In Health, a reflection on Ken Nye

by Bill Stamey, M.D.

In my exam room there is a piece of writing hanging on the wall which I often find people reading as I enter. Many feel moved by the words. Some knew Ken Nye, who put these feelings down for us to read. In those cases, I am treated to a story about Ken. They tell me how he helped them, how he was there when needed, how he taught them, how he was just a great guy, or a great friend. "He cared ... he turned my life around ... he listened ... he was a mentor." I have heard so many good things standing there. These days the stories and comments are always in the past tense, because Ken himself passed in October 2012.

Ken was someone worth knowing.

I met Ken and his wife Ann in 2007 when I first moved to Maine. They came to my old office in Westbrook for a consultation regarding a diagnosis he had carried since ten years earlier, when he was 55. I remember that Ken told me he had enjoyed singing with the church choir, and had been having difficulty with his voice. The falsetto, in particular, was breaking up, and this was not normal for him. He thought it might be related to aging. Over the next year, however, his left arm did not swing while walking. There was a gradually worsening stiffness in his muscles. When he would extend the arm to reach for something, he felt a ratcheting sensation in the elbow. He learned he had Parkinson disease and began medications, which did help symptoms, but did not slow the progression.

Ken had been a very active person all of his life and did not want this disease to slow him down. He was educated, and an educator. He taught high school in Evanston, Illinois; was department chair of one of their four schools (combined studies department - English and social studies); and became principal of Rumford High School, before taking the same post Yarmouth High School. During that stint, he was diagnosed with PD.

One of his first concerns with PD was what this would mean for others. He worried about his wife, Ann, his family, his students. However, he quickly decided to be open about PD. He would still go on to become an assistant professor in the graduate program at USM, where he taught teachers who wanted to be principals and athletic directors. Ken started the advisor/advisee program, independent study, and community service, which many other high schools picked up and still use. He was a Renaissance man. For about five years in Yarmouth, he co-taught a senior English class while he was principal. While he was at USM and after he retired, he gave lectures on poetry at middle and high schools, and gave area schools class sets of some of his books for poetry units. Student poetry and writing awards at Yarmouth and Freeport High Schools are known as the Ken Nye Awards. Ken and Ann were very involved in Maine PD community. They were members of the Young Onset PD Support Group, and initially attended meetings at the Curtis Library in Brunswick. The group moved to the Nye residence in Freeport for a time, before heading on to South Portland.

Ken's disease progressed, and he became less able to do some of the things he enjoyed. He had always been the kind of guy who would climb up on the roof and fix something himself. Ann and Ken had raised their family on a 20-acre farm in Rumford Center with Black Angus cattle, bees, and gardens. During the last 30 years of his life, living in Freeport, Ken was just as ambitious. With PD, he eventually had to find interests that conformed to some of his limitations with balance, and the newest devil to find him, insomnia. In the sleeplessness of night, he found poetry. I remember him telling me he attended a weekend meeting and took a chance by sharing his writing with others. He found that he was able to express himself. Ann looks back now and tells me that his four books of poetry probably happened because of Parkinson disease. It was an outlet he could use, and it meant a lot to him. I would say it meant a lot to others too.

Ken also enjoyed casting and painting small figures of soldiers, primarily of the British Empire, but also of the U.S. Civil War. He had collected similar figures as a boy, and took up creating his own in the mid-1980s after learning the craft from a friend and former professor. As time went on, he produced these for family, always trying faithfully to define the uniform, the character, the era. I saw some of the tiny soldiers and was impressed by the skill and patience each had taken. I was more impressed by the body of knowledge needed to create them. He loved history, and enjoyed not just the factual side, but reading historical fiction, twice through the 20 novel set of the Aubrey-Maturin series of nautical novels by Patrick O'Brian. I would often find him in an exam room with book in hand, Ann at his side, her own book open. The two were a perfect pair.

Ken inspired others. He was a peer mentor, was active with PD support groups, attended fundraisers, and was involved with planning and coordinating events. He attended PD exercise classes at Ocean View and supported exercise as an intervention. Even after he had disease for many years, he built a shed at his home, was the president at his church, and head of the Steeple Committee. Ken enjoyed sailing and when it became too unsafe, regretted that loss in a rare moment of sadness with his condition.

I think the disease was particularly hard in the last few years, but there were complications from other very serious health concerns. Falls, spills, injuries, and concurrent illnesses took their toll, and at times Ken was hospitalized or met acutely with the medical profession. Still, Ken was an inspiration to me. He took most of this in stride and with dignity. He laughed once recounting an ER experience for a painful fracture, and truly appreciated those who had cared for him. This was a recurrent theme. Many times I have thought about this, and hope I have learned from him something about how to face illness.

I have to give a small confessional as well. I fumbled about on the day he told he was close to dying. There were tough decisions, and rather than briefly extend his life with difficult treatments for an illness outside of PD, he had decided to stop and take only comfort measures. I thought he was feeling desperate, and said so. I thought he was giving up. I had it wrong. With Ann and their daughter Amy at his side, he told me "No, I am not desperate. I am taking control." And he was. At Ken's celebration of life a few months later, I was moved again, to hear about the life of Ken Nye from his closest friends and family. He touched so many, and we all better for it.

In Sickness and In Health

by Ken Nye (included with permission from Ann Nye)

Eleven years ago, when the doctor looked me in the eye and said, "Mr. Nye, you have Parkinson's disease," it never occurred to me that the "you" to whom he was speaking included another person who wasn't even in the room.

But as the years have passed, and this chronic, progressive disease has become more of a nuisance, I have come to understand that "you" meant not me alone, but Ann, too.

There are buttons I cannot button, zippers I can't get started. There are organizational tasks I used to do routinely that have now been passed to Ann. There is medication to be sorted into little square cups of a daily pillbox, ready to be imbibed every three hours through the day so I can function almost normally. There is a joint pocket calendar maintained by Ann to be sure I get where I am supposed to be, when I am supposed to be there - and she is the driver. There are foods that I shouldn't eat, but sometimes do; things I shouldn't do, but threaten to. So she watches over me, like a nurse working a 24 hour shift that never ends.

But she never complains. And when I get angry or frustrated that we have to deal with all of this pain-in-the-neck crap, she brushes it off, cools me down, never offering pity or even sympathy, just acting as if this is all part of life and the promise we made to each other a long time ago. She meant every word of that "in sickness and in health" stuff 45 years ago.

"Mr. Nye, you and your wife have Parkinson's disease."

She never blinked an eyelash.

Am I a lucky man or what?



PD and diet

by Bill Stamey, M.D.

Recently, at a talk regarding PD in Brunswick, a common question came up as to whether diet influences the disease. I gave sort of a stock answer, in that no dietary intervention has been proven to treat the condition, but that there are several points to consider about diet (see, for example, the article in MPDN about constipation). Here, I will review some other issues.

Not surprisingly, the data seems to favor eating a healthy diet. Many ask, "What is a healthy diet?" That is a complex question. Most studies seem to point to something akin to the **Mediterranean diet**, which contains significant olive oil, grains, vegetables, fruits, potatoes, seeds, nuts, legumes, and fish; and generally lower intake of red meats, poultry, dairy, and alcohol (though small amounts of red wine can be beneficial). Numerous observations have been made regarding longevity and the reduction of cardiovascular or metabolic diseases among those who eat this way.

These observations have led investigators to study diet for improving health and preventing disease. The **DASH** diet (Dietary Approach to Stop Hypertension) is based on the Mediterranean diet, though using relatively more low-fat dairy and less fish. The **MIND** diet (Mediterranean–DASH Intervention for Neurodegenerative Delay) takes elements from both diets and increases consumption of berries, nuts, and beans. A meta-analysis, which is an in-depth review of multiple similar studies, looked at 14 prospective trials totaling thousands of participants in the U.S., Greece, Europe, and Australia (1). In these trials, people were followed from 3.7 to 18 years and had lower rates of Alzheimer disease. One of

the studies, conducted by the World Health Organization Study Group, followed more than 130,000 health care professionals for 16 years, and showed that those who ate a Mediterranean diet had lower rates of PD (2). Studies of older people who followed the MIND diet showed less cognitive decline at a 4.7-year follow-up (3, 4).

The PREDIMED study included 522 people aged 55-80 who were at high risk for cardiovascular disease (5). These people were randomly assigned to one of three diets: a Mediterranean diet with supplemental extra-virgin olive oil (EVOO), a Mediterranean diet with supplemental mixed nuts, or a regular diet with reduced dietary fat. Heart attack, stroke, and death from cardiovascular causes were all reduced in those eating the diet with EVOO, and those people scored higher on the Mini-Mental State Examination (MMSE) and the clock-drawing test at 6.5 years.

In a four-month study, 124 participants with high blood pressure started either a DASH diet, or aerobic exercise and a DASH diet (6). Those with the combined approach had better psychomotor speed (basically, a measure of the time for the connection between thought and movement). Though none of these patients were noted to have PD, the finding is interesting because one of the problems with PD is a decrease in psychomotor speed.

In a large population study of 1,260 people with cardiovascular risk factors for dementia, the Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGER) (7), participants were chosen who had

average, or slightly lower than average, cognitive performance for age, and were randomly assigned to a combination of diet, exercise, cognitive training, and vascular risk monitoring, or to a health advice control group. The diet included fruit, vegetables, whole-grain cereals, low-fat milk, low-fat meat, low sugar, margarine instead of butter, and two or more portions of fish per week. Participants underwent a group of 14 neuropsychological tests. During the 24-month follow-up period, composite scores were 25% higher in the intervention group than those who received advice alone. Executive functioning, which includes problem solving, critical analysis, and processing speed, were better in the intervention group.

There is some data that **caffeine** consumption may decrease risk of PD. Over 8,000 men were followed for 30 years in the Honolulu Heart Program (8). Incidence of PD decreased with amount of coffee intake: the more coffee consumed, the lower the risk of PD. Similar data was found among those that consumed caffeine from sources other than coffee, such as tea. The National Institutes of Health-AARP Diet and Health Study prospectively examined whether caffeine intake was associated with lower risk of PD among over 300,000 men and women (9). The effect was equal, with no gender difference. Again, the more caffeine consumed, the less likely one was to develop PD. Multiple other studies have shown similar results. Caffeine is hypothesized to protect dopaminergic neurons by antagonizing a neuronal receptor known as adenosine A2A (10). Animal models have shown that chemicals which inhibit A2A can protect dopamine-containing neurons, and caffeine has been shown to improve some motor function in PD (11). The effect on A2A receptors has led to a new class of

investigational drugs, such as istradefylline. Of note, caffeine is also a CNS stimulant which may help with daytime sleepiness, alertness, and cognitive function.

Curcumin is an active ingredient of turmeric. In volumes used in cooking it is non-toxic. It is able to cross the blood-brain barrier. Curcumin binds to mutant α -synuclein (see the article in MPDN about alpha-synuclein), and thus may prevent aggregation and formation of Lewy bodies (12, 13).

Mucuna pruriens, the velvet bean, aka cowhage, has long been used in traditional Ayurvedic medicine for Parkinsonism. In 1937, researchers isolated levodopa from the beans (14), though this was prior to a scientific understanding of the link between levodopa and Parkinson disease. From 1978 to 2000 there were at least three open label studies (in which patients knew they were taking the study drug instead of taking a blinded pill, which might be treatment or placebo) (15, 16, 17). These studies reported significant improvements in Parkinsonism for up to 20 weeks. In 2004, London researchers demonstrated with eight PD patients that single doses of immediate release 50/200 mg carbidopa/levodopa were not as fast in onset of effect as a 30 g *mucuna* preparation (34.6 v 68.5 min), and this was consistent with time to peak blood concentration (18). Average ON time was 37 minutes longer with 30g *mucuna* than carbidopa/levodopa, and plasma concentrations 110% higher, implying the amount of levodopa was much higher in the *mucuna* preparation, essentially double the dose of the carbidopa/levodopa. Each of the eight patients were trialed with *mucuna*, and two complained of mild nausea, whereas one dropped out of the study due to "short lasting vomiting." Acute side effects of levodopa are

known to include nausea and vomiting. This is the reason carbidopa is combined with the drug in tablets. The authors suggested that domperidone might be combined with *mucuna* to prevent these side effects, and that larger randomized trials should be undertaken to evaluate *mucuna*. *Mucuna* is unfortunately still not endorsed by such trials, and no standard measurement of levodopa derived from the bean is as yet available.

Fava beans, *Vicia fava*, aka the broad bean, have been known to contain levodopa since 1913 (19). One open-label study comparing 250 g of cooked fava with 100 mg synthetic carbidopa/levodopa showed lower peak plasma concentrations after eating the beans, though the effect was similar to synthetic levodopa (20). Unlike *mucuna*, however, the concentration of levodopa in fava beans is very low, and therefore would require a large number of beans to reach a benefit. Likewise, there is no standard for the amount of levodopa in fava beans, making dosing unpredictable. In addition, some people with a genetic deficiency of glucose 6-phosphate dehydrogenase may react to eating fava beans with favism, a form of hemolytic anemia.

Levodopa competes with proteins for absorption and should be taken one hour before, or two hours after meals that contain protein.

Finally, because levodopa, the active ingredient in Sinemet, Duopa, Stalevo, Rytary, and Parcopa competes with amino acids for absorption in the GI tract, it should be taken one hour before, or two hours after, meals containing protein. This creates certain problems with timing of medications and foods. It is better to stick with a consistent time to dose meds, and plan meals around this. Often, patients report they have stopped eating proteins, or moved all protein to the nighttime. There are problems with this approach as well, because we need proteins, but the all at once approach may not be the right path.

I discussed this issue with Alison Fernald, RD, LD, CDE notes that "an average person can only digest 25-30 grams of protein at a time, and a 140 pound person needs at least 50 grams of protein a day. So, they have to break it up, and not eat it all at once." For those who need detailed instructions, Alison suggested a way to tackle this

might be the protein redistribution diet, which is included below in the next article.

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Protein Redistribution Diet

by Alison Fernald RD, LD, CDE



Protein needs vary depending on height, weight, gender and age, but generally, 1.2 grams of protein per kilogram (2.2 pounds) of weight is adequate to preserve lean muscle mass. The diet below provides approximately 85 grams of protein, enough for a person weighing about 160 pounds.

7:00 a.m. Take medication – coffee or tea if desired, which have no protein unless milk is added

7:45 a.m. Breakfast of 2 eggs or ½ c. cottage cheese, or nuts if tolerated; a piece of fruit; a slice or two of whole grain toast with 2 tsp. butter or vegetable oil spread; and 6 ounces milk (cow, almond or soy). (22 g protein)

11:00 a.m. Take medication

12:00 p.m. Lunch with 1 cup of vegetables of choice, including beans if desired; 2 oz. tuna or other protein of choice; 2/3 c. brown rice, stone ground bread or quinoa (a whole grain that has extra calcium, is less refined, and has more fiber than white bread). Don't forget to add some fat to the meal – mix tuna with olive oil mayo, or drizzle oil and vinegar on vegetables - 1/2 to 1 tablespoon is fine. (about 20 g protein)

4:00 p.m. Take medication

5:00 p.m. Meal with up to 4-5 ounces of protein: chicken, fish, beef, pork (lean) or a bean dish like chili with lots of chopped vegetables (at least 1 c.), and a salad, or sautéed onions with the protein (or any vegetable you like), and ~1 c. of whole grain/starch of choice – ex: pasta, peas and corn, brown rice, whole grain bread or roasted red skin potatoes, or a baked potato. Add a little healthy fat – cold pressed oils like grape seed, walnut and extra virgin olive oil have potential to be slightly better than the mass produced vegetable oils (we all need some fat in our diets, just avoid deep fried choices, or pre-deep fried items like tater tots and chicken nuggets). (about 35 g protein)

8:00 p.m. Snack on 1 ounce sharp cheese and/or nuts and fruit, some non-starchy vegetables - marinated cucumbers, celery and carrot sticks, or some whole grain crackers like "Hint of Salt" Triscuits or other fiber-rich cracker. (about 10 g protein)

It generally takes 3 to 4 hours for a meal, including its protein, to be completely digested. If you take a fourth dose of medication at night, you will need to time the snack accordingly, i.e. have the snack at 2:00 p.m., or put it off until 10:00 p.m., after you have taken your nighttime dose at 9:00 p.m.

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Parting thoughts

I want to extend a thanks to all those who contributed in articles, interviews, or insights. Also, I would like to thank readers. Your kind words have told me that you find this useful. If there is some topic in the realm of Parkinson disease you want to know more about, please let me know. If it is something I can address, I will be happy to try.

Visit MPDN online at www.maineprdnews.org to see upcoming events.

If you know of a PD event in Maine, please let me know.

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Be well, and stay out of trouble,

Bill Stamey, M.D.

