

Genome

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Issue 17

May 05

DUKE Institute For Genome Sciences & Policy

Seize the Day

Goldstein and McNamara Combine Forces Against Epilepsy

It's like an all-star history team: Socrates, Alexander the Great, Joan of Arc, Napoleon, Lord Byron, Pope Pius IX, Dostoyevsky, Alfred Nobel, and van Gogh, just to name one imaginary starting line-up. What did they have in common? Epilepsy. According to the Epilepsy Foundation, 2.5 million Americans are afflicted with epilepsy, and ten times that number will experience a seizure at some point during their lives.

Despite the relatively high incidence of epilepsy, our understanding of what causes the electrical signals in the brain to become disrupted and provoke seizures remains incomplete. We are also unable to prevent it: current therapies treat the symptoms – not the causes – of epilepsy, much as insulin treats the symptoms of diabetes. What's more, 30 percent of patients don't respond to anti-seizure medication. Recent and ongoing work at Duke suggests that those treatment-resistant patients may have cause for hope.

Epilepsy: Of Genes and Drugs

Dozens of studies have demonstrated that genes are involved in epilepsy. However, as with other complex diseases like cancer and heart disease, single-gene versions of epilepsy account for only a tiny fraction of the disease burden. From a genome scientist's perspective, the rest is kind of a mess.

"That's exactly how I'd characterize it," says David Goldstein, Director of the IGSP's Center for Population Genomics & Pharmacogenetics. "But I would make one additional point: the nice thing about studying epilepsy is that you



have access to the relevant tissue."

Goldstein is referring to patients whose epilepsy does not respond to medication and must be surgically treated. In such cases (~ 30 per year treated at Duke University Hospital), the brain tissue where the seizures originate is removed. "What that allows us to do is look at those cells and see what's different about them. That might help us to understand what's going on."

Building up large numbers of tissue samples for study takes time. For now, Goldstein is using a pharmacogenetic approach to get at the molecular basis of epilepsy and, he hopes, to influence the way anti-epileptic drugs are used.

Epilepsy (continued on pg 3) >



Message from the Director

Quick. Where's your genome from? Sounds like an easy enough question, and most of us wouldn't hesitate before answering. England, Scotland, Spain, Senegal, China. But do we really know?

This is the premise behind the National Geographic Society's recently launched Genographic Project, which aims to amass the world's largest collection of DNA samples in order to map how human migration led to the population of the planet.

As a genome scientist interested in the evolutionary and migratory history of my species, it's hard not to get excited by the prospect of the Genographic Project and what it might tell us about our genomes and ourselves. Part of the appeal is the scale of the project. For population studies of any kind, bigger is almost always better: the more data one can analyze, the more likely one's conclusions are valid. The thought of collecting a hundred thousand or more genomic snapshots from the remotest corners of the globe to reconstruct human population history is a tantalizing one that fits well with some of the aims of the IGSP, especially our Center for Population Genomics and Pharmacogenetics.

However, there are at least two concerns about the Genographic Project. The first is the question of fair access. In the early 1990s, an initiative known as the Human Genome Diversity Project ran into trouble when it set out to sample a variety of indigenous populations for clinical and evolutionary research purposes and failed to make its case to many of those populations it wished to study. The result: the HGDP was delayed for about 10 years.

The Genographic organizers – some of whom were integrally involved in the HGDP – say they've learned their lesson. They say they will continue to seek

advice and counsel from indigenous leaders as they proceed. They say their project will not involve medical research of any kind and will therefore be free from disputes over commercialization. They say they will give back to the communities they study through educational activities and cultural preservation projects. But it is notable that the National Geographic Society's press announcement was followed closely by one from a group called the Indigenous Peoples Council on Biocolonialism that sharply opposed the Genographic Project as an "unwanted intrusion" and just another form of biopiracy.

Not only is the Genographic Project turning a blind eye to the medical implications of its work, it seems also to be ignoring the other elephant in the room: race. You've read about the issues in these pages before: is race nothing but a social construct or does it have a biological basis? Can populations be distinguished on the basis of race? Should race be considered when prescribing medications? These issues are central to many of us within the IGSP. Not so the Genographic Project: "There is no desire to look at race," according to one of the project leaders. For its part, the project will only tell participants what migratory routes their ancestors took and which "branch" of the human family tree they belong to.

Genomes gather traces of their travels through history and have stories to tell, whether we want to hear them or not. In many populations, admixture between genomes is increasingly evident, whether reflecting the African diaspora, wars and invasions by hostile nations, or simply chosen relationships between peoples of different ethnic or racial origins. The concept of mixed genomes may cause confusion and much angst among participants. What will it mean to be told that 20 percent of your genome comes from a different origin than you expected? That'll make for some interesting discussions around the dinner table or around the classroom...

Is our current understanding of genetic diversity up to the task? Perhaps, but perhaps not. Our knowledge of the relative minority of genome variants that are specific for certain geographic origins is incomplete at the moment, based largely on studies of a limited number of "parent" populations – western European, west African, east Asian and indigenous American. The public may not settle for a vague answer, not when the Genographic Project is charging \$100 to participate.

There is evidently much more to learn about genome variation, both shared among and private to particular population groups. One hopes that the Genographic Project, like other large-scale studies of different populations, will contribute to that knowledge and provide useful databases for study of human history and human health. The story of human migration and the circuitous paths our genomes have taken can't help but be a fascinating one. Here's hoping it can be told in ways that are inclusive, equitable and enlightening to all. ▶

Huntington F. Willard, Director

Epilepsy (continued)

In a recent paper published in *The Proceedings of the National Academy of Sciences*, Goldstein and his former colleagues at University College in London identified variants in two genes that are strongly associated with the maximum dose needed to control seizures with two common medications, carbamazepine and phenytoin.

How did he decide which genes to check for such an association? “We looked at the very first genes you would look at if you were starting a pharmacogenetics project: the genes encoding the primary drug-metabolizing enzymes, the ones encoding the transporters that keep the drug out of the brain, and the ones encoding the targets that the drug physically interacts with.”

For Goldstein, the take-home message is to not overlook the “obvious genes,” many of which will harbor variants in them that may be clinically meaningful. And because the majority of prescription drugs’ modes of action are well understood or at least strongly suspected by pharmacologists, he expects that the approach he took with carbamazepine and phenytoin will be

“There’s a minority of drugs where we just don’t know how they work,” Goldstein says. “But in most cases we have a very good idea where to start looking.”

broadly applicable to other conditions and other drugs. “There’s a minority of drugs where we just don’t know how they work,” Goldstein says. “But in most cases we have a very good idea where to start looking.”

A Real Knockout

Goldstein’s work on epilepsy finds him in good company. Jim McNamara, the Carl R. Deane Professor and Chair of Neurobiology, has been studying epilepsy for over 30 years. In the 1990s, his lab discovered genes that, when knocked out in mice, either caused epilepsy or made the animals more seizure-prone. Last year, he was somewhat taken aback to find a gene called *trkB* (pronounced “track B”) that, when knocked out, actually prevents mice from becoming epileptic.

“Epileptogenesis is the process by which a normal brain becomes epileptic,” he says. “And this is the only gene that’s been demonstrated to be required for epileptogenesis in a mouse model. I figured that there would always be adaptive mechanisms in the mammalian brain that would circumvent epileptogenesis – you might slow the process but you’d never be able to stop it altogether. [The existence of] *trkB* really surprised me.”

McNamara is hopeful that interfering with human *trkB* might offer a way of preventing epileptogenesis, be it in patients predisposed to sporadic epilepsies or in those with familial varieties. But first, he will need to determine whether disruption of *trkB* can prevent it in mice known to be epileptic, i.e., mice carrying mutations in a gene that has made them susceptible to spontaneous seizures. “If so, I think that would give us a good sense of the importance of the *trkB* pathway.”

Synergy and Overlap

McNamara’s interests are not limited to mouse models. He has been collecting material on human epilepsy families for some time and is looking forward to pooling his resources with Goldstein’s. “We have [identified a genetic] locus and now we have to track down the genes within that locus. We’re going to do this by analyzing additional families. It’s a nice convergence because David was interested in this independently [before coming to Duke].”

Goldstein sees the collaboration on family studies with McNamara dovetailing seamlessly with his own work in pharmacogenetics. “It makes perfect sense to do those two things together because there’s a great deal of overlap in the genes you test. If you think about it, one of the best sets of candidate genes for predisposition in epilepsy are precisely those genes that drugs act on and that [reside in] the pathways they affect. You can easily imagine that dysfunction in those pathways is one of the things that contributes to epilepsy.” ▶



Neurobiology Chairman Jim McNamara in his lab

Let's Get Small

Duke Scientists Use DNA in Computing and More

In 1994, Associate Research Professor of Computer Science Thom LaBean was a young postdoc in Biochemistry at Duke when a paper was published that changed the trajectory of his career. The paper, by University of Southern California computer scientist Len Adleman, described a way in which DNA could be used as a calculator to solve the classic “Hamiltonian path” problem that involves finding whether a path exists that visits a number of stops (e.g., cities) exactly once. As the number of cities increases to as few as seven, so does the number of possible paths; even a supercomputer can get stumped as to whether such a path truly exists.

“I was at a meeting when the paper came out, and it was all the buzz,” remembers LaBean.

Not long after that, Professor of Computer Science John Reif approached LaBean and wanted to know more about DNA – he had been inspired by the Adleman paper, too. Reif soon lured LaBean to Computer Science. The seeds of biomolecular computing at Duke had been planted.

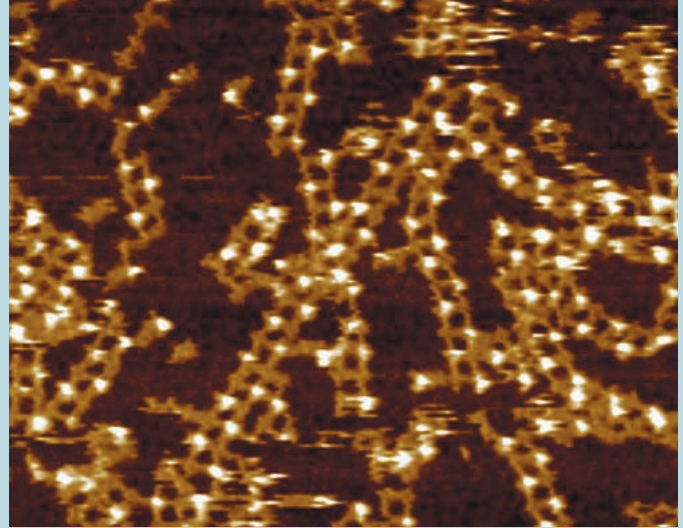
Honey, I Shrunk the Computer

DNA's ability to propagate genetic information faithfully is well known. But what makes it so amenable to performing calculations? First and foremost is that DNA behaves according to a well-understood set of rules. The four distinct bases in DNA – A, G, T and C – pair up in a predictable fashion: A with T, and G with C. The intrinsic tendency for DNA to pair or “hybridize” with its partner has led to the process of DNA computing to be referred to as “self-assembly.” DNA's adherence to predictable rules means that it can be programmed to solve complex problems like Adleman's path problem.

DNA has other potential advantages: it is far more compact than even the tiniest microprocessor. A single gram of dehydrated DNA can encode as much as 10^{21} bits of information. Moreover, DNA computers can operate in “massive parallel” and as much as a million times faster than conventional computers.

So it's just a matter of time before we trade in our Macs and Dells in favor of tiny DNA-based machines? Hardly, says Reif. “The difficulty in solving hard computational problems [using DNA] is that they don't scale.” In the case of Adleman's Hamiltonian path problem, for example, a tiny drop of DNA produced trillions of answers as to what might be the likeliest route through a large number of cities. Unfortunately, most of them were wrong. To ferret out the right answer meant performing a series of labor-intensive follow-on lab steps.

To help overcome the limitations of the original self-assembly methods, LaBean, Reif and their colleagues have begun to use and develop “DNA tiles,” an approach pioneered by Erik Winfree at Caltech. DNA tiles are akin to dominoes 15 nanometers (billionths of a meter) long: they are essentially nanoscale building blocks of DNA that can store data and be programmed to perform mathematical operations depending on how they fit together.



Microscopic image of DNA nanostructure resembling a railroad track. The nanotrack (amber) self-assembles from synthetic DNA, then binds and organizes protein molecules (white dots). Thom LaBean and colleagues are working to replace the proteins in this system with electrically active materials for use in nanoelectronics.

Tiny House Calls?

Recently, LaBean and colleagues have used the tiles to make a computational lattice that counts in binary. And in a recent paper in *Science*, they demonstrated how a more complex tile could be used in a programmable self-assembly of protein arrays. For genome and proteome scientists, there are clear implications for the ways in which protein-laced tiles might assist in the work they do, whether it's experimental or in the realm of clinical diagnostics. “Those proteins could be designed to be detectors,” says Reif. “They could detect distinct molecules and you could have a response programmed into these types of devices.”

Reif goes on to describe work from Israel's Ehud Shapiro, whose goal is “a doctor in a cell.” In one scenario, a DNA computer is deployed in a cell, looks for sequences associated with cancer and then releases a drug if it finds them.

Another possibility is to use DNA computing to miniaturize and streamline lab protocols. “There could be new protocols in biochemistry for doing standard things,” speculates Reif, “but in ways that are surprisingly more self-contained.”

Reif is quick to caution that while he is enthusiastic, all of this will take time. “I tend to be fairly realistic about the expectations. Molecular electronics for biological applications is probably 10 years down the line.”

Nanoscience Goes Macro on Campus

However far biomolecular computing applications are from the marketplace, the science has established a clear foothold at Duke. As one example, LaBean points to the soon-to-be opened ‘clean room’ in CIEMAS where a special bay will be available for DNA nanostructure work. And in an effort to bolster training on campus, Reif has developed a graduate certificate program in nanoscience involving 13 departments; it was approved a year ago and is now matriculating students.

“I feel like we're building a lot of bridges among different departments and schools,” says LaBean. “A bunch of little pieces are coming together. This might be the best way for it to happen: it's self-assembly.” ▶

Against Autonomy

A Philosopher Considers an Emerging Problem Raised by Genomics

As a philosopher of biology, Alex Rosenberg notes that one of the principles that have been frequently challenged in the history of genetics and genomics is individual autonomy or self-determination. Rosenberg, the R. Taylor Cole Professor of Philosophy, cites the state-mandated eugenics movement of the 20th century as one telling example where autonomy was subverted with dire consequences. But he says that autonomy, too, can pose questions in a genomic age. “In most cases, the morally central idea has been to preserve individual autonomy whenever it has come under threat as it did during the time of eugenics.”

What Rosenberg wants to know is: if people are autonomous, what will they do with their genomic information? What will be the worst-case consequences of their choices among the startling new options genomics puts at their disposal? And how should we design our institutions to protect ourselves?

Cover Me

In one case, Rosenberg sees autonomy as leading to what he calls “the insurance problem.” He wonders how private insurers can equitably cover people known to be at risk for costly, life-threatening genetic diseases. From Rosenberg’s point of view, if we insist on privacy of our genomic information above all else, insurance will eventually become unaffordable to all but a select, wealthy few.

“Increasingly, people will have an incentive to find out about their own genetic fate,” he says. “Those who find they carry an increased likelihood of severe health risk in the future will secure extra insurance to pay for the costs associated with that. In the long run, there are going to be financial incentives for insurance companies to exclude people on the basis of their increased genomic vulnerabilities. If they can’t, the price of health care will increase for everyone.”

deCODING the Future?

But, Rosenberg imagines, if individual autonomy were not the overriding concern, events might play out differently. If citizens could be assured that they would not be discriminated against on the basis of their genomes, and if the social stigma attached to genetic disease could be lessened further, he thinks it might be possible to convince people that it would be in their own best interests to relinquish their private genomic information for the greater – and more affordable – public good. The consolidation of population-wide genomic data into a public database has enormous potential, says Rosenberg.

“The pooling of what is now private genetic information into large bodies of data could enable us to expedite the discovery of say, pharmacogenetic relationships between drugs and genes. We might reduce the cost of delivery of drugs to some patients with certain genetic backgrounds for whom these drugs don’t work as well, or for whom other drugs work better.”

As a model, Rosenberg points to the Icelandic firm deCODE Genetics, which has convinced most of the population of Iceland to donate its DNA for gene and drug discovery purposes. In return, the company has agreed to provide any commercial drug or gene-based diagnostic test stemming from its research to all Icelanders free of charge. Citizens may also opt out of the database. “DeCODE is securing valuable genomic information with the cooperation of the citizens of Iceland,” says Rosenberg.

A Call for Collective Action

DeCODE has succeeded, he says, because it has overcome what he calls “the collective action problem” resulting from the demand for privacy of genomic information. “The problem is that everybody has an incentive to hide their own private genetic information, while encouraging everybody



Alex Rosenberg

else to give their genetic information to the medical establishment so that each individual can benefit.”

And what of the discrimination and stigma issues? “In the long run, we will address them,” Rosenberg says. “Discrimination against gays and discrimination on the basis of race certainly haven’t been abolished, but they’ve been mitigated. If we could similarly mitigate discrimination against genetic proclivities and the stigma attached to them, then people would be more willing to make their genomic information public in a way that’s beneficial to everybody.”

Raising the Stakes

At one level, Rosenberg sees the privacy issue as just another version of the old philosophical conflict between individual rights (autonomy) and the collective good (beneficence). In the past, he says, these conflicts have almost always been decided in favor of autonomy. But in this case, he believes we choose autonomy at our peril.

“What genomics does is raise the stakes for individual and collective human welfare. The improvement in genetic endowments it promises is so great that individuals will certainly exploit current commitments to personal autonomy in their attempt to secure these benefits for themselves, no matter how great the cost for the community.” ▶

Back to the Mines

Haystead Scours Proteome for Better Malaria Drugs

It's hard to imagine that a single-celled parasite transmitted by mosquitoes could cause so much trouble. But what *Plasmodium falciparum*, the causative agent in most cases of malaria, has managed to do to the human population is undeniably horrific: 500 million infections and as many as three million deaths each year, most in sub-Saharan African children. And things are getting worse. Why? There are many reasons – lack of money, market forces, politics, logistics. According to a UN representative, even getting simple solutions such as mosquito netting to at-risk populations has proved to be a monumental task.

But another major reason for malaria's toll is the declining effectiveness of many antimalarial drugs. Just as first- and second-generation antibiotics have lost much of their ability to fight bacterial infections due to resistance, so too have classic antimalarials become increasingly ineffective. As summarized in a recent review in the *American Journal of Tropical Medicine & Hygiene*, in some regions of the world, older drugs such as chloroquine are now essentially useless. Moreover, newer therapies tend to be more expensive, have more difficult compliance regimens and cause more side effects.

Digging into the Proteome

Associate Professor of Pharmacology & Cancer Biology Timothy Haystead believes proteomics may offer a way to identify drugs that, if they don't overcome resistance altogether, can at least buy another 20 years of effective treatment. His lab's approach, called "proteome mining," is to look at a subset of all human proteins (the proteome) and try to find those targets that would be most apt to bind small-molecule drugs and therefore likely to respond to new therapies.



In setting off on their fishing expedition several years ago, Haystead's group opted to use adenosine triphosphate (ATP) as bait, as ATP is one of the most frequently occurring proteins in the proteome and is capable of binding small, drug-like molecules. "If you immobilize ATP in a specific orientation," says Haystead, "then you can capture all of the enzymes that bind it."

Since *Plasmodia* infect the blood, in this case the "pond" to be fished was the blood proteome.

Whatever proteins the researchers were able to reel in with their ATP-laden matrix could then be screened against known malarial drugs. If those known drugs had an affinity for ATP-binding enzymes in the blood proteome, Haystead and company would be able to isolate those enzymes.

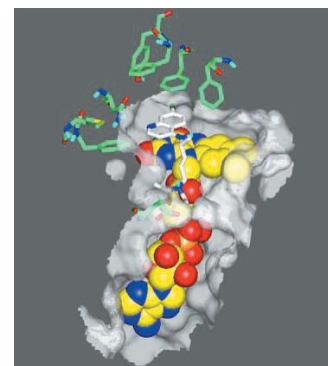
QR2: Super Target

Haystead says the most intriguing target revealed by the proteome mining experiments was the enzyme quinone oxidoreductase 2 (QR2), which is present even in simple bacteria and chemically reduces a variety of compounds known as quinones. Subsequent work with Assistant Professor of Biochemistry Johannes Rudolph showed that existing malaria drugs such as chloroquine act by inhibiting QR2. In Haystead's words, chloroquine and the enzyme fit together "like hand in glove."

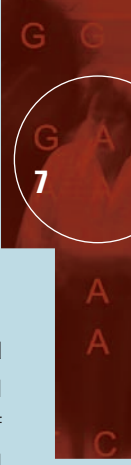
Chloroquine worked in humans for 40-odd years by targeting QR2. Can the next generation of antimalarials work by targeting the same enzyme? Haystead thinks so. A series of QR2 inhibitors his lab has identified in collaboration with Rudolph are strongly antimalarial and show no toxicity in mice. And transgenic mice which have had their QR2 genes knocked out show no phenotypes whatsoever. Haystead finds that particularly encouraging.

"If you look at the way resistance to malaria happens in humans, each population develops its own means of resistance, though [we know that] it always involves mutations in a human enzyme. QR2 would be a great target for an antimalarial drug because we know [from the knockout mice] that we can live without it."

The next step will be to try to infect the QR2-knockout mice with malaria. Haystead's group is collaborating with researchers at the London School of



The antimalarial drug chloroquine (color) bound to the active site of the human enzyme quinone oxidoreductase 2 (QR2). By demonstrating that chloroquine acts via QR2, Tim Haystead and colleagues may have made it possible to identify more potent antimalarial agents.



Hygiene & Tropical Medicine to do just that. If the proteome mining data are correct, a mouse that lacks a functioning QR2 enzyme should not be able to get malaria. "If we can infect those mice, then we're just plain wrong," says Haystead. "But if we can prove that the mice cannot be infected, the next thing is to look for human populations that can't be infected, either." Chances are such populations carry a defective version of QR2 that renders them essentially immune to malaria.

In evolutionary terms, Haystead believes the QR2 gene is important. He notes that two groups have identified variants in the gene that appear to be associated with the development of the blood disorder agranulocytosis (suppression of white blood cells) in schizophrenics of Mediterranean or African origin being treated with the antipsychotic medication clozapine. "It may be that those patients' QR2 genotypes give them a selective advantage against malaria," speculates Haystead. He hopes to find a collaborator in Psychiatry who can assist in testing that hypothesis.

The Case for Drugs

Haystead realizes that malaria is unlikely to attract much interest from the pharmaceutical industry, given that most of the patient population is poor and from developing countries. But that fact notwithstanding, he remains convinced that developing better drugs will be a necessity in the battle against the disease. Why not a vaccine? A malaria vaccine "seems like a crazy idea to me," says Haystead. He points to recent data from the *P. falciparum* genome and notes that the only place the parasite has the ability to change its protein profile is in immune evasion. Thus, he contends, any vaccine program would quickly be overwhelmed by resistance.

"You'd have to have seasonal malaria vaccines. You might have 40 strains going at once. One mutation and suddenly your antibody's no good – that's all it would take. You're telling me you can beat that? I don't think you can."

He concedes that drugs aren't a perfect solution, but believes that by targeting human proteins (like QR2) rather than parasitic ones, it's possible to stave off resistance and develop products that are suited to use in the remote parts of the developing world. "The myth about drugs is that they're too expensive," he says. "I don't think that's true. If you can come up with a good target, and develop something that's potent and long-acting, you can make a tanker full of it. Then you shove it out the back of an airplane and people only have to take it once."

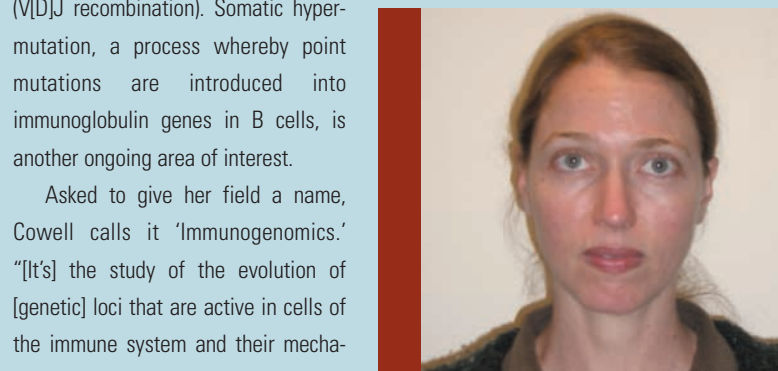
If Haystead is right, it may be that malaria's alarming trend line can finally be reversed.

Further Reading:

Graves PR, Haystead TA. A functional proteomics approach to signal transduction. *Recent Prog Horm Res.* 2003 58:1-24.

Faculty Profile: Lindsay Cowell, PhD

Assistant Professor of Biostatistics & Bioinformatics Lindsay Cowell joined the Duke faculty and the IGSP Center for Bioinformatics & Computational Biology in the fall of 2003, following a postdoctoral stint in Professor of Immunology Garnett Kelsoe's lab. While there, she developed a statistical model for understanding the combinatorial process by which immune system specificity is generated from a limited amount of genetic material (V(D)J recombination). Somatic hypermutation, a process whereby point mutations are introduced into immunoglobulin genes in B cells, is another ongoing area of interest.



Asked to give her field a name, Cowell calls it 'Immunogenomics.' "[It's] the study of the evolution of [genetic] loci that are active in cells of the immune system and their mechanisms of genetic diversification."

Recently, Cowell has been studying the ways in which genetic diversification is achieved in the immune systems of older, jawless vertebrate species such as the eel-like lamprey and hagfish. "In the last few years, mechanisms of genetic diversification have been discovered in these organisms that are very different from the ones we're familiar with in jawed vertebrates. But they confer on these organisms immune capabilities similar [to higher vertebrates]."

"The genomics of the immune system is quite different from other parts of the genome," she says. "The genes that are active in white blood cells use mechanisms of genetic diversification that are not [present elsewhere]. As they evolve, we see a lot of gene duplication, expansion and contraction of these loci. They're fascinating mechanisms to study." ▶

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Further Reading:

Cowell LG, Davila M, Ramsden D, Kelsoe G. Computational tools for understanding sequence variability in recombination signals. *Immunol Rev.* 2004 200:57-69.

Genomes@4

Not just another seminar series! The IGSP invites you to a biweekly series, "Genomes@4", held on Thursdays (at 4 o'clock naturally). This is an opportunity for all IGSP faculty, collaborators, students and any other interested parties at Duke to hear presentations and engage in discussions on various topics relevant to the genome sciences, ethics, and policy. All seminars take place in CIEMAS Auditorium B unless otherwise noted.

- May 5** **Greg Wray**, Director, IGSP Center for Evolutionary Genomics; "Rewiring gene networks: Is evolution a tinkerer or an engineer?"
- May 19** **Geoff Ginsburg**, Director, IGSP Center for Genomic Medicine; "The Road To Personalized Medicine." Note: Will take place in Bryan Research Auditorium 103.
- June 2** **David Goldstein**, Director, IGSP Center for Population Genomics & Pharmacogenetics
- June 16** **Kelly Marcom**, Department of Medicine, Division of Medical Oncology; "Clinical Application of Germline Cancer Genomics"



GenomeLIFE, the newsletter of the Duke Institute for Genome Sciences & Policy, is published monthly and edited by Misha Angrist and Denise Haviland. We welcome your input! Please direct all inquiries, suggestions, and ideas to genomelife@genome.duke.edu



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