HEALING ORACLE

GCMAF
IMMUNOTHERAPY

Gcmaf destroys 100% of all immunity diseases, including Cancer & Autism

preface: GcMAF and Nagalase: two amazing proteins.

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In the pages that follow you are going to learn about two amazing proteins: GcMAF (glycoprotein macrophage activating factor) and Nagalase (alpha-N-acetylgalactosaminidase). These two natural bioidentical protein molecules (made by our bodies using our own genetic program) have the potential to prevent a lot of human suffering and save millions of lives.

Research studies have shown how we can use GcMAF and Nagalase to:

- identify the presence of—and reverse—metastatic cancer
- cure HIV and other chronic viral infections
- detect and reverse early cancers long before imaging can identify them
- determine whether a cancer treatment program is working

Nagalase screening, coupled with early treatment using GcMAF, has the potential to rid the world of the scourge of cancer. This is a strong statement, but anyone who carefully examines the science behind GcMAF, Nagalase, and the molecular biology of macrophage activation will realize it is true.

This book has two primary goals, both of which are attainable. The first is to further the cause of making GcMAF available to all who need it. The second is to promote the establishment of Nagalase cancer screening programs for all adults and all high risk populations. With Nagalase testing we now possess the technology to identify cancer when it is just a handful of cells and then easily reverse it with a few GcMAF injections. For me, the Holy Grail of cancer eradication is finding it early and nipping it in the bud.

Introduction: Routine Nagalase testing finds cancer early and GcMAF cures it

If we can accelerate research on Nagalase testing and GcMAF treatment, the following conversations might happen five years from now:

Joe has an appointment with Dr. Jones, his family doctor for the past two decades, to discuss Joe's annual lab test results.

"Hi Joe. Good to see you!" says Dr. Jones. "We ran some screening tests on your blood and I've got some good news and some bad news."

"It's been a long day, doc. How about the good news first, if you don't mind," says Joe.

"Sure. Your blood tests tell me you are a picture of health. Your cholesterol and other heart markers are all normal. Your vitamin D level is excellent at 70. Your PSA is low. Your thyroid is in balance. Your complete blood count and other metabolic parameters are perfect."

"Sounds good, doc, and that all fits perfectly with the fact that *I feel great!* …so what's the bad news?""

"Well, Joe, your Nagalase level is elevated. and that's got me a little concerned."

"My *Nagalase* is elevated? What is that and what does that mean?" says Joe cautiously.

"Nagalase—short for alpha-N-acetylgalactosaminidase—is an enzyme made by viruses and cancer cells. We can test for it. An elevated level tells me you have the very earliest beginnings of either a cancer or a virus.

Nagalase testing is a lot like cholesterol testing. Elevated cholesterol is associated with increased risk of arterial plaque and heart attack. An elevated Nagalase tells us you are at increased risk of cancer.

"Cancer or virus? I don't understand. What does that mean, doc?"

"Both cancers and viruses make Nagalase, and at this point I can't tell you which it is. But there's nothing to worry about here, Joe. You don't have any symptoms, so it probably isn't a virus. We'll get some antibody tests to rule out that possibility, and we'll repeat your Nagalase level to see whether it is going up or not. For now, all we need to do is keep an eye on everything. If the elevation is caused by a virus, the level will go back down. If the elevated Nagalase is caused by an early cancer, the Nagalase will keep going up, and we'll treat it and make it go away. Here's your lab order. See you in a month, okay?"

“Should I be worried, doc?”

“No.”

“I might have cancer, but not to worry? How does that work?”

“Five years ago, Joe, I’d be very concerned. But back then we didn’t have Nagalase testing for detecting early cancers, so I wouldn’t have even known that a cancer was getting started in you. At least not until it got a lot bigger. And five years ago, if I *did* have some way to know you had early cancer, I wouldn’t have had a way to reverse it. Now, I have both. And I must tell you, as a doctor who has seen a lot of people suffer and die from cancer, we are extremely fortunate that these tools are now available. Millions of olives have already been saved by catching cancers early and reversing them before they got out of control. So, not to worry, Joe. If it’s cancer, we have the tools to get rid of it. See you in a month.”

“Okay.”

One month later

“Joe, your viral antibody tests all came back normal, so now we know you don’t have a viral infection. Your Nagalase, however, continues to climb higher. That tells me you have a very small early stage cancer growing in you somewhere.”

“I’ve got *cancer*? Yikes! You *did* tell me this was a possibility. *Now* should I be worried?”

“No, Joe, you really have nothing to worry about. We have a treatment that will make this cancer go away. And we can accomplish that long before it gets big enough to show up on a CAT scan.”

“Does it matter what kind of cancer it is?”

“Nope.”

“Does it matter where it is located?”

“Nope.”

“Shouldn’t I get a CAT scan so we can find it?”

“Since your Nagalase is only slightly elevated, I know that your cancer’s still way too small to see on imaging. But the cancer cells are definitely in there, making the Nagalase that we see on your blood test.”

Couldn’t there be something else that’s making the Nagalase?” says Joe.

“No, Nagalase is only made by cancers and viruses, and we’ve eliminated the possibility of a virus, so it has to be cancer.”

“How soon can we start treating it?”

“In one month. I want to get a third Nagalase level, just to make sure—and if it is still going up, then we can be certain it is cancer, and we’ll start the treatment.”

Another month goes by…

“Well, Joe, your Nagalase is still climbing, so we can start treatment today.”

“Okay. What do we do? What is the treatment?”

“Weekly injections of GcMAF. We’ll keep on measuring your Nagalase once a month, and when it gets back down to normal, we’ll know the cancer has been cured, so that’s when we’ll stop giving you the shots.”

“How long does that take?”

“Usually three to six months.”

“What *is* GcMAF, anyway?”

“GcMAF—group specific component macrophage activating factor—is the protein your body makes to activate your anti-cancer immune activity…

“Anti-cancer immune activity. What’s that, Dr. Jones?”

“Joe, your immune system contains some very large cells called macrophages that, when *activated*, will track down, attack, devour, and kill viruses and cancer cells. GcMAF—an immune protein made by your lymphocytes—is what literally turns them on. Without GcMAF, your macrophages remain in a state of suspended animation—sort of like zombies.”

“You mean if I don’t have enough GcMAF they stop killing off the invaders? That can’t be good.”

“No, it isn’t, Joe. This is not a subtle slowdown, either. In terms of killing power, an activated macrophage is about 40 times more aggressive than a *de*-activated macrophage. Without GcMAF, they just go to sleep.”

“Wow.”

“Your cancer cells have figured out that if they block the production of GcMAF, they can disable their archenemies, the macrophages, and get the upper hand. Without GcMAF to activate them, the macrophages don’t have a chance.”

“So, doc, how did they do it? How did my cancer cells disable my macrophages.”

“By making Nagalase.”

“The same stuff you’ve been testing?”

“Yup. Nagalase blocks GcMAF production, so when we see a rising Nagalase, we pay very close attention. It is an incredibly sensitive test, and it tells us cancer is on the move. As cancers grow, they generate more and more Nagalase.”

“So the rising Nagalase numbers told you my cancer cells were winning the war.”

“Yes.” replies Dr. Jones.

“And that the Nagalase had put my macrophages to sleep by blocking my body’s GcMAF production.”

“Right. There may be a future for you in molecular biology, Joe.”

“So how do we *re-*/activate my macrophages so they get back to work destroying my cancer cells?”

“By giving you weekly injections of GcMAF, to bypass the production blockage caused by the Nagalase.”

“Makes sense to me. How big are these shots, doc?”

“They’re incredibly small. Just 100 nanograms—that’s 100 *billionths* of a gram, dissolved in a few drops of water. In fact, if you removed the water, the GcMAF alone would be too small to see.”

“Amazing.”

“And this stuff really works?”

“In early cases like yours, Joe, it works every time. That’s why we like to catch it while it’s still small using Nagalase screening.”

“So you are going to give me some GcMAF shots that’ll perk up my macrophages so they can get back to work finding and gobbling up these cancer cells, right?”

“Right. Now, please roll up your sleeve.”

How GcMAF works: GcMAF is the protein that activates macrophages and jump-starts the entire immune response. To sabotage the immune system and put the macrophages to sleep, all cancers and viruses make Nagalese, the enzyme that blocks production of GcMAF. In the absence of GcMAF, cancers, HIV, and other viruses can grow unimpeded. Dr. Nobuto Yamamoto demonstrated that GcMAF administration bypasses the Nagalese blockage and re-activates the macrophages, which then proceed to kill the cancer cells and HIV viruses: See this short video

GcMAF cures Mary’s metastatic breast cancer

“Hi Mary.”

"Hi Doc."

"Good to see you. As you already know, we had found that small breast cancer on your mammogram, and Dr. Humphrey has already taken it out."

"Yup."

"As you also know, I got a Nagalase level before the lumpectomy, and then again after surgery, and then monthly levels since. Because the Nagalase level is always directly proportional to the amount of cancer in your body, it really helps me to track your cancer and make sure it doesn't get out of control."

"Yes, and remember how high it was right before my surgery? Then, right after the surgery, it dropped a lot, because there was less cancer to make the Nagalase. But it was still elevated after the lump was taken out, so we knew there still had to be some cancer left behind after the surgery, darn it. And then my Nagalase started climbing again, so we knew the cancer, wherever it was—was growing. That part was pretty depressing, doc! That's why you started me on the GcMAF, right?

"Yes. And as you know, Mary, we've been testing your Nagalase levels monthly since we started the GcMAF shots, and it has been gradually coming back down. This told me that the GcMAF was activating your immune system and that your macrophages—if you'll excuse the expression—were in there kicking butt on your cancer cells."

"No problem, doc, it *is* pretty exciting.

"I've got some really good news for you today, Mary. Your Nagalase level has finally, after 20 weeks of injections, reached zero. You are now officially cancer-free, so we can stop the injections. And we can celebrate! If you don't mind my saying so, YIPPEE!!!

"Wow! That's incredible. You know, Doc, I might have been dead right now if it weren't for you …and the GcMAF. Thank you so much!

"Mary, it is sincerely my pleasure."

1: A Cure for Metastatic Cancer?

Can it be true? A cure for early metastatic cancer and HIV that appears to work 100% of the time—that almost nobody knows about? My search for answers. Solving the riddle of GcMAF. Why GcMAF remains obscure. Please don't be intimidated by cell biology! The war analogy.

On November 19, 2008 something touched my life and changed it forever. I had just completed my second book, *Outsmarting The Number One Killer* (about how to prevent and reverse atherosclerotically-driven heart attacks and strokes) when I came across three seminal studies published earlier that year by internationally recognized research immunologist and molecular biologist, Nobuto Yamamoto, Ph.D. These pivotal papers—which will, I believe, change the course of medical history—blew me away: Yamamoto had apparently discovered a way to "outsmart" cancer, and was using the body's own natural healing systems to do it.

“Holy cow!!!” I said to myself (using a word that’s less bovine and more graphic). “This guy has discovered a cure for early metastatic cancer that appears to work 100% of the time!!!” And this was no “black box” model or statistical study (you know, the kind that make observations but don’t address underlying causal mechanisms. Medicine A cures disease B, but nothing about how it actually works). All the basic science, all the necessary molecular biological information—an impressive display of published research studies from a *quarter century* of work—was right there for all to see. No smoke and mirrors. Anyone who has perused Dr. Yamamoto’s research papers would have to agree he published an impressively extensive series of serious science masterpieces.

As I learned more I became committed to bringing this powerful new set of ideas into public awareness. It became clear to me that we need to find a way to make GcMAF available to all cancer, HIV, and chronic virus patients, and we need to institute routine annual Nagalase testing to find cancers much earlier than imaging now allows.

If all new cancers were detected early by regular Nagalase testing, we could reverse them with GcMAF—long before X-rays could find them—and put cancer out of business once and for all. This may seem like a rash statement, but I believe it is supported by the facts.

Yamamoto’s three studies showed that incredibly small weekly doses (100 billionths of a gram—an amount that is invisible to the naked eye) of GcMAF had cured early metastatic breast, prostate, and colon cancers in 100% of (nonanemic) patients. In a fourth paper, he used the same treatment to cure 100% of nonanemic HIV-infected patients.

For the three cancer studies, Dr. Yamamoto had chosen patients who had recently received the standard mainstream triad of surgery, chemo, and radiation. Despite these treatments, every patient had evidence of metastatic disease, which means that despite the best efforts of conventional medicine, their cancers were out of control and still growing. Their prognoses were poor at best. Nevertheless, this patient group had one thing in common: their tumor mass (also known as tumor burden) had been drastically reduced by the therapies they had received, and this in turn dramatically increased the likelihood that GcMAF would remove the few remaining cancer cells.

These studies underscore the importance of the unique circumstance in which GcMAF is most likely to be effective: very low tumor burden. Low tumor burden occurs in just two situations. The first is the earliest stage of any cancer, when the number of cancer cells is still very small. The second is immediately after a diagnosed tumor has been maximally debulked by standard therapies—i.e., that which prevailed in Dr. Yamamoto’s studies. Conversely, the situation in which GcMAF is *least likely* to be effective is when there is a large number of tumor cells. Although results will vary greatly from one patient to another and further study is needed, clinical research experience to date suggests that tumors larger than one cm. in diameter are unlikely to respond to GcMAF therapy. (See Chapter 18: ‘The Cancer continuum and the Point of no return’_)

Curing metastatic cancer *at all* is rare. Until Professor Yamamoto discovered and administered GcMAF, no one had ever cured every single case. These are the patients oncologists give up on, the ones that get “palliative” care. Perhaps another round or two of chemo or radiation in the slim hopes of a long-term reversal or a little extra (probably not very high quality) time—but with metastatic disease there is no serious expectation of an actual cure. The numbers are profoundly dismal.

Granted, all of Yamamoto's patients were in the earliest phase of metastatic disease and received GcMAF shortly after the Big Three had failed. For these patients (though a sprinkling might have been saved by additional radiation and/or chemo), the assumption would usually be that their cancers would grow and eventually kill them. GcMAF, remarkably, saved every single one. This is an exceptional outcome and deserves greater scrutiny that it has received.

A fourth study, published in January of 2009, showed Yamamoto—using the same treatment protocol—had removed all signs of viral activity in 100% of HIV infected patients. All patients were free of HIV within 18 weeks.

Remarkably, Yamamoto accomplished these cures relatively rapidly. The breast and prostate cancer patients were all cured in less than 6 months of weekly GcMAF injections. The colorectal cancer study took about a year to cure all subjects. Five to seven years of careful followup revealed no recurrences in any of the patients. Anyone who is familiar with cancer research would have to find this remarkable.

This was not a "one off," a "lucky strike." Yamamoto's four papers were the culmination of decades of trailblazing research in which he had already proven—via basic science and animal studies—exactly how GcMAF and Nagalase work. The 2008 human trials were just the frosting on a phenomenal cake that took a quarter century to bake. The breadth and depth of the underlying research is important here because misinformed critics whine about GcMAF being "unproved." Had these naysayers read the dozens of Yamamoto papers published in peer-reviewed journals between 1979 and 2008 that lay down an unimpeachable foundation for his final proof? I doubt it.

My search for answers

When I first read Yamamoto's studies, I couldn't believe it either. A cure for early stage metastatic cancer that's effective in every single case? Absurd. Published in peer-reviewed journals? No way. I figured there must be some hitch, a mistake, a logical error, a weak link, a fatal flaw, and I was determined to find it, but the deeper I delved the more convinced I became that GcMAF was for real!

Then I started wondering why I seemed to be among the very few who "got it."

At first, I spent a huge amount of time enhancing my understanding of the relevant molecular biology, genetics, and immunology. I learned a lot more than I ever thought I would about cancer, macrophages, oxidative bursts, adhesion molecules, antibodies, phagocytosis, protein chemistry, cytokines, messenger molecules, receptors, N-acetyl-galactosaminidase (Nagalase), and GcMAF. At times I felt as if I had stuffed so much new information into my head that it was going to explode. I needed to understand exactly how it all worked, how all the pieces fit together. I poured over research articles and molecular genetics texts until I felt I had a reasonable grasp of what Yamamoto was doing and saying. I developed the ability to visualize—in great detail—the workings of macrophages battling cancer cells and viruses in this brutal microscopic cannibalistic war.

The more I learned the more it sunk in: using impeccable science, Yamamoto had found a powerful means of enhancing our bodies' own anti-cancer, anti-viral weaponry! That's what cured the cancers.

The more I learned the more I asked the question: why had the medical community—much less the average person—never heard of GcMAF? No article in the print media. No video, no book, no research articles other than Yamamoto's. No serious scientific web discussions (which is truly extraordinary, because *everything* is on the internet). No nothing.

Googling on GcMAF does generate some hits, but nothing substantial, no serious science beyond Nobuto Yamamoto's own original papers. "Hmmm. This is truly remarkable," I thought.

Next I turned to what I call "human browsers." I called a bunch of my physician buddies and molecular biology colleagues—good scientists all—but there, too, I drew a blank every single time. Not a single one of them had ever heard of GcMAF.

Not easily dissuaded, I contacted several immunology researchers at major institutions, and again no one had heard of GcMAF. Maybe someone in the government or research establishments, the FDA (Food and Drug Administration), the NCI (National Cancer Institute), the NIH (National Institutes of Health), American Cancer Society (ACS) knew something? Nope. No one there had ever heard of it either. (Or if they had, they sure weren't talking.)

In those early months, my level of frustration gradually escalated. Proof of a natural cure for advanced (metastatic) cancer (not to mention HIV and other chronic viral infections) and nobody seemed interested? I just couldn't understand it.

Finally, a sense of surrealism set in. I had spent hundreds of hours on this, with literally nothing to show for it. Nobody knew anything. And when I tried to explain Yamamoto's work to some of the people I called, I could *hear* their eyes were glazing over. I could hear them thinking, "C'mon now, doc. A cure for all cancers? Oh, sure. One that works 100% of the time? On metastatic cancer? Give me a break. It sounds like smoke and mirrors to me." I started questioning my grasp on reality. No Oprah? No Larry King? No New York Times article? No article anywhere? No media coverage of any kind! No scientific recognition? What the heck is going on here?

Solving the riddle of GcMAF

After writing an entire book on the subject, I can still honestly say I don't know why the average person—not to mention the average physician or the average molecular biologist—has never heard of GcMAF. It seems to me that that should have happened a long time ago. I hope that sharing this information will create the critical mass we need to overcome the obstacles. So: Hello out there! Here's a cure for cancer and AIDS!!! Even more significantly, here's a way to rid the planet of the scourge of cancer. I am passing on what I have learned about it to enable you to chip in and work with me to transform GcMAF from a set of abstract concepts to a lifesaving reality. Millions upon millions of lives would be saved if we could make GcMAF—a harmless protein—available to the masses of cancer and HIV patients who desperately need it. And countless cancers would be prevented using Nagalase screening and GcMAF therapy on all adult human populations.

Please help! This is a two-way street. I've chosen a reader-editable format (actually, truth be known, my web genius friend and cyberguru, created it specifically for this book) whereby anyone who is interested can contribute their ideas. The beauty of this approach is that it

facilitates collective development of ideas by an organized community. In sharing results , you will have participated in a process that has the potential to help a lot of your fellow humans and to alleviate a huge amount of suffering.

And—as if that weren't enough of a reward— you'll also (if you so desire) be listed in the Acknowledgments.

Why GcMAF remains obscure

Here are a few key facts that provide a partial answer to the fascinating question: Why has GcMAF gone unnoticed?

- Understanding Yamamoto's work requires a firm grasp of some pretty advanced molecular biology, which most people—even most doctors and researchers—don't possess. It's kind of a language problem: if someone shouts "Cancer Cure!!! Cancer Cure!!! Cancer Cure!!!" in Swahili, it is quite possible that earthshatteringness of it all won't get through, and everyone will go on about their business as if nothing happened. (In this book I have translated these ideas into everyday language. It's not *that* complicated.)
- To a stodgy medical community that's resistant to change, GcMAF is just another "unproved therapy." And an "alternative" one at that. Unproved therapies are not to be trusted. (Even if they're harmless and bioidentical.)
- "Proving" this discovery the conventional way would involve developing and promoting a lucrative drug. Doing that takes about a decade and costs over 100 million dollars. Beyond time and money, it requires a lot of biochemical know-how and some sophisticated equipment. Brewing it up in your basement lab with a chemistry set and a bunch of buddies is *not* an option. However, a motivated pharmaceutical company could do it overnight.
- Big Pharma isn't interested because there's no cash cow at the end of this rainbow. GcMAF—like all chemicals our body is programmed to make—can't be patented because it fits the FDA's definition of "natural" (translation: "unpatentable"). We are thus confronted with the supreme pickle: is it possible to conduct open-minded, non-profit driven research in an era of corporatized and politicized medical science? I yearn for the olden days when science was done for the sake of science. It wasn't *that* long ago.)
- The cancer industry does not really want cancer to go away. This may seem harsh, but it's true. Many incomes would be interrupted if cancer and HIV suddenly ceased to exist. Government agencies would have to be closed, oncologists would have to be retrained, researchers redirected, cancer treatment centers shut down or converted to screening and prevention facilities—and that's just the tip of the iceberg. We're talking profound social upheaval here. Cancer is entrenched and institutionalized, and vanquishing it would cause major fireworks. These fears are largely unfounded, however. For optimum effectiveness, GcMAF and Nagalase testing will need to be integrated into the existing cancer care system, so we need the system.
- To understand GcMAF and Nagalase we must embrace an entirely new model—a completely different approach to cancer and chronic viral infections. There *is* no super drug, no magic bullet. Our bodies already know how to cure cancer and viral infections; we simply need to enhance these systems using natural medicines. That's how GcMAF works. The scientific community, however, is deeply resistant to the idea of natural medicines bolstering the immune system.

- If we are going to commit to stopping these epidemics our new direction must be annual screening (with Nagalase or AMAS testing) for early detection, then nipping cancer in the bud with GcMAF. The old "wait until its gotten so big we can see it on imaging and then slash and burn it out" approach has really got to go.

A couple "brief asides" here, and then—in the next chapter—we'll get into how GcMAF works.

Brief aside I:

Please don't be intimidated by cell biology!

To the outsider, the world of molecular biology and biochemistry may seem bewildering and the language we use to describe cellular events often appears foreign and incomprehensible. Just because we use inscrutably complex words like glycoprotein macrophage activating factor (GcMAF) or alpha-N-acetylgalactosaminidase (Nagalase) doesn't mean the concepts are inaccessible. They aren't. Please don't be intimidated: this material is not as complicated as it might seem. My self-imposed job description has been to translate this arcane, esoteric, imposing, and highly technical science into concepts that are easily understood by the lay person—and, in the process, to bring it to life. If you find the material hard to understand, I have failed at that task.

Brief aside II:

The war analogy: I use it because it works

For the same reason that language is the most popular and appropriate analogy for describing DNA and genetics, I have chosen the war analogy to depict the nano-scale drama that unfolds on the immunological battlefield: *it works*.

Please don't think that this means I am pro-war. Quite the opposite. I think most wars are bad and stupid, and I detest them. Though I believe that some wars have been justified (a recent example would be World War II, in which control of the planet by despots was at stake), I am one who is hopeful that members of the human race can learn to work things out without resorting to needless slaughter. We will have reached a high point in our evolution when we learn to put war behind us once and for all.

Speaking as a humanoid member of a cell-based life form, however, it is crucial to acknowledge the necessity of this inner war against cancer and microbes, those evil forces that are out to destroy us. Pacifism won't work here; we really have no choice but to fight back or die.

2: Professor Yamamoto and Real Science

There are three kinds of research studies. (Actually, there are many many kinds of research studies, but for the purpose of this article, I'm making it three.)

First, there's what I will call *"black box"* studies. In these, you try something out on experimental subjects and see what happens. The actual internal mechanisms driving the results are not necessarily known: "Oh, when we fed the rats mushrooms and green tea, some

of their cancers went away." Interesting and useful outcomes, but cause and effect are ignored.

Second are *population* studies in which (not necessarily accurate) conclusions are drawn from statistical studies involving large groups of people: "We found that 50% of people who had heart attacks had normal cholesterol levels. We therefore conclude that…" Again, underlying causative mechanisms are not part of the picture.

With both black box research and statistical studies, the importance of cause and effect are effectively dismissed: the underlying cause of an observed effect is never known. This is a fundamental flaw; we can't trust the evidence we get from black box and statistical studies because they don't address causality. For this reason, I pay very little attention to these first two kinds of research studies.

The third type of research is *basic science*, where ideas are developed and tested from the ground up. Insights gained in one study become the driving force for designing the next one. Professor Nobuto Yamamoto did—and still does—*basic science* research. He spent a quarter of a century examining—in minute detail—the basic molecular biology and immunology supporting his discoveries, alpha-N-acetylgalactosaminidase (Nagalase) and glycoprotein macrophage activating factor (GcMAF). There's no black box, no statistics, just a complex trail of experimental insights that collectively form a colossal infrastructure upon which his final conclusions are based. He didn't try out a new, unknown and potentially toxic drug on a bunch of people; he did the basic science research and figured out—exactly, precisely, down to the submolecular level—how cancer breaks our bodies, and then he showed how to fix it using the body's own systems as his therapeutic tool. It's a brilliant, Herculean, masterpiece of work, and Dr. Yamamoto deserves a Nobel Prize for his efforts. He also deserves to have his ideas widely understood and taken seriously—an outcome that has not yet happened.

Dr. Yamamoto is healthy and active at age 84. I feel that for him the best reward of all would be to see, in his own lifetime, the world's cancer and HIV patients benefit from his work.

3: Your Incredible Immune Army

Your immune army uses an amazing array of weapons to protect you from cancer and microbes.

There are good guys (our white blood cells) and bad guys (cancer and microorganisms) in us, and they are fighting it out for control of the territory that is *you*. I am going to put you in front of a very powerful microscope so you can see—in vivid detail—this constant vicious struggle going on inside every one of us. The opposing armies conduct complex military maneuvers and deploy a fantastic and unimaginably small array of war machinery, all coordinated by a sophisticated communications system.

The bad guys—the foreign invaders—are constantly attacking us; I speak of allergens, infectious agents, various toxins, and cancer. Our immune army, our guardian, is comprised of dozens of cell types with hundreds of different functions. In any single person, immune cells number in the hundreds of billions—several times more then the number of stars in our galaxy or galaxies in our universe! They routinely sacrifice their teeny (but incredibly complex) lives to defend us.

Healthy immune cells are essential to our survival in this ongoing war, and they are relentless in their pursuit of cancer and other foreign invaders. When activated, they make Hitler, Stalin, and Mao look like neighborhood bullies. Immune cells attack in extremely large numbers—far larger than any army ever. Come to think of it, every single human has far more immune warriors than all the armies in history put together. Their onslaught is superbly coordinated by an extremely sophisticated communication and command system; human military intelligence operations are kindergarten games in comparison. Using a complex language comprised of molecular words (proteins, glycoproteins, cytokines, cell-signaling molecules, neurotransmitters, etc.), your immune cell soldiers release a barrage of chemical messages that identify foreign invaders, provide their location, estimate numbers, and coordinate the attack.

Macrophage engulfs and phagocytizes a cancer cell. From "The Immune System" published by The Upjohn Company.

And what an awesome attack! Our immune cells sport an arsenal that would make Star Wars look puny. Immune cells can blow up enemy invaders. They can shoot out beams of ionized particles that literally rip holes in the outer cell membranes of infectious microbes and cancer cells. They release spurts of corrosive chemical poisons. They surround, cannibalize, and digest enemy cells—and then recycle the parts. They launch guided missiles from great distances that land and explode with incredible precision. They even smother their enemies in sticky goo (called complement) like the Marshmallow Man in Ghostbusters.

Your immune army deploys these remarkable weapons in multiple wars on several fronts. The skin, respiratory system, intestinal tract and bloodstream sustain the largest exposures and thus contain the most immune cells. Our largest exposure to pathogens—by far—is in the gut. Because this is the largest and most frequently breached barrier, roughly 80% of your white blood cells are embedded just beneath the intestinal mucosal surface. Microbes—in the form of bacteria, fungi, parasites, viruses, and the occasional helminth —are persistently knocking on the door and must be fended off on a non-stop basis. As you will see, we fend with some pretty big sticks. Imagine an army with hundreds of billions of white blood cell soldiers, each fully-equipped to take on cancer cells, viruses, and bacteria *mano a mano* in a struggle to the death.

Cancer cells are forming continuously in all of us, but alert, *activated* (you'll soon see why I italicize this word) immune cells will give them the instantaneous hatchet. If your constantly vigilant and aggressively protective immune system took a coffee break, you'd be dead by the time it was over.

I am going to tell you a lot more about the wondrous, though violent, molecular biological world inside of us. But first, let's take a look at the "big picture," an overview of our "War on Cancer."

4: The War on Cancer Inside Us

Cancer is a war waged on a submicroscopic molecular scale. Taking a keen interest in this war wouldn't be a bad idea, as its outcome will eventually determine whether about a third of us will live or die. The advent of Nagalase testing and GcMAF therapy gives us reason to hope and expect that we can soon eradicate the specter of cancer. There'll be problems along the way.

Cancer is many things to many people. From the point of view of the cancer patient, cancer may be seen as a fear-inducing, life-threatening nightmare. From the perspective of a family member, who may be called upon to provide emotional, physical, and/or financial support, how could cancer be anything other than stressful? From the viewpoint of the doctor, who must find and treat the cancer to the best of his or her ability, cancer is both a backbreaking, sometimes thankless, job *and* an opportunity to provide exceptional, loving patient care.

The molecular biologist, however, views cancer as a vicious and protracted battle waged between cancer cells and immune cells. This cancer war—a strategic dance of cellular activities—is breathtaking in its elegance and sophistication.

Two cell types—cancer cells and macrophages—comprise the main forces of the opposing armies. Both have massive numbers of personnel and awesome weaponry at their disposal, and both have a strategy for winning.

Remember, this is WAR!!! Cancer cells are genetically programmed to survive and spread. On the other side our immune system consists of enormous numbers of crafty white blood cells doing their best to defend us.

Taking a keen interest in this war wouldn't be a bad idea, as its outcome will eventually determine whether about a third of us will live or die. Hundreds of billions of cellular warriors on both sides will die before it's all over. The stakes don't get much higher. If the cancer (or virus) wins, you will cease to exist.

GcMAF treatment and Nagalase testing will dramatically change the way this war is fought. With some hard work and perhaps a few lucky breaks, we now have reason to hope—and even expect—that we can eradicate cancer. Not just in our lifetimes, but in the next few years.

When I said "a few lucky breaks" above, I was referring to our medical care delivery system, including the federal government, Big Pharma, insurance companies, cancer care facilities and cancer specialists. For a multiplicity of reasons—including but not limited to the flow of cash—this gargantuan beast resists change. GcMAF and Nagalase are the two new elephants in the room. Accepting that these benevolent behemoths have moved in for good—and making space for them within our current cancer care system—is going to be the challenge of the century.

People will continue to die because of the reluctance of modern medicine to embrace change. If we started screening everyone for elevated Nagalase today, and giving GcMAF to those with high levels, we could eradicate cancer literally overnight. Left behind in the rubble would be some some very unhappy drug salesmen and more than one bored but retrainable oncologist with an overdue Mercedes payment.

5: Your Immune Cells Versus The Pathogen Army: A Nanoscale War

About the nano-scale war fought inside us. Some important features of cancer cells. It's the cancer army vs our immune army. How macros tell friend from foe. Macrophages are the ultimate fighting machine, but they're naturally indolent and remain idle

without GcMAF activation. Nagalase, made by cancer cells and viruses, puts macrophages to sleep by blocking GcMAF production. Without GcMAF, macrophages remain comatose. GcMAF injections bypass Nagalase, re-activate the macros, and this jump-starts the entire immune system.

What is cancer?

Cancers are made up of cells that carry damaged genes. These cells have stopped cooperating with the rest of the cellular community, and are growing out of control. Once a useful part of us, and still harboring our genome, these deranged cells have now mutated into our archenemy, hell-bent on destroying us. Left to their own devices—they will. They grow, forming an isolated local renegade community made up of malignant cells. This tumor can eventually send its cells through the bloodstream (we call this metastasis) to establish satellite communities at remote locations in other organs.

The disease we call cancer is actually a nano-scale war fought in each of us every moment of every day. We now know that we are all developing cancer cells all of the time, but that a healthy immune system devours them as fast as they are made. Anything that weakens the immune system can shift that delicate balance in favor of cancer, allowing it to grow.

Some important features of cancer cells

- *Cancer cells don't stop reproducing* – Unlike normal cells, cancer cells do not stop reproducing after they have doubled 50 or 60 times. This means that a cancer cell will go on and on and on doubling. So one cell becomes 2, then 4, then 8, then 16. The cancer cells may be able to stop themselves self -destructing. Or they may self - destruct more slowly than they reproduce, so that their numbers continue to increase. Eventually a tumor is formed that is made up of billions of copies of the original cancerous cell. Scientists describe cancer cells as being 'immortal'.
- *Cancer cells don't obey signals from other cells* – Something in the cancer cells overrides the normal signaling system. This may be because the genes that tell the cell to reproduce keep on and on firing. Or because the genes that normally tell the cell to stop reproducing have been damaged or lost. So the cancer cell keeps on doubling, regardless of the damage the extra cells cause to the part of the body where the cancer is growing.
- *Cancer cells don't stick together* – Cancer cells have lost the molecules on their surface that keep normal cells in the right place. So they can become detached from their neighbors. This phenomenon also helps explain how cancer cells spread to other parts of the body.
- *Cancer cells don't specialize; they stay immature* – Unlike normal cells, rather than maturing, cancer cells become more and more primitive (we call this *undifferentiated*) and tend to reproduce ever more quickly and haphazardly.

The cancer army

To prevent bumping into one another, normal healthy cells regulate their growth rate by communicating with neighboring cells via a mechanism called "cell recognition" in which each cell sends out multiple chemical messages in an ongoing biochemical "conversation"—a heated discussion about who gets to grow (or do other things cells do), and when. There is an etiquette all cells subscribe to, and this very civilized molecular exchange definitively settles

matters to everyone's satisfaction. Cancer cells, however, have "opted out." They no longer agree to do this. They no longer feel the need to work out an agreement about territorial issues. They want it all now, and refuse to stop growing. Having regressed to barbarians, they just push everyone else aside. It's what would happen if a group of complete social idiots crashed a civilized dinner party, elbowed the guests aside, and started grabbing all the food for themselves. Respect for the rules that govern cooperative society is not on their radar. Say hello to the cancer army.

The cancer army's most effective weapon—by far—is Nagalase, an enzyme made by all cancer cells (and viruses) that shuts down GcMAF production, thus disabling the entire immune system. Macrophages need to be "activated" by GcMAF; without it, they just can't fight. By blocking GcMAF production, Nagalase effectively cripples the immune army by putting its soldiers into a deep sleep. (Later on, I'll be going into much greater detail about how this all works.)

Your immune army

Your body also has an army. It's made up of your fearless immune cells, ready to square off against the cancer army and wage a protracted war. Each side's fighters can number in the hundreds of billions, but the battle itself consists of hand-to-hand combat between individual macrophages and cancer cells in a battle to the death. The macrophages attack and engulf (phagocytize) the cancer cells. The cancer cells are trying to grow as fast as they can, while preventing the macrophages from killing and eating them.

Macrophages are the principal cellular soldiers of our human immune army. They exist to protect and defend us from threats like cancer. Stationed at critical strategic locations around your body (and embedded in every tissue type), these cellular behemoths stand guard, ready to deploy sophisticated weaponry should an invader—a virus, bacteria, fungus, toxin, or allergen—or a cancer cell—dare to …er, … invade.

As cells go, macrophages are massive: roughly 30 times the size of an average body cell. If a cancer cell were the size of a motor scooter, a macrophage would be bigger than an 18-wheel semi. But this would be no ordinary truck. More like a Sherman tank, the macrocyte comes armed to the teeth with a multiplicity of nasty weapons. It also knows how to tell friend from foe—and if you're foe, prepare to be more or less instantaneously liquidated.

In their quest to kill cancer cells and HIV virions, macrophages aren't shy about deploying their array of high-tech devices. They extrude clusters of long skinny powerful octopus-like arms that grab onto and then drag their victim in, whereupon they surround, engulf, and digest it. They emit free radical rays that fry the outer cell membranes of cancer cells and microbes they've tracked down. Then they casually spit out the dead parts and move on to the next cancer cell, bacteria, or virus.

Utilizing sophisticated surveillance and communication systems, macros exchange complex messages with B and T lymphocyte cells. Outgoing messenger molecules tell lymphocytes where and how to aim their antibodies and inflammatory responses, and incoming messages help the macros zero in on the enemy, providing specific directions about where and how to direct their intimidating weaponry.

How macros tell friend from foe

Because it is very important that these killing machines not accidentally attack our own cells ("friendly fire"), they are equipped with a system for distinguishing "self" from "other." It's an ID check, not unlike like police use when they inspect a driver's license at a routine traffic stop. First the macrophage sends out a "cell extension," a long protoplasmic arm that wraps around potential targets (which are often identified by a cloak of sticky IgG antibodies), putting them into a strong "headlock." The arm then contracts a little, pulling the intruder closer, in order to be able to "frisk" it. (Like a cop grabbing a fleeing suspect.) "Self"-cells have a molecular surface protein (their "drivers license") that tells the macrophage, "Friend here; please let go of me." The macrophage responds by switching off the headlock, allowing the self cell to move on. Foreign cells, i.e., those without the protein "license," suffer the ignominious fate of being not just apprehended, but also summarily eaten alive. No ticket, no trial, no judge, no jury; this is raw cellular vigilante justice.

Imagine, if you can, literally *billions* of these macrophage war machines simultaneously tracking down, apprehending and dispatching malignant cells inside of a cancer patient.

The outcome of this microscopic war will be decided by one simple strategic fact: whether or not the cancer army can successfully incapacitate our macrophage soldiers, because if this happens, the cancer will prevail.

Macrophages are cunning and scary—but naturally indolent

Macrophages are the ultimate fighting machine. But they are not actually a machine: they are alive; they "think;" they make well-informed decisions. Each possesses a copy of your DNA, complete with all 25,000 (or so) genes, and this means they have the capacity to synthesize tens of thousands of chemicals—whatever they need to fight the war.

But, as we shall see in a moment, macrophages are natural slackers; without a kick in the *derriere*, they'll stay asleep. That kick, the motivation to get up and get going, comes in the form of GcMAF, a protein synthesized by our lymphocytes. GcMAF attaches to receptors on the surface of the macrophages and sends a powerful message: "Get to work … NOW!!!"

Two macrophages (left, brown) ensnaring bacteria (blue) in their long pseudopods. (Encyclopedia Britannica)

Nagalase (made by cancer cells and viruses) puts macrophages to sleep

The main weapon deployed by cancer cells and virus particles to sabotage our macrophage-driven immune response is an enzyme with a tongue-twisting name: alpha-N-acetylgalactosaminidase. We call it *Nagalase* for short. Nagalase defeats our immune system by blocking GcMAF production. Without GcMAF, macros remain comatose. Without macrophages to stop them, pathogenic invaders can grow at will. With Nagalase on their side, cancer cells and viruses will multiply and they will spread. In chapters 9, 10, and 11 I'll explain how Nagalase accomplishes the remarkable feat of outmaneuvering and zombifying our otherwise intimidating macrophage warriors.

Macrophages remain idle without GcMAF activation

In order to realize they are supposed to go out and destroy cancer cells, macrophages must be "activated." GcMAF (stands for glycoprotein macrophage activating factor) is our bodies'

principal macrophage activating factor. GcMAF is a protein that is made and released into the bloodstream by your T and B lymphocytes. Macrophage surface receptors monitor incoming chemical messages, patiently waiting for specific orders to activate. Much as a tiny key fits into a very small lock, GcMAF molecules locate and lock onto specific GcMAF receptors on the outer surface of your macrophages. Inserting the key (GcMAF) into the lock (GcMAF receptor) unleashes a powerful alarm that is instantaneously heard everywhere inside that cell (even though the cell is millions of times larger that the GcMAF molecule!) Though there are some weaker chemical messengers that can give a little nudge toward activation, when GcMAF comes along, macrophages really pay attention. With volume turned all the way up, GcMAF shouts, “”Get going!!!! Track down and kill all cancer cells. Kill all viruses!!! DO IT NOW!!!” It’s kind of like a drill sergeant barking commands in boot camp. Or agent Jack Bauer mobilizing Counter Terrorist Unit field operations in “24.”

Based on size alone, this Lilliputian GcMAF dictator has more power than the director of the FBI or CSI. And the “signal” is not a request; it’s a command. This is power and leverage—like the single switch that turns on all the lights in a football stadium. When GcMAF talks, macrophages listen.

When a macrophage is under the control of GcMAF, its activity level increases by a factor of 30. If it were an automobile, the macro would accelerate from 5 to 150 miles per hour. But we’re not just talking velocity here; we are, more importantly, talking about an explosion of activity. The safety is off; “Big Mac” is now armed, ready to do battle, and actively seeking engagement with the enemy. The rate at which this now *activated* macrophage can seek, grab, and phagocytize alien life forms (and other nonliving foreign intruders, like toxins and allergens) is awe-inspiring.

GcMAF bypasses Nagalase and re-activates macros.

Once activated by GcMAF, macrophages morph into relentless killers. But when *de*-activated by Nagalase, they slow to a crawl and refuse to deploy their awesome array of weaponry They start losing the war on cancers and microbes. If macrophages remain deactivated, the patient will eventually die. Injecting GcMAF bypasses the production blockage, and *re-activates* the macros.

The following charts from Dr. Yamamoto’s early metastatic cancer and HIV studies illustrates how GcMAF injections bypass the Nagalase production blockage and cure cancer. Patients received weekly injections of 100 nanograms of GcMAF. The GcMAF-activated macrophages destroyed the pathogens (cancer cells and virus particles), thus lowering the amount of Nagalase they make. Weekly Nagalase reaches healthy control levels in all cases, indicating the patients are cured.

6: Your Awesome Macrophage Killing Machine

Macrophages are big and smart white blood cells that chase, capture, engulf, and digest intruders. They trap and phagocytize (literally, “eat”) their enemies. They can multiply rapidly when necessary. However, they’re naturally indolent and need to be activated by GcMAF. Opsonin “super glue” helps them stick to their prey. Their electron-driven free radical death ray (AKA “oxidative burst”) blasts holes in microbes and cancer cells. Once a microbe or cancer cell has been phagocytized by a macro, it is encapsulated inside a “phagolysosome” (the intracellular “death chamber”), where it is

then killed (if it isn't dead already), and then dissected down into its component parts, which are then recycled.

Although I have already described macrophages, these large immune cells are so important in terms of understanding how GcMAF works that I need to go into a little more detail about them. Besides, they are truly fascinating critters!

A macrophage

If you could imagine a living, breathing, oozing, cunningly horrific humongous sticky blob that combines the dangerously diabolical features of King Kong's intimidating size, Hannibal Lechter's cannibalism, Darth Vader's lightsaber, The Terminator, Bruce Lee, and the Marshmallow Man that takes over New York City in *Ghostbusters*—all rolled into one giant killing machine—you'd have some idea of what a macrophage is all about. They're big. They're nasty. If you were a bacteria, virus, or cancer cell, you would do your darnedest to avoid them.

As cells go, macrophages are huge

How big is a "Big Mac"? By way of comparison, red blood cells, white blood cells, and typical cancer cells are about 7 microns (micrometers or millionths of a meter) in diameter and have a volume of about 250 cubic microns. At about 20 micrometers (20 millionths of a meter) in diameter, macrophages are about three times as wide as regular cells. But, because a little extra width translates into a lot of extra volume, macrophages—at around 4000 cubic microns—have about 16 times the volume of normal sized cells. If a cancer cell were the size of a Toyota pickup, a macrophage would be bigger than an 18 wheeler.

Not a dumb truck

But this behemoth is not a dumb truck. Bristling with weaponry, it's stuffed to the gills with a daunting array of high-tech systems programmed for a singular purpose: take out the enemy as quickly and efficiently as possible. We call this "tumoricidal capacity."

Here's how it works. When it isn't swimming in the blood stream, a macrophage can slowly "walk" through tissues using self-generated stumpy little (one micron) "legs" (about ten of them sprout at a time). The macrophage ambles over to and snuggles up alongside a "foreign invader" (e.g., cancer cell or virion), quickly identifies it as foe, sprays it with membrane-frying free radical-laden Darth Vader death beams, grabs, engulfs, smothers, kills, and digests it. If the enemy is further away, or trying to escape, the macro chases after it, extrudes a cluster of long thin sticky spaghetti-like tentacles that wrap around and ensnare the fugitive cell, clutching it in an unbreakable strangle hold.

In a process known as phagocytosis, the macro draws in its victim, engulfs and smothers it, then encases it in a small bubble-like cyst (called a phagolysosome) inside its cytoplasm. The phagolysosome then secretes a cocktail of corrosive free radicals and enzymes that rapidly digest its victim down into its component parts (amino acids, nucleic acids, fatty acids, etc.). The macrophage then spits out these pieces into the intercellular "soup. " Because the remnants of viruses and cancer cells are fundamental cellular building blocks, the body quickly recycles them using the "spare parts" to build brand new healthy cells.

I finding it totally amazing that this complex and truly violent scenario is unfolding in you and me billions of times per minute.

A review of the macrophage's most important weapons:

Pseudopods

Literally "false legs." These can be short and stumpy, e.g., like the ones macros make in order to "walk" along the inner lining of blood vessels. For chasing and grabbing fugitives, however, macros can make much longer pseudopods that extend out relatively large distances (perhaps 60 microns). Imagine a macro the size of a Volkswagen with the capability to extend hundreds of long thin arms (each about the diameter of an exhaust pipe) out to 50 feet or more. Once out there, they can weave themselves into a net that tangles around and traps the hapless enemy. If that target were a cancer cell, it would be about the size of a motor scooter. If a bacterium, it would be about the size of a roller skate.

Macrophage ensnaring bacteria

Phagocytosis and phagolysosome formation

Once the pseudopods have ensnared their victim, the engulfing process ensues. The outer membranes of the pseudopods nearest the microbe or cancer cell simply merge into one another so that the victim is completely surrounded and encapsulated in what is called a *phagolysosome*. ("*Phago* " means "eat," "*lyso*" means "digest," and "*some*" means "cell" or "body.") Amoeba-like, the macrophage has reshaped itself such that the phagolysosome lies deep inside. Then the membrane that makes up the wall surrounding the phagolysosome shoots more death rays at its captured prey (just to make sure it is dead), and proceeds to digest it with an array of corrosive enzymes. More about phagolysosomes in a minute.

To see a cool video of a white blood cell (a neutrophil) chasing and phagocytizing a bacteria, go here:

http://www.youtube.com/watch?v=MgVPLNu_S-w&NR=1

(This is a neutrophil, not a macrophage; a macrophage would be about 16 times larger.)

Opsonins: Super Glue "binding enhancers" that help macros latch onto enemies

To help them grab and hold their victims, macrophages send signals to nearby lymphocytes, instructing them to spray a thin coating of sticky proteins onto potential prey. Then, when the macro's long thin arms make contact with the microbe or cancer cell, this "super glue" coating hardens, making it impossible for the desperado to shake loose.

Typically, a macro sends out a cluster of (say twenty or so) sticky pseudopods that surround the enemy cell, encasing it in a mesh like affair, not unlike a large fish net, in which the microbe or cancer cell becomes ensnared. Like a fly in flypaper, the enemy cell is both stuck *in* it and *to* it, so there is no way to get loose. Then the prey is gradually surrounded and engulfed, ending up snugly inside the macro as a *phagolysosome*, in which it spends its last few moments as a life form before being digested down into its component parts by various free radicals and enzymes.

The sticky proteins are called “binding enhancers” or “opsonins.” The gluing process is called “opsonization.”

Interestingly, when a macro grabs an enemy this way, it wants its fellow phagocytic soldiers to know prey is nearby, so—like an isolated soldier who has stumbled upon a group of enemy troops and is calling for backup— it sends out protein signals telling nearby macrophages to make more of the receptors that specialize in grabbing specifically that kind of enemy. There’s safety in numbers. (Technically this is called “upregulating expression of complement receptors on neighboring phagocytes.”)

Electrons death ray beams from the “oxidative burst”

Because it is so diabolically sophisticated—and right out of *Star Wars*—my favorite macrophage weapon is the “oxidative burst” (also widely known as the “respiratory burst”). This is the Darth Vader death ray. An enzyme (called NADPH oxidase) stationed in the macro’s outer membrane sprays out a beam of highly reactive free electrons, like bullets from a machine gun.

Remember those old TV sets with picture tubes? The NADPH gun is kind of like that. At the back of the tube an electron gun aimed particles that hit phosphorescent particles on the screen. When the electron beam hit the particles, the screen lit up, creating a picture. Likewise, NADPH also emits a particle beam. But instead of playing Howdy Doody, it’s blasting tumor cells and microbes to smithereens.

The electrons in the beam emerge one at a time, but they really really don’t want to be “free,” so—as fast as they possibly can—they snatch another electron to form a stable pair (we are talking nanoseconds here). A chain reaction of electron-snatchings triggered by the oxidative burst literally vaporizes molecules in the outer wall of a cancer cell or viral capsid, ripping holes in it. Now the membrane that held the victim together literally falls apart, spilling out its contents. Without an intact outer membrane, a cancer cell can’t survive for very long.

Oxidative bursts don’t happen all of the time. That would be a waste of firepower. The “trigger” that turns it on is the perceived proximity of a “foe,” a cancer cell, HIV virus, hepatitis virus, or a bacterium. When a macro comes into immediate contact with “enemy,” then—and only then—does it turn on the electron death beam.

There are lots of oxygen (O2) molecules everywhere in our bodies. (We need plenty of oxygen and glucose, the “fuels” from which we generate the “energy” that drives all of the cellular chemical reactions that make life possible.) When released, most of the electrons in the death ray beam crash into one of these omnipresent oxygen molecules, from which they quickly grab the electron they need to make a stable pair. The oxygen molecule now is missing one of *its* electrons, and is thus transformed into the violently corrosive free radical known as “superoxide” (O2-). Now superoxide is the one wanting an electron, and it will destroy anything in its path to get one. That “anything” would be the virus, bacterium, or cancer cell our macro has grabbed with its pseudopod. Suddenly the invader finds itself with a huge hole in its outer membrane. It’ll die soon.

The free electrons and superoxides also trigger chain reactions forming other reactive free radical species. One of these is the hydroxyl ion (OH-). This is hydrogen peroxide, just like

the stuff that comes out of that brown bottle, but 33 times as potent—a locally generated intercellular dose. Perfect for frying microbes and tumor cells.

By oxidizing omnipresent chlorine atoms, the electron beam also generates noxious hypochlorous acid (HClO), which can poke a hole in an enemy membrane in nothing flat. Now we have a toxic soup of free radical oxidizing agents that can do tremendous local damage to our enemies.

"Wait a minute," (I can hear you saying,) "how come our own cells aren't damaged by friendly fire? How do they escape the death rays?" Great question. We have a protective shield that prevents the free electrons and free radicals from damaging our own cells. It's called SOD (superoxide dismutase) and it's an enzyme (a large protein molecule) that specializes in neutralizing superoxide and other free radicals before they can damage our own cells. For maximum protection, SOD is positioned right next to the NADPH death ray generator proteins in the outer cell wall (or membrane) of our macrophages.

The "barrel" (the NADPH molecule) of the macro's electron-generating death ray gun is aimed toward the outside of the cell and sticks out of a little hole that is surrounded and protected by molecules of SOD, forming a kind of "bunker" to protect the electron gun and your macrophage cell. As long as we keep making SOD (and we'd be dead in minutes if we stopped), we'll be safe; the electron beam can't harm us. It's a pretty cool combination: a ridiculously deadly weapon with built-in safeguards for the user (that would be you).

GcMAF-Activated Macros and the "Oxidative Burst"

You've heard this before, but I have to say it again: only *GcMAF- activated* macros are going to deliver oxidative bursts that are potent enough to be effective. If Nagalase from viruses or cancer cells has put the macros to sleep, the oxidative burst degenerates into a piddly potato gun that's not going to hurt anybody. Firepower—or lack thereof—is what we are talking about here. Remember those old westerns in which six-shooters were the main weapon? There'd be a shot here, long pauses, and then another shot over there? There was a long enough gap between shots that you could actually hear the ricochets. That's a *de*-activated mac: slow at the draw and not getting very many shots off. Reloading after every six shots. No wonder the Indians creamed Custer. *Activated* macros fire the atomic equivalent of millions of rounds a second and never have to pause to reload. Some newer movies have so many bullets flying from so many directions that it is hard to understand how anyone could survive. That's firepower of the sort only GcMAF-*activated* macrophages could deliver.

The Phagolysosome execution (and dismantling) chamber

If, somehow, a microbe or cancer cell has survived the oxidative burst and phagocytosis, it will *not* survive the death chamber. Once eaten, internalized, and embedded in the macrophage's cytoplasm, the enemy is imprisoned in a round cyst-like bubble inside the macrophage (called a *phagolysosome*) into which are squirted all sorts of digestive enzymes and many more rounds of oxidative burst, just for good measure. Pretty things do not happen inside of phagolysosomes. If the cancer cell or microbe is not already dead, the phagolysosome "death chamber" will certainly polish it off. ("Phago" means "to eat." "Lyso" means "to dissolve." "Some" means "sack" or "bag.")

Once the dismembering process is complete, the phagolysosome slides over and makes contact with the outer cell membrane, merges with it, then disgorges the now harmless breakdown products (nucleic acids, fatty acids, amino acids, etc.) out into the extracellular fluid. They are then taken up by nearby cells and recycled into new body parts. The ecologically-minded among us should find the efficiency of this process commendable. Nothing is wasted. Scary toxic bad guys are killed, dismantled, and transformed into spare parts for the good guys: *us.*

A sophisticated communication system

Talk about communication systems! Immune cells—macrophages and lymphocytes— carry on a constant blather, like a huge town hall chat room where everybody is talking at once. However, since the talking is a release of "messenger molecules" and the listening is done by protein receptors, immune cells can actually *listen while they are talking*!! No need to complain about being interrupted! It's weird, and foreign to us humans, but this simultaneous talking and listening makes for a far faster exchange of messages than if you had to stop and listen every time the other guy was talking (like we humans usually do).

There is so much activity, what with the constant molecular chatter coupled with a madhouse of cellular scrambling to grab and kill enemy cells as rapidly as possible, that the casual observer might get the impression of chaos. But she would be sadly mistaken. There are no wasted efforts here. Like a Beethoven symphony, everything is extremely well-organized and perfectly coordinated.

The chemical chatter among macrophages and other immune cells is so rapid and efficient that it would make a sophisticated military communications system look like a bunch of kids with tin can phones. Macros release clouds of messenger molecules (cytokines, interferons, leukotrienes, and other small molecules)—at rates of up to thousands of molecules per second per cell. Each molecule carries a specific request or command. Like "Bring me this," or "We need some of that over there," or "Kill everything that looks like this." "We need an inflammatory response over here." Or "We don't need to do that anymore." They discuss what the enemy looks like and how aggressive he is. They tell each other how hard to work. They label targets for other cells to identify and kill. They talk about where the enemy is hiding. They discuss current enemy strategy and how best to outmaneuver it.

Exponential self-cloning: the ultimate weapon

Last, but definitely not least, macros—if outgunned—play the population card: they multiply rapidly. When they find themselves in an area of high cancer cell or viral particle density, they don't have to call up the draft to get more troops; they simply clone themselves, which they can do on very short notice. More macros automatically translates into more of all the other weapons enumerated above. But, again, this multiplication process occurs only in activated *macros*.

GcMAF Activation

Without GcMAF, macros languish. In the presence of GcMAF, their activity level increases exponentially. Once activated, macros multiply rapidly and attack ferociously. In the following chapter, I explain why…

7: Macrophages Need GcMAF to Thrive

Despite their intimidating size and multitude of nasty weapons, macrophages languish without GcMAF "activation." Quantifying immune activation. Macros use "antigen presenting" messengers to reverse immunosuppression and activate other immune cells. Elevated Nagalase blocks GcMAF-driven macrophage activation, resulting in immunosuppression.

I think you'd have to admit that macrophages are pretty amazing—and intimidating—characters. Expanded to human scale, they would be far superior to any existing weapon. They'd be able to blow their way through cement walls, devour moose-sized animals whole and grab stuff that was fifty feet away with a cluster of their long strong *ad hoc* arms. But—as with most things that seem too good to be true—there's a hitch. Despite their incredible potential to protect and defend us, macrophages are naturally indolent. These slackers sleep until noon (figuratively speaking), and hang out in the blood stream like a beach bum at the seashore. It might be more generous to say that in their natural state they are "off duty." Macros accomplish absolutely nothing beyond eating enough to stay alive, and indulging in the occasional "replication experience." An inactive (or deactivated) macrophage lacks the killer instinct and might float right past a huge gathering of cancer cells or some partying virus particles, oblivious to their presence. Not exactly a killing machine; more like a gargantuan lifeless blob of cold gooey marshmallow.

To get motivated, these loafers need a swift kick in the butt. Because we are serious scientists, however, we have to call this "macrophage activation." It is accomplished by specific "activator proteins" that attach to receptors on the macrophage's surface, not unlike the toe of a boot (the activator protein) being embedded in the butt (the receptor) of a slacker (the macrophage). Though several types of molecules are known to give macros a gentle shove, GcMAF is by far the most potent macrophage activating factor. In fact, nothing comes close.

When GcMAF activates macrophages

As GcMAF docks on its receptors on the outer surface of the macrophage, it sends a signal to the entire cell, telling it to become aggressive, accelerate all activities to warp speed, and prepare for battle. Our laid- back beachcombers morph into frenzied kamikaze warriors. We are not talking about a nudge here; the word "activation" may be too weak to describe a phenomenon that consists of a 30-fold increase in macrophage activity. Imagine a Model T putting along at 20 mph suddenly transforming into a jet plane doing 600!!! You get the idea.

GcMAF activation transforms macrophages—more or less instantaneously (okay, it takes about 3.5 hours)—into a super conqueror that vanquishes cancer cells and virions at a phenomenal rate. GcMAF restores the "tumoricidal capacity" of macrophages—it's ability to rapidly scarf and kill viruses and cancer cells—that had been obstructed by the Nagalase. Battalions of activated macros now track down and chew their way through enemy armies, spewing out dead parts along the roadside behind them so fast they make it look easy.

Quantifying macrophage activation

Dr. Yamamoto quantified several specific measures of macrophage activation. Comparing deactivated to GcMAF-reactivated macros, he observed a 30-fold increase in rate of tumor

cell ingestion (phagocytosis), a 15-fold increase in the "oxidative burst" (a sudden release of superoxide (O2-) ions that zaps cancer cells, bacteria, and viruses), a 40-fold increase in systemic macrophage counts, and an exponential 180-fold rise in macrophage levels in inflamed lesions. (The latter because activated macros, via a process known as *chemotaxis*—are attracted toward, and migrate, to areas of inflammation—e.g., cancers and viral infections.)

Weekly injections of GcMaf unleashes armies with billions of these GcMAF-activated cloned macrophages that relentlessly attack and gradually annihilate small tumors within about six months. (Dr. Yamamoto's metastatic breast, colon and prostate cancer cases were all put into a five year remission—if not cured—within this time frame.)

Larger tumors—a bigger meal for the macros, so to speak— present more of a challenge. Here, results will certainly vary from one patient to the next, and some will not be curable. We really don't yet have hard data on larger masses. The macros will certainly do their best—if activated—but for each patient there is going to be a point of no return where the tumor has gotten so big (or the viral load so large) that it can successfully fend off and stay ahead of the best efforts of the activated macros.

Activated macros use "antigen presenting" messengers to reverse immunosuppression

Like that proud mouser cat who insists on dropping dead rodents at his master's feet to prove he is earning his keep, macrophages, in a strange, microscopic sense, are similarly proud of the fact that they have identified, trapped, phagocytized, killed, and dismembered a desperado, be it cancer cell, bacterium, or viral particle. So, to spread the word of their success to their immune cell comrades, macros release triumphant clouds of messenger molecules.

Macrophages also want to pass on identity information to other immune cells so that everybody else can more easily identify the bad guys. To accomplish this, macros biochemically paint the molecular biological equivalent of a huge luminescent "X" on scraggly pieces of leftover detritus that (prior to dismemberment in its phagolysosome) had been part of a cancer cell or a virus. Once released into the intercellular fluid, these brightly-labeled parts float off like a message in a bottle that, when discovered by another immune cell, tells it to "Keep an eye out for anything resembling this guy—and when you see it, kill it."

Other immune cells find the bottles, read the messages, and instantaneously know—and remember—that this is the kind of cell they should be targeting. Word spreads fast among phagocytes. Since living cancer cells contain these same parts as those marked with the "X," lymphocytes, other macrophages, and other immune cells now can instantaneously recognize the enemy and take them out without further ado. We call this "antigen presenting," and it serves a dual purpose: first it spreads the word about whom to target, and second, it transfers the activation of macros to all other immune cells.

Elevated Nagalase is synonymous with immunosuppression

If macros aren't activated, antigen-presenting (and all other immune functions) slows to a crawl. Cancers and viral infections deactivate macrophages by manufacturing and releasing Nagalase (alpha-N-acetylgalactosaminidase), the enzyme that prevents GcMAF production.

Without GcMAF (and *with* Nagalase), the entire immune system has dropped out. They've reverted to a collection of stagnant, indolent, ineffectual cells—all "gone fishin'." The end result is stagnation—general immunosuppression—and an environment in which cancers and viruses get a huge green light.

Deactivated macrophages malfunction in numerous ways. Phagocytic activity dramatically declines. Now no invaders are being eaten, so no luminously labeled tumor or bug parts are being spit out, so 'antigen presenting' screeches to a halt.

Without antigen presenting, the rest of the immune system won't be warned that invaders are nearby, so it remains dormant. As you can see, the deactivation itself perpetuates more deactivation; it's a vicious cycle. So all the other immune cells sit idly by waiting for marching orders that never come. The entire cascade that leads to ramped up immune activity is blocked when macros are not being activated by GcMAF.

From the macrophage's perspective, this is a nightmare scenario. Like Gulliver, it has been tied down and immobilized by hordes of Lilliputian Nagalase molecules. Big Mac wants to get up and go out and kick some butt, but it can't. Immune activity grinds to a halt. Dr. Yamamoto puts it this way: "Macrophages are the major phagocytic and antigen-presenting cells. Because macrophage activation for phagocytosis and antigen presentation to B and T lymphocytes is the first (and) indispensable step in the development of both humoral and cellular immunity, lack of macrophage activation leads to immunosuppression." (Immunotherapy of Prostate Cancer with GcMAF. Yamamoto et al., Translational Oncology Vol. 1, No. 2, 2008.)

8: How Your Body Makes GcMAF

An illustrated description of the biochemical transformations involved in the synthesis of GcMAF from Vitamin D Binding protein.

How your body makes the GcMAF that activates macrophages and protects you from cancer and viruses

GcMAF and Nagalase are both proteins, so let me start with a brief—and hopefully painless—primer on proteins. You know those birthday present bows made of clusters of curly ribbons? Under a very powerful microscope, proteins look like that. The ribbons are long chains of hundreds of amino acids that make up a protein molecule. Our DNA is programmed to make tens of thousands of different proteins, and what makes them different is the ordering of the amino acids. Each strand (usually there are three or four of them) of curled ribbon in our birthday bow is one of those chains. The curly ribbons are all attached together where the bow is fastened to the present. They may look like a big blob of randomly-placed bands—and in the ribbon, they are. But in a protein, there is a very specific three-dimensional structure, such that even though the curly ribbons look randomly placed, they are, in fact, very precisely positioned—and even slight positional changes will significantly alter the nature of the protein.

Vitamin D-binding protein (DBP) is the *precursor* protein out of which our immune cells make GcMAF. Up close DBP looks kind of like a small Brillo pad, but the convolutions are not sharp-edged; they're actually quite soft and sticky. DBP contains 458 amino acids, one of which is very special and quite different from all the others. This is a threonine amino acid,

the 420th amino acid in its chain. Attached to this threonine is a group of three sugars. The presence of these sugars defines the purpose of the entire DBP protein molecule. To keep things simple, I am going to name the three sugar molecules after candy bars.

Because Vitamin D Binding Protein comes with sugars attached, we can now refer to it as a *glyco*protein. Most of the immune system's "messenger molecules" are glycoproteins.

Now imagine DBP as this large protein with three sugars (or candy bars) attached. The first is a Hershey's bar, the second is a Milky Way, and the third is a Snickers. All three are attached to one another, as shown in the diagram, in an upside-down "Y"-shaped configuration.

Vitamin D-Binding Protein (DBP) is the starting point in GcMAF production.

DBP is the protein from which we are going to make GcMAF.

The dashes (–) indicate chemical bonds (pairs of electrons that hold atoms together to form chemicals) that attach the sugars to each other and to the protein.

Making GcMAF from DBP

Now let's transform our candy bar model of DBP into GcMAF. There are two steps in this process. The first step is to snip off the Milky Way bar. (This is performed by the enzyme beta-galactosidase which is embedded in the outer cell membrane of B-lymphocytes.) You can go ahead and eat it; we won't need it anymore. If you don't want it, your body will just recycle it.

Intermediate in GcMAF production.

The second step is to snip off the Snickers bar. (This is performed by the enzyme sialidase, which is located in the outer membrane of T-lymphocyte cells.) (You can have that one too, if you want to get on the fast track to diabetes.)

Now we're left with a huge protein that has just the remaining Hershey's hanging off of it. Guess what: this *is* GcMAF.

By snipping off two of the three sugars (first the Milky Way and then the Snickers bar), we have transformed the Vitamin D-Binding Protein into GcMAF.

It's fully formed and ready to float off, find a macrophage, lock onto its receptor, and then send a powerful message to the entire cell, telling it to stop watching reruns of Desperate Housewives and get to work beating up microbes and killing cancer cells. And, as you know, when GcMAF talks, macros listen.

Candy bar identities revealed

Just for the record (and for you biochemists in the house) my Hershey's bar is alpha-N-acetylgalactosamine (GalNAc), the Milky Way is D-galactose, and the Snickers bar is sialic acid (also known as N-acetylneuraminic acid).

Now that we have synthesized some GcMAF, we'll see—in Chapter 9 how Nagalase sabotages it…

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9: Nagalase: Friend *and* Foe?

What is Nagalase?

Nagalase is a protein made by all cancer cells and viruses (HIV, hepatitis B, hepatitis C, influenza, herpes, Epstein-Barr virus, and others). Its formal, official chemical name is alpha-N-acetylgalactosaminidase, but this is such a tongue-twisting mouthful of a moniker that we usually just call it "Nagalase." (Sometimes, when I want to impress friends with my brilliance, I'll say the entire word real fast: "*alpha-N-acetylgalactosaminidase*." I have found that it's important to practice beforehand if one doesn't want to embarrass oneself.)

Why is Nagalase important?

1. Nagalase *causes immunodeficiency*. Nagalase blocks production of GcMAF, thus preventing the immune system from doing its job. Without an active immune system, cancer and viral infections can grow unchecked.
2. As an extremely sensitive *marker for all cancers*, Nagalase provides a powerful system for early detection.
3. Serial Nagalase testing provides a reliable and accurate method for *tracking the results of any therapeutic regimen for cancer, AIDS, or other chronic viral infection*.

Nagalase proves that cancer cells break all the rules

Normal healthy cells cooperate with one another in a concerted effort to further the good of all. Cancer cells refuse to play ball. Their disdainful attitude toward the rest of our cellular community is appalling. For example, these cellular scofflaws ignore clear messages to stop growing and spreading and encroaching on their neighbor's space. How would you like it if your neighbor moved his fence over into your backyard?

Of all the rules cancer cells break, none is more alarming than the production of Nagalase, the evil enzyme that completely hog-ties the immune system army's ability to stop cancer cells.

Virus particles also make Nagalase. Their goal is the same as that of the cancer cells: survival by incapacitating their number one enemy: the immune system.

Nagalase precision

Like a stealth bomber, the Nagalase enzyme synthesized in and released from a cancer cell or a virus particle pinpoints the GcMAF production facilities on the surface of your T and B lymphocytes and then wipes them out with an incredibly precise bomb. How precise? Let me put it this way: Nagalase locates and attacks one specific two-electron bond located at, and only at, the 420th amino acid position on a huge protein molecule (DBP), one of tens of thousands of proteins, each containing millions of electrons. This is like selectively taking out

a park bench in a major city from six thousand miles away. More astonishing, if that is possible, Nagalase *never* misses its target. There is no collateral damage.

As you already know, GcMAF is a cell-signaling glycoprotein that talks to macrophages, enabling them to rapidly find, attack, and kill viruses and cancer cells. By activating macrophages, GcMAF triggers a cascade that activates the entire immune system. Blockage of GcMAF production by Nagalase brings all this wonderful anti-cancer and anti-viral immune activity to a screeching halt, allowing cancer and infections to spread.

What does Nagalase actually do? How does it destroy immune functioning and deactivate macrophages?

Once synthesized and released into nearby tissue or into the bloodstream, Nagalase, like that drill sergeant at boot camp, shouts harsh commands at the vitamin D binding protein (DBP) that is about to be turned into GcMAF. Nagalase demands that DBP not, *under any circumstances*, attach itself to a specific sugar molecule (galactosamine). If DBP has already grabbed (i.e., connected to, using a two-electron, "covalent" bond) a galactosamine sugar molecule, it is commanded to immediately let go. "Leave galactosamine alone, or you'll be in big trouble!!!" is the Nagalase sergeant's command. We'll probably never know whether or not, on some deeper level, DBP knows that Nagalase's motives are dastardly—but it doesn't really matter: DBP will definitely always obey. Like the army private, the DBP literally has no choice. Because of the way hierarchies work in cellular biology, proteins must do the bidding of their enzymes. The enzymes, like Nagalase, are the drill sergeant and the proteins, like DBP, are the privates. That's just the way it is. Obeying the drill sergeant's command means DBP can't do its assigned task, that of becoming GcMAF. It is rendered useless. For DBP, on a molecular level, life no longer has meaning.

Unfortunately for cancer and viral patients, DBP had been on its way to becoming GcMAF until the Nagalase drill sergeant so rudely interrupted. Now GcMAF—the one protein our bodies need in order to activate our immune systems—can't be made. Immune activity screeches to a halt. The defense system protecting us from cancers and viruses has been snuffed out.

Nagalase, using this astonishingly simple yet cunningly subversive technique, emasculates the GcMAF precursor protein (DBP) by knocking off its three sugar molecules. One quick whack by Nagalase and the DBP protein that would have become a GcMAF molecule now limps off into the sunset, permanently disfigured and disabled. With one simple, swift maneuver, Nagalase has brought the entire immune system to its knees.

Here's how Dr. Yamamoto put it (for clarity, I've replaced some of the technical words):

"Serum vitamin D3-binding protein (DBP) is the precursor for the principal macrophage activating factor (GcMAF). The precursor activity of serum DBP was reduced… These patient sera contained alpha-N-acetylgalactosaminidase (Nagalase) that deglycosylates (removes the sugars from) DBP. Deglycosylated DBP cannot be converted to GcMAF, thus it loses the GcMAF precursor activity, leading to immunosuppression." (Microbes Infect. 2005 Apr;7(4):674-81. Epub 2005 Mar 22. Pathogenic significance of alpha-N-acetylgalactosaminidase activity found in the hemagglutinin of influenza virus. Yamamoto N, Urade M.)

Nagalase testing: former mass murderer now works for the good guys

It's easy to be a little schizy about Nagalase. On the one hand, this nasty protein's behavior toward us has been reprehensible and disastrous. Working in cahoots with cancer and HIV—not shy about getting into bed with our mortal enemies—Nagalase can rightfully claim direct responsibility for billions of human deaths. And it would just as soon add you to the list, so we don't have to be shy about placing Nagalase in the "genocidal murderer" column.

With the advent of Nagalase testing, however, this bad actor now will be harnessed to a useful purpose. By providing us with precise and reliable advance information about enemy operations, Nagalase blood level testing becomes a "Deep Throat" double agent for cancer. He helps us by giving us an early warning sign.

Early detection (using AMAS or Nagalase) saves lives

You don't want a cancer to have gotten out of control by the time you find and start treating it. When cancers are still young and small, gentle natural therapies are the most effective. Alternative treatments work best on early small cancers by enhancing immune functioning and removing the source of the inflammation that is causing the cancer in the first place. Cancers that have become large enough to see on imaging pose a much more significant threat, and the big guns now become necessary.

The current method for diagnosing most cancers requires us to wait until a mass shows up on imaging (e.g., a mammogram, chest X-ray, or colonoscopy). This approach wastes valuable time and causes needless deaths. But long before imaging can find it, a positive Nagalase (or AMAS test) can tell us that early stage cancer exists somewhere in the body. By enabling earlier and therefore less invasive treatment options, this information provides a huge head-start.

Normally present at only trace levels, Nagalase shows up in the blood when a cancer or virus appears

The malignant and viral entities that make Nagalase are not normally present, so its appearance is a big deal from a diagnostic perspective. When Nagalase shows up, even in very small amounts, we have the earliest glimpse of a new cancer or viral infection. The old adage, "Where there's smoke, there's fire" applies here. A positive Nagalase test notifies us that a cancer (or a nasty virus) lurks within.

Nagalase appears in the blood stream when a nascent cancer is just a minute cluster of abnormal cells, long before conventional diagnostic methods can detect it. Through blood testing, we can find this red flag, even when present at exceedingly low levels. Providing us with this early warning sign might not quite qualify Nagalase for the "Good Samaritan" award, but I could go with "extremely useful." Like a rehabilitated criminal on parole, the potential for harm is still there. For now, however, he's staying out of trouble and doing community service. Turn your back and he's a mass murderer again.

Using Nagalase testing to track cancer treatment

Rising Nagalase levels indicate a cancer or virus is growing and spreading. Conversely, Nagalase levels will decrease if the cancer or infection is being effectively destroyed.

Any treatment that lowers cancer cell (or viral numbers) will lower Nagalase levels. Nagalase will, for example, always drop after surgery (whether or not the entire tumor was removed). Chemotherapy and radiation also reduce Nagalase levels. So does GcMAF. If, after these treatments, the depressed level begins to rise again, this is the warning sign that the cancer was not completely removed, and/or that metastatic disease is hiding out somewhere. With viral infections, increasing Nagalase levels indicate return of the infection.

Consecutive rising Nagalase levels are therefore a red flag, warning us it may be time to entertain new treatment options. Conversely, if levels are going down, stay the course: the cancer or virus is going away.

Flat-earth medicine

Many medical professionals don't feel comfortable with "nonspecific" tests like Nagalase. It drives them nuts to discover that a cancer is lurking somewhere inside without knowing exactly *where* it is located. "How," they ask, "do you expect me to treat a cancer I can't see? Why, I'm not going to tilt at windmills!" This may be a signal that you need to find a different doctor, perhaps one who works in an alternative cancer clinic. Here you will find highly-trained professionals who understand the concept that cancer is a molecular biological change long before it presents visually (by this I mean becomes viewable on imaging).

When GcMAF becomes available, the answer will be easier: a six month course of weekly 100 ng GcMAF intramuscular injections with monthly Nagalase level tests to follow the Nagalase level as it goes back down to baseline. The cancer can be declared cured, even though it never reached life-threatening proportions. (We have a long way to go before this kind of medical behavior will be commonplace and acceptable. The sooner the better, however.)

Nagalase role "under-appreciated"

Nagalase, arguably our most immunosuppressive protein molecule, poses an enormous threat in terms of cancer perpetuation and viruses' ability to continually defeat us. Yet cancer researchers have not shown any interest in it. (Maybe I'm being a little too generous here; perhaps "clueless" would be more a more accurate depiction.) Why don't they get it that blasting cancer cells into oblivion with chemo and radiation is usually not sufficient to stop advanced disease and does nothing to address the *cause*: immunosuppression. Even if we ignore for the moment the excessive collateral damage caused by chemo drugs and radiation, the patient also needs—requires—a healthy immune system to finish the job. If we don't revive immune function by disabling Nagalase, the cancers and viruses will just keep roaring back. Restoring immunocompetence by negating the stultifying effect of Nagalase should therefore become a primary research goal.

10: How Nagalase Blocks GcMAF Production

Cancer cells and all viruses manufacture and release Nagalase, the enzyme that sabotages GcMAF. Without GcMAF, macrophages remain in a deep slumber. This chapter answers the question: "How does Nagalase do it?"

Nagalase sabotage

As you know by now (because I have reiterated it *ad nauseum*), the Nagalase spewed out by cancer cells and virus particles neutralizes the immune-activating effects of GcMAF. For millions of years GcMAF (and our immune systems) have had no defense against Nagalase. Then Professor Nobuto Yamamoto came along and figured out how Nagalase paralyzes immune functioning. He then went on to demonstrate that replacing the GcMAF deficiency dramatically restores immune function, effectively circumventing the roadblock set up by Nagalase.

The ultra-miniature Nagalase power play that I'm about to describe has leverage far beyond its trifling size. This relatively simple molecular biological enzymatic reaction has cost billions of humans their lives. Who would've thought that chipping a few sugars off of a glycoprotein molecule could have wreaked such carnage?

A *saboteur* is one who intentionally causes the destruction of property in order to hinder the efforts of his/her enemy. The word derives from 15th century Netherlands, where, fearing that automated machines would render their occupations obsolete, Dutch workers threw their *sabots* (wooden shoes) into the gears of the textile looms in an attempt to break the cogs. Likewise, Nagalase, the *saboteur*, "throws a boot" into the biochemical machinery that produces GcMAF.

Like taking candy from a baby

Now I will describe—on a molecular "blow by blow" basis—how Nagalase blocks GcMAF production. Recall from Chapter 8: How Your Body Makes GcMAF that our lymphocytes make GcMAF by enzymatically removing two of three sugars from a Vitamin D Binding Protein (DBP) molecule:

Vitamin D Binding Protein is converted to GcMAF.

To appreciate how Nagalase interferes with GcMAF production, imagine that DBP is the baby that is about to be blocked from becoming GcMAF. Baby GcMAF (AKA DBP) is trying to hold onto its Hershey's bar (AKA N-acetylgalactosamine). Nagalase comes along and snatches all three sugars away from the "baby." (See illustration below.) The sugars float away, off into the cellular soup, never to be heard from again. (Oh, I imagine sooner or later—as with other sugars—they'll be burned up in a mitochondrial factory to generate a little cellular energy—but as far as DBP is concerned, they're Gone.)

Nagalase blocks GcMAF production by deglycosylating Vitamin D-Binding Protein.

When the dust settles, we are also left with a worthless "deglycosylated" DBP molecule. With all of its sugars removed by the Nagalase, there's no way DBP can become GcMAF. Thanks to the saboteur Nagalase, all that remains is a useless, sugar-free protein that can't be converted into *anything* of value. It too drifts off into cellular obscurity and eventually gets recycled.

In its underhanded effort to subvert the immune army, Nagalase has literally stooped to snatching candy from a baby. The fetus (DBP) that would have become GcMAF has died in utero. And, Voila! The pathogen has neutralized it's enemy's immune system. The cancer cell or virus that made the Nagalase has succeeded in neutralizing its archenemy, the macrophage. And (as pointed out earlier, but bears repeating), when macrophages are de-

activated, cell signaling is simultaneously deactivated, so other immune cells (primarily B- and T- lymphocytes) stop functioning too. Anti-cancer and antimicrobial immune activity have been effectively disabled.

Nagalase is remarkably efficient

Because it is an enzyme—a catalyst—Nagalase performs this malicious ritual over and over and over again, and each time it comes away unscathed and unchanged. One Nagalase molecule can thus destroy a huge quantity of GcMAF precursor molecules.

To appreciate how precisely Nagalase (Alpha-N-acetylglucosaminidase) targets GcMAF production, imagine a Stinger heat-seeking ground-to-air missile tracking a fighter jet. Evasive maneuvers—even by the best pilots—won't outsmart the Stinger, which rapidly adjusts its course to track the plane down and blow it up. Nagalase works the same way: it tracks down and then pulverizes the (DBP) precursors of GcMAF molecules.

Nagalase has no natural enemies. No bodily process, no drug, no treatment could outsmart this diabolical killer. Sure, high-powered drugs and radiation will take out many of the cancer cells and in some cases produce a cure, but until Dr. Nobuto Yamamoto came along and outed Nagalase, we had no idea as to the actual *cause* of the immune shutdown that let cancers and viruses go wild.

Dr. Yamamoto's schematic diagrams for the production of GcMAF. FIG. 1A: The formation of GcMAF from Gc protein (vitamin D binding protein. FIG. 1B: The deglycosylation of Gc protein by Nagalase (alpha-N-acetylgalactosaminidase).

11: If Cancer Cells Could Talk…

Wistful ruminations of a cancer cell

"For a long time we cancer cells had a great job. Our Nagalase enzyme Stealth bomber squadrons made it easy to keep cancer going. Sneaking in under the radar and demolishing GcMAF production facilities was our specialty, and—if I say so myself—we were good at it. I never missed my target—and I never caused any collateral damage. We shut those GcMAF factories down big time. Blasted them into oblivion. And we had billions of cancer kills.

"Without their daily dose of GcMAF, those macrophages couldn't get out of bed. They didn't really have a chance against us. It was a romp! Cancer ruled!

"I'm surprised at how long it took the scientists to unravel our scheme. Our killing spree lasted hundreds of years! Try and match that!!! Lives lost in wars pales in comparison. In fact, we killed many times more humans than all the wars in history combined.

"Even when human molecular biologists finally figured out how we operate—'cracked the code,' so to speak—and Dr. Yamamoto outed us by explaining to the world how we did it, it still took them another decade to pay attention and finally shut us down. The cancer establishment didn't want to sacrifice their cash cow so, instead of implementing these new discoveries, they still just kept lobbing those silly useless drugs at us. As usual, that slowed us down a little, but it took another ten years before they finally started routine Nagalase screening, and then giving weekly GcMAF to everyone with an elevated Nagalase. Why did

it take them so long to see that fixing the immune system was infinitely preferable to their mostly futile attempts to "shut the barn door after the horses had gotten out," so to speak. Given half a chance, the body can cure itself. Because of that mistake—and it was a huge one—we killed millions more before they sidelined us.

"So how did I personally escape the GcMAF and activated macros? Well, I'm one of the lucky ones, and I must say there are very few of us. When Dr. Yamamoto discovered Nagalase, I switched sides and changed my identity. Now I'm in a—sort of—witness protection program. The only reason they don't kill me is because I'm useful to them as a mole, a whistle blower …they call it a cancer marker—however you want to put it. The docs test levels of me to see wheteher (and how much) cancer or virus is present in my host's body.

"I'm a fortunate guy. Before GcMAF, the cancer cell community adored us Nagalase molecules. Now, post-GcMAF, I am still a hero, but to the other side. Now I help doctors find new cancers and track known ones, so suddenly I went from bad guy to good guy. Now, as a spy, I provide you guys, my former enemies, with early warnings that cancer is on the move. And I let you know whether your treatments are working. Here's how that works: More of me means more cancer (or virus), for sure. And less of me means the cancers (or virus) are going away. If I appear to be going away but then come back, you'll know the cancer or HIV has returned (i.e., wasn't fully killed off) and my owner person needs another round of GcMAF.

It's a job, and somebody had to do it. Considering the alternatives, well— like I said—I'm a lucky guy!"

Looking at Nagalase from a future perspective: "Back in The Day…"

It is 2020. Two cancer cells, Jimmy and Jack, are having a chat. Jimmy's the young go-getter and Jack the seasoned but terminally cynical veteran.

Jack, in animated tones, reminisces about the good old days when cancer could wreak havoc…

"I want to tell you about Mickey, the legendary cancer cell who discovered Nagalase. Mickey's discovery catapulted us cancer cells from a minor nuisance up into one of the all-time greatest threats to the survival of humanity. Of course, those days are over now, but man, we had quite a run. Millions of years and billions of kills! Not bad, huh?

"Oh, there've been other famous cancers. Henrietta Lacks' immortal cervical cancer cell line comes to mind. But even though he's less well-known, Mickey was by far the greatest. With Mickey's Nagalase in our arsenal, we were unstoppable. Like Bruce Willis or The Terminator. The oncologists and surgeons and radiologists could aim their peashooters at us all day long — the chemo, the surgery, the radiation —but, man, we just shrugged them off and kept on comin.'

"But it's all changed now. Right, Jack? I mean, they're testing just about everybody for Nagalase now. We don't have a chance." Jimmy tentatively intones. He knows a story is coming.

“Mickey discovered Nagalase back in the ancient history, Jimmy, in the stone age of cancer. Before written history, so we don’t know exactly when. And how he did it, I don’t know—in fact nobody knows. Probably a mutation in his genome that generated a protein that stopped cancer in its tracks. Of course Jimmy’s contribution—and it’s not one to be sneered at—was recognizing its value. A lesser cancer cell might have received the gift of Nagalase production, but failed to appreciate its crucial importance—but not Mickey. He saw that Nagalase stopped the macros in their tracks…

“How does it work, Jack?”

“It’s so simple. Nagalase simply breaks a pair of electrons apart. This particular pair of electrons make up a bond that holds a crucial part of the GcMAF precursor molecule (the molecule GcMAF is made out of) together, so the GcMAF can’t be manufactured. Aalakazam!!! No more GcMAF! Can’t be made because the precursor disintegrated. Bottom line: no more macro activation, no more macros eating my pals. End of story. We win.

“Wow, Jack. That’s amazing.”

“And Mickey also figured out how to spread the word and make a lot more Nagalase.”

“How?”

“By teaching all his friends and neighbors, our distant relatives, how to make it. They just shared the genes for it. No big deal. After a while, due to Darwinian survival of the fittest, all of our cancer cell brethren that couldn’t make Nagalase just died off. May they rest in peace. Well, actually, they didn’t exactly die—they were eaten by the macros. But the ones that could make Nagalase survived and passed on the gene. We use the humans to carry it around in their DNA. The power to set us free (and make us dominant) remained in there, locked into perpetual existence in the human genome, the blueprint for life …and death. It was replicated and passed on from unsuspecting generation to unsuspecting generation inside the humans, and it just sits there in all of their genomes, lying in wait, ready to be exploited by us cancer cells whenever we get the chance to start growing. And by smoking, eating pesticides and spewing an enormous array of toxic chemicals into the environment, they gave us plenty of opportunities.

“Then, back in 2010, along came GcMAF. The docs finally figured out that if they gave people GcMAF, it would bypass the Nagalase and activate the macros. Now, instead of ruling the roost, we are on the run. The macrophages are everywhere, buzzing around, ganging up on us, and there’s nowhere to hide like in the old days. They can take out brigades of our cells with no effort at all. And they target the younguns—that’s not nice!

“If you ask me, GcMAF is cruel and unusual punishment to us cancer cells. We were just doin’ our thing man, if you know what I mean.”

“But those were the good old days, Jack. Now it’s different and we have to adjust…”

“You’re right, Jimmy, a good thing can’t last forever. We have to accept that our run is over, our time has passed. Nagalase testing has become more popular than cholesterol used to be. Nowadays, everybody gets tested for Nagalase, starting—can you believe it, in high school? They find us so early now—and then they pour on the GcMAF. We don’t have a chance

anymore. Usually the macros nail us so early that we can't even metastasize. Adding insult to injury, then they monitor us with Nagalase testing until they're sure we're gone. Normally, we can't get a serious tumor going. Our current host, however —man, this dude is clueless. For some reason or other, he hasn't gotten tested. That's the only reason we're alive here, Jimmy.

"Oh, we still have pollution, toxicity and chemicals on our side, so we'll be hanging around in small numbers, generating the occasional new malignant colony, but unless we come up with something really good like Nagalase used to be, we are basically toast. It's only a matter of time.

Then, startled, Jimmy screams: "Oh, oh. Yikes!!!! What's that huge blob coming around the corner of the building over there? Is that what I think it is?"

"Dang! Yup. That's a macrophage, son. Well, I figured they'd come after us sooner or later. This guy must have finally gotten tested. We're dead meat now, Jimmy. It's coming to eat us!!! Dodge those superoxide radicals, if you can! Watch out for that pseudopod! It's been nice knowing you, Jimmy. Argggggh…."

12: GcMAF and HIV/AIDS

Dr. Yamamoto treated 15 HIV patients with GcMAF, which eradicated the virus in all cases. These patients went into remission and remained disease free during 7 years of followup.

To sabotage the immune system and put macrophages to sleep, all viruses make Nagalase, the enzyme that blocks production of GcMAF. Without GcMAF (the protein that activates macrophages and jump-starts the entire immune response) HIV and other viruses can grow unimpeded. Nagalase puts the immune system to sleep. Dr. Nobuto Yamamoto demonstrated that GcMAF administration bypasses the Nagalase blockage and re-activates the macrophages, which then proceed to kill the HIV viruses and cure the infection.

Something to cheer about?

People infected with the Human Immunodeficiency Virus (HIV) have something to get excited about. They just don't know it yet. In 2009 Dr. Nobuto Yamamoto published a landmark paper entitled: "Immunotherapy of HIV-Infected Patients With Gc Protein-Derived Macrophage Activating Factor (GcMAF)" in the *Journal of Medical Virology* in which he demonstrated that GcMAF cured 100% of nonanemic HIV infected patients. After seven years of followup, there were no recurrences. All patients maintained healthy CD+ counts.

Of course, this is just one study. And it had the disadvantage of containing some complex molecular biological chemo-speak. If the reader weren't familiar with Yamamoto's decades of background research (all of which was published in journals that HIV researchers and patients wouldn't be likely to read), this study would fall on deaf ears. But Professor Yamamoto's HIV study was no quirk. Based on a quarter-century of solid research that predicted success long before the actual human trials, it presented all the science one would need to understand exactly why these HIV patients were cured.

Though this study was published in 2009, there has been no informed discussion on this topic, no further GcMAF research as a therapy for HIV, and no media chatter. It's as if this study never happened. Why is this?

How GcMAF destroys HIV

HIV—like all viruses—makes Nagalase, the enzyme that blocks GcMAF production. Without GcMAF, macrophages become indolent and the anti-viral immune response shuts down. This allows the HIV infection to spread. To remedy this situation, Dr. Yamamoto simply gave these patients GcMAF. This reactivated the sleeping macrophages, which then proceeded to phagocytize all of the viruses.

The precise molecular biological pathways and mechanisms involved with HIV, Nagalase, and GcMAF are identical to those for cancer cells, and need not be repeated here.

In his HIV study Yamamoto first showed that HIV patients had high Nagalase levels which correlated with their high HIV RNA levels (a way to measure the amount of HIV infection). Then, as he administered GcMAF (100 ng. once a week for 18 weeks), all patients' Nagalase levels gradually went down to control levels, and, in tandem with the Nagalase, viral load went down to zero. Yamamoto wrote that these data "suggest that these patients were free of both HIV virions and HIV-infected cells."

Professor Yamamoto followed these patients for seven years, and their viral load (HIV-1 RNA), CD4 counts (helper cells, a type of lymphocyte used to evaluate immunocompetence), p24 antigen (HIV-specific antigen), viral culture, and Nagalase levels remained normal. All patients continued to be free of disease. (Note: anemic HIV patients were excluded from this study. Anemia is common in HIV patients. The effect of GcMAF on anemic HIV patients is thus unknown.)

13: The AMAS Test-An Alternative To Nagalase Testing

The AMAS test is useful both as a screening test for early cancer and for monitoring cancer therapies. AMAS is elevated when cancer is present and goes down below baseline when cancer is gone. AMAS is over 99% accurate (when done twice) and can be used instead of Nagalase to find and follow cancers. (Unlike Nagalase, AMAS does not detect the presence of viruses.)

The AMAS test:

- a naturally occurring antibody present in serum of all people, including children
- accurately detects early cancer of all types
- positive if any type of cancer exists anywhere in the body
- greater than 95% accuracy; repeat testing greater than 99% accurate; false positive and false negative rates less than 1%
- the earliest anticancer antibody to appear
- detects cancer very early; your doctor may not be able to find the cancer
- detects cancers long before they appear on imaging
- early detection dramatically increases the possibility of a permanent cure
- goes down with successful cancer treatment
- normal levels in successfully treated cancer patients indicates absence of malignancy

The AMAS cancer test: an alternative to Nagalase testing

Until Nagalase testing becomes available, I recommend the AMAS (Anti-Malignin Antibody, Serum), a test that definitively determines whether or not cancer is present. In this broad-based study (click on ref 13) dozens of researchers and medical centers conclusively demonstrated the value of AMAS as a screening tool for finding cancers early.

All cancers make anti-malignin antibody. Because we are making cancer cells all of the time, anti-malignin antibodies are present at low levels in everyone. Normally, a healthy immune system (one with *activated* macrophages) is destroying these cancer cells as they are formed. An AMAS level that rises beyond the baseline of 135, however, tells us that the immune system is not getting rid of those new cancer cells in a timely way, and their numbers are therefore increasing. Cancer is afoot.

AMAS is both a cancer *screening* test and a cancer *monitoring* test. In other words you can use it to determine whether cancer is present, and you can use it to track therapy.

The brainchild of neurochemist Samuel Bogoch, M.D., Ph.D., AMAS is similar to the PSA for prostate cancer and CEA for colorectal cancer, except that AMAS simultaneously screens for all cancer types, not just one.

A positive AMAS will tell you that cancer is present, but it won't tell you what kind of cancer it is, and it won't tell you where that cancer is located. (Not knowing a cancer's name and location tends to drive doctors nuts. "How can you treat a cancer when you don't know what it is or where it is? they mutter.)

How AMAS works

Our immune system recognizes an antigenic protein on the surface of cancer cells. In 1988 Dr. Bogoch discovered this antigen and named it "malignin." When our immune system "sees" malignin, it starts making an antibody named anti-malignin antibody. Dr. Bogoch then developed the AMAS test to identify the presence of cancer by identifying the presence of anti-malignin antibody.

Using the AMAS for monitoring cancer

When a treatment shrinks a cancer, the AMAS will go down. Whether that treatment is surgery, chemo, radiation, an alternative cancer therapy like GcMAF, or spontaneous remission—if the cancer is smaller, the AMAS goes down. If cancer remains, the AMAS is positive. When a cancer is gone (again, regardless of cause), the AMAS reverts to normal.

A rising AMAS tells us cancer is growing

AMAS is an extremely precise immunoassay. Levels above baseline indicate the presence of cancer with 95% accuracy on the first testing and over 99% accuracy when tested twice.

The cutoff point for a positive AMAS is 135. More than 99% of patients with cancer have AMAS levels above 135. AMAS levels below 135 are seen in normal individuals who do not have cancer.

Sequential AMAS levels can also be used to track tumor advancement and the effectiveness of therapies. AMAS levels will always increase if cancer is growing and spreading. Conversely, AMAS levels will decrease if the cancer is being effectively destroyed (whether by surgery, radiation, chemotherapy, GcMAF, or other alternative cancer therapies).

AMAS and breast cancer

AMAS has identified breast cancers as small as a pencil dot (too small for any form of imaging).

Clinical research data shows that breast cancer can only be presumed cured if the AMAS returns to normal (<135) after treatment and that breast cancer cannot be presumed to be in remission unless AMAS returns to normal.

The usual followup methods for breast cancer include imaging (CT scans, MRIs, or x-rays) and hormonal blood tests, looking for signs of cancer after treatment. AMAS testing provides a much more accurate way to know whether cancer is still present, and at a fraction of the cost. Not to mention the inconvenience.

Although these studies were done only on breast cancer patients, there is no reason to believe that the results would not apply to all types of cancer.

Cancer patients who no longer have evidence of cancer on imaging, but do have a positive AMAS test could nip a returning cancer in the bud by utilizing natural alternative cancer therapies (including GcMAF when it becomes available).

Avoiding unnecessary biopsies

If a patient has a mass on imaging, the AMAS will tell whether it is malignant or not. A negative AMAS means the mass is not cancerous, and therefore a biopsy is not necessary. Use of the AMAS test could thus reduce the pain and suffering of needless biopsies. Not to mention the cost.

AMAS compared to Nagalase

AMAS differs from Nagalase in that AMAS is specific for cancer, while Nagalase identifies an enzyme made by both viruses and cancer cells and is therefore unable (by itself) to distinguish between the two. Because Nagalase testing cannot distinguish the difference between cancer and virus, AMAS is a better screening test for cancer.

How to order the AMAS test

To order a free AMAS kit, call 1-800-922-8378 or order online at: http://www.oncolabinc.com. AMAS is a product of Oncolab, Inc., 36 The Fenway, Boston, MA 02215. Phone: 617-536-0850.

Please note

According to Oncolab: The AMAS test “should be used in the context of good clinical judgment by a physician experienced in the treatment of cancer.”

"A normal AMAS level can occur in non-cancer, in terminal cancer, and in successfully treated cancer in which there is no further evidence of disease; clinical status must be used to distinguish these states."

"As in all clinical laboratory tests, the AMAS test is not by itself diagnostic of the presence or absence of disease, and its results can only be assessed as an aid to diagnosis, detection or monitoring of disease in relation to the history, medical signs and symptoms and the overall condition of the patient."

Possible future developments

Dr. Bogoch has shown that purified AMAS kills cancer cells in a test tube. From this research a future treatment can be developed in which injections of anti-malignin antibody would be used to treat cancer.

Dr. Bogoch's contribution to science and medicine will hopefully someday be recognized with a Nobel Prize.

14: Biomarker Testing

This chapter will show you how to use AMAS testing, Nagalase testing, and viral antibody testing to determine whether you have cancer. If you do have cancer, you'll learn how to track your treatment program to make sure it is working.

It is important to appreciate the fact that *both cancers and viruses make Nagalase*. Therefore, a positive Nagalase test result tells us both cancer and/or virus could be present, but it doesn't tell us which. This problem can be easily solved. Since the AMAS test is specific for cancer (and not viral infections), a positive AMAS tells us cancer caused the Nagalase to go up. Viral antibody testing can also be used to rule specific viruses in or out. Thus the combination of AMAS and viral screening will effectively sort through the possibilities presented by a positive Nagalase test.

Here is a summary of what each test will tell you:

- A positive *Nagalase* test indicates presence of either a a cancer or viral infection, or both.
- AMAS testing (now available) indicates presence of cancer (only).
- Viral antibody testing (now available) indicates presence of specific viruses (see list below).
- Appropriate combinations of these three tests will sort through the possibilities and identify the actual cause of the disease.

Using Nagalase to screen for cancer

In the (hopefully not-too-distant) future, once Nagalase testing and GcMAF are available, all people at risk of cancer—i.e., everyone over the age of 40—will get an annual Nagalase test along with their other routine blood tests (complete blood count, comprehensive metabolic panel, vitamin D, lipid panel, etc.).

Nagalase will become our standard marker for early cancer detection. Those with an elevated Nagalase (after ruling out a viral cause; see below) will be treated "presumptively" (i.e., we know it's in there even though we can't actually see it) with GcMAF and/or other alternative cancer therapies.

Followup Nagalase testing will document the patient's progress. Once the cancer is gone, as documented by a return of Nagalase to baseline, subsequent testing will provide an early warning if cancer starts growing again.

Using Nagalase to track effectiveness of cancer therapy

The more cancer cells present in the body, the more Nagalase they generate. Thus, Nagalase is the perfect marker for determining response to cancer therapies because, in a given patient, tumor burden will always be proportional to the Nagalase level.

In cancer patients, a declining Nagalase level reflects a reduction in total tumor "load" or "burden." We would expect a lower Nagalase after chemo, radiation, or surgery because these treatments all reduce tumor burden. Having used these therapies does not mean the disease is gone, however. A very low (baseline) Nagalase level would indicate the cancer has been cured.

If there is even a remote chance of metastatic disease—as indicated by an elevated Nagalase level—it would be wise to use GcMAF to activate the immune system so it can find and polish off any remaining cancer cells. Repeat testing with declining levels would indicate that the treatment is working.

Using AMAS to screen for cancer

The AMAS test (Anti-Malignin Antibody in Serum) measures serum levels of AMA, an antibody found to be elevated in 99% of patients with active malignancies.

Until Nagalase is available, I recommend the AMAS as a screening test for all people over 40. See Chapter 13 for a complete description of the AMAS test.

Using AMAS to track effectiveness of cancer therapy

Tumors produce AMAS in direct proportion to their size, so declining AMAS levels indicate the cancer is shrinking and that the treatment program is working. Increasing AMAS levels, on the other hand, indicate cancer growth and that a different treatment approach should be considered. (At least three AMAS tests are required to establish a pattern.)

Using AMAS to determine whether a positive Nagalase indicates presence of cancer or virus.

Since all viruses and all cancers make Nagalase, an elevated Nagalase level could be caused by either. AMAS testing, however is specific for cancer (i.e., tells us nothing about viruses). So an elevated Nagalase and a normal AMAS indicate a viral infection. If, conversely, both Nagalase and AMAS are elevated, you know you are dealing with cancer.

Using viral antibody testing

A negative AMAS coupled with a positive Nagalase indicates the presence of a viral infection. Anti- viral antibody testing will identify the specific virus. Each virus generates its own specific antibodies. The most common chronic viral infections are listed below. Antibody testing is available for each:

- Herpes zoster
- Herpes Simplex I
- Herpes Simplex II
- Epstein-Barr virus (mononucleosis)
- Hepatitis B
- Hepatitis C
- Cytomegalovirus (CMV)
- Human Immunodeficiency Virus (HIV)

Presumptive treatment of "occult" cancer

Conventional medicine continues to prefer to wait until imaging reveals both the presence *and location* of a cancer before acknowledging its existence and instituting therapy. For "occult" cancers—the ones that can't be "seen," but testing says they're in there somewhere—the best course of action is to treat "presumptively." In this situation, a positive Nagalase and/or AMAS testing has told us cancer is present, so we "presume" the presence of cancer, and then go ahead and treat it even though it's still too small to be seen on imaging. An AMAS test done twice and positive both times identifies the presence of cancer with 99% accuracy. A positive Nagalase (once this test becomes available) coupled with a positive AMAS would also constitute *convincing evidence* of an occult cancer.

If the treatment works, we may never actually see the cancer. This may trouble some physicians, but ask yourself this question: would you rather remove a small cancer with natural, harmless medicine, and never get to see it—or would you prefer to wait until the malignancy enlarges to the point where it'll show up on a CAT scan, which means you'll now need a biopsy, surgery, radiation, and chemotherapy? Hello?

15: Eradicating the Scourge of Cancer from the Face of the Earth

We can get rid of cancer once and for all. We now possess the scientific understanding necessary to accomplish this.

I want to abolish cancer. This goal may seem impossible, but it's not.

Early stage cancers are a lot easier to treat, so catching a malignancy early (when it is < 5 mm, too small to be seen on imaging) is a crucial piece of the eradication strategy. By the time a cancer is big enough to be seen on a CAT scan, it's too late to rely solely on natural medicines. The conventional options have become necessary: some combination of surgery, radiation, and/or chemotherapy. (Of course, alternative methods still can and should play an important *adjunctive* role, but no responsible alternative oncologist would recommend bypassing the usual mainstream therapies.)

So the trick to reversing cancer is to catch it early. That's when the gentle, natural, non-toxic, non-invasive, immune-supportive options work best. That's when GcMAF works best. When

GcMAF becomes available, we have every reason to expect that it's effectiveness in small early cancers will approach 100%. We just need to find them.

How? Nagalase screening. Elevated Nagalase levels (repeat testing is necessary to make sure the level is going up) reveal the presence of cancers when they are still extremely small. At this early stage, a few months of weekly GcMAF injections will be all that is needed to reactivate the macrophages and eliminate the cancer.

Applying the strategy of 1.) annual Nagalase testing in large populations with 2.) weekly GcMAF therapy for those who test positive has the potential to obliterate the scourge of cancer from the face of the earth.

Let's do it.

16: Retro-Docs and Nano-Medicine: A Rant

Conventional doctors are going to have to let go of their need to see a cancer on an X-ray before treating it. In the nano-world of molecular medicine we can now contemplate a patient's "metabolic landscape" to spot subtle biochemical markers—signposts alerting us to current or future disease. Several examples are provided. Tests like AMAS (now) and Nagalase (in the future) track the molecular biological footprints of malignancy, enabling diagnosis and initiation of treatment much earlier in the evolution of the disease—long before imaging could detect a mass.

Molecular biology: rewriting the rules for medical practice

Let me take a brief detour here to address the issue of doctors who are uncomfortable treating a cancer that is too small to see on imaging. In order to feel okay about treating, these guys really need visual—and just aren't comfortable with biochemical—evidence. In this, the dawning new age of molecular medicine, however, the zone between no disease and clear-cut, palpable, imageable disease is becoming increasingly blurry. Thanks to sophisticated testing and a deeper understanding of the molecular biology of pathological processes, we are now able to "see" the earliest stages of disease—and even the preexisting biochemical landscape that sets the stage for it—long before we can pin down its location with imaging (X-rays, CAT Scans, and MRIs).

My advice to doctors who need visual—as opposed to biochemical laboratory—evidence of disease is this: *get over it!* We have entered the nano-age of molecular medicine, where we can detect the earliest stages of disease via biochemical changes. In the olden days (ten years ago) imaging and palpating disease were the best we had, but these modalities have become increasingly archaic as genetics and molecular biological testing pave the way to earlier identification of pathological changes, allowing us to reverse disease while it is still in the formative stages, before symptoms appear.

As a prime example of this trend, Nagalase testing will become the new cholesterol, the screening test for cancer that will be part of regular blood testing. Using elevated Nagalase and AMAS levels, we will be able to treat and reverse tumors we never get to actually *see*—much less biopsy.

Via molecular medicine testing we can now contemplate a patient's "metabolic landscape" examining subtle genetic and biochemical markers that are signposts pointing to a currently developing or even a future disease. For example:

A low DHEA-S (an adrenal hormone; the most prevalent hormone in the human body) level, for example, tells us this person is not capable of managing stress very well, and thus prone to cancer, allergies, autoimmune disease, and other immunological disorders. Because of their compromised ability to manage all kinds of stress (toxic, inflammatory, infectious, allergic, chemical, traumatic, emotional) individuals with low DHEA levels have shorter lifespans.

An elevated MCV (mean corpuscular volume above 90) on a simple CBC (complete blood count) tells us the patient has a vitamin B12 and/or folic acid deficiency. Untreated, this significantly raises risk for a host of diseases including cardiovascular disease (heart attack, stroke, senile brain disease),neurological diseases (depression, dementia), and all cancers.

The vitamin D deficient individual is a sitting duck for cancer, diabetes, autoimmune disease, osteoporosis, and a host of other afflictions. Vitamin D deficiency very common (over 70% of Americans have one) and is a sure sign of dramatically higher risk of neoplasm. Ideal level is 50-100. Levels below 30 put one at 400% baseline risk for *all* types of cancer. Testing is the only way to determine deficiency. Though 10,000 IU a day would be a reasonable dose for someone who is deficient (below 50), testing is required to determine optimum dose.

A positive AMAS (Anti-Malignin Antibody, Serum) test tells us cancer is absolutely present. (See Chapter 13.) AMAS measures antibodies the immune system makes in response to cancer cell antigens. This test is 95% accurate—99% on repeat.

An elevated Nagalase (enzyme made by cancer cells and viruses that protects them by shutting down the immune response) tells us a cancer or virus is brewing.

A double standard

The lipid panel is a really lousy marker for cardiovascular disease. Half of all people with an elevated cholesterol never have a heart attack or stroke. Half of people who get a stroke or heart attack have a normal cholesterol. Nevertheless, doctors pay literal homage to an elevated cholesterol by whipping off the obligatory prescription for a statin drug; treating it gives them the feeling they are nipping something in the bud, even though they have not a clue about where that bud might be located. So why do doctors ignore—even fear—the AMAS and Nagalase tests that would do the same thing for cancer that cholesterol does for cardiovascular disease? It just doesn't make sense. (Sometimes, in my darker moments, I wonder if the availability—or lack thereof—of a drug for the abnormal marker might have something to do with it?)

Earlier detection allows earlier treatment

Treating abnormal lab results (rather than positive imaging or physical exams) allows us to begin treating earlier and earlier in the disease process. As in the examples above, we can detect and reverse a disease with diet, exercise, and nutritional supplements if we catch it early enough. The longer we wait to get the "old-time" diagnosis, the more advanced the disease and the more extreme the treatment measures must be.

In terms of cancer, the bottom line here is that practicing the best possible medicine will force us to dispense with the luxury of palpating the mass or seeing it on an X-ray. Lives will be lost if we stay stuck in that groove. The best doctors will be using newer biochemical techniques to find disease early.

When rising Nagalase levels expose these early occult cancers, the first line of therapy will be GcMAF (100 ng/week, intramuscularly), along with immune strengthening, anti-cancer nutritional medicines. If the GcMAF program works, the Nagalase level (which should be checked monthly) will go down and the doctor and patient can rest assured that the cancer is going away. We'll sleep better at night.

As mentioned above, it may seem bizarre and surreal to be treating a cancer that is still invisible, but this is exactly what is happening—and it works. Dr. Yamamoto's studies showed that with GcMAF it works 100% of the time.

Once Nagalase testing is available (and physicians have learned how to use it), the rules for treating cancer will change. Nagalase testing will not only permit extremely early detection of the presence of cancer cells, it'll also give us a handle on how much cancer there is (we call this "load" or "burden"). And it can accomplish these daunting tasks *before* the tumor has expanded to the point where imaging can blow its cover.

Guidance in this process by a trained professional is absolutely crucial. Trying to be your own plumber may work at times—a leaky faucet probably won't kill you—but in matters of life and death, it is absolutely essential to have experienced guidance. In the early days of GcMAF availability, until proper accreditation is established, finding a doctor who is experienced in the use of GcMAF and Nagalase testing will be challenging.

17: Differentiation and Cancer Grading

Cancer cell "differentiation." About cancer grading. Macrophages easily find undifferentiated cells because they display a higher degree of cell surface abnormality. GcMAF cure rates depend on the degree of cancer cell membrane abnormality, which corresponds to the grade of differentiation of malignant cells.

Under a microscope cancer cells don't look like normal cells

In order to understand how cancers are graded, we need to have a short talk about cancer cell *differentiation*. Suppose you have a ripe banana. As it gradually goes bad, first a few minor brown spots appear on the surface. Then, as it approaches inedibility, it *looks* more and more rotten and it becomes progressively easier to make that determination. Cancer cells are like that. As the cancer progresses, the cells look (to macrophages) increasingly weirder and weirder, and as a consequence they become progressively easier to identify—and kill. Here's how it works…

As they mature, normal cells "*differentiate*" into specialized cells. Cancer cells are different: they do *not* differentiate into mature specialized cells; they remain immature-looking. Cancer specialists therefore call them "*undifferentiated*."

Well-differentiated (i.e., early stage) cancer cells look almost normal, but poorly-differentiated (more immature) cells do not look at all like normal cells. They look like more

and more like cancer cells. Advanced cancers consist mostly of *poorly differentiated* (immature looking) or *undifferentiated* cells.

When macrophages are activated, they develop very large numbers of surface receptors that are programmed to spot cancer cell surface irregularities and then latch onto them. Since undifferentiated cancer cells have more surface irregularities, they are easier to locate. When the activated macrophages find them—well, by now you know the drill.

Cancer "grading"

The above facts are useful when trying to understand cancer *grading*. Degree of differentiation provides information about cancer aggressiveness and progression because the more normal (i.e., differentiated) a cancer cell looks, the lower its grade. The more abnormal or less well developed (i.e., undifferentiated) a cancer cell appears, the higher its grade. There are several grading systems, depending on the institution doing the grading and the tumor type. Here is a description of a typical three-tier grading system:

- Grade 1: *low grade or well differentiated* cancer cells still look a lot like normal cells. These cancers are usually slow growing.
- Grade 2: *intermediate/moderate grade or moderately differentiated* cancer cells do not look like normal cells. They are growing somewhat faster than normal cells.
- Grade 3: *high grade or poorly differentiated* cancer cells do not look at all like normal cells. They are fast-growing or "aggressive."

There is never any absolute certainty about how cancer cells will behave, but grade is a useful indicator. A low grade cancer will grow more slowly and be less likely to spread than a high grade one. Oncologists take cancer grade into consideration when pondering treatment decisions.

Poorly differentiated (immature) cancer cells are colored blue.

Why is differentiation important in terms of GcMAF?

The more undifferentiated a cancer cell is, the easier it is for GcMAF-activated macrophages to find it.

Like an advanced cancer cell, Waldo (of *Where's Waldo?*) stands out in a huge crowd because he looks different. If Waldo were a cancer cell, activated macros would find him easily and he'd be toast in minutes; his little red and white striped shirt and funny glasses would be all that would be left behind.

The weirder the cancer cell looks, the easier the macros can recognize and destroy them. Undifferentiated (i.e., advanced or aggressive cancer cells) have more abnormalities on their cell surfaces, so—to activated macrophages—they look more "foreign" than earlier stage, well-differentiated cancer cells with fewer surface abnormalities. Therefore, undifferentiated cancer cells are more rapidly killed by activated macrophages because they "stand out" (like Waldo) in the crowd.

When macros are activated by GcMAF, their genome dramatically upregulates expression of the receptor proteins that identify cancer cells. More receptors translates into more "detectives" looking for Waldo, so he is lots easier to find.

Dr. Yamamoto showed that cancer cell surface abnormalities manifested a high correlation with GcMAF effectiveness. He also showed that macrophage activation caused dramatic increases in numbers of macrophage cell surface receptors that recognize a wide variety of cancer cell surface abnormalities.

in his 2008 paper on breast cancer Dr. Yamamoto describes these phenomena as follows: "Thus, the macrophages activated by GcMAF developed enormous variation of receptor that recognize a variety of microbial agents (e.g., bacteria and viruses) and abnormalities in malignant cell surfaces. This fundamental nature of macrophages to recognize cell surface abnormality (nonselfing nature) is universal to all types of cancers. In fact weekly administration of 100 ng GcMAF to cancer patients showed curative effects on a variety of cancers. Types of cancer so far tested are prostate, breast, colon, stomach, liver, lung (including mesothelioma), kidney, bladder, uterus, ovarian, head/neck, brain cancers, melanoma and fibrosarcoma. Efficacy of GcMAF therapy for cancers depends on the degree of cell membrane abnormality. Precision of measurement of Nagalase activity allowed us to determine the degree of cell surface abnormality by the curative rate during GcMAF therapy. Undifferentiated tumor cells are killed more efficiently than differentiated cells. In fact adenocarcinoma such as breast and prostate cancer cells are undifferentiated and killed rapidly by the activated macrophages whereas well-differentiated cancer cells such as squamous carcinoma cells are slowly killed by the activated macrophages. This curative rate appears to depend on both the amount of receptors for the particular antigen on macrophages and the amount of antigens on each cell." (Int. J. Cancer: 122, 461–467 (2008). Immunotherapy of metastatic breast cancer patients with vitamin D-binding protein-derived macrophage activating factor (GcMAF). Nobuto Yamamoto, Hirofumi Suyama, Nobuyuki Yamamoto Naofumi Ushijima)

"Administration of 100 ng GcMAF per human results in the maximal level of macrophage activation which develop an enormous variation of receptors that recognize abnormality in malignant cell surface and kill cancerous cells. All malignant cells have abnormalities in their cell surface. A series of glycolipid, glycoprotein and mucin antigens have been identified and designated as tumor-associated antigen (TAA) on the cell surface of a wide variety of tumor cells. When human macrophages were treated in vitro with GcMAF (100 pg/ml) for 3 hr and a breast cancer cell line MCF-7 was added with effector/target ratio of 1.5, 60% and 86% of MCF-7 cells were killed in 4 hr and 18 hr incubation, respectively." (Int. J. Cancer: 122, 461–467 (2008). Immunotherapy of metastatic breast cancer patients with vitamin D-binding protein-derived macrophage activating factor (GcMAF). Nobuto Yamamoto, Hirofumi Suyama, Nobuyuki Yamamoto Naofumi Ushijima)

Again in his 2008 prostate cancer paper Dr. Yamamoto comments on the connection between cell surface abnormalities, degree of differentiation, and effectiveness of GcMAF: "Efficacy of GcMAF therapy and curative rates of various cancers by GcMAF therapy depend on the degree of (cancer) cell membrane abnormality, which corresponds to the grade of differentiation of the malignant cells." (Translational Oncology. 2008 July; 1(2): 65–72. Immunotherapy for Prostate Cancer with Gc Protein-Derived Macrophage-Activating Factor, GcMAF. Nobuto Yamamoto, Hirofumi Suyama, and Nobuyuki Yamamoto)

18: The Cancer continuum and the 'Point of no return'

Stages in the life of a cancer. How Dr. Yamamoto chose his patients had a lot to do with getting 100% cure rates. The optimum point in the cancer continuum for GcMAF therapy. The bigger a cancer gets the less likely GcMAF will work. Tracking effectiveness of all cancer therapies with Nagalase and AMAS. An expanding tumor sooner or later reaches a "point of no return."

The Cancer Continuum

One could view the life of any cancer as a continuum starting with one cell (on the far left side) and continuing through growth and metastasis and finally (to the far right side) as a massive complex of metastatic tumors comprised of many billions of cells, occupying multiple locations in the body —a cancer that has become large enough to overwhelm and kill its host. Cancer specialists "stage" cancer according to where it lies on this continuum.

How cancer grows and spreads

Malignant tumors begin with one cancer cell. This cell multiplies by dividing to become an expanding tumor mass. At first, like termites in your house, cancers persistently gnaw away, expanding and encroaching. They capture territory by spreading locally and pushing aside healthy tissues. Eventually they break through the barriers that keep them local and spill over into the local lymph channels (which try to contain them). Again, if unchecked, the cancer cells next manage to crash the barriers to the bloodstream, which, like enemy submarines, they silently navigate to far- flung regions of the body where they spawn new colonies. Up to this point the body's cancer containment barriers have worked. By breaking out, the cancer has become the insidious, most feared, most deadly form: *metastatic* cancer.

Prior to metastasis, almost all cancers are considered curable. After metastasis, the odds of a cure plummet. But even now, at this, the metastatic stage, oncologists continue lobbing chemotherapeutic bombs and radiologists still blast away with particle beams. In the current system, these well-intended efforts are for the most part futile: metastatic cancer may be slowed but is rarely cured. But as you will see, the standard therapies that reduce tumor burden greatly improve the odds that GcMAF will be effective.

Optimum treatment depends on the stage of the cancer

Optimum treatment strategies vary depending on the type and stage of the cancer. In the earliest stage, prior to detection on imaging (but positive on AMAS or Nagalase), alternative treatment approaches work best. This is true because natural therapies enhance the body's own anti-cancer systems rather than damaging them. By the time a cancer has grown to the size where it can be seen on imaging, however, the "toxic triad" (surgery, radiation, and/or chemotherapy) have become necessary.

Early detection is the Holy Grail

The effectiveness of any therapy, including GcMAF, has everything to do with the point along this continuum at which treatment is initiated. Early detection has justifiably become the gospel of cancer therapy. The sooner a cancer is detected, regardless of type, the better the prognosis. Catching a cancer before it metastasizes is especially important. We want to

move the point of detection as far to the left as we can. AMAS and Nagalase testing will do that.

Metastatic cancer

In "metastatic" disease the tumor has spread beyond the reach of surgery, and chemo and radiation have not succeeded at containment. Pandora's Box is now wide open. These are the patients on whom Dr. Yamamoto chose to focus: the ones with demonstrable *early* metastatic disease (defined by the presence of Nagalase) but for whom the standard therapies had failed. Weekly intramuscular injections of 100 nanograms (100 billionths of a gram; you'd need a powerful microscope to be able to see it!) for 3-6 months cured every single one of these "incurable" patients.

Yamamoto's work pushed the envelope in that he cured carefully chosen *early stage* metastatic cancers. As tumor size and metastases increase, the chance of success with GcMAF declines. With larger (greater than 2 cm.) metastatic tumors, GcMAF may not work or a longer duration of treatment may be required. The probability of success with GcMAF can be improved by reducing tumor burden via chemo, radiation, and surgery. Sooner or later, however, as a cancer continues to grow, even large numbers of highly activated macrophages won't be enough to carry the day.

Choice of cancer stage had everything to do with Yamamoto's success

Why did Dr. Yamamoto choose to treat patients at the point in the cancer continuum where conventional therapies had failed? Obviously, if conventional therapies had *cured* these cancers, there would be no further need for GcMAF or any other treatment. Yamamoto chose this group of patients because he knew that GcMAF would be most effective in patients with low tumor burden who had flunked the mainstream approach and still had cancer growing somewhere within them.

Elevated Nagalase levels told him that metastatic disease continued to lurk somewhere in these patients' bodies. All cancer cells make Nagalase (and normal cells never make it), so the presence of Nagalase is synonymous with the presence of cancer.

Yamamoto also knew that larger tumors would be harder for GcMAF-activated macrophages to vanquish, so he chose patients in whom the bulk of the cancers had been removed and in whom there was great reason to believe that the "tumor burden"—though metastatic—was at its lowest possible point.

Yamamoto's research patients had special advantages. First, they were in the *earliest stage of metastatic disease*. Second—because they had endured the appropriate conventional combination of therapies—their "*total tumor burden*" (the number of cancer cells remaining after surgery, radiation, and/or chemotherapy) was *very low*. As a consequence, GcMAF-activated macrophages were given a huge advantage. The ratio of activated macrophages to cancer cells was high, and so the macros had no trouble polishing off their cancer meal.

Yamamoto's stroke of brilliance was this: he chose patients at the point on the cancer continuum where GcMAF would exert the greatest impact. This point—early metastatic disease with recently minimized tumor burden—thus provided GcMAF with a tremendous likelihood of a cure. And that's exactly what happened—in every single case.

If he had treated earlier—before standard therapies had had their chance to succeed or fail—he would not have known for sure whether or not these patients even had metastatic disease. If he had waited longer to treat, he would given the shrunken tumors time to grow back ("re-bulk"), and thus would have risked losing some of the patients.

The "point of no return"

Individual cancers are difficult to categorize and impossible to quantify, so we are dealing with probabilities here. At some point, however, the cancer and/or its metastases will gain enough mass to be able to thwart the best efforts of debulking followed by GcMAF. This is where the cancer shrugs off the GcMAF effect because it has now grown to that size at which it is now adding new cells faster than the activated macrophages can devour them. The meal is just too big. I call this the "point of no return." It's a cancer cliff, so to speak, beyond which no amount of GcMAF could rescue the patient.

In some cases surgical removal, radiation and/or chemotherapy might debulk the tumor back down to a size where the GcMAF could be effective. These kinds of decisions would obviously have to be made by an oncologist experienced in GcMAF therapy.

Is GcMAF effective in early cancers too small to show up on imaging?

Success rates should approach 100% in these cases. (A good reason to implement Nagalase screening for the masses as soon as possible. For now, go get an AMAS cancer screening test.)

Is GcMAF effective in early small cancers large enough to show up on imaging?

We also don't know how effective GcMAF would be in early disease, but there is excellent reason to believe—based on Dr. Yamamoto's studies—that all, or almost all, early small tumors that have not yet metastasized would respond to standard therapies followed by GcMAF. Based on the outcomes reported in Dr. Yamamoto's studies, effectiveness would approach 100% for cancers that have been recently debulked and are in the early stages of metastasis when GcMAF therapy has been begun.

Is GcMAF effective in advanced (large metastatic) cancers?

Dr. Nobuto Yamamoto's human studies focused only on the early group. We have no research-derived data addressing the issue of GcMAF effectiveness in more advanced cancers. The studies have not yet done, and we don't know how advanced a cancer might be and still respond to GcMAF. There may well be great variability from one patient to another.

Tracking effectiveness of all cancer therapies with Nagalase and AMAS

If cancer is present, Nagalase and/or AMAS will be positive.

Nagalase and AMAS are not only qualitative markers, however, they are also quantitative—by which I mean rising or falling levels tell us what the cancer is up to. Nagalase/AMAS can therefore be used to monitor the effectiveness of *any cancer treatment*, including both drug and alternative therapies, separately or in combination.

Nagalase, AMAS and the "point of no return"

Normally, with GcMAF treatment, serial Nagalase (or AMAS) levels will decrease over time. This tells us the treatment is working (i.e., a declining cancer cell population is making less Nagalase). If, however, the malignant tumor mass had achieved the "point of no return" before GcMAF therapy had been initiated, the Nagalase and AMAS levels might drop initially but eventually would continue rising, indicating the GcMAF-activated macrophages have not overcome the cancer, and that it continues to grow.

19: GcMAF Therapy Guidelines

Dosage. Frequency of administration. Route of administration. Duration of treatment. Side effects and toxicity. Contraindications. Possible GcMAF inhibitors.

GcMAF Dosage

for individual protocols please contact Mary who has effectively worked with GcMaf for many years mary@healingoracle.net she will be willing to advise on a personal protocol.

Route of administration

All patients were given intramuscular (IM) injections of pure GcMAF.

IM injections are necessary because oral administration would expose the GcMAF protein molecule to gastric hydrochloric acid and pancreatic protease enzymes, resulting in degradation and deactivation.

Duration of treatment

In Dr. Yamamoto's studies the duration of treatment varied from patient to patient and disorder to disorder:

- 100% of nonanemic HIV patients were cured in less than 18 weeks.
- 100% of nonanemic early metastatic breast cancer patients were cured in 16-22 weeks.
- 100% of nonanemic early metastatic colorectal cancer patients were cured in 32-50 weeks.
- 100% of nonanemic early metastatic prostate cancer patents were cured in 14-25 weeks.

Side effects and toxicity

There has never been an adverse reaction, side effect, or toxic reaction to pure GcMAF. Because its molecular structure is identical to GcMAF made by the body (i.e., *bioidentical*), there is absolutely no reason to expect that pure GcMAF would cause any kind of problem. Our genetic program contains the code for building GcMAF molecules. In normal, healthy bodies, the genes for GcMAF production are "expressed," and ongoing GcMAF production makes sure our macrophages are continuously activated so that they can effectively address the ongoing onslaught of bacteria, viruses, parasites, and newly forming cancer cells. When viruses and cancer cells obstruct GcMAF production by releasing Nagalase, this finely tuned

mechanisms breaks down, GcMAF dwindles, and macrophages slow down and stop. Now the welcome mat is out, the door is open, and the intruders come marching right in. Replacing the deficient GcMAF with *exactly* the same molecule that is missing guarantees there will be no adverse reactions.

The administration of impure (non-bioidentical or contaminated) GcMAF carries a high potential for side effects and adverse reactions. It also might not work. In Chapter 21 I address the challenging problem of bootlegs, counterfeits, knockoffs, wannabes—and certification to ensure purity. The importance of a establishing a GcMAF certifying authority cannot be overstated; a market flooded with ineffective products would provide authorities with a good reason to shut down all production.

Diseases for which Dr. Yamamoto has indicated he feels GcMAF would be effective:

- Tuberculosis
- Cancers (all)—including prostate, breast, colon, stomach, liver, lung (including mesothelioma), kidney, bladder, uterus, ovarian, head/neck, brain cancers, melanoma, and fibrosarcoma
- Influenza A and B
- Herpesviruses
- Hepatitis B and C
- Human Immunodeficiency Virus (HIV) All other retroviral infections
- Epstein-Barr Virus

Potential Obstacles to GcMAF effectiveness

Protease inhibitors – GcMAF might not work for HIV patients taking protease inhibitors. In macrophage phagolysosomes, proteases work to digest cancer cells and viruses. Protease inhibition might interfere with phagolysosomal protease activity, slowing digestion of phagocytized invaders, thus indirectly obstructing the macrophage activating effect of the GcMAF.

Opiates – According to Dr. Yamamoto, opiates (morphine, Demerol, opium, oxycodone, hydrocodone, Percodan, Percocet, Vicodin, Norco, etc.) may block GcMAF's macrophage activating effect.

Anemia – A lack of sufficient red blood cells may compromise GcMAF effectiveness. Anemic patients were excluded from all of Dr. Yamamoto's studies.

Insufficient macrophages – If a person doesn't have enough macrophages or monocytes (the precursor cells that become macrophages), they might not respond as briskly to GcMAF as a person with normal macrophage/monocyte levels. Patients with low white cell counts (or compromised immune functioning) should take hydrolyzed whey protein which will encourage the bone marrow to make more white blood cells. Make sure you purchase *cold processed* "hydrolyzed" whey protein marketed by a company that specializes in supplying nutritional medicines to physicians (Pure Encapsulations would be an excellent choice). The whey protein found in health food stores is processed differently and would not be effective.

Medical monitoring required

Good intentions coupled with poor training can lead to disaster. Anyone who allows a novice to administer GcMAF is making a big mistake. Once available, GcMAF administration must always be monitored by a physician trained in its use.

Rather than a substitute for conventional cancer therapy, GcMAF is an adjunct to it. GcMAF effectiveness depends on optimum administration of conventional mainstream cancer therapies. Substituting GcMAF for conventional therapies for an imageable cancer could result in a preventable death.

Medically unsupervised administration of GcMAF would be a grave error for the following reasons:

- GcMAF effectiveness depends on optimum administration of conventional mainstream cancer therapies. Tumor debulking using surgery, chemotherapy, and/or radiation—is crucial because GcMAF works best on smaller cancers. Debulking moves the patient back from the point of no return.

- Physicians are necessary to diagnose and treat concomitant medical disorders.
- Your doctor will need to monitor progress by checking AMAS or Nagalase levels (when available) in order to determine whether or not the GcMAF is working, and to know when it's time to discontinue it.
- Cancer imaging, ordered and interpreted by physicians, provides crucial information about cancer progress.

20: Why Not Skip Conventional Cancer Therapies and Just Take GcMAF?

Why not just skip the surgery, radiation, and chemo and use GcMAF all by itself?

Some cancer patients—in the hopes of sidestepping the slash, burn, and toxic discomforts of surgery, radiation, and chemotherapy—might consider opting for GcMAF therapy all by itself. If GcMAF works, they ask, why bother with the nasty stuff?

Not so fast. For most cancer patients this certainly would be a very bad idea. Here's why: GcMAF works better on smaller tumors. Despite the many drawbacks of the "big three," they all reduce tumor mass. The basic idea here is that activated macrophages gobble up cancer cells, but—as with your lunch —the less you have to eat, the quicker and easier you can finish the meal. And just as there is an upper limit to the size of a meal you could consume, there is likewise a limit to the amount of tumor material even the most aggressively activated macrophages can polish off.

Think about it: the bigger the mess, the more sponges you'd need to clean it up. Same's true for cancer. Surgery, radiation, and chemo all diminish the size of the tumor and/or its metastases (this is called "debulking"). Smaller tumors are easier targets for the GcMAF-activated macrophages because with fewer cancer cells to devour, their workload is lightened. So, you cancer patients dreaming of a quick, simple, easy cure, listen up: avoiding the recommended conventional treatments for your cancer would not be a wise move. Using GcMAF on a debulked, smaller tumor may make the difference between cure and no cure.

Another faulty line of reasoning might go something like this: "I could try the GcMAF alone, and if it doesn't work, then I can still go back and do the surgery, chemo, and/or radiation."

This approach might prove foolhardy because waiting could allow cancer growth beyond the point at which it is still reversible. Again, it's best to go with the "debulk first" strategy.

Dr. Yamamoto debulked first

It was no accident that Dr. Yamamoto chose to limit his groundbreaking human research to cancer patients who had recently received optimum "debulking" procedures. Each of the breast, prostate, and colorectal cancer patients to whom he administered GcMAF had recently completed the appropriate mainstream course of treatment for their cancer. Surgery, radiation, and/or chemo (in whichever combination was indicated in that particular patient's cancer) had been done. Because these patients still had elevated Nagalase levels, he knew they had metastatic disease. The conventional therapies had not cured them. The patients had, however, been "debulked," and this made a huge difference in the ability of the GcMAF to do its job. Yamamoto thus proved that combining mainstream debulking therapies with GcMAF could produce a cure in 100% of early metastatic cancer patients in whom the conventional therapies alone had failed.

Had Yamamoto chosen identical patients (in terms of cancer type and stage) and administered the GcMAF prior to the conventional therapies, he almost certainly would have had some failures. Had he waited months or years for these uncured cancer patients' tumors to return (to "re-bulk," as it were), again, his cure rate would certainly have been lower. Debulking works, is an important feature in the curative process, and should not be avoided or delayed in the hopes that GcMAF will accomplish the job all by itself. One who proceeds down this avenue risks losing the chance at a cure that early debulking therapies—followed by GcMAF—might have provided.

Reducing viral "load" in HIV

The above logic also applies to HIV. Using appropriate drug cocktails to reduce HIV viral load will give GcMAF a big head start in terms of devouring the remaining viruses, and this could make the difference between cure and no cure, between life or death. (Because protease inhibition might interfere with GcMAF effectiveness, it will probably prove necessary to discontinue protease inhibitors while on GcMAF therapy. HIV patients—once GcMAF is available—will need to discuss these concerns with their physician.)

The exception to the debulking requirement: early cancers too small to image

In the earliest stages of cancer, when the mass is still very small (< 5 mm), the "debulk first" requirement does not hold. If a patient knows from an elevated Nagalase level that he or she has a cancer growing somewhere, but it is not big enough to be seen on imaging and there are no localizing tests like a positive PSA (prostate), BrCA1 or 2 (breast), or CA-125 (ovarian) to identify its whereabouts, then using GcMAF alone would be advisable. If, over time, the Nagalase (or AMAS) levels drop back to normal, one can assume the cancer was "nipped in the bud," cured before it got big enough to be seen on imaging. If I had a tumor that was big enough to see on imaging, however, I'd want a surgeon to take it out. I'd still use GcMAF (if it were available), just in case the surgeon didn't get it all.

Since Nagalase testing is not yet available, using it to find cancer early remains a purely theoretical notion. Meanwhile, AMAS testing can be used to detect early cancers and to track cancer therapy.

“How can I treat it I can’t see it?”

Some physicians find it disturbing to be treating an “invisible” disease that has no symptoms, no physical signs, and even eludes state-of-the-art imaging. These old-schoolers remain hesitant to treat if they cannot make a formal diagnosis using the tools they understand best. Fear not, this is an outmoded, vanishing breed that in the age of molecular and genetic medicine is headed for extinction.

Using state-of-the-art biochemical markers, the molecular medicine early warning system enables earlier diagoses. Detected in its infancy, a developing disease can usually be reversed using non-toxic nutritional medicines (diet, phytonutrients, herbs. vitamins, minerals, amino acids, essential fatty acids, hormones, enzymes, and homeopathic medicines). Once the disease has progressed to a heart attack or large tumor, however, reversal becomes far more difficult, natural medicines are less often an option, and the need to resort to strong drugs looms large.

So here’s my unsolicited advice to those doctors who would resist identifying and treating the earliest manifestations of disease: study molecular biology and natural healing methods. Good things will happen!

21: Knockoffs, Wannabes, Bootlegs, Counterfeits–and Certification

Hype and misinformation about GcMAF. Ineffective and potentially toxic products. Pure, real GcMAF vs. phonies. Types of GcMAF knockoffs. About purity. GcMAF production. Contamination. Certification. Caveat emptor!!

Be careful!

The emergence of GcMAF therapy and Nagalase testing as viable methods for treating and preventing cancer will inevitably be accompanied by genuine concern and debate about how best to put these valuable tools to work. Paralleling these positive efforts to prevent suffering and save lives, negative forces will exude from the swamp of diabolical human greed. Slick hucksters will offer stuff called “GcMAF” that doesn’t work—and may even be harmful. Ineffective imitations and counterfeit products will appear, including knockoffs, copycats, phonies, bootlegs, and wannabes. There will be nay-sayers and disinformation campaigns trying to convince you this is all a big hoax. And, as we all know, the internet is rife with hype and misinformation; GcMAF/Nagalase will be no exception.

What’s a person desperately in need of cancer or HIV treatment to do? The urge to buy into false hope may be difficult or impossible to overcome.

Pure, real GcMAF vs. the phonies

Because it is identical to that which the body makes (i.e., *bioidentical*), pure GcMAF will never cause symptoms of any kind. When symptoms appear in an individual taking GcMAF, the cause is impurity, contamination, or both.

The phonies will come in packages that look exactly like quality-certified products. Packaging replication technology has advanced to the point where even brand-name manufacturers can’t tell the difference between their own products and the knockoffs without

chemically testing the ingredients. People will die needlessly because they put their faith in phony GcMAF products.

Types of GcMAF knockoffs

A huge market for GcMAF will emerge as it becomes a household word. The already substantial risk of impure, contaminated, and inactive products will then skyrocket. Hucksters will ooze from the woodwork, hawking "GcMAF" that will range from inactive and harmless to toxic and dangerous. There will be different types of knockoffs:

Bootleg or *wannabe* versions: serious attempts to make GcMAF that have fallen short and are impure, contaminated, and/or potentially toxic. Bootleg versions can be pure but "dirty," containing contaminants that cause muscle aches, flu-like symptoms or fatigue. A bootleg could also be pure (in the sense of clean), but non-bioidentical, and therefore too weak in its macrophage activating power to be effective.

Phony versions: packaged to look exactly like the real thing, but totally inactive. The "GcMAF" might be powdered sugar, starch, or any (hopefully) harmless white powder that, like GcMAF, disappears when put into solution with water. The phony product might simply contain nothing at all: several hundred nanograms of GcMAF in a vial, sans water, would be about the size of a small speck of dust.

As mentioned above, nowadays it is possible to so exactly copy a bottling and packaging process that even the authentic manufacturing company can't tell, by examining the packaging alone, whether or not the product inside is real. Chromatography testing is required to make that determination.

I'll never forget an experience I had several years ago. A patient told me she had been injecting human growth hormone (hGH) that she had been obtaining from a source in in Tijuana, via San Diego. She showed me the package and it looked real enough, as if it had come directly from the manufacturer: perfect box, red plastic-capped vial inside, flawless label, nothing suspicious. I suggested we test her blood for hGH, and when we did, there was none there! She had been injecting very expensive water! Obviously this was a perfect knockoff, and when I contacted the manufacturer (of the real product—a major brand name household word drug company), they told me that even *they* were unable to tell by the packaging alone whether or not a given product was the real thing. They had to test the ingredients to be sure it was theirs! Perfect packaging tells you nothing.

Purity and GcMAF production

A "pure" protein contains only molecules of a single protein, and no other molecules of any kind. Impurities cause compromised effectiveness, adverse reactions, and symptoms of toxicity. Making "pure" GcMAF is not a simple undertaking. So far, the only human being to have succeeded in making pure GcMAF is Professor Nobuto Yamamoto. The process is not that complicated, however. Dr. Yamamoto provides specific instructions in his research papers. A good biochemist with proper equipment could easily make GcMAF. The raw materials are inexpensive and a large scale manufacturing operation should make for a price tag that's within reach of the average person.

Bravo is not active GcMaf. Beware of this product portrayed as the real GcMaf, it is a far cry from the real thing.

GcMAF can be made via recombinant DNA technology by inserting a small section of DNA into the bacterium E. coli. This piece of DNA reprograms the bacterium to make GcMAF. An *impurity* can be introduced at any step in this multistage protein manufacturing process.

For example, once these reprogrammed bacteria are incubated and have generated the GcMAF from their revised genetic program, the newly made GcMAF must be separated from the E. coli and then purified. If some of the E. coli (or any of the many other chemicals generated by E. coli) are left behind with the GcMAF, you have a troublesome impurity. Endotoxins are an especially common E. coli byproduct impurity; these typically cause flu-like symptoms. Solvents and surfactants can likewise be left behind in the purification process.

Impurities don't necessarily affect the macrophage activating power of the GcMAF product, but they can and do cause side effects such as fatigue, weakness, malaise, and muscle and joint pain. These symptoms can be so uncomfortable for the patient that it becomes impossible to tolerate the beneficial effects they would otherwise obtain from the GcMAF.

Contamination

Contamination occurs when a foreign agent gets into the GcMAF product from the *outside* (rather than being introduced as a part of the manufacturing process). Contaminants can cause symptoms similar to those due to impurities (fatigue, weakness, malaise, and muscle and joint pain). Contaminants can be airborne, or carried into the (otherwise sterile) production lab environment by hapless humans—on their hands, feet, or clothing. They can enter via the lab's water and air supply. A cough from a person with a bacterial respiratory infection could cause big problems. Contamination via molds, viruses, and bacterial species is very common in biotech production facilities and requires hygiene precautions so elaborate they make surgical scrubbing look like a cakewalk. Entering a biotech production facility is like dressing for a walk in outer space: cap, gloves, full body gown, mask, booties. You don't need oxygen tanks, but the air you breathe must be filtered to remove dust, toxic chemicals, and pathogenic microorganisms.

Potency (quantified as macrophage activation)

Assuming we have a pure GcMAF product, then what about activity or *potency*? In the world of macrophages, potency is activity. The degree to which GcMAF activates macrophages is our measure of potency. Only pure, bioidentical GcMAF will provide the level of optimum macrophage activation necessary to reverse cancers and viral infections.

Extremely sensitive receptors on the surface of macrophages are happiest when they are stimulated by pure GcMAF. Rearrange even a few atoms, much less a few amino acids, and the "GcMAF" will not look the same to those receptors and either they won't recognize it at all, or their response will be halfhearted. So, beyond purity, the GcMAF product must also be tested for macrophage activating power. This test literally asks the macrophages, "Do you recognize this particular batch of GcMAF?" And, "How much does it activate you?" Because this involves working with live human macrophages, it is not easy. To determine the macrophage activating power of pure bioidentical GcMAF, Professor Yamamoto quantified

rate of phagocytosis of cancer cells (a 30 fold increase), rate of generation of superoxide radicals (15 fold), and rate of macrophage proliferation (40 fold).

Certification

It will be necessary to establish a consumer protection certification program whereby product offerings—when they do become available—will be subjected to rigorous testing and then certified for both purity and activity.

Where to purchase quality GcMaf:

1. **Healing oracle: mary@healingoracle.net**
2. **Order information click here**
3. **Contact@saisei-mirai.or.jp**

Q. Where can I find a clinic using GcMaf therapy?

A. Please contact mary@healingoracle.net

Appendix 1: About Dr. Nobuto Yamamoto

Dr. Nobuto Yamamoto was born in Japan on April 25, 1925. Though accepted to three medical schools, he chose to focus on biochemistry in graduate school.

After graduation he served as associate professor at Gifu Medical School, Japan until 1959.

Visiting scientist in the Microbiology Group at the Institute for Cancer Research (Fox Cancer Center), Philadelphia, PA, from 1959 to 1961; studied the genetic evolution of bacterial viruses.

1964: joined the faculty at Temple University, Philadelphia, PA, as Head of Virology and Genetics of the Fels Cancer Research Institute where he served until 1980.

In 1980 Dr. Yamamoto was appointed professor of Microbiology and Immunology at Hahnemann University School of Medicine, where he continued to study viral evolution and revived his graduate study of immunology from 35 years before. His immunological studies emphasized mechanism of macrophage activation and discovered GcMAF.

When Dr. Yamamoto retired from Hahnemann University in 1990, he was asked to return to Temple University Medical School as a Research Professor of Biochemistry. There he studied the tumoricidal capacity of macrophages activated by GcMAF and cancer therapy with GcMAF.

In 1994 Dr. Yamamoto became the founder and director of the Socrates Institute for Therapeutic Immunology, where he continues to study the therapeutic efficacy of GcMAF for a variety of cancers and HIV.

To make a donation supporting Dr. Yamamoto's work, you could send a contribution to: Socrates Institute for Therapeutic Immunology, 1040 66th Ave Philadelphia, PA 19126-3305.

Appendix 2: The Yamamoto Papers

Dr. Yamamoto's 2008 breast cancer paper entitled: "Immunotherapy of metastatic breast cancer patients with vitamin D-binding protein-derived macrophage activating factor (GcMAF)." (This is an abstract. You can access the full paper by clicking in upper right-hand corner.)

Dr. Yamamoto's 2008 prostate cancer paper entitled: "Immunotherapy for Prostate Cancer with Gc Protein-Derived Macrophage-Activating Factor, GcMAF."

Dr. Yamamoto's 2008 colorectal cancer paper entitled: "Immunotherapy of metastatic colorectal cancer with vitamin D-binding protein-derived macrophage-activating factor, GcMAF." (This is an abstract. You can access the full paper by clicking in upper right-hand corner.)

Dr. Yamamoto's 2008 HIV paper entitled: "Immunotherapy of HIV-Infected Patients With Gc Protein-Derived Macrophage Activating Factor (GcMAF)."

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