

Dancing with Ned

By Patrick Maguire
LRG Member

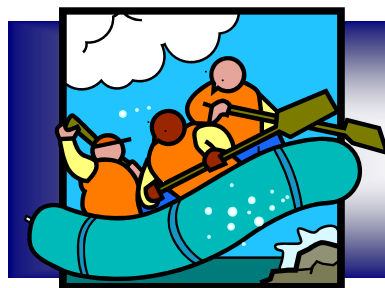
I was persuaded into taking West Coast Swing dance lessons a few years ago. Despite my two left feet, I eventually “graduated” beyond the beginner level, but I admit I enjoyed the experience no matter how goofy I probably looked attempting it. However, I’ve been fortunate to be taking another type of dance lessons for almost seven years now. To tell you this story properly I have to start at the beginning -so I thank you in advance for your patience.

In 1997 I began to experience extreme fatigue, headaches and anemia. These symptoms had come so gradually, and coupled with my strong ability to deny things I don’t want to deal with, by the time I couldn’t ignore or dismiss them any further I was quite ill. In my defense my denial was well-founded; I was only 34 years old, I never got sick—I didn’t even have a doctor. I ate correctly, exercised four to five times a week, and on weekends climbed “14ers” (Colorado has 55 peaks above 14,000 feet) or did other physical activities to make the most out of



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Battling gastrointestinal stromal tumor



LIFE RAFT GROUP

February 2010

In memory of Laura Kukucka, Clive Harrison, Tommy Crutchfield, Rita Patient & Gail Orso

Vol 11, No. 1

Bench to bedside and back again

By Drs. Margaret von Mehren
& Andrew K. Godwin
Fox Chase Cancer Center

GIST is a great example of how science can impact outcomes in cancer patients. As a malignancy, GIST has fewer changes in its genes than many other cancers. Thus, the discovery by Hirota and colleagues in 1998 that GIST contained mutations in the KIT gene was a breakthrough for the treatment of GIST. Tyrosine kinase inhibitors such as imatinib and sunitinib that target KIT, as well as PDGFRA, have significantly improved the outcomes for patients with GIST. Clinicians at the bedside of their patients have seen the impact of these drugs and have also had to face the challenges of what to do when therapy

doesn’t work. How might we do better?

One approach is figuring out before therapy what the optimal treatment for an individual patient should be. This is a form of care that uses information about a person’s genes, proteins, and environment to prevent, diagnose, and treat disease, i.e., personalized medicine. Much work has been done looking at the different sites of changes in the KIT and PDGFRA genes and correlating the benefit of imatinib and sunitinib with different mutations. Studies have also demonstrated that the imatinib dose can affect benefit in some cases, particularly the



VON MEHREN

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2009 Executive Director’s report

By Norman J. Scherzer
LRG Executive Director

There are a number of reasons to put the year 2009 behind us. The global financial crisis put retirement plans on hold, and lost jobs and homes added to the stress of families struggling to survive cancers like GIST. Fundraising for critical research was set back, and the hopes of patients for easier access to quality medical care were far from realized.

Americans continued to argue over the universality of health care, British GIST patients continued to fight for access to second-line treatments, Canadian GIST patients waged their battle for adjuvant treatment, and too many GIST patients in too many countries struggled for access to any treatment at all. GIST



SCHERZER

patients continued to die either because they could not access treatment or because known treatments had failed them.

Despite these setbacks, the Life Raft Group continued its work on many fronts. We maintained our funding to the ten investigators on our world-class research team which continued to make progress in a number of areas including preclinical validation of KIT switch pocket inhibitors and improved understanding of cell mechanisms that normally destroy activated KIT and

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More Life Fest 2010 updates!

By Erin Kristoff
Newsletter Editor

Plans are moving right along for Life Fest 2010, and our gala event: *GIST 2010: A Decade of Difference*.

Registration is now open for the June 25-27 event and we have updated our Life Fest page so that you have a “one-stop shop” for all of your Life Fest news at www.liferaftgroup.org/members_lifestest.html.

Here, you can register for Life Fest, view pictures, video and presentations from past Life Fests, download a nomination form for GIST Clinician of the Year and download materials for our new project: Tree of Life.

Register for Life Fest

Registration is now open and can be done at: www.liferaftgroup.org/members_lifestestregistration.php

You can also book a hotel room under the special Life Raft Group rate of \$129+ tax at the Hyatt Regency in Jersey City, where Life Fest will be held. Because this location is in such close proximity to New York City, we expect the hotel might sell out quickly. Please reserve your room as early as possible.

If reserving a room by phone:

Call 1-800-233-1234 and ask for a room for the Life Raft Group event at the Hyatt Regency in Jersey City. Tell the person assisting you that the code is G-LRAG.

If reserving a room online:

Go to <http://www.jersecity.hyatt.com>

You can book it on the main page, as well as the “Rooms & Rates” page. Enter the code G-LRAG in the box labeled “Group/Corporate#”

Clinician of the Year

On Saturday, June 26, 2010, the LRG will be giving out the GIST Clinician of the Year award... and you are going to help us choose the winner!

If you think that your doctor or nurse is the best of the bunch then we want to hear about it.

- Go to the Life Fest page and download and print a nomination form
- Tell us why your doctor should be the GIST Clinician of the Year and send it back by February 15
- The winning entry will be decided by a panel of GISTers and the winner will get a free trip to Life Fest where s/he can personally present the award in front of all the Life Fest attendees

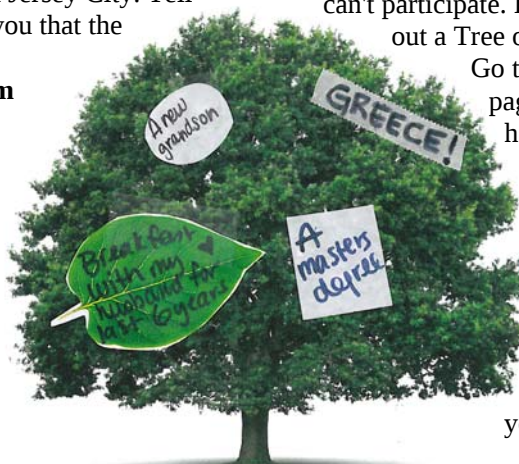
“Tree of Life”

Your “Tree” will represent all the good things that have come into your life since your GIST diagnosis.

You hear many stories in the LRG about survivors getting to see a grandchild that they never thought they would, finally going on that European vacation with their spouse, or even simply getting to retirement. This is a chance to tell the world just what this time has meant to you.

If you are a loved one of someone who has passed on, don't feel as though you can't participate. Feel free to fill out a Tree on their behalf.

Go to the Life Fest page to find out how to participate in our “Tree of Life” project, as well as view a sample completed “Tree” (pictured, left) and options to personalize your “Tree”.



The Life Raft Group

Who are we, what do we do?

The Life Raft Group (LRG) directs research to find a cure for a rare cancer and help those affected through support and advocacy until we do. The LRG provides support, information and assistance to patients and families with a rare cancer called Gastrointestinal Stromal Tumor (GIST). The LRG achieves this by providing an online community for patients and caregivers, supporting local in-person meetings, patient education through monthly newsletters and webcasts, one-on-one patient consultations, and most importantly, managing a major research project to find the cure for GIST.

How to help

Donations to The Life Raft Group, a 501(c)(3) nonprofit organization, are tax deductible in the United States.

You can donate by **credit card** at www.liferaftgroup.org/donate.htm or by sending a **check** to:

The Life Raft Group
155 US Highway 46, Suite 202
Wayne, NJ 07470

Disclaimer

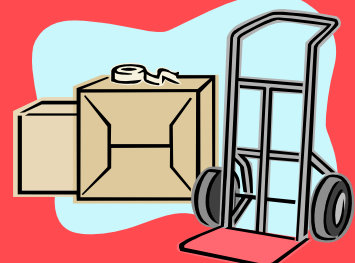
We are patients and caregivers, not doctors. Information shared is not a substitute for discussion with your doctor.

Please advise Erin Kristoff, the Newsletter Editor, at ekristoff@liferaftgroup.org of any errors.

THE LRG HAS MOVED!

PLEASE MAKE A NOTE
OF OUR NEW ADDRESS:

155 US HIGHWAY 46
SUITE 202
WAYNE, NJ 07470



GIST Research: LRG Articles on the Science of GIST

It's time to consider mutational status for resistant GIST patients: KIT exons 9 & 11

By Jerry Call
LRG Science Coordinator

This is the last article in a series discussing mutational status and resistant GIST. In this issue, we will discuss KIT exon 11 and exon 9 GIST. For an introduction and overview of this topic, see the first article which appeared in the September 2009 Clinical Trials Bulletin (You can find it at www.liferaftgroup.org/docs/ClinicalTrials/September2009.pdf)

In the not too distant future, we may have newer KIT inhibitors that overcome most types of GIST resistance. But for the present, it is becoming increasingly clear that GIST can be divided into four main types based on mutational status; KIT exon 11, KIT exon 9, PDGFRA D842V and wild-type GIST. In addition, there is another group comprising the "rare"

mutations (KIT exons 13 & 17, etc.). The different types have different initial responses to Gleevec and resistance occurs via different mechanisms. GIST patients and doctors can use this knowledge to their advantage in choosing a clinical trial or, in some cases, to consider off-label treatment options.

KIT exon 11 and exon 9 mutations represent the two most common types of mutations (wild-type GIST is technically not a type of mutation but a lack of mutations) found in GIST patients. About 60 to 65 percent of GIST patients have a KIT exon 11 mutation and about 10 to 15 percent have a KIT exon 9 mutation.

Gleevec/Sutent resistance in KIT exon 11 and exon 9 patients is different than resistance in wild-type or D842V mutations. The primary difference between



CALL

exon 11/9 types and other types is that exon 11 resistance is driven primarily by secondary mutations. Exon 9 type tumors may not develop secondary mutations as often as exon 11 tumors, but in some cases multiple secondary mutations have been noted in exon 9 patients, especially upon resistance to Sutent.

Note: In this article, resistance refers primarily to resistance after both Gleevec and Sutent. It is already fairly well established that exon 9 tumors respond poorly to standard doses of Gleevec, respond fairly well to high-dose (800 mg) Gleevec and respond well to Sutent.

Secondary mutations create a problem in GIST because they prevent Gleevec from binding to KIT and thus they cause resistance to Gleevec. Sutent effectively inhibits secondary exon 13 and exon 14 mutations, but it is not effective when

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February 2010 clinical trials update

By Jim Hughes
Clinical Trials Coordinator

Updated five year adjuvant Gleevec trial: Phase II now open at 21 sites in the United States. NCT00867113

Updated MP470 trial in solid malignancies: Phase I Updated to Ongoing as of 11/3/2009. NCT00894894

Added GDC-0980 trial in solid tumors: This Phase I trial of a combined PI3K & mTOR inhibitor is open at Dana-Farber Cancer Center (DFCI), Boston, Mass., Vanderbilt University, Nashville, Tenn. and in Sutton, Surrey, United Kingdom. NCT00854152

Added GDC-0980 trial: This Phase I

trial of a combined PI3K & mTOR inhibitor is recruiting at DFCI, Ville Juif, France and Manchester, United Kingdom. NCT00854126

Added PLX108-01 trial: Phase I study is recruiting in Scottsdale, Ariz. and Spokane, Wash. The drug is also called PLX3397. It is a combined fms KIT and Flt3 inhibitor NCT01004861

Added Gleevec trial: This Phase III study of dose escalation versus no dose escalation in metastatic GIST patients is sponsored by the Sarcoma Alliance for Research through Collaboration NCT01004861. It is now recruiting at Sarcoma Oncology Center in Santa Monica, Calif. NCT01031628.

Added STA-9090 trial: This Phase II trial of a second generation small molecule HSP-90 inhibitor is now recruiting at Dana Farber Boston, MA NCT01039519

Added Sutent trial: This phase II trial evaluating patients with bulky GIST is now recruiting at University of Alabama. This trial administers neo-adjuvant Sutent prior to prospective surgery at 14-16 weeks after trial entry. NCT01054911

Solely International

Updated Sutent trial: This phase 4 trial in China has changed to ongoing and is no longer recruiting patients. NCT00793871

Tasigna shows greater efficacy over Gleevec for CML: Will GIST be next?

By Belinda Ehrlich
LRG Staff Writer

For the past eight years, Gleevec has been the first-line treatment for adult patients with c-Kit (CD117)-positive, unresectable and/or metastatic malignant GISTs.

But for some patients, Gleevec is not the answer to their prayers. A small percentage, five to 15 percent, do not benefit from Gleevec from the outset. Others develop a resistance to the drug or cannot tolerate the side effects.

Could there be another drug with greater efficacy against GIST that is better tolerated?

In December, Novartis announced that in a large phase III clinical trial, Tasigna (nilotinib) showed greater efficacy over Gleevec in treating adult patients with newly diagnosed Philadelphia chromosome-positive chronic myeloid leukemia (PH + CML).

Tasigna surpassed Gleevec in every measure of treatment efficacy designated in the trial including prevention of disease progression at 12 months.

But what does this mean for GIST patients?

Dr. Jonathon Trent of MD Anderson Cancer Center in Houston said he believes Tasigna is equivalent to Gleevec in treating GIST. He plans to open a front-line preadjuvant and adjuvant trial in early 2010.

In the study, titled "Preoperative plus Postoperative Tasigna for patients with resectable or potentially resectable GIST," patients will receive Tasigna before and after surgery.

"The only way to know whether one is better than the other is to do this trial," Dr. Trent said. "The alternative trial would be Tasigna versus Placebo, and I strongly discouraged that trial design

from the beginning. I would not ethically put patients with advanced GIST on a study where they get placebo."

Dr. Trent said the side effects of Tasigna versus Gleevec are also important and being examined.

"If Tasigna is better then it will mean that patients were found to have a longer time until the drug quit working (longer progression-free survival) or more patients benefited for the same amount of time (percent of patients progression-free at one year)," he said.

"The side effects of Tasigna are also being looked at closely and will be compared directly head-to-head with Gleevec. If the side effects of Tasigna are acceptable, and it helps more patients for a longer period of time, then it may be approved as a new front-line therapy for advanced GIST."

Recruiting is underway for a phase III clinical trial comparing Tasigna to Gleevec as first-line

with Gleevec-resistant GIST, Tasigna was found to have promising clinical activity. Tasigna is also able to achieve a higher cellular concentration than Gleevec in tumors. It is also much more potent against wild-type KIT (tumors with non KIT or PDGFRA mutation), and therefore may work better against wild-type GIST.

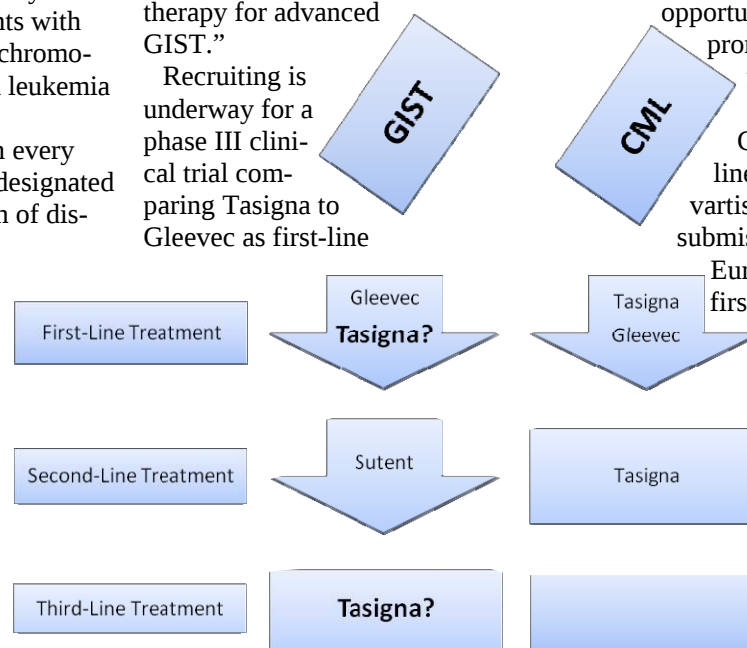
Sutent is often prescribed for patients with GIST that have stopped responding to Gleevec or who were unable to tolerate Gleevec.

"Resistance to Gleevec and Sutent is proving difficult to overcome," said Jerry Call, Life Raft Group Science Coordinator. "Preventing resistance with better first-line therapies may prove to be superior to trying to overcome resistance, at least for the near future. The opportunity to participate in trials for promising first-line therapies offers patients another treatment path."

Currently, Gleevec is the first-line treatment for CML, but Novartis is planning to file regulatory submissions in the United States and Europe for approval of Tasigna as first-line treatment. Tasigna received FDA approval in 2007 for CML patients who fail other treatments such as Gleevec.

In the CML study of 846 patients, Tasigna showed statistically significant improvement over Gleevec in every measure of efficacy, including major molecular response (MMR), complete cytogenetic response (CCyR) and prevention of progression to accelerated or blastic phase.

Tasigna was also well tolerated, so fewer patients discontinued use. The most frequent side effects were rash, itchiness, nausea, fatigue, headache, constipation and diarrhea, but most were mild to moderate in severity.



therapy for the treatment of adult patients with unresectable or metastatic GIST. Visit http://www.liferaftgroup.org/treat_trials.html to learn more about this and other GIST-related clinical trials.

In a phase I study of Tasigna alone and in combination with Gleevec in patients

Magic Cancer Bullet: Behind the scenes

With our GIST 2010: A Decade of Difference gala event approaching. We decided to look back ten years and as the question, "What's the fuss about?"

The following excerpt was reprinted from Magic Cancer Bullet: How a Tiny Orange Pill Is Rewriting Medical History by Daniel Vasella, M.D., Chairman of Novartis Pharmaceuticals.

Why was there such a fuss about Gleevec?

After all, other breakthrough drugs have surfaced over the years, but none received the massive media attention this drug did; an American cabinet member had never called a news conference to announce FDA approval for any other drug. Research scientists and physicians alike have never rallied behind a drug with such public enthusiasm as did those who were Gleevec's official cheerleaders from the day of its FDA approval. Company employees rarely give up their weekends and vacations to help expedite a product's journey to market, as was the case with Novartis personnel.

Why did the media demonstrate almost universal praise and awe for this drug at the time of its "victory in May"? Usually quick to join critics, normally proud of its fiery independence, the media this time joined Gleevec's other cheerleaders, and happily proclaimed this tiny orange capsule a "magic bullet" of sorts.

No editor of a major news organization thought it too early to do in-depth stories on the drug. In those first days after FDA approval, no editor thought it worthwhile to criticize Department of Health and Human Services Secretary Tommy Thompson or Acting FDA Commissioner Bernard Schwetz for parading Gleevec in public as a drug that could well be the breakthrough drug we have all been waiting for in cancer therapy.

Why all the fuss?

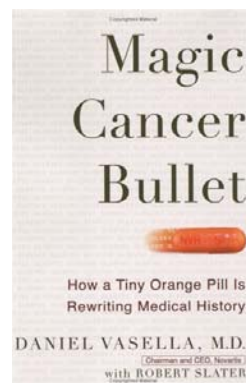
Plain and simple: the early results were nothing short of spectacular and the mechanism of action opens new horizons of cancer-targeted treatment.

Still, none of us at Novartis could have predicted such an outpouring of goodwill and enthusiasm for Gleevec as occurred in those early days after FDA approval in May 2001. Of course, from the very first hours that we saw the stunning results of the Phase I patient trials in April

1999, we began to believe that STI571 might have some very interesting positive effects on cancer patients.

And now that we have all witnessed the outpouring of excitement for the drug, expectations for its future have grown dramatically. And that is not surprising either. Gleevec did not come to life in the dark shadows; it was swept to prominence by a giant wave of media exposure that increasingly exposed all of us to questions about its possibilities.

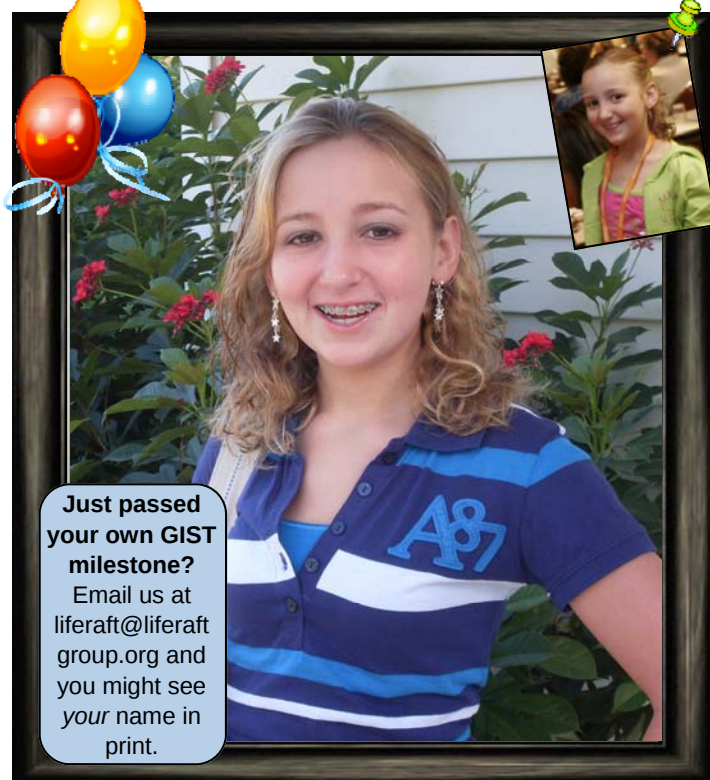
We were asked to read the future. We were asked to say precisely how this drug would work on other cancers.



And of course we could not. We could only talk about the first four years of Gleevec and even then we felt an obligation to be exceedingly cautious. We were barraged with the same question: Which of the other

cancers will Gleevec help? We had to say that we did not know, that we were intensely studying the question.

HAPPY CANCER-VERSARY TO JOSALIN DUNN!



Congratulations to 12 year old Josalin Dunn, who stole hearts at our 2006 Life Fest in Dallas (pictured above right). She just celebrated her five year GIST cancer-versary! She has come along way from where she was at seven years old— misdiagnosed with Ewing's Sarcoma and on her seventh round of chemotherapy.

GIST Research: LRG Articles on the Science of GIST

Synta opens phase II trial STA-9090 in GIST

By Jim Hughes

LRG Clinical Trials Coordinator

On December 23, 2009, Synta Pharmaceuticals announced a phase II clinical study of STA-9090 in patients with advanced gastrointestinal stromal tumor (GIST), the sixth clinical trial of STA-9090 in cancer.

STA-9090 is a synthetic, small molecule HSP90 inhibitor. It has a unique chemical structure different from earlier HSP-90 inhibitors that, like IPI-504, are first generation drugs based on a family of antibiotics called ansamycins. As a result, STA-9090 is likely to have a different toxicity profile.

HSP-90 inhibitors in GIST represent a new approach to managing resistance to

traditional therapy. A quote from George Demetri, of Dana-Farber Cancer Center (DFCI) provides the rationale, "Once those two standard drugs (Gleevec and Sutent) fail, patients have a poor prognosis and very limited treatment options. HSP90 inhibition is a promising therapeutic approach for these patients

because the mutated kinase proteins that are the cause of resistance to both Gleevec and Sutent depend upon being chaperoned and protected by the function of HSP90. STA-9090 can potentially inhibit the HSP90 function and disrupt the mutant signaling in multidrug-resistant GIST. Based on the preclinical and early clinical results seen to date,

STA-9090 has the potential to unlock the true potential of HSP90 as a therapeutic target in GIST."

In Synta's lab, STA-9090 has been shown superior to both Gleevec and first generation HSP-90 inhibitors in cell

"The most common side effects of STA-9090 observed to date have been fatigue and gastrointestinal toxicities, which were manageable and reversible." Synta Press Release

lines expressing typical Gleevec sensitive mutations. In addition, in the slide from Dr. Jonathan Fletcher of Brigham and Women's Hospital (page 9), STA-9090 is shown to be roughly 10 times stronger than first generation HSP-90 inhibitor 17AAG in a GIST cell line

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KIT

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the secondary mutation occurs in exon 17 or 18 (the activation loop). Compounding the problem, resistant patients frequently develop more than one secondary mutation. In one study of patients undergoing debulking surgery by Leigl and colleagues, 83 percent of patients with KIT mutations had secondary KIT mutations and in 2/3 of the patients there were two to five different secondary mutations.

While secondary mutations are a major problem with exon 11 and exon 9 patients, wild-type patients do not develop secondary mutations. At the present time, secondary mutations are probably not much of a problem with D842V patients either, because secondary mutations form, or become dominant, over time and patients with D842V mutations are resistant to drug treatment right from the start (primary resistance). Patients

with the D842V mutation probably don't have enough time on effective drug therapy for secondary mutations to become dominant. This may change in the future if patients have long-term responses to effective D842V inhibitors.

So, in contrast to wild-type GIST and D842V mutations, the first thing that a drug will need to do to be effective against secondary resistance in exon11 and exon 9 patients will be to *target secondary mutations*. There are several possible ways to do this and two of these are a little more developed. The two most developed approaches are:

- Inhibit KIT with a KIT tyrosine kinase inhibitor with *very broad activity* against exon 11/9 mutants with secondary mutations.
- 1. Sorafenib (Nexavar) is an example of an approved KIT inhibitor (approved for kidney and liver can-

cer) with better, but still not good enough, activity against secondary mutations.

2. The switch pocket kinase inhibitors being developed by Deciphera Pharmaceuticals are an example of very potent KIT inhibitors that also have wide-spectrum activity against secondary mutations.

- Destroy the KIT protein instead of merely blocking the signal. Hsp90 inhibitors use this method. Since the KIT protein is dependent on Hsp90, inhibiting Hsp90 results in destruction of the KIT protein, regardless of secondary mutations in KIT. HDAC inhibitors also use this method, but also have other effects.

1. STA-9090 is a very potent second generation Hsp90 inhibitor. A phase II trial for GIST is due to open very soon.

A third approach that is slightly less developed is to target critical pathways downstream of KIT. The leading candi-

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Chicago-area GISTers meet!



Last week, The Life Raft Group's very first local chapter met again to discuss coping and GIST education. Those in attendance were: (Front row left to right) Bill Buchanan, Fred Chamanara, Vince Coccia, Lucy Madsen, Robert Madsen. (Back left to right) Jacqui Cuellar, Salvador Lazaro, Leigh Borland, Beth Sanchez, Margi Hughes, Nestor Sanchez, Jennifer Johnson, Pat Johnson, Dick Kinzig. Taking the picture: Jim Hughes

MAGUIRE

From Page 1

living in Colorado.

After months of waiting rooms, medical tests, consultations, a couple of bad doctors and a few hospital stays, I was diagnosed with a golf ball-sized tumor in my small intestine. The surgeon informed me that whatever it was (and it wasn't my mother's lost steak knife), it had to come out; so surgery was swift.

Like so many GIST patients during that time, the pathology of the tumor labeled it a leiomyosarcoma. It was "almost" benign, hadn't spread anywhere yet, and although they weren't sure what caused it, the oncologists told me the likelihood of a recurrence was slim to none.

A person having just been diagnosed with cancer pretty much couldn't have gotten better news.

Healing was painful but in just a couple weeks I was on my feet again, and in time back in the gym and the outdoors. A follow-up CT scan in 1999 verified I was healthy.

In May of 2001, I returned to Denver from The White Rim in Canyonlands National Park— my favorite place. I attributed my complete exhaustion to the long drive and three days of biking in the Utah desert.

Two weeks later I was still exhausted. I would sleep all night, wake up, eat breakfast and sleep into the afternoon.

Eventually, I realized this was more a physical issue than an attitude problem!

A CT scan revealed a football-sized tumor in my abdominal area (well no wonder that White Rim ride was so hard!) coupled with two metastatic tumors on my liver. By the grave look on the oncologists' faces I could tell things were bad, and that "thing" was me.

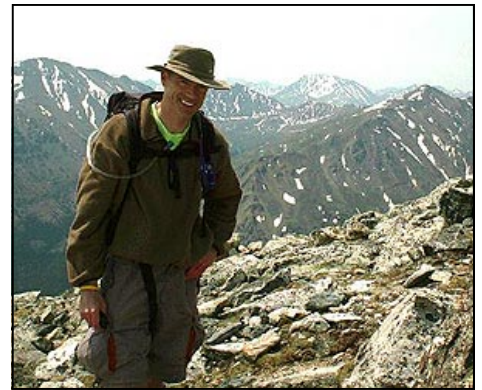
The doctors said due to the size and location chemotherapy would not work (we still didn't know it was GIST), and also due to the large size, it was basically inoperable— and they didn't know of a way to shrink it. They added that performing surgery now would be so damaging to me (as they would have to remove so much of "me" to obtain clear margins around the cancer) that even if I did survive the surgery I would "not have much of a quality of life."

They hadn't said the dreaded, "You need to get your affairs in order," but stated I only had a couple of months left to do something about "it" or "it" would be "too progressed."

So this time, having just been diag-



On a group ride in Boulder, Colo.



Patrick climbing Mount Massive, the second -highest peak in Colorado.

nosed again, I pretty much couldn't have gotten any worse news. As you can imagine, hearing all of this was devastating. This was the worst situation to be in; each day the cancer was progressing inside me while I was getting weaker. And I had no clear plan of how to begin to fight it, let alone conquer it, and neither did the experts.

This was my darkest hour... but I also thought it could be my finest hour. So I would cry alone in my bedroom at night, but afterwards collect myself, and vow to be brave, persevere, and accept whatever the future would bring and face it with as much dignity and courage I could muster.

Anyone that is really ill needs someone to advocate for them and fortunately for me, one of my sisters, a former National Institutes of Health nurse man-

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Insomnia is common, likely under-diagnosed among people with cancer undergoing chemo

The following article was reprinted from ASCO Cancer Advances with permission.

Researchers have found that 43 percent of patients undergoing chemotherapy for cancer in a clinical trial met the clinical criteria for insomnia syndrome and an additional 37 percent had insomnia symptoms, suggesting that the majority of patients (80 percent) experience sleep difficulties. This rate is approximately two to three times higher than that seen in the general population. Insomnia syndrome is defined as difficulty sleeping three or more times per week for at least a month, and can cause significant distress or impairment in day-time functioning. The study was published in the *Journal of Clinical Oncology*.

Prior studies have identified increases in sleep disturbances among people with cancer, but this is the first investigation to show an elevated risk of insomnia

syndrome in people undergoing active cancer treatment with chemotherapy.

In this study, researchers performed a subset analysis on data from a prospective trial of 823 patients undergoing chemotherapy for a range of cancer types enrolled between 1997 and 1999. Researchers found that 43 percent of patients experienced insomnia syndrome and an additional 36.6 percent reported insomnia symptoms during cycle 1 of chemotherapy; during cycle 2, 35.2 percent experienced insomnia syndrome and an additional 33.1 percent reported insomnia symptoms. By comparison, the prevalence of insomnia syndrome in the general population ranges from 16 to 21 percent.

The prevalence of insomnia syndrome was highest among patients with lung cancer (50.8 percent) and among patients under age 58 (the median age of patients in the study). Patients with insomnia syndrome reported more depression (32.3 percent) and fatigue (45.5 percent) than those who reported sleep-

ing well (10.4 percent and 30.8 percent, respectively).

The authors speculated that the side effects of cancer treatments as well as the stress and anxiety of a cancer diagnosis may contribute to insomnia, but this study did not examine the causes of patients' insomnia. They also added that some chemotherapy drugs used today are different than those received by patients in this study and may therefore influence sleep patterns differently among patients today.

What This Means for Patients

This study shows that insomnia is very common among patients undergoing cancer treatment and is likely under-diagnosed. Patients who have difficulties sleeping should talk to their doctors, who can provide helpful tools and treatments, such as insomnia medication or referrals to a sleep clinic. Cognitive behavioral therapy for insomnia, exercise, and yoga are all beneficial, when they are feasible.

Southern California GISTers meet!



The Manse on Marsh Street was the location of the first GIST gathering on January 10, 2010 in San Luis Obispo, California. Chris Skiff owner/developer of The Manse on Marsh Street, an upscale senior living center, co-sponsored the event with Mike Glenn. Chris is new to GIST and Mike has been challenging GIST for over five years. They made a great team. There were ten GIST patients, looking mighty fine we might add, and an additional 10 spouses and family members attending the gathering. The main purpose was to provide a forum for new GIST people to meet the warriors, talk and share their stories and consider some new ideas on handling GIST treatment. Rick Sword reminded us to pay attention to the amount of radiation GIST patients receive after years of treatment. This gathering was so successful and inspiring; a yearly meeting is already in the works.

San Luis Obispo is a great place to meet as it is centrally located to many of our west coast GIST patients. Our first gathering was all about meeting one another. In the future, there may be speakers on a variety of topics of interest. We found that our fight with cancer did not keep us from laughing and talking about common issues and putting faces to names we'd only met online so far.

That was fun. Everyone complimented Chris and Mike's effort to reach out and connect one on one with their fellow GIST partners.

NIH Pediatric & Wildtype clinic continues with success

The fourth bi-annual Pediatric & Wildtype GIST Clinic at the National Institutes of Health on January 20 was, once again, a great success.

To view a video or listen to a podcast of Dr. Su Young Kim's presentation on "Utilizing Results from the NIH GIST Clinics to Plan for the Future", go to <http://videocast.nih.gov/Summary.asp?File=15559>.

The next clinic will be held June 17-18, 2010.



Behind the scenes at the NIH: (In head order) Dr. Michael P. LaQuaglia, Dr. Alberto Pappo, Tricia Mcaleer, Dr. Maya Lodish, Phyllis Gay, Dr. Katherine Janeway, Dr. Jennifer Wright, Dr. Jonathan Trent, Dr. Margaret Von Mehren, Dr. Suzanne George, Dr. Su Young Kim, Dr. Constantine Stratakis, Evan Ball

SYNTA

From Page 6

containing a Gleevec resistant exon 11/17 mutation.

Dr. Fletcher is quoted in the press release about these results and the rationale for moving from the lab to the clinic for resistant GIST.

"We have shown that as many as eight different secondary KIT Gleevec-resistance mutations can occur in different metastases from a single GIST patient whose disease has progressed after treatment with Gleevec, which poses a significant challenge for treating drug-resistant GIST," said Dr. Fletcher.

"Importantly, all of these different resistance mutations were still sensitive to STA-9090. In these studies, STA-9090 was also 5-15 fold more potent than 17-AAG, a first-generation, ansamycin-family HSP90 inhibitor. Additionally, STA-9090 was active against GIST cells that were resistant to 17-AAG."

To participate, patients must have failed both Gleevec and Sutent. Prior HSP-90 inhibitor treatment is allowed.

Synta planned sites include Dana-Farber Cancer Institute, Fox Chase Cancer Center, Oregon Health & Science University and UCLA. ClinicalTrials.gov currently lists a trial contact at Synta: Robert Bradley, 781-541-7984, rbradley@syntapharma.com.

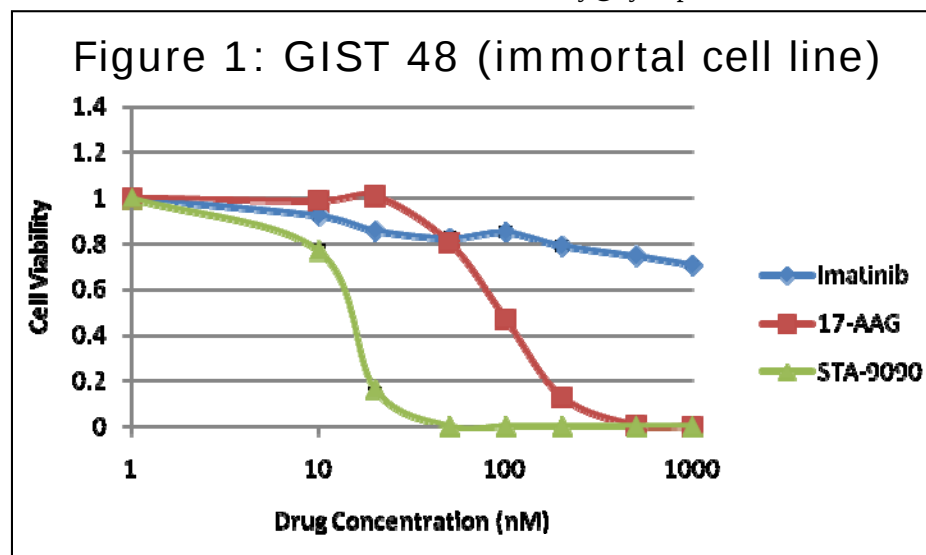
STA-9090 is an intravenous (IV) drug administered once weekly. Patients will therefore be required to be at the trial site weekly. According to Synta, "Subjects may request reimbursement for appropriate study related travel expenses incurred during participation. The details of this should be discussed with the Investigator and/or Study Coordinator at the participating institution."

Patients will continue on the study until disease progression is measured on CT scans using the RECIST criteria.

Synta reported three GIST patients to date have taken STA-9090 in the phase I trials: This quote from Dr. Vojo Vukovic, Senior Vice President and Chief Medical Officer of Synta Pharmaceuticals refers to one of those three, "A particularly encouraging observation was that a GIST patient on our once-weekly dosing Phase I solid tumor study, who experienced disease progression while on multiple prior therapies, including Gleevec and Sutent, experienced substantial tumor shrinkage and stabilization of disease following treatment with STA-9090."

Synta expects to report data from the ongoing STA 9090 phase I and II trials in the first half of 2010.

While these are encouraging early indications for STA-9090 the purpose of a phase II trial is to determine the effectiveness of a new treatment in a larger sample of patients.



Slide provided by Dr. Jonathan Fletcher and used with permission.

Patients with rare cancer get second chance to fight back

The following news release was re-printed from various new sources.

January 27, 2010— When Morty Wagman underwent surgery to remove a gastrointestinal stromal tumour (GIST) in 2008 with his wife Judi by his side, he was faced with a concerning 55 per cent chance that his cancer would return. Then, his oncologist told him about Gleevec (imatinib), a treatment that could greatly reduce the risk of his cancer returning, and Wagman felt empowered and hopeful. Today, he and his wife applaud Health Canada for approving Gleevec for patients with this potentially life-threatening condition, at an earlier stage in the disease.

“At first we thought my only option after surgery was do nothing but live in fear of my cancer's return, and we knew that if it came back, it could be a lot worse,” says Wagman, 71, of Toronto. “I was so relieved to learn about Gleevec following my surgery. For me, it represents new hope; a second chance to keep this disease in check and to go on living my life, spending valuable time with my family.”

GIST, a rare cancer of the gastrointes-

tinal tract, is difficult to diagnose, causing few symptoms in the early stages of the disease. While surgery is the mainstay of treatment for primary GIST, tumours will often recur, months to years after surgery.(i) Recurrent tumours are often more aggressive than primary tumours, and are more likely to be fatal.(ii) In addition to treating inoperable, recurrent or metastatic GIST, Gleevec is now approved by Health Canada as the only post-surgical treatment for patients with primary GIST.

“As experts in this area, we are confident of the preventative benefits of Gleevec for primary GIST, and are now able to treat patients in an appropriate way, at the appropriate time,” says Dr. Shailendra Verma, Medical Oncologist, The Ottawa Hospital Cancer Centre. “Research has shown that 98 per cent of patients receiving Gleevec had not experienced a recurrence of the disease one year after surgery. This freedom from relapse has significant meaning to patients and the physicians who care for them.”

While the true incidence of GIST is difficult to determine, a Swedish study has estimated an incidence of primary

GIST of 14.5 and a prevalence of 129 individuals per million population. This represents approximately 500 newly-diagnosed Canadians per year.(iii)

“Data recently published in The Lancet show that Gleevec reduces the risk of recurrence of GIST by 89 per cent after surgery. Its approval means that many patients can alter the course of this life-threatening disease at an earlier stage, when it can make a huge difference in their lives,” says David Josephy, president of GIST Sarcoma Life Raft Group Canada. “Given the strength of the data for post-surgical Gleevec, we urge provincial governments to act quickly and provide reimbursement to patients who need access to this important treatment option.”

Global GIST Network adds new GIST representatives



Global
GIST-Network



Nicaragua
Maria Teresa Ponce
Maria.teresa.ponce@aeienergy.com

HAPPY CANCER-VERSARY TO CAROLINA PONCE-WILLIAMS!

Not only did Carolina just celebrate a cancer-versary, she has been blessed with the one thing she has always wanted— a child.

“I am so thankful to be able to celebrate my four year NED anniversary this year, especially because I am still a wife, a teacher, a daughter, a friend and now I am also a **MOM!!**”



“Choosing not to have my own children, so I can stay on Gleevec was not easy. The last couple of years were a roller-coaster ride for us, since we started the adoption process. Now we know that it was all worth it and we are blessed with our little milagrito (little miracle)...Antonio Marcelo.”
Congratulations twice, Carolina!



BENCH

From Page 1

finding that patients with changes in exon 9 of KIT have improved tumor control with higher doses. Similar to findings in chronic myelogenous leukemia, information is emerging on the importance of achieving a specific level of imatinib in the blood for best outcomes, and this concept will be investigated further. Utilizing all these pieces of information, we can choose how best to treat patients. However, this is only the tip of the iceberg in regards to why some GIST patients benefit from imatinib or other second-line therapies and other patients do not.

Our group has worked to explore molecular markers of response to imatinib. To do so we have obtained tumor biopsies from patients throughout the U.S. and Europe who respond differently to therapy and have applied state-of-the-art approaches, such as DNA gene-expression microarray and SNP (Single Nucleotide Polymorphism) array analyses to map out differences between these tumors. We believe that by knowing what is “wrong” with a given tumor we can help choose the correct method(s) of treatment and apply them early in the course of disease to significantly improve a patient's chances of survival. As an example, we explored the changes seen in gene expression in tumor sam-

ples before treatment and correlated expression patterns with the likelihood of benefit from drug therapy. We were able to do so in a study run by the Radiation Therapy Oncology Group led by Dr. Burton Eisenberg: RTOG 0132. This study treated patients with large primary or resectable recurrent or metastatic disease with eight to twelve weeks of imatinib, followed by surgery and then adjuvant therapy for two years; patients with recurrent and metastatic disease were allowed to continue adjuvant imatinib beyond that. All patients had a diagnostic biopsy before they started treatment. During treatment prior to surgery, patients had CT scans to assess response to therapy.

We asked the question, is there a difference in the gene expression patterns in patients whose tumors shrank by 25 percent or more after the initial therapy, compared to those whose tumors did not. Intriguingly, we uncovered 38 genes that were expressed at very low levels in tumors that subsequently had very good response to imatinib. Eighteen of these were members of a family of genes called “Krüppel-associated box (KRAB) domain containing zinc finger (ZNF) transcriptional repressors”. Interestingly, 10 KRAB-ZNF genes were located on one chromosomal segment (Rink, 2009). The function of KRAB-ZNF family members is just being explored, but they generally work to stop genes from being expressed and their



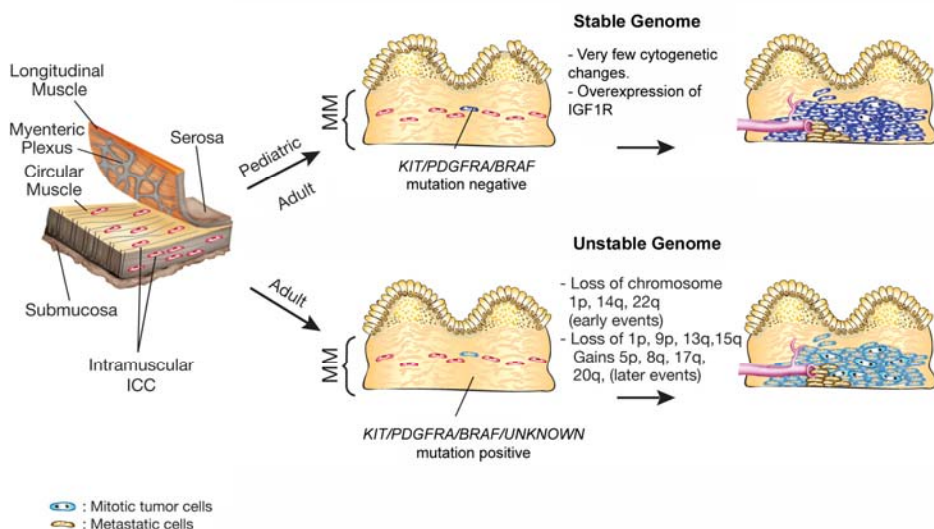
GODWIN

corresponding proteins from being made. We have further shown that if we can shut off expression of some of these genes, we can make GIST cells much more sensitive to imatinib.

We are intrigued by these findings. Testing for the presence of a high level of these genes that in our study were associated with an increase in tumor size or slow tumor response is not difficult and does not require large amounts of tissue. First, we wish to confirm this observation and are seeking collaborators that have access to tumor tissue from a different set of patient samples. Potentially, one might use this information to treat a patient with a large primary tumor that would require a complicated operation to remove. For example, since the level of expression of these genes is correlated with imatinib being less effective, is there a way of inactivating their function or alternatively increasing their destruction within tumor cells? Targeting these genes with drugs along with imatinib may prevent tumor growth and avoid increasing the complexity of the surgery or a situation where surgery is no longer technically an option. We also believe this is an opportunity to study this new class of proteins to better understand what they do and how they do it, particularly in GIST. Our results suggest this may lead to better efficacy of imatinib in GIST.

In other studies we have shown that not all GISTs that lack mutations in KIT or PDGFRA, the so-called “wild-type” tumors are alike. Using whole-genome SNP-arrays we have been able to obtain high-resolution maps of the GIST's genome. We used high-density SNP arrays containing roughly 1.8 million markers genome-wide to analyze GISTs lacking KIT/PDGFRA mutations as well as mutant GISTs. Importantly, our studies found that the majority of KIT/PDGFRA mutation-negative, both pediatric and

Figure 1: GIST pathway



REPORT

From Page 1

PDGFRA. In-person meetings in Boston and Portland continued the ongoing collaboration and information sharing that distinguish the Life Raft Group team from the unfortunate reality of competition and disunity that characterizes most of the cancer research world.

We rebuilt the data structure of our patient registry and published a breakthrough study on the relationship of Gleevec dosage to progression-free and overall survival (Go to <http://springerlink.com/content/346011445r10um38/> to see the published article). In collaboration



with Stanford University and a group of key research laboratories we launched a comprehensive tissue bank which enables researchers to access and share research results

on hard to obtain GIST tissue and to connect these results with the clinical histories maintained within the registry. To provide patients and their doctors with diagnostic and treatment summaries, we began to distribute medical histories aptly named GISTories.

Toward the end of 2009 we formally launched our Latin American Initiative to facilitate the flow of information about GIST throughout Latin American in both Spanish and Portuguese and to help develop viable patient organizations to create culturally relevant grass roots support to patients, their families and their doctors. I've just arrived in Monterrey, Mexico where I'll join a planning group of patient representatives from ten Latin American countries.

Not a week went by that we did not provide one-on-one assistance to numbers of GIST patients and caregivers seeking the latest information on treatment, including clinical trials. Our websites, webcasts and pamphlets continued

to provide information to patients and medical professionals around the world.

Our local groups continued to meet and provide

What if cancer were a contagious disease?

After the initial panic the cancer research community would:

- *Create an action plan instead of individual researcher requests*
- *Agree to cooperate instead of compete*
- *Agree to share information instead of withholding it for publication*

Why not do this anyway?

personal support through a combination of hugs and critical information. And we continued to advocate both publicly and behind the scenes on behalf of GIST patients.

In 2010 we plan to accelerate our battle to find a cure for GIST and to keep patients alive until we do so.

In addition to maintaining our core commitments to our world-class research team, we plan to build upon their unique coordinated approach to implementing a continuously refined strategic plan by providing new incentives to exploit promising new ideas to identify and overcome pathways of treatment resistance.

We will also continue to expand our efforts to make sure that no patient dies because of the ignorance or inexperience of his or her physician or because of a lack of urgency in applying cutting-edge knowledge to support the battle for survival. We will insist that every GIST patient receives a mutational test for his or her primary tumor and, if on Gleevec, a plasma level test on an ongoing basis.

We remain concerned that 400mg of Gleevec may be inadequate over the long term to provide maximum pro-

tection to many patients against the development of resistance. We believe that it is currently easier to prevent resistance by optimizing Gleevec dosage levels than it is to reverse resistance by raising such levels after the fact. We have gained a great deal of experience in avoiding serious side effects on higher doses of Gleevec by employing gradual dose escalation and a growing array of techniques to manage side effects should they occur.

Although we do not yet have accepted reference levels for plasma testing, we think nonetheless that such testing is a critical tool for the physician to utilize now, particularly in the event that the patient demonstrates downward trends. At a minimum, this should create an occasion for the physician to discuss treatment compliance and to do so in a non-threatening manner. The issue of patients not taking all of their oral medications remains an ongoing concern for us, and we plan to intensify our efforts to address this issue.

We will continue to utilize every method at our disposal for providing information, education and support to create empowered patients who seek a mutually respectful partnership with their physicians. This combination of well-informed patients and physicians reinforces the battle for survival. We believe that competent physicians will welcome this partnership.

Given the rapidly growing amount of information about GIST and the fact that it is a comparatively rare cancer, we will encourage patients to seek out physicians who have practical experience gained through seeing a significant number of such rare patients. We will continue to expand our online directory of GIST specialists and are planning to better define and identify physicians and centers of excellence.

Should our tone seem a bit strident or impatient we submit that our intention is to encourage thought and collaboration and that our urgency is driven by the very human struggle for survival.

The struggle to find a cure, and to help patients stay alive until then, continues.

Too many candles have been lit not to succeed.

BENCH

From Page 11

adult GISTs which are clinically more resistant to imatinib-based therapies, generally do not possess the large-scale chromosomal aberrations that are commonly found in mutant GISTs (Belinsky, 2009). We refer to these tumors as KIT/PDGFR mutation negative/**genome stable** GISTs, while the remaining tumors (including both KIT/PDGFR mutation positive and some mutation negative GISTs) are **genome unstable** (see Figure 1). Furthermore, we have shown that these KIT/PDGFR mutation negative/genome stable GISTs greatly overexpress the insulin-like growth factor receptor-1 (IGF1R) relative to other GISTs. In our previous studies, first reported in 2008, we demonstrated that IGF1R may be an interesting therapeutic target to consider in GIST patients, especially in pediatric and adult KIT/PDGFR mutation negative cases in which treatment options are extremely limited. This important finding has subsequently been confirmed by others as discussed in the last LRG newsletter by Dr. Mike Heinrich. We found that KIT/PDGFR mutation negative GISTs greatly overexpress IGF1R (both at the protein and mRNA levels) and drugs that target IGF1R can inhibit the ability of GIST cells to grow (Tarn, 2008; Godwin, 2008; Belinsky, 2009). We have seen that the mutation

negative GISTs that have a stable genome also generally overexpress IGF1R (Tarn, 2008; Belinsky, 2008; Belinsky, 2009). Clinical trials are being developed to explore IGF1R as a target based on these and other molecular studies. Finally, we have shown that a portion of previous classified “wild-type” GISTs that demonstrate genome instability have mutations in the BRAF gene (Belinsky, 2009). There are now several additional reports indicating that the BRAF gene is mutated in roughly four to 13 percent of GISTs without KIT or PDGFR mutations (e.g., Agaram 2008). However, the genetic changes in GISTs without KIT/PDGFR/BRAF mutations is not known, so we are currently using modern deep sequencing approaches to further explore the GIST genome in these patients to uncover additional clues to help define therapy.

These are a few examples of how work at the bench can be translated into improved therapeutic modalities influencing how patients will be treated at their bedside. Advances in GIST care involve all of us, the patients who participate in clinical trials and share their tissues, the doctors and nurses who care for them, and the scientists who help increase our understanding of this disease and provide new insights to take back to the patients.

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Mark your calendars!

- The Latin America GIST Coalition is meeting for the first time in Monterrey, Mexico **January 2-4!**
- The American Association for Cancer Research (AACR) conference will be held **April 17-21** in Washington DC.
- The St. Louis GIST foundation is planning a meeting for **May 15**. Email Ally Klein at aklein@gistl.org for more information.

**Registration is open for
Life Fest 2010 June 25-27!**

Register at www.liferaftgroup.org/members_lifestestregistration.html

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Show the LRG some love!

*Valentine's Day is coming up,
a perfect time for your friends
and family to show GISTers some love!*

*Please send out your “A Cure Is In Our Reach”
campaign note cards. All donations go directly to
GIST research.*

*You can also go to www.acureisinourreach.org,
upload a photo (you and your sweetie, perhaps?)
and tell the world what a cure would mean to you.*

Won't you be our valentine?



KIT

From Page 6

date in downstream pathways appears to be PI3-K. There are a number of PI3-K inhibitors in phase I clinical trials.

Other approaches to overcome secondary mutations are to prevent the production of the KIT protein, for example to inhibit KIT transcription (bortezomib and flavopiridol), and to prevent activation of KIT by blocking receptor dimerization. These approaches seem to be less developed than the first two.

While secondary mutations form the major mechanism of resistance for exon 11 tumors and a smaller, but still significant percentage for exon 9 mutations, they are not the only mechanism that cause resistance in these tumors. The mechanisms affecting exon 9 tumors are not as clear as those affecting exon 11 or other types. KIT amplification (too much KIT protein) as well as activation of an alternate kinase has also been noted in resistant GIST. In particular, AXL kinase has been noted to be upregulated in some GISTs that have lost KIT expression and focal adhesion kinase (FAK) may also play a role in GIST tumor cell survival. Of note, MP470, a multi-tyrosine kinase inhibitor in phase I clinical trials, is reputed to be a wide-spectrum KIT inhibitor and also an AXL inhibitor.

For resistant exon 11 tumors, overcoming secondary mutations with a wide-spectrum KIT inhibitor or via HSP90 inhibition is the first priority. Though perhaps not as frequent, secondary mutations also appear to be a problem for resistant exon 9 tumors. Other mechanisms of resistance have been noted for these mutations including KIT protein overexpression and activation of alternate kinases. The relative infrequency of KIT overexpression and alternate kinase activation combined with a lack of clinical testing to identify these mechanisms makes targeting them difficult at this time, although very potent KIT inhibitors that also have a wide spectrum of activity (such as the Deciphera compounds in preclinical development) may naturally target protein overexpression as well as secondary mutations.

Beloved wife, mother, grandmother & friend enjoyed all of life's blessings

A beautiful spirit & consummate caregiver, Rita Catherine Joyce Patient touched the lives of all she knew. Proud wife, mother & grandmother, she passionately supported her family in all of their endeavors. She enjoyed & treasured the blessings of many lifelong friends in Minnesota & Texas & her travels to points beyond.

Rita earned her nursing degree from the College of St. Catherine & offered comfort & support to all placed in her care, especially elementary school children. She always greeted strangers with an encouraging word & a kind smile.

Rita passed away peacefully on 11/29/09 surrounded by her loving family. Rita is survived by her devoted husband of 39 years, Greg; her 3 children,



PATIENT

Amy (Bjorn) Larsen, Greg (Noreen) & Melissa; her 4 grandchildren, Samuel, Katherine, Emma & Olivia; her mother, Leona Joyce; 9 siblings, Mary Joyce, Patrick (Joan) Joyce, Paul (Betty) Joyce,

Thomas (Judy) Joyce, John (Linda) Joyce, Sheila (Gordon) Bonine, Michael (Cheryl) Joyce, Monica (Gregg) Schaner & Francis (Lisa) Joyce; her sister-in-law, Marianne Mohrlant; brother-in-law, Paul Patient; & many cousins, nieces, nephews & friends. Memorials preferred to www.liferaftgroup.org, an organization supporting those with GIST or to St. Stanislaus. WULFF GODBOUT 651-224-4868.



Wife, mother and friend to all passes away after courageous battle with GIST

Laura Samantha Kukucka, born March 8, 1977, died Tuesday December 1, 2009 peacefully ending her battle with cancer. She was born in Oberlin, OH and graduated from Olentangy High School. She received her B.A. in communication from The Ohio State University. She worked for the State of Ohio in the Dept. of Ad-

ministration Services. Her hobbies were reading, crosswords and New York Yankee Baseball. Survived by husband, Phillip Michael Kukucka; son, Jacob Benjamin Cooley; mother, Patricia "Poopsie" Sheffield; father, Clinton Lawrence Sheffield.

Laura fought a very courageous battle against cancer. She never would give in, and loved everyday to its fullest. She was always full of life, even to her last days. She was always kind, supportive and understanding, and would put others' feelings first. She will be missed beyond what words can describe. We all love you very much and will be always thinking of you. In lieu of flowers, contributions may be made to www.LifeRaftGroup.org. To sign and view the on-line register, visit www.MaederQuintTiberi.com.



Laura with her son, Jacob.

MAGUIRE

From Page 7

ager, offered to do so. By now, the tumor had become so large it was restricting my kidneys so stents (not-so-flexible plastic tubing) were placed in my ureters to keep them open and draining properly.

We divided the days into “hard days” and “easy days.” Hard days consisted of waiting rooms, filling out medical history and consent forms and appointments. Easy days meant sleeping for me while my sister researched avenues for treatment options.

A month passed.

One day, while searching online, my sister found a posting for a clinical trial

at all and the cancer was actually progressing. There was also a chance of side-effects so severe that I would have to be taken off of it. It needed to work — but not work *too* well.

I called these ‘The Dog Days of Gleevec’ because each day consisted of me sleeping 16-18 hours interspersed with flu-like side-effects of nausea, upset stomach, soreness and fatigue. The stents caused back pain and bleeding if I stood, sat or slept in one place for too long or walked too far. I was too sick to work or even do much exercise beyond walking around the block. I had weekly blood tests and consults with my oncologists, more than I can even recall. I’d lie on my bed and read or listen to music. I meditated and thought about the battle going on inside of me, hoping for the best and mentally forcing the cancer out of me. I wished I was healthy enough to go for a hike in a red-walled canyon, climb a mountain or take a simple bike ride.

I couldn’t believe my life had come to this but I felt a slight relief because at least I now had a path to follow, albeit the only one available, and a tool or weapon to use against the disease.

The day finally came and I was lucky enough to be told the tumors were shrinking!

About two years passed consisting of my daily doses of Gleevec, more medical appointments for blood work, CT scans every three months, and four more stent surgeries. The primary tumor, located in the center of my pelvis, eventually shrunk to the size of a baseball. The stents finally came out for good and one day my oncologist recommended sur-



Patrick shows off his battle scar after a successful surgery.

gery.

In June 2003, after five hours in surgery, an excellent surgeon removed all of my cancer. Since that time, I have wondered why I survived while others who fought just as hard— or harder— died. If my cancer would have been as progressed in 1997 as it was in 2001, I would not be here to convey my story to you.

I had a great amount of support from my family, friends and medical team for which I am forever grateful. I could not have done it without them, but mostly I believe I was just lucky.

People often want to label survivors of a traumatic ordeal as a “hero.”

What I know is that anyone who has cancer and is brave enough to fight it, whether they are lucky enough to live, or dies trying— especially if they die trying— is a hero. Everyone who fights this disease, whether as a caregiver, working in the medical field, or contributing to research is a hero, since it takes all of these to win against cancer.

I still take my Gleevec each day as I have for almost nine years, keeping my fingers crossed and staying humble regarding a possible recurrence.

GIST is different than other cancers because although we’ve been given an enormous second chance, I feel, probably as many do, that I only get so far away from it, and then it’s time for another dose or another scan.

It’s a daily reminder of a dark time but also how truly fortunate I am, and to appreciate the important things in life and see the beauty in this world.

I hope our paths cross while hiking a trail in a remote desert canyon, climbing a 14er or biking on a summer day.

Note: Almost five years have passed since my surgery and I am happy to report my follow-up scans have been clear, showing “No Evidence of Disease,” or NED — or from the perspective of cancer survivors — I am “dancing with Ned,” no matter how goofy I look.



Patrick gets some oar time on the Green River in Utah last summer.

for an experimental drug called Gleevec. Gleevec had shown promise in patients with certain types of leukemia and in one or two very limited GIST trials, shrinking tumors in about half the patients. If a biopsy of my cancer proved it to be of the “stromal” type, and I met other requirements, I could apply to be accepted into the trial before the closing date only a month away.

In late August 2001, less than two weeks before the trial closed, I was accepted into the trial at Dana-Farber Cancer Institute in Boston and began taking eight orange 100 mg capsules each day of Gleevec.

Three months would pass before I would know, by CT scan, whether Gleevec was shrinking my tumor and stabilizing my liver mets, or not working

THE LIFE RAFT GROUP

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