MAGNIFICENT SEVEN

Meet seven Manitoba researchers who are working to answer some of the world's toughest medical questions

By Joel Schlesinger

Numerous Manitobans have made important contributions to the field of medical research over the years. One of the earliest contributors was Dr. Bruce Chown, best known for developing a vaccine for RH (Rhesus Factor), a potentially fatal blood condition that affects women and their fetuses. Licensed in 1968, the vaccine is credited with saving tens of thousands of lives and is considered by many to be the most important medical discovery made in Manitoba.

More recently, Dr. Allan Ronald was inducted into Canada's Medical Hall of Fame in April for his work in pioneering the University of Manitoba's world renowned infectious disease research program. In addition, he has mentored a number of key Manitoba researchers, including Drs. Frank Plummer, Stephen Moses and Jamie Blanchard. All three, in turn, have made their own important contributions to HIV research. This rich tradition of excellence continues today, thanks in large measure to the support of the Manitoba Health Research Council. Every year, the council provides more than $5 million to further health research in the province. A key area of focus is to recruit and retain the best and brightest talent via the Manitoba Research Chairs program. Each year, the program provides two grants totalling $1 million over five years to researchers in this province. These grants are making it possible for Manitobans to conduct research that will lead to better suicide prevention programs, enhanced protocols for managing asthma, better care for children with disabilities, and potential cures for cancer and HIV. All of this work will improve the delivery of care, not just in Manitoba, but around the world. This special report, sponsored by the Manitoba Health Research Council, highlights the work being done and the people who are doing it.

GROUND ZERO

Manitoba research may hold key to fighting a new type of HIV infection

Manitoba is ground zero for a new type of HIV infection.

And Dr. Keith Fowke, a microbiologist at the University of Manitoba, believes he has found one of the reasons why. If he's right, it would be a discovery that would have an impact in Manitoba, and around the globe.

In the last few years, doctors at local community health clinics have encountered patients with rapidly progressing HIV disease that leads to AIDS – the collapse of the immune system – in months rather than years.

"Normally, without the use of anti-HIV drugs, the disease progresses in about 10 years to AIDS," says Fowke, Professor of Microbiology at the University of Manitoba. "These people are losing all their CD4 cells and dying within a matter of months."

Fowke, a leading HIV researcher, is interested in how the virus infects people differently in countries around the world. His work has been wide-ranging, from studying sex trade workers in Nairobi, Kenya, who, despite being repeatedly exposed to the virus, do not become infected, to learning why HIV progresses so rapidly in solvent abusers in Manitoba.

"One of the underlying themes of all of our research is that in order for HIV to replicate efficiently in a cell, that cell has to be activated," says Fowke, a 2008 Manitoba Health Research Council Chair award recipient.

HIV infects CD4 positive T-cells, a type of white blood cell that regulates the body's immune system. When viruses, bacteria or fungus invade the body, these CD4 cells activate and "act as generals sending orders to the killer cells to destroy the invading organism."

But once activated, these "generals" become susceptible to the HIV virus, which ultimately destroys them. "HIV actually goes straight to the general of the army rather than just to the foot soldiers."

Once the virus has a hold in the body, even when treated with drug therapies, the disease can still progress because partial protein particles of the virus may actually be interacting with non-activated CD4 cells, and activating them and thereby making them more susceptible to HIV.

"But the cell knows it hasn't been turned on through the right series of steps and dies automatically," he says. "So the main reason we see CD4 cells decline over the years is because even HIV proteins are able to turn on CD4 cells improperly, and once an activated cell is turned on improperly, it can either be infected by the virus or it gets destroyed."

Fowke says the sex workers in Kenya who do not get the virus may have immunity because the CD4 regulator cells of their immune systems are not as easily activated and, as a result, less susceptible to infection when exposed to the virus. Fowke calls this "immune quiescence."

"What we're finding is that people who are able to resist infection or are able to control their infection have very, calm, cool, quiet immune systems," he says. "They don't have a lot of activating cells." They still respond to an infection when necessary, but their immune system is very efficient, limiting the number of activated cells targeted by HIV.

In contrast, the group of solvent abusers in Manitoba are likely experiencing a rapid progression of the disease because their immune system is over-activated. Solvents destroy users' mucosal lining, mainly found within the respiratory tract, but also in gut mucosal lining.

Because the mucosal lining serves as a barrier to infection, users may be more prone to illness or exposure to bacteria. As a result, their CD4 cells become highly activated. When they are exposed to HIV through unprotected sex or sharing needles, they are easily infected and the disease progresses very quickly.

"Our immediate focus is looking at solvent abusers in Winnipeg and establishing relationships with that community, but we know that there's solvent abuse in Colombia, India and Kenya amongst very poor people, especially orphans and children, because it's an appetite suppressant," he says. "We predict it will cause huge increases in risk of people getting infections, including HIV."

Fowke says he is optimistic he's on the right track. The evidence gathered so far supports his "immune quiescence" hypothesis, and some day it may lead to a vaccine or a drug that will effectively quiet the immune system response.

But that will be a task easier said than done, he says. The treatment would have to balance between dampening the activation response and still maintaining an immune system that will fight off illness.

"You have to try and modulate it and flip the balance into a more favourable scenario to fight off HIV." The trick is making sure you don't tip the balance too much, he says.

"If you do, you might not get HIV but you'd get all sorts of other kinds of infections, and that would be probably a balance that people wouldn't be willing to go for."

KILLING CANCER

Researchers look for effective ways to eradicate disease

Cancer cells are tough.

So tough, in fact, that's what makes them so deadly. Unlike the body's normal cells that die when they become damaged and stressed, cancer cells have a molecular makeup that allows them to survive.

This hard-to-kill nature is the focus of cancer researcher Dr. Spencer Gibson and his team at the Manitoba Institute of Cell Biology at the Department of Biochemistry and Medical Genetics at University of Manitoba.

"We are trying to understand on a very fundamental level what is the process that makes that cancer cell survive when a normal cell would die," says Gibson, a 2008 Manitoba Research Chair award recipient. "If you understand that, then you can design chemotherapy that can not just induce the death response in these cells, but you can also prevent the survival response in the cancer cells."

And by inhibiting the survival mechanism, once cancer is treated, it won't come back.

Despite all the recent advances in cancer treatments, the perpetual problem with many therapies – in particular, chemotherapies – is they often fail in completely eradicating the disease.

"You can treat the cancer and it will respond to it, but then three months, six months, a year or five years down the road, the cancer returns," says Gibson, who is the Director of Translational Research at the Institute.

Just as bacteria can become resistant to antibiotics after repeated exposure, cancer cells exposed to chemotherapy can become resistant to treatment. Basically, the cancer evolves and becomes increasingly difficult to treat.

But researchers understand that within each cancer cell are mechanisms that enable cancer cells to survive.

"If you can target a therapy with a chemical agent to interfere with those signals that allow them to survive, and then use the chemotherapy that we have today, you will drive those cells into further cell death so they don't come back," he says. "And even if they don't die, you're inhibiting them to a point where they won't be able to come back and repopulate."

So far, the research team has been working with CancerCare Manitoba patients suffering with chronic lymphocytic leukemia – a form of cancer that is notorious for returning after chemotherapy treatment.

"We've gone from the bench to the bedside with the project," he says. Patients were treated with an additional agent called valproic acid (VPA), which has been used to treat depression and epilepsy in the past.

It has turned out to be a potentially effective agent to increase the potency of chemotherapy treatments, he says. "We have seen clinical responses with these people." But patient response to the treatment varies, and the study still needs more patients to understand how the mechanism works. "In other words, we didn't cure these patients yet."

Nonetheless, the study will provide a greater understanding of how to make cancer cells more susceptible to chemotherapy. And learning from patients – the very basis for translational research – is an integral part of the research, he says. "It's from the test tube to the patient and back again, because patients have a lot of information that we can use in our research."

The aim is to get a firmer understanding of the biochemistry of cancer cells and how each patient's cancer is slightly different from the next. Gibson says eventually they will be able to develop cancer treatments that are more effective because they're developed not just based on the cancer's biology but also the patient's biology.

"We're not quite there yet," he says. "But I think that within the next decade, you will see customized therapies toward not just a type of cancer but for the individual."

FLIPPING THE SWITCH

Biochemist aims to starve cancerous blood tumours

If cancer has an on/off switch, Dr. Jeffrey Wigle may have found it.

The biochemist based at the Department of Biochemistry and Medical Genetics, Faculty of Medicine and the St. Boniface Hospital Research Centre is studying how the growth of both blood and lymphatic vessels in the body play a vital role in the spread of cancer.

"Tumours can only grow so much before they have to grow their own blood vessels," says the 2008 Manitoba Health Research Council Chair award recipient. "A new way that we're looking at to stop cancer growth is by blocking blood vessel growth."

Basically, his research team is developing potential treatments that would starve cancerous tumours by cutting off their blood supply.

So far, the team has uncovered a gene in vessel cells – called Meox2. When that gene is switched on, it blocks cell division and growth of new cells required to build new vessels. Wigle says if they can understand the mechanisms that switch the gene on and off, they may be able to stop growth of new vessels in tumours, effectively preventing tumours from growing and thus making chemotherapy and radiation therapy more effective.

And there is more. Wigle's team has discovered a gene in the lymph cells that controls lymphatic vessel growth.

If the cardiovascular system is the body's watering system bringing life-giving oxygen to the cells, the lymphatic system would be its plumbing, removing fluid that leaks out from the blood vessels.

"Our blood vessels are a little bit leaky by design so fluid leaks out from them, and if you had no other way to bring it back, you would get swelling in your limbs," he says. "The lymphatic system takes that fluid and brings it back into the blood system."

But the lymphatic system also has another important role: protecting our body. It's the immune system's superhighway to fight infection. The lymphatic system has lymph nodes throughout the body that produce and store white blood cells to fight infection.

The lymph vessels provide transportation for these disease fighters to attack infections throughout the body. "That's why when you get sick, your lymph nodes get swollen," he says. "It's because the immune system is activated."

But when cancer cells are present, the lymph system can turn out to be the body's worst enemy. "The cancer hijacks the lymphatic vessels to spread through the body," he says.

Due to the lymph vessel's porous nature, cancer cells can enter and travel throughout the body. This is one of the most common ways that cancer metastasizes and spreads throughout the body.

Wigle's research has uncovered a gene in lymphatic cells – called Prox1 – that controls lymph growth, and, eventually, research may lead to a way to use that gene to prevent a tumour from spreading via the lymphatic system.

"There isn't a magic bullet that will treat all cancers," he says. "This is another way to help fight it. If we can keep the cancer from growing and spreading, it will make it easier to kill."

Wigle says cancer treatments based on his research are at least five to 10 years away, but his research could also lead to treatments for other diseases and disorders.

In the future, they may be able to stimulate blood vessel growth to help restore blood flow after a heart attack, and they may also be able to prompt lymphatic cell growth in people who are born without a developed lymphatic system.

"We're looking at certain situations where we want to stimulate vessel growth, and in others, we want to block that growth."

HEALING HEARTS &MINDS

A Winnipeg psychiatrist examines the roots of mental illness for two different yet equally high-risk groups: Aboriginal youth and soldiers returning from Afghanistan

It could be called psychiatry's million-dollar question.

Why do some people exposed to atrocities of war, child abuse, neglect, poverty or physical abuse carry on healthy and productive lives relatively free of depression, anxiety and other mental disorders, when others cannot?

It's a question mental health experts have yet to definitively answer, says Dr. Jitender Sareen, a psychiatrist and professor at the University of Manitoba's Faculty of Medicine.

But it's one that lies at the very heart of understanding the causes of mental illness, he says. And it's why Sareen has been working for more than a decade with high-risk groups for mental illness.

"The vast majority of the people who go through a traumatic experience do fine," says Sareen, a leading specialist in studying anxiety disorders in Canada. And yet many others do suffer. They struggle with depression, sleeplessness, anxiety, drug and alcohol problems, and in extreme cases, they become suicidal.

Sareen has worked with two groups of Canadians in particular who are at higher risk for mental illness than the general population to better understand mental disorders and ultimately develop more effective care.

One area of study is Canadian soldiers returning from peacekeeping missions and, more recently, combat in Afghanistan – some of whom may suffer from post-traumatic stress disorder (PTSD), anxiety or depression.

"We've been trying to understand what kind of level of need there is for mental health services in the Canadian military, and what are risk

factors for mental health problems in the military," says Sareen, recipient of a 2009 Manitoba Research Chair award.

The other group is First Nations youth, both here in Winnipeg and on rural reserves, who have suicide rates three times the national average. "It's an epidemic in some communities," he says.

In both cases, Sareen says his research aims to better understand past and present stresses experienced by these groups, so mental health professionals can provide better care.

Yet each study takes a different approach. "First Nations people have felt researched and researched, and they don't want another survey or another study," he says. "So the difference between our study and previous work is that it's an ongoing partnership."

With help from elders and other members of First Nations communities, they have developed programming aimed at suicide prevention and community building.

"A youth who is suicidal on a reserve is not going to come to a psychiatrist in Winnipeg and say, 'I'm suicidal,'" he says. "They're going to go to their friends, their cousins, and their uncle."

The prevention initiative is a two-day seminar for youth and their families on reserves. It aims to help them identify suicidal behaviour in friends and family, and provide them with techniques to get their loved ones the help they need.

"The second thing that is repeatedly brought up by First Nations communities is that there is a loss of connection and identity of the youth to their elders and culture," Sareen says.

Referred to as anomie, this loss of connection to others is a risk factor for suicide. "So the idea was to create a two-day resilience retreat so youth and elders can come together and have group discussions," he says.

Initial pilot studies have shown both programs are successful early on, but further study will provide a clearer picture, which many members of First Nations communities want before putting the two-day prevention course in place.

"One of the challenges with awareness and prevention training in the communities is that there is concern among some community members that talking about suicide might lead to more people thinking about suicide and making a suicide attempt."

But Sareen says prevention programs have been shown to have positive effects for other groups in society, and he expects the pilot will have the same results. "We expect the prevention and awareness program will do better in directly reducing incidence of suicide than the resilience retreat, while the people who got the resilience retreat might actually do better as far as their own quality of life," he says.

And that can be equally as important over the long run, he says.

In the other study, Sareen is working with soldiers returning from Afghanistan to determine their need for mental health services. He says the need is there. They're just not sure to what extent and how best to serve that need.

"We've done a number of studies and found that combat and witnessing atrocities are a risk for mental health problems," he says.

The most widely talked about problem is PTSD, caused by exposure to traumatic events. Its sufferers experience a range of mental health issues, including fatigue, irritability, depression, suicidal thoughts, anger, anxiety and drug abuse. Sareen has been trying to determine what – if any – additional factors may put some soldiers at higher risk.

"Right now, we're looking at childhood adversity and how that interacts with deployment stress and risk for mental health problems," he says. "We know that one of the most important risk factors for adult mental health problems is a history of child abuse, so we're trying to disentangle what's the impact of child abuse, and what's the impact of combat stress on the mental health of soldiers."

So far, studies in this regard on Canadian soldiers have been inconclusive, but Sareen says he expects they'll have a clearer picture as research with returning soldiers unfolds over the next two years.

At the very least, he says it could lead to increased funding and expansion of Canada's network of Operational Stress Injury Clinics, which are already operating in locations across the country. "These are places where they can meet with psychiatrists, psychologists and nurses who provide and facilitate treatment if they experience mental health problems after returning from deployment," he says, adding he works weekly with soldiers at a clinic in Winnipeg at the Deer Lodge Centre.

THROUGH THE EYES OF A CHILD

Researcher gains important insights by talking to children about their care

Dr. Roberta Woodgate will be a busy researcher over the next five years. That's what happens when you end up juggling many studies all essentially asking the same question.

"What is it like as a child or youth to have an illness and live day-to-day?" asks the Professor of Nursing at the University of Manitoba.

While it sounds simple enough, Woodgate says the reality is that medicine's understanding of a child's experience of health care is an often ignored area of study.

But the goal of her work is not just to improve care for children. It's also to help parents and caregivers get the support they need to provide the care they want for their children.

To accomplish this wide-ranging task, Woodgate, the recipient of a 2010 Manitoba Research Chair award, has four studies on the go aimed at improving child and youth health care.

She is presently surveying families caring for children with illness or disability to find out what challenges they face in providing ongoing care and how it affects their ability to participate in daily life.

"Most children with complex needs are cared for in the home now, not in a hospital or institution, which is a good thing. But these families have a lot on their plate, so we want to help them to find the best way to care for their child."

What research has uncovered so far is that current support within the system falls short.

"I don't think the general public understands – unless they have a relative or friend going through the same situation – what these families really give up," she says. The complex care that the child requires affects the participation of all family members. The decision to choose to participate becomes much more dependent upon accessibility, and the availability of resources and the family's ability to harness them.

Woodgate says the best comparison to provide people with some understanding is caring for a newborn. "Only with a child with a severe disability, it's all their life. And there's that fear when the child turns 18: What happens to their child then?"

A related study she's working on looks more specifically at the needs of First Nations families caring for children with disabilities. "A severe disability in itself for a child is quite problematic. But for First Nations families, it's often one of many issues," she says. "You have housing issues, for instance, where the child is in a wheelchair but because the house is small, they can't use the chair in certain sections of the house."

While much of the research in both studies involves families, Woodgate's main focus is to discuss with children their experience with care as they grow into adulthood.

In fact, another study she is leading looks specifically at teenagers with chronic illness and to what degree they should be involved in decisions over their own care.

"One of the reasons we're conducting this is because teenagers are transitioning into adulthood, we have to prepare them to make decisions for themselves when they go into adult care," she says. Pediatric and adult medicine are two different worlds. "If you're not prepared for adult care, it can have an impact on your health."

Ultimately, Woodgate says these studies will lead to better care and new programs to help children and their families deal with the psychological, emotional and physical toll of illness.

But her last study is actually piloting a new program for sick children to help them deal with the psychological and emotional challenges of living with an illness. "It's basically a video game but it's a psychosocial intervention that I'm going to be testing out on children with cancer."

Here's how the game works. Children play in an online world with other children and participate in a various activities, creating music, writing stories and drawing pictures, to name a few. "It helps children deal with the different ways they feel."

If the pilot is successful, Woodgate says the project will likely be expanded to help children with other illnesses. Not only will that help children, it will also offer health-care providers a unique view into a child's world. And that, in turn, will improve care in the long run.

"I think there's a perception that until you become an adult, you don't know enough to contribute," says Woodgate. "But I think we have a lot to learn from children and youth."

A BREATH OF FRESH AIR

Asthma affects millions of Canadians, yet the only existing medication fails to help its worst sufferers. One U of M researcher aims to fix that

For millions of Canadians, catching their breath is not to be taken for granted.

They have asthma, a chronic inflammation of the airways that can, in extreme cases, make it impossible to breathe and results in a trip to the emergency room.

In fact, it's one of the biggest costs to the health-care system, says Dr. Abdelilah Soussi Gounni, a Professor of Immunology at the University of Manitoba's Faculty of Medicine.

Asthma costs the health-care system $600 million every year, according to the Canadian Lung Association. It's estimated that more than eight per cent of adults suffer from asthma, and the prevalence of the disease has been increasing over many decades, Gounni says.

The basic causes of the disease are known: it's the result of chronic inflammation of the lung's airway tissue from allergens. But doctors and other health specialists who study and treat the disease still do not know why people develop asthma and, even more importantly, why some people develop severe asthma.

And it's these severe sufferers who are most at risk of visits to the emergency department, sometimes barely able to breathe. Even more problematic is the fact that the treatment doesn't work that well for severe sufferers.

"The drugs which are available right are not very effective for dealing with the severe form of asthma," says Gounni, who received a 2009 Manitoba Research Chair Award.

Gounni says the current treatment involves corticosteroids that dampen the inflammation in the lung's airway cells. This stops the airway tissue from constricting and limiting the lung's ability to take in air to oxygenate the blood. The steroids have a mild effect in some cases and none at all in others.

But Gounni's research is designed to find asthma's root causes. In pursuing this line of inquiry, Gounni hopes his work and that of his team will lead to the development of medications that would target those root causes instead of simply treating the symptoms, as the current treatments do with varying results of effectiveness.

So far, the research has focused on two molecules present in high levels in people suffering from the disease. These molecules likely cause sufferers' immune systems to mount a higher inflammatory response to allergens. This is important because the more irritated their airways get, the more damage is done to the airway cells by the immune response. And if the inflammatory response continues untreated, eventually it causes scarring. "That's what we call airway remodelling," he says. "Once the scar tissue is established, it is very difficult to get rid of."

Gounni says individuals with severe asthma all show signs of remodelling. At this point, he's not sure when this remodelling occurs. But Gounni says children have higher rates of asthma than adults, meaning some outgrow the disease while others don't. This is called the "allergic march."

"There is a chance that after your immune system matures, you walk out of the disease," he says, adding that children have higher rates because their immune systems are still sensitive while developing.

Those who do remain asthmatic likely have developed the scar tissue during childhood for one reason or another. Gounni hypothesizes that high levels of the two suspect molecules play a role.

He says he hopes to test his molecular theory soon on lab mice who have had one of the molecules removed genetically from their cells. If his team finds these mice do not develop asthma when exposed to high levels of allergens, Gounni says they may have found one of the disease's possible root causes – and that may eventually lead to better treatments.

"With the lab-work, we're trying to understand how those mechanisms work, because if we can do that, we will be able to design better drugs that provide effective relief and avoid trips to emergency."

UNRAVELLING THE SECRETS OF SEPTIC SHOCK

Research team tackles leading cause of ICU deaths

Most people have heard of toxic shock, a catastrophic condition in which a blood infection causes the body's organs to shut down.

Also referred to as septic shock, it's one of the leading causes of deaths in our intensive care units, says Dr. Jude Uzonna, Associate Professor of Immunology at the University of Manitoba's Faculty of Medicine. In fact, during the 2009 H1N1 pandemic, many of the fatalities were actually caused by septic shock.

It's long been known that septic shock results from a bacterial blood infection that runs rampant when the body's immune system fails to eliminate the invader from the blood.

But Uzonna, a 2010 Manitoba Research Chair award recipient, and his team of researchers are studying a hypothesis that suggests the cause of septic shock is actually the other way around.

"Our working model is that septic shock results from excessive and unregulated immune response to blood pathogen – like using a sledge hammer to kill a fly where you also cause collateral tissue damage in the process," Uzonna says.

Central to their focus is the role of a special type of immune cells known as regulatory T-cells that control the magnitude of how the body's immune system fights infections. "Their job is to knock other immune cells on the head and thereby dampen or prevent them from going haywire and initiating excessive and deleterious immune response," he says.

The results of the research so far have supported their theory. Tests have demonstrated that mice die very quickly when these regulatory cells are taken away from their system because they lose

the ability to regulate their immune response even when injected with bacterial products that normally do them no harm.

Uzonna says they're still trying to understand whether it's a lack of regulatory T-cells that leads to septic shock or whether they simply stop working. The results of the research could lead to better care for patients in ICUs, and even help reduce deaths in future influenza pandemics.

In the future, Uzonna says treatment for sepsis/septic shock could be similar to platelet transfusion for patients with clotting disorders. "We could forsee a situation where a patient with septicaemia receives a transfusion of regulatory T-cells," he says.

For Uzonna, however, his research at the Department of Immunology doesn't just involve understanding toxic shock. He is also leading research into treatment for parasitic diseases that affect millions of people in some of the world's poorest regions.

Leishmaniasis is a single-celled organism that is spread by sand flies found in South America, Africa, the Middle East and Asia, and infects more than two million people a year. "But I can tell you this is a gross understatement because the disease affects very poor countries and very poor people," he says. "Most people, when they get sick, they don't go to the hospital."

Depending on the species of the parasite, leishmaniasis can cause death or leave its hosts with disfiguring scars. While medication to treat the disease – also known as dum-dum fever – has been available since the 1940s, it has not been effective. Not only is it expensive, but its toxic nature can lead to fatalities in some instances.

Uzonna and his research team have been studying how to genetically manipulate the organism. They are developing an attenuated parasite that doesn't cause illness but can still infect people. Much like a vaccine containing a virus that doesn't cause illness, the attenuated parasite would prime a person's immune system to quickly recognize and destroy future infections by Leishmania parasites. So far, he has signed a memorandum of understanding to test his vaccine on monkeys, who also are also susceptible to infection, at the Institute for Primate Research in Nairobi, Kenya.

Uzonna, who studied veterinary medicine in his home country of Nigeria, is also involved in researching a vaccine for another parasite, only this time to save cattle. In fact, this parasitic infection, which is widespread in his homeland, is the reason he became involved in parasitic and immunologic research in the first place.

African trypanosomiasis is commonly known as sleeping sickness. It's a curable disease that affects the brains of those it infects, making them sleepy "zombies" and is fatal if left untreated.

Uzonna wants to uncover why this parasite is so virulent in exotic cattle imported to Africa.

"It's the number one problem that prevents rearing cattle in a lot of African countries because the hybrid European and North American breeds of cattle do not survive in those areas," he says. The indigenous cattle are too small to be commercially viable. "Solving the problem in cattle will not solve the problem of human disease, but it will have a huge economic impact." And that, in turn, will help improve living conditions among some of the poorest regions of the world, he says.