Connecticut Parkinson's Working Group Newsletter

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This edition is an eclectic collection of articles. The lead article describes a new MAO-B inhibitor, which is about to be released; this should interest everyone. The next piece explains the difference between PD and parkinsonism, often a confusing issue. Next we learn of the status of GDNF clinical trials; here we need to come to a personal decision on what is right.

Jeff has provided us with an interview with Dr. John Seibyl of MNI, and we revisit the important question of compulsive behavior by Mirapex takers. Finally, a piece taken from *Sports Illustrated* on a well-known biker who has PD. — ***Stan***

RASAGILINE—SECOND-GENERATION MAO-B INHIBITOR FOR THE TREATMENT OF PD

Rasagiline(RSG) is a second-generation irreversible monoamine oxidase type B (MAO-B) inhibitor for the treatment of Parkinson's disease (PD) and the product of joint development of the Danish pharmaceutical company Lundbeck and Teva Pharmaceutical of Israel.

Following successful completion of phase III trials in patients with early and advanced PD, regulatory approval for RSG was filed for in both the US and Europe. In February 2005 the European Medicines Agency (EMEA) gave final marketing authorization for its use as initial monotherapy in patients with early PD and as adjunctive treatment in those with moderate-to-advanced PD. EMEA approval followed earlier approval in Israel at the beginning of 2005.

RSG will be jointly marketed under the brand name ***Azilect*** in Europe and is expected to start in the second half of 2005. In the US the drug will be promoted under the brand name ***Agilect***.

IMPROVED TREATMENTS ARE NEEDED FOR PD: PD is a disease of the basal ganglia arising from the loss of dopamine (DA)-producing cells in the substantia nigra. The loss of DA, a major neurotransmitter, produces the characteristic motor symptoms of PD. Current treatments aim to address the deficiency in DA production. Levodopa, a naturally occurring amino acid, has been the mainstay of treatment for PD for the last 30 years. Although it remains the gold standard, over time as many as three-quarters of patients on levodopa develop long-term side effects such as dyskinesias (involuntary movements), "on-off" motor syndromes and mental disturbances such as hallucinations.

While the ultimate goal of treatment is to cure the disease, new drugs that can offer improved symptomatic relief are greatly needed. MAO-B inhibitors are an important new class of drugs for symptomatic relief of PD. As their name implies they inhibit MAO-B, an enzyme responsible for the breakdown of DA. By preventing DA breakdown, MAO-B inhibitors may help reduce DA loss and delay the need for treatment with levodopa, thus increasing its clinical utility.

Currently only one MAO-B inhibitor, selegiline

(Eldepryl), is approved for treatment of PD and so approval of RSG, a second-generation compound, would expand treatment options with this class of agents. Compared with selegiline, RSG exhibits more potent in vivo inhibition of MAO-B, and because its metabolism is different it appears devoid of the undesirable amphetamine-related effects seen with selegiline. These include increases in blood pressure, sleep disturbances and euphoria.

CLINICAL TRIALS SUGGEST RSG MAY DELAY PROGRESSION OF PD. The clinical efficacy and safety of RSG has been evaluated in a series of phase III trials, in which it was administered alone or in combination with standard levodopa therapy. In the 26-week TEMPO trial, in which RSG monotherapy was compared with placebo in 404 patients with early PD (not on levodopa), treatment with RSG was significantly more effective than placebo with respect to change in the Unified Parkinson's Disease Rating Scale (UPDRS) score from baseline to endpoint. When treatment was continued for a full 12 months, patients in the two active treatment arms (1mg and 2mg RSG) of the TEMPO trial showed a smaller decline in total UPDRS compared with those whose switch to active treatment was delayed by 6 months (placebo group). These findings suggest that early initiation of RSG may slow the progression of impairment associated with PD.

Efficacy has also been demonstrated when RSG is given in conjunction with levodopa. In the 26-week placebo-controlled PRESTO trial involving 472 PD patients, RSG 1mg/day or 0.5mg/day given in conjunction with levodopa significantly reduced off-time compared with placebo as well as improving motor function. More recently data was presented from the 697-patient LARGO trial, in which PD patients were randomized to 18 weeks of treatment with either RSG, entacapone (a COMT inhibitor) and placebo in addition to levodopa. Consistent with the PRESTO trial, treatment with RSG reduced off-time, with the magnitude of response similar to that seen with entacapone. In this study RSG was noted for its long duration of action.

Interestingly, in an ancillary analysis of data from the LARGO trial, which looked specifically at efficacy against freezing, RSG achieved a significant improvement in freezing compared with placebo.Overall the clinical trial results suggest RSG is a well-tolerated drug with an incidence of adverse events similar to placebo.

MARKETING COMMENTARY. Although there have been some improvements in the treatment of PD over the past decade, there remain many unmet needs for patients with PD. Significant market opportunities therefore exist for drugs that can offer greater symptomatic improvement over existing agents as well as conferring some degree of neuroprotection. The 12-month data from the TEMPO trial suggest RSG has potential to slow PD-related impairment, which if sustained with long-term use would be an important development. Analysts are optimistic that the drug, which is administered orally and does not require titration, will be an important addition to the range of drugs available to treat patients with PD.

CPWG Golf Outing
Tom Sullivan

On a day where the temperature reached the mid-90s, CPWG held its first golf outing at Portland West Golf Course, which was the best possible host. While the course record was never threatened, the 12 golf participants acquitted themselves well. A large number of pars were recorded and water hazards collected few golf balls. In addition, 8 non-golfers joined the event and shared drinks and conversation, after the play finished. Thanks to Jeff Lincoln, Pat Sullivan and Jackie Dorwin for their assistance in planning the event. Special thanks to Gary T. for being the official photographer. Thank you to Erica Bradley of Schwarz Pharma for her support and Jack Diamond for donating prizes. Hope to see you next year.

APDA Offers Parkinson Tulip

You can purchase the "James Parkinson Tulip" from the APDA / CT Chapter. This is a beautiful red tulip with white edges. The cost is 10 bulbs for $5.00. A special price can be arranged if you wish to plant the bulbs as a project in your town (perhaps in a distinct location with a ceremony and publicity.) For more information contact the Chapter at 203-288-0546 or *gladkt@hotmail.com.*

PARKINSONISM: NOT ALWAYS CAUSED BY PARKINSON'S DISEASE

BY MAYO CLINIC STAFF • SEPTEMBER 01, 2005

The term "parkinsonism" refers to a disorder with any combination of the movement abnormalities seen in PD resulting from the loss of dopamine-containing nerve cells. PD is the most common cause of parkinsonism. But not everyone who has parkinsonism has PD. Other causes of parkinsonism include:

- Stroke
- Encephalitis, inflammation of the brain usually caused by infection
- Meningitis, inflammation of the membranes covering the brain and spinal cord
- Progressive supranuclear palsy, a rare degenerative brain disorder
- Multiple systems atrophy, a degenerative disorder that destroys the nervous system
- Corticobasal degeneration, a rare neurological disease
- Certain medications, such as some antipsychotics and metoclopramide

Signs and symptoms of parkinsonism include:

- Tremors
- Slowed movements
- Impaired speech
- Muscle stiffness
- Loss of automatic movements such as blinking

There is no definitive test for parkinsonism or PD. A doctor may be able to make a diagnosis based on medical history, signs and symptoms, and a physical exam. However, in the early stages of a disease, it may be difficult to know if an individual with parkinsonism has PD or another condition that mimics it. The development of additional signs and symptoms and the progression of the disease may help with the correct diagnosis.

Treatment of parkinsonism is directed at the underlying cause when possible. Treatment may also include medications to manage signs and symptoms of parkinsonism.

JUDGE REJECTS PATIENTS' SUIT TO GET TEST DRUG

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A federal judge has denied a request by two people with PD that he order Amgen to continue giving them a drug they used in a clinical trial that the company discontinued.

The lawsuit raised questions about the rights of patients in clinical trials. The patients accused Amgen, the world's largest biotechnology company, of treating them as ''mere guinea pigs'' and argued that the company had a legal and moral obligation to continue the treatment, which they said had eased their symptoms.

But Judge P. Kevin Castel of United States District Court in Manhattan ruled that Amgen was under no contractual obligation to continue supplying the drug. He said that the informed consent forms signed by the patients before participating in the trial explicitly acknowledged Amgen's right to terminate it.

While it is not illogical for participants in a trial to assume the company would continue testing the drug, Judge Castel wrote in a 21-page opinion, ''that is a far cry from establishing a contract by which Amgen bargained away the freedom to terminate the research trials in its sole discretion.''

Alan Milstein, the lawyer representing the patients, said he was considering options, including an appeal. Last September, Amgen stopped giving the drug, called glial cell line-derived neurotrophic factor, or GDNF, to all clinical trial participants, about four dozen people. The company said the drug had not proved meaningfully better than a placebo and might even be dangerous.

But some of the patients, backed by PD patient advocacy groups, implored Amgen to keep supplying them with the drug, saying that in some cases it had immensely improved their condition, even given them their lives back. Some doctors who treated the patients in the trials took the patients' side, arguing that the drug worked and the safety risks were overblown. But other doctors involved in the trial agreed with Amgen.

Two patients, Robert Suthers of Greenlawn, N.Y., and Niwana Martin of Harpers Ferry, W. Va., filed the lawsuit in April. They sought a preliminary injunction that would have required Amgen to supply GDNF while the legal case proceeded.

But Judge Castel denied the preliminary injunction, saying the plaintiffs were not likely to prevail in the case. He wrote that the patients' agreement was not with Amgen but with NYU, which recruited them for

the trial and treated them. Such separation of the patients from drug companies is a standard way of trying to keep the companies from interfering in trials, he wrote.

''In this case, there is no basis in fact or law to impose a fiduciary duty running from the sponsor of an independent study to participants who it does not select, has not met, and about whom it might not know the details of their medical condition,'' he wrote.

Kristen Suthers, the daughter of Mr. Suthers and an organizer of the patients' efforts to get the drug, said the decision did not seem fair. ''Amgen drove this study from beginning to end with a heavy hand and significant involvement,'' she said by e-mail, ''yet they have no responsibility to the participants?''

Andrea Rothschild, a spokeswoman for Amgen, said the company was pleased with the decision and continued to believe that withdrawing the treatment was the proper decision, made with patient safety in mind.

TWO PART INTERVIEW WITH DR. JOHN SEIBYL—PART I

Dr. John Seibyl is the President of Molecular NeuroImaging (MNI) and the Executive Director and Senior Scientist for the Institute for Neurodegenerative Disorders (IND). This is a two-part interview. In this installment, we will discuss Dr. Seibyl's background and how he came to be involved in PD research at IND / MNI. For the next newsletter we'll explore the process of scanning, techniques, science and how it relates to PD (Don't worry, we will take it slow and easy). But first, Dr. John Seibyl's (JS) background as told to Jeff Lincoln: (JL)

JL: Could you tell us how you came to be at the Institute for Neurodegenerative Disorders (IND) and Molecular NeuroImaging (MNI)?

JS: It was a long circuitous route. I hope you have enough tape with your recorder. I took my undergraduate degree at Yale in philosophy. Knowing that I was going to medical school, I thought it would be a colossal waste of time not to take advantage of the offerings of a great liberal arts university as an undergraduate. I love kids, and as an undergraduate I ran the student volunteer effort in the Yale New Haven in the Child Life Department. I organized students to spend time with the children on the wards working with the Child Life people. At this time my ambition was to become a pediatrician. I then worked for a year after graduation doing community research at the John Pierce Foundation Laboratory in New Haven doing psycho-physics research, essentially research in perception. In this particular position I was doing studies of the sense of taste. That gave me a "taste" of the research life. It was exciting to me. As a doctor-to-be, I thought that it might be something I could see myself doing somewhere down the line.

Next, I went to Case Western Medical School in pediatrics. The Dean of the Medical School was a pediatrician, and Case Western had a famous pediatric hospital. So I was all set. By the time I got to my second year of medical school, I became interested in brain functions; in particular, psychiatric disorders. I was intrigued by patients who had schizophrenia and other psychiatric disorders. I liked it so much that I decided to do my training in psychiatry rather than pediatrics. So I returned to Yale and did my residency here in psychiatry. When I finished my residency, I went to work on a schizophrenia research unit. I also became an Assistant Professor in the Yale School of Medicine. I had a wonderful experience working at the Inpatient Unit at the VA Hospital in West Haven. We were developing new treatments and medications for psychiatric disorders. This gave me a real flavor of clinical research.

Interestingly, there was a faculty member at Yale in the radiology department named Paul Hoffer, a nuclear medicine physician who was going around to psychiatrists and neurologists saying that he had new techniques for imaging the brain. Fifteen years ago, when I first saw these techniques, they were exciting to me because these were potentially important tools to understand motion disorders, which doctors don't know much about. We have medications for a number of neurological and psychiatric disorders. Some of them are effective, but it's astounding how little we know. Dr. Hoffer's imaging techniques could lead to better understanding of these disorders.

I realized early on that I had to get some additional training, so after two years of working as an Assistant Professor, I went back and did a second residency in nuclear medicine here at Yale for two years, and during that time I did the sort of standard things that nuclear medicine residents do, reading bone scans and lung scans and everything else outside of the brain. I was still working intensely with the group in psychiatry that was developing new ways to image the brain and in particular specific target sites. One of those sites happened to be a protein that sits on dopamine neurons and is called "Dopamine Transporter". We knew

from the beginning that this protein was going to be good for evaluating Parkinson's Disease (PD) because unlike all of these other disorders, we have a long history with PD, almost 100 years to understand some of the changes in the brains of PD patients Armed with that knowledge, I knew these nuclear medicine tests were designed to help us to either diagnose the disease or help to evaluate medications taken by people with PD (PWP's) . By a quirk of fate, Dr. Paul Hoffer eventually was diagnosed with PD. He was one of the first patients that we imaged with this particular technique [that he had developed – ed.] I remember sitting in front of the computer after we had completed the scan and looking at it for the first time. We saw that the scan was effective in showing us some changes. I watched my mentor look at his own brain with a scientific curiosity that was quite refreshing. He was excited as a scientist, but also as a PWP. He saw it from different angles. That series of studies took off for us.

Meanwhile, I joined the radiology department as my primary appointment in the Yale School of Medicine with a secondary appointment in psychiatry. I also continued to do clinical nuclear medicine and ran a research lab called the NeuroSPECT Center, entombed deep in a basement with no windows. That lab was started by Dr. Hoffer, and when he stepped down from the lab, I took it over. We were fortunate to be able to expand into new space. About the time that we were beginning to image PD, I realized that I needed to have an expert neurologist working together on the project because my expertise at that time was in the technical aspects of the imaging. The critical and key questions from the standpoint of how can these tests be used in the clinical world to help PWP's had to come from a movement disorders expert, and that was Dr. Ken Marek.

We joined up and collaborated early on in these efforts. As time went on, we were beginning to see the value of these brain imaging techniques for not only early diagnosis of PWP's who were just showing their first symptoms, but also in evaluating the progression of PD.

It was a real mystery for us that some patients progress rapidly and others more slowly. It was a clinical phenomenon that we didn't understand. We wanted to apply imaging to show some of the subtleties that would complement the information gathered in the motor exams. [such as the Unified Parkinson's Disease Rating Scale or UPDRS – ed.]. We launched a series of large disease progression trials. These studies involve imaging PWP's from all over North America in the NeuroSPECT lab at IND. Typically they were scanned once, put on a regimen of medication and then scanned several times later on. We used imaging to track changes. As the number of patients studied grew, it was clear that the logistics were getting complicated. We had people assist us in transporting people from and to airports, making travel arrangements. It got to the point where Dr. Marek and I felt that we could do the research more efficiently in the context of a private research foundation. We called this the Institute for Neurodegenerative Disorders (IND) and Molecular NeuroImaging (MNI).

IND is kind of our academic base and we continue to do these large imaging studies using patients from across North America and Europe. MNI is a company that we created to interface with pharmaceutical companies who want to develop medications for symptomatic improvement or for slowing the course of progression in patients over time. That was about 4 years ago, and it's been a successful bit of work scientifically. We are now doing the largest studies in the world in brain imaging including an 800 patient study called the Precept Study. Another study uses a medication called GPI 1485, and at this stage the value of the imaging is quite well demonstrated. It gives us another window into the brain as we move away from symptomatic improvements to medications that might target some of these ongoing processes of nerve cell loss over time. That's where our energy is. The imaging that we have developed is exquisitely sensitive and allows us to interrogate the system in a different way that complements clinical evaluations.

JL: I think most PWP's spend a little time looking back to see what happened before they were diagnosed, and realize in the end that they were symptomatic long before they thought they were.

JS: I've heard a number of PWP's tell me that, and I think it's interesting. There has been a lot of speculation on how long the process goes on before symptoms occur and how long before a definitive diagnosis is made. We don't know the answer to that. At present we have tools that are more sensitive and better able to detect these changes and a medication or treatment of some sort that can slow down or reverse the progression. That's not a bad place to be with PD.

— Jeffrey Lincoln

The second part of this interview will be in the next Newsletter.

PD DRUG CAN CAUSE COMPULSIVE GAMBLING By Mayo Clinic staff July 15, 2005

People who had never gambled before suddenly were losing thousands of dollars at casinos—sometimes more than $100,000. Men who had been content with once-a-week sex began having sex three or four times a day. Some overate, gaining 50 pounds in just a few months.

What did all these people have in common—besides their compulsive behavior? They were all taking a type of medicine, called dopamine agonists, to treat PD. Most of these people were taking one specific dopamine agonist: pramipexole (Mirapex).

M. Leann Dodd, M.D., a psychiatrist at Mayo Clinic, Rochester, Minn., was the lead author of a recent study that shed light on the surprising link between dopamine agonists and the sudden onset of compulsive behavior. In this interview, she answers a few questions about the topic.

How did this connection come to light? It started coming up at doctor appointments. Patients or their family members would bring it up. It was mostly gambling, but over half of our patients also manifested other compulsive behaviors, such as hypersexuality or overeating.

It started out as a curiosity, but then we found case reports that showed there might be some connection between this compulsive behavior and a particular type of medication. When people were tapered off these medications, the compulsive behavior would go away. It was a dramatic resolution of the behavior in many of the cases.

What's special about this drug? People who have PD are deficient in a brain chemical called dopamine. Dopamine agonists are a synthetic version of dopamine that binds to dopamine receptors in the brain. Pramipexole binds to one particular type of dopamine receptor—the dopamine receptor D3—much more than to other dopamine receptors.

These D3 receptors are highly concentrated in the area of the brain devoted to mood, behavior and rewards. It's exciting to think that future studies may reveal more about how the D3 portion of the brain may be associated with addictive behavior. Ultimately, such studies could lead the way to development of medications that curb addictive behavior in general.

How often does this side effect occur? A study of 529 people taking pramipexole for PD symptoms revealed that 1.5 percent of them had developed compulsive gambling behaviors. Our study evaluated only 11 people, all with PD, and all of whom had started gambling compulsively while being treated with dopamine agonists.

Studying this select group demonstrated the connection between the drug's strongly selective binding to the D-3 receptors and the development of compulsive gambling. We also measured how long it took for the behaviors to appear after starting on the drug. Most of the people who developed gambling behaviors did so within a month or two. Some took longer.

This drug was approved in 1997. Why are we only seeing this connection now? Compulsive gambling, hypersexuality and overeating—those are things that are not normally associated with PD medications. And they're pretty embarrassing behaviors. A lot of people are just not going to share these things with their doctors. Even their doctors may not have been aware of this possible connection.

Our key point is to let the public know that they need to tell their doctors when something's amiss. If you start behaving in a way that's out of character for you, you need to talk to your doctor, even if it's embarrassing. We also want to educate physicians about this connection, so they can let their patients know that this side effect is a possibility—it is crucial to make this association because these effects are potentially reversible.

Can people get hurt by stopping the drug abruptly? This is a very good medicine for the treatment of PD, and people should not just stop taking it on their own. This side effect is very rare. If people think they are experiencing compulsive behavior because of the drug, they should talk to their doctors about it. It might be best to taper off the drug. However, several people in our study abruptly stopped taking the drug, and they didn't experience any worrisome problems.

Has there been a lot of media attention? Yes, it's been a bit overwhelming. We even got mentioned on "The Tonight Show." Jay Leno made a joke about how Las Vegas was going to start stocking these drugs in the casinos. But this isn't a joking matter to the people it's affected. One lady, from a family with a modest income, lost $100,000. Her husband and children left her. She remarried and her second husband was about to leave her. She stopped taking the drug and within two weeks the gambling compulsion disappeared.
In a few tragic cases, people have lost their financial security and their relationships. My e-mail box has been flooded with people telling their stories. One man said his wife woke him up, crying, with a newspaper article about this study in her hand. She's had a gambling addiction for two years and now they're hopeful that they've found a possible answer.

STILL RIDING: FACING THE TOUGHEST CLIMB OF HIS LIFE,

Davis Phinney, the winningest U.S. cyclist, wages a brave battle against PD

Austin Murphy From: *Sports Illustrated,* Sept 12, 2005 v103 i10

The evening was billed as a roast of Davis Phinney, the winningest bike racer in U.S. history, but you had to wonder: Would people really zing a guy with PD? Several hundred people gathered at the Italian Athletic Club in San Francisco last Friday night to raise funds for the Davis Phinney Foundation, through which the charity's chairman intends to afflict his affliction. The thought of this once ferocious competitor ravaged by a disease for which there is no cure was sobering and sad. Surely no one would make sport of his condition.

Missing that memo, apparently, was Robin Williams, who strode to the dais and embraced Phinney. Williams proceeded to auction off the sleek Griffen road bike he'd donated between impersonations of Paris Hilton, Marlon Brando and James Brown, among others; taking bids on the bike; and sharing his slogan for an imaginary cycling squad called Team Viagra—"Ride hard, ride long!"—Williams noticed that Phinney was sipping coffee. "What are you doing with PD drinking coffee?" he shouted, backing away in feigned alarm. "That's a ballsy move!"

No one laughed harder than the 46-year-old Phinney, whose condition was diagnosed five years ago but whose hand, on this night, held steady as he drank—a marked improvement over the night I met him two years ago. He's now on meds that go a long way toward controlling some symptoms of his disease. But they don't attack its source.

When he stood to speak, the man who won 328 races in an 18-year career chose to dwell on a day he finished last. It was in the Alps in 1990, in his final Tour de France. Phinney was fried. Dropped by the peloton on the first major climb, he still had two monster passes to get over, the second being the notorious Alpe d'Huez. He was in danger of missing the time limit. (Stragglers who fail to finish a stage within a certain percentage of the winner's time are tossed from the race.)

Grinding alone up the Col de Glandon, Phinney came close to quitting. Instead, he challenged himself. He thought, Everything I've learned over all the years, I'm going to put into the rest of this ride. I'm going to get to the top of the Alpe, and I'm going to make this time limit.

And what had he learned over all the years? To question authority, for starters. After Phinney failed to finish at the 1976 U.S. Nationals—he got a flat in the first 100 meters—his high school history teacher told him, "You'll never make it as a bike racer."

Wrong. Phinney won a jaw-dropping 32 races in 1982 alone. That was the year he hooked up with the 7-Eleven team. In '86 he became the first American to win a stage in the Tour de France, a feat he repeated the next year. In '88 he won the Tour of the Americas and the Coors Classic—the latter a race he flat-out owned, with 22 stage victories and overall points wins from 1981 through '88—but was also involved in a horrific crash in the Liege-Bastogne-Liege race, plunging face-first through the rear windshield of a team car parked on the course. Though he severed a tendon in one arm and would require 35 stitches and 160 microstiches to repair cuts on his face, Phinney would return to competition 10 days later.

In the latter years of his career, when Phinney felt inexplicably fatigued on the bike, he wondered if that crash was somehow to blame. The answer would come up in 2000, when doctors discovered the PD. He retired from cycling in '93 but kept a hectic schedule as a TV commentator. In '02 he and his wife, Connie, and their two children moved to Italy for three years. It was there, while slowing the pace of his life and "getting a handle on my situation," he says, that Phinney learned to redefine victory. "When my daughter comes running up to me and jumps into my arms," he says in a video clip shown at the roast, "I note it in my mind as a victory."

Coming in last that day on the Alpe d'Huez: How's that a victory? After taking the final turn, 400 meters from the finish, Phinney tells the audience at the roast, he saw . . . workers taking down the bleachers. Crossing the line, he collapsed into the arms of team manager Jim Ochowitz, who Phinney recalls whispering in his ear, "Two minutes. You made it by two minutes."

At this the crowd erupts, but Phinney isn't finished. "Those two minutes are sort of what I'm facing now," he tells the rapt audience. "When you have a neurological disorder that has no known cure, you face continuous degeneration. We can find a solution, but we have a time limit. It's going to be tough; it's going to take everything we have to get from the bottom of the Glandon to the top of the Alpe d'Huez, before we're out of the race. But we can do it."

Living with his condition, this once fierce competitor has learned to REDEFINE VICTORY.

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Articles in this newsletter are for information only.
Any questions of treatment should be discussed with your physician.

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