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SciLifeLab, Science for Life Laboratory, is a Swedish research center within molecular biosciences with focus on health and environment. To further strengthen the research environment at SciLifeLab the center regularly recruits young, talented research leaders to become SciLifeLab fellows. Each fellow is recruited by one of the center host universities and receives funding from them.

One of the SciLifeLab fellows is Paul Hudson whose research focuses primarily on metabolism of photosynthetic cyanobacteria. The idea is to manipulate the bacteria to make chemicals and fuels from carbon dioxide, water and light, which are all free abundant resources. Paul’s aim is always to link fundamental science with an application.

“Our dream is to create a microorganism that can simultaneously reduce greenhouse gases and produce something of value, like a fuel or chemical that we right now can only get from oil”. Paul said.

Paul did his PhD at the University of California, Berkeley, US, and then moved on to a Post Doc position in proteomics at KTH Royal Institute of Technology in Sweden before he applied for the SciLifeLab fellows program.

“There was this concept at SciLifeLab of building up expertise in high throughput genomics and systems biology and I thought it would be interesting to apply these new technologies to study and engineer an ancient organism like cyanobacteria. The start up-package offered lots of financial support, which was appealing of course.”

“Right now we are applying systems biology tools to cyanobacteria in a way that I think is only possible at SciLifeLab. Being here has changed our scientific approach to old problems; as a result I have started thinking about cellular processes in a different way. I also get a lot of great input from the other SciLifeLab fellows. For example, I sit next to Vicent Pelechano who is an expert in RNA sequencing techniques. Applying these makes our research unique in the field of metabolic engineering.”

Recently, Paul’s group also expanded to study other bacteria, such as those that use hydrogen gas as their energy source. That is very relevant for Sweden because the country has an abundance of sources for hydrogen like the forest industry and hydroelectric power where electricity is used to split water and make hydrogen.

“Sweden is good for me professionally because there are many avenues of support for environmental research. The government and the industry in Sweden are unified in this and are consistent and serious in wanting to reduce greenhouse gases. I can definitely see myself staying in Sweden.”

SciLifeLab – a national resource

SciLifeLab is a Swedish research center within molecular biosciences with focus on health and environment. It is also a national center with the mission to develop, use and provide advanced technologies. The center infrastructure encompasses a multitude of biomolecular technologies and bioinformatics services. National funding makes SciLifeLab’s services and expertise available to researchers in all of Sweden.

The center is a joint effort by four Swedish universities (Karolinska Institutet, KTH Royal Institute of Technology, Stockholm University and Uppsala University). Founded in 2010, the center today encompass more than 1 200 researchers mainly located in and around the two center nodes in Stockholm and Uppsala.
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Cancer immunotherapy comes of age

Oncologists have long rested their treatment plans on three so-called “pillars”—chemotherapy, surgery, and radiation. But in recent years, scientists have been busily erecting a fourth pillar: immunotherapy. The idea of harnessing the immune system to fight cancer has already moved from the lab to the clinic, thanks to technologies such as checkpoint inhibitors and genetically engineered immune cells. By Amber Dance

Fifteen years ago, Renier Brentjens returned from a vacation and rushed to his lab at Memorial Sloan Kettering Cancer Center in New York. A month earlier, he’d treated mice with genetically engineered immune cells that he hoped would combat cancer. And when he got to the lab, he found that all of the mice were still alive.

Amazed, Brentjens thought to himself, “This stuff might actually work.” And it did—in 2013, he and his colleagues reported that they used this kind of cell therapy to treat five people with B-cell acute lymphoblastic leukemia, and all five achieved total remission, though one later relapsed. That success ignited a “firestorm” in the development of engineered immune cells, says Brentjens.

The idea behind immunotherapy is to harness the system the body normally uses to attack pathogens and encourage it to go after cancerous cells instead. The field has exploded in recent years, with approval of a handful of medications and nearly 1,500 cancer immunotherapy trials listed on the U.S. National Institutes of Health ClinicalTrials.gov registry.

Two approaches getting plenty of attention are checkpoint inhibitors and modified cells known as “chimeric antigen receptor (CAR) T cells.” The former approach takes the brakes off of anticancer immune cells. The latter, used by Brentjens, involves genetically engineering immune cells to allow them to home in on cancerous cells.

But those are just two of many ideas under the immunotherapy umbrella, which also includes approaches such as vaccines. Those developing such therapies use a variety of techniques and tools, including antibodies, gene editing, and viral gene transfer. Unfortunately, these treatments don’t usually work for all cancers, and can cause serious side effects and even death—meaning there is still plenty of work to do to improve them and to eliminate potential risks.

The answer to all cancers?

While one should be cautious about the word “cure,” there are certainly patients from early trials who are still alive 10 years later with apparently little or no cancer in their bodies, according to Alan Korman, vice president of immuno-oncology discovery at Bristol-Myers Squibb in Redwood City, California, who has been involved with developing two of the checkpoint inhibitors now on the market, nivolumab and ipilimumab.

Indeed, cancer immunotherapy is not a new idea. The late-19th century surgeon William Coley found that deliberately inducing bacterial infections in his patients could sometimes mysteriously eliminate cancer. Though he didn’t understand how at the time, it’s now believed that the bacteria or bacterial products Coley used activated his patients’ immune systems. As radiation—which was easier to apply and offered more consistent results—became a popular therapy, Coley’s toxins fell by the wayside.

Another early hint of immunotherapy’s potential came in the late 20th century, when clinical trials showed that treating melanoma with interleukin-2 (IL-2), an immune cell regulator, yielded survival beyond five years for many patients.

Checkpoint bypass

In the bodies of many people with cancer, there are already immune cells that can recognize and attack the tumor. But tumors defend themselves by producing compounds that activate biological “checkpoints” to stifle those protective cells.
Now medications have been developed to bypass those checkpoints. The first such medication to undergo testing was ipilimumab, an antibody to the inhibitory receptor CTLA4. Ipilimumab sits on the surface of immune T cells and blocks CTLA4’s activity, allowing the T cells to attack tumors. Soon after its success, scientists also achieved favorable results with antibodies that block either PD-1, expressed on immune cells, or its suppressor, PD-L1, found on tumors and some immune cells. Today, four such checkpoint inhibitor antibodies are on the market—nivolumab (Opdivo) and pembrolizumab (Keytruda) against PD-1; atezolizumab (Tecentriq) against PD-L1; and ipilimumab (Yervoy) against CTLA4—and other potential checkpoint targets are being actively pursued.

Checkpoint inhibitors have already changed cancer treatment, says David Kaufman, executive director of translational immuno-oncology and lead for oncology clinical research at Merck Research Laboratories in North Wales, Pennsylvania, which makes the checkpoint inhibitor pembrolizumab. “What it’s done is displace chemotherapy in many settings where chemotherapy was either the only option or the best of a handful of less-than-ideal solutions,” he says.

Checkpoint receptors are just one type of immune molecule that scientists hope to take advantage of. “Almost anything on the surface of a T cell is now a potential target for activating the immune response,” says Korman, adding that there are also molecules on T cells that, when bound, shun up the immune response. These molecules are called “costimulatory receptors,” and companies are already testing whether binding and activating them with antibodies could improve immune activity.

**Different patients, different responses**

For some patients, treatment with checkpoint inhibitors can destroy cancer, or at least keep it in check, leading to “a new détente between the tumor and the immune system,” says Kaufman. Around 20% of all cancers respond to this type of treatment, he estimates. Those tend to be the people who already have cancer-targeted T cells waiting in their tumors before they even start immunotherapy. All their T cells need is for the checkpoint inhibitors to unfetter them. But for other patients, checkpoint inhibitors don’t work.

There are probably multiple reasons for the different response patterns, and researchers at Merck and elsewhere are trying to understand them. It might be that certain tumors have antigens—that the molecules immune cells recognize as foreign or dangerous—that are hard for the immune system to identify, Kaufman explains. Or perhaps T cells are present but are unable to reach the cancer cells, he adds.

Another issue is that sometimes patients respond to checkpoint inhibitors at first, then develop resistance. Researchers are just starting to figure out why that might be, says Kaufman. In some cases, the tumors seem to change, making themselves resistant to the attacking molecules produced by T cells. Or they may undergo mutations rendering them invisible to those T cells, and thus evade attack.

For those unlucky patients who don’t respond to checkpoint inhibitors, others are working on cancer vaccines as a way to wake up the immune system and bring those T-cell “soldiers” to the tumor site. The idea, explains Elizabeth Jaffee of Johns Hopkins University School of Medicine in Baltimore, Maryland, is to generate new T cells specific to the cancer, so follow-up treatment with checkpoint inhibitors can set them to work. She is now planning for trials with a fast genetic-sequencing technology that defines unique mutations in tumor cells—called “neoantigens”—to create tailored vaccines.

**Riding in CARs**

Checkpoint inhibitors may also work in combination with cell-based therapies. Normally, the body eliminates T cells that would attack its own, “self-” tissues and cause autoimmune disease, leaving only immune cells that attack anything “nonself.” That gives cancer an advantage, since it’s also a self-tissue. The idea of CAR T-cell therapy, explains Brentjens, is to “re-educate” certain T cells to identify the tumor as nonself.

T cells use T-cell receptors (TCRs) to identify antigens. Researchers add a gene to a T cell to manufacture modified TCRs, or CARs, on T-cell surfaces. These specialized receptors contain an antibody-like part that binds to a specific protein (antigen) on a cancer cell. Further inside the cell, the CARs have a domain that mimics the signals activated by antigen-attached or “bound” TCRs. A transmembrane domain, and a flexible hinge that allows the antigen-binding portion to reach its target, round out the chimeric protein. Researchers frequently use lentiviruses or retroviruses to deliver the genetic payload to the T cells. Once inside a patient, when the CAR-bearing T cell binds a cancer cell, it should respond as if it’s seen an invader and attack.

So far, CAR T-cell trials have focused primarily on blood cancers. All B cells in the blood, including any cancerous ones, express a marker called “CD19,” so researchers designed CARs that bind to it. In a recent trial, Novartis announced that 89% of children with acute lymphoid leukemia were alive after six months. Without the treatment, one would expect that...
number to be much lower, says immunologist Bruce Levine of the Perelman School of Medicine, University of Pennsylvania, who collaborates with the company. For this treatment, in addition to adding the CAR, researchers activate the cells with a costimulatory antibody. Then they grow the engineered cells in bioreactors before returning them to the patients.

Unfortunately, receiving CAR T-cell therapy is no Sunday drive. One of the signs it’s working is that the patient gets miserably, dangerously sick. Amping up the immune system causes the cells to release signaling molecules called “cytokines,” and can lead to a “cytokine storm.” This causes symptoms such as nausea, fatigue, and fever—a handful of patients have died as a result.

But when it works, it works wonders. During an early trial in 2010, Levine and his colleagues calculated that each of their two patients lost between 2.5 and 8 pounds of leukemia cells. Two of those patients are alive today.

Seeking the “Holy Grail”

Despite these successes, CAR T-cell therapy remains immature. “We have a Model A Ford,” says Brentjens. “We need a Ferrari.” Reducing toxicity is a key goal. One backup system researchers are exploring is to include a self-destruct gene in their CAR T cells, such as a caspase cell suicide gene, that can be turned on by a medication, so they can delete the engineered cells if necessary.

Another major challenge is to take CAR T-cell therapy beyond blood cancers. Even though the treatment attacks all of the cells expressing CD19—cancerous and healthy ones—patients can live for a time without those kinds of cells. That’s not the case with the body’s organs.

“The Holy Grail” would be a molecule expressed on all tumor cells that is not expressed on any healthy cell in the body,” says John Maher, an immunologist and clinician at King’s College London.

CAR aficionados have limited choices for targets because the CARs can only access molecules on the surface of cancer cells. That’s why some scientists prefer to work with natural TCRs, which recognize snippets of internal proteins displayed on a cell’s surface. “In a way, the TCRs dig inside the cancer cells,” says Chiara Bonini of San Raffaele University and Hospital in Milan, Italy. She is working on a procedure to take a patient’s T cells, remove their TCR genes with zinc-finger nucleases, and use a lentivirus to add in new, tumor-specific TCRs.

A team led by Steven Rosenberg at the National Cancer Institute (NCI) in Bethesda, Maryland, has found that the natural TCRs on T cells already resident in a tumor are often pretty effective. In one experiment, they collected immune cells from a patient’s tumor, grew them in culture, and selected the ones that recognized cancerous cells. Then, they gave these chosen cells back to the patient. Doing this, the group obtained “dramatic” results with melanoma, says Stephanie Goff, a member of Rosenberg’s team. Up to 70% of patients saw their tumor load decrease substantially; in one trial, 40% had their tumors disappear for at least five years after treatment.

Other roadblocks for CAR T-cell therapy

Researchers are also beginning to load their CAR T cells with additional factors that should help them cut through the tumor microenvironment. Brentjens, for example, has engineered CAR T cells that make their own IL-12, which amplifies immune responses in a solid-tumor environment.

Yet another issue with CAR T-cell therapy is its personalized nature. Making every batch of individualized T cells currently takes “a lot of labor,” says Levine.

Collectis thinks it has the answer to making off-the-shelf, universal CAR T-cell treatments, says Julianne Smith, vice president of CAR-T development at the Paris-based company’s New York City branch. The company uses gene editing, based on precisely targeted transcription activator-like effector nuclease (TALEN) enzymes, to delete part of the TCR complex from donor immune cells, so they shouldn’t attack a new host. These CAR T cells will eventually be rejected by the recipient, but Smith thinks they’ll last long enough to perform their duty. The cells are in clinical trials now.

Back to the bench

The normal progression of biomedical science is to translate an idea in the lab into a treatment in the clinic. But with so much still unclear about how immunotherapies work, which approach to take, and how to improve the available treatments, lab scientists are busy.

Researchers want to understand the tumor microenvironment, and how a person’s microbiome might influence immunotherapy. Moreover, they are eagerly searching for biomarkers that would tell them if immunotherapy—which can take time to show definitive results—is working in a patient, says Jaffee. And they are also poring over tissue samples from patients who were treated, trying to differentiate those who respond to a given immunotherapy from those who don’t. That, says Kaufman, involves sequencing DNA and RNA, examining epigenetic markers, and visualizing tissues via immunohistochemistry.

Nonetheless, immunotherapy has already handed cancer physicians a powerful new weapon, not to mention an entirely new area of biology to master. “The oncologists are becoming the new immunologists,” says Maher.

Amber Dance is a freelance writer living in Los Angeles.

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