



Genome LIFE

Code Finders

In Search of Meaning in DNA Once Thought to be Junk

The sequencing of the human and many other genomes may be finished, but our understanding of how those sequences can produce a complex functioning organism like a human being remains far from complete. Take our genome, for example; of its three billion base pairs of DNA, genes that code for enzymes, structural components and signaling molecules account for just two percent. So what about the other 98 percent? For many years, scientists had essentially disregarded the runs of As, Ts, Cs and Gs in the genome that didn't code for proteins as genetic gibberish, meaningless syntax that had no relevance to the understanding of our genomic blueprint.

However, many scientists are realizing that using the classic 1960's notion of the genetic code – the translation of triplet codons of sequence into the amino acid building blocks of proteins – to judge whether a piece of DNA is functional or not leaves out many of the sequence elements needed for a gene to function. “That genetic code is really just one of many,” said Greg Wray, director of the IGSP's Center for Evolutionary Genomics. “You need another kind of genetic information in order to make, process and package the genetic message; otherwise it would just sit there.”

Wray is among several investigators at the IGSP searching for these other genetic and genomic codes among the vast uncharted sequences of DNA. Such codes could regulate how genes become active, how DNA is replicated, how chromosomes are inherited, and how two meters of the DNA double helix gets packed into a tiny three-dimensional sphere within each cell; the list goes on and on. “The first genetic code, which quite deservedly resulted in a Nobel Prize, was actually easier to find,” Wray said. “But there is no single thing that every gene uses in its regulation – it is all highly gene specific. That is why it has taken so long to figure out these other codes. It is not a single other code that just happens to be hard to crack; it is myriad modifications on a very loose theme.”

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Code Finders (continued)



Comparisons between the human and other primate genomes can point the way to conserved and important sequences.

Great Expectations

With the Human Genome Project concluded, it is as if scientists have all of the nouns in place and now just have to insert the proper verbs and adjectives, along with some punctuation. This additional genomic language includes the promoters, enhancers, repressors, and other regulatory sequences that determine when and where any one of the predicted 25,000 genes is expressed or ‘turned on.’ IGSP Investigators Terry Furey and Greg Crawford are part of a broader effort to identify each and every one of these functional elements as part of the international ENCODE (**E**ncyclopedia **O**f **D**N**A** **E**lements) Project. Their specific approach looks for regions of the genome that are not tightly wound up or packaged into the protein-DNA complex known as chromatin, with the idea that these areas of open chromatin contain sequences that control the activity of other genes.

Currently, their interdisciplinary team is scanning these regions for known regulatory codes, such as the binding site of a classic transcription factor – the protein responsible for turning genes on or off. But they are also trying to uncover novel patterns of sequence that lead to other important factors that have never been discovered before. The researchers are following their quantitative analysis with work in the wet lab to determine exactly what influence each discovered factor has on gene regulation.

“It is a very daunting task,” said Furey. “I think we are just beginning to shed light on the whole complexity of the regulatory machinery. And I imagine the more we learn, the more complicated it is going to get.”

Furey and Crawford have turned to Wray to help them whittle down their hundreds of thousands of sequences to ones that have functional meaning. Wray has been lining up their human genome sequences with ones from chimpanzees, gorillas, and other species to map the elements that have been conserved through evolution. Such highly conserved blocks of sequence are a genomic road sign for what nature has considered important for the last 50 to 100 million years, and thus far their studies suggest that over five percent of the

genome is actually ‘important’. Wray says these evolutionary comparisons are revealing certain elements that appear to be critical for all life, and others that may underlie species-specific traits.

“It enables us to tease apart what you might call the fine-tuning dials of evolution – the regulatory regions – versus the master volume control, the protein-coding regions that you can’t change without messing everything up,” said Wray. “We are finding these little blocks of conserved sequence that are specific to a particular species and are essentially the codes that might tell you how you make a mouse different from a monkey.”

Filling in Blank Pages

Despite the fact that the human genome is said to be fully sequenced, a few blank pages do remain, especially in the repetitive sequences flanking the centromeres, found near the middle of chromosomes. These sequences – called satellite DNA – are so notoriously difficult to assemble that only a couple of labs are even attempting the task, one of them that of Hunt Willard, director of the IGSP.

Ignored for decades as trivial text in complex genomes, the repetitive sequences around the centromere are increasingly being recognized as critical to genome function. Willard’s lab finally established their relevance about 10 years ago when they succeeded in making functional artificial chromosomes using satellite DNA. “The key question now,” says Willard, “is not *whether* these sequences work, but rather *how* they work.” To address this question, researchers including IGSP Investigator Beth Sullivan are exploring how centromeres are organized and regulated. She has discovered that centromeres have a unique organization whereby certain histone proteins – those involved in packaging and compacting DNA – are alternated in a regular pattern. If that pattern is changed or the proteins are removed, the centromeres fail to do their job of separating chromosomes during cell division.

“We have good evidence that regions of the genome that are highly repetitive – like at the centromeres – are regulated more by the three-dimensional chromatin structure than the underlying DNA sequence,” said Sullivan. “Surprisingly, it appears that they contain not only the tightly packaged regions that we know to be repressive to gene expression, but also more open regions where genes can actually be actively turned on. Centromeres are much more complicated than we originally thought.”

Sullivan is now studying what happens when chromosomes fuse to each other to create one chromosome with two centromeres, a rearrangement found in one in a thousand people. By building such dicentric chromosomes from scratch in the laboratory, Sullivan’s group has analyzed which chromosomes – from numbers 1 to 22 – are most likely to fuse, and how they behave in the cell once they are linked together. Sullivan’s experiments have demonstrated that any two chromosomes will fuse together, but only the centromere from one of them will ultimately remain active in the dicentric chromosome. Her laboratory is now looking at the sequence differences between the centromeres to see if there are any genomic cues for choosing one centromere over another.

“That [1960's] genetic code is really just one of many.

You need another kind of genetic information to make, process and package the genetic message; otherwise it would just sit there.” —Greg Wray

Kristin Scott, an IGSP Associate Investigator, is also interested in the genomic elements needed to establish a functional centromere. She has been using fission yeast as a model system to investigate the codes that are required to assemble the tightly compacted form of DNA – called heterochromatin – that is so characteristic of centromeres. Scott has discovered sequence-specific elements that basically behave like molecular stop signs, allowing heterochromatin to form in some regions surrounding the centromere, but not in others. She is currently looking into whether the sequence she identified acts alone or recruits other molecules that might accumulate and behave as a roadblock to additional packaging.

Scott and graduate student Bayly Wheeler in the Willard lab tinkered with several different genomic sequences – such as those necessary to establish, maintain or spread heterochromatin – to see what will happen when they are taken out of the tightly packaged heterochromatin and placed in more open expanses of DNA. Wheeler has found that she can remove the sequence elements that are required to wind up the DNA in the first place, and the DNA still remains tightly compacted for hundreds of generations.

“It is just fascinating to consider how such memory can be maintained in the absence of DNA sequence through so many rounds of mitosis and meiosis,” Scott said.

Around the Genome in 60 Minutes

If DNA is the book of life, then replication is the process of making precise copies of that text, so that every cell gets the same book to read. Just like the genomic codes that determine what genes are active or how chromosomes are packaged, DNA replication requires specific sequences, called origins of replication, to initiate the process. Until recently, surprisingly little has been known about how these start sites in the genome are selected. As part of the model organism arm of the ENCODE project, Dave MacAlpine, an IGSP member in the Department of Pharmacology, is currently using gene chips to systematically map all of these sites in the *Drosophila* genome.

“It turns out replication is quite a huge feat,” said MacAlpine. “You have three billion base pairs of DNA, and every cell cycle you’ve got to copy them all not only accurately, but just one time – because if you underreplicate or overreplicate the genome it will lead to catastrophic problems. Somehow you have to coordinate thousands of potential start sites across the entire genome in order to finish a cycle of replication in the right amount of time.”

MacAlpine has crunched the data from the model ENCODE project and found that chromatin structure plays a key role in specifying where start sites are located. Apparently, the same genomic modifications that promote the unwinding of the DNA helix to allow genes to be activated may also be involved in opening up the chromatin for the DNA to be copied. In the end, MacAlpine

doesn't think that one single code will be driving the DNA duplication machinery. Rather, he predicts that the signals will come from a combination of sequence elements, activated genes and chromatin structure.

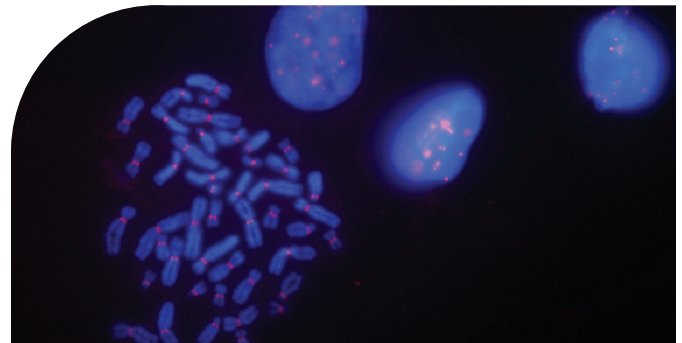
Translation, Interrupted

One of the biggest surprises to be uncovered in recent genome studies has been the existence of a class of new ‘genes’ that do not follow the language of the classic genetic code. These non-coding RNAs – sequences of DNA that are transcribed into functional RNA molecules but are not translated into proteins – were initially discovered in the 1990's by both Hunt Willard's laboratory and Shirley Tilghman's laboratory at Princeton. Now it appears that there are many thousands of such non-coding genes in the genome, ranging from the short microRNA genes involved in the regulation of development to much longer non-coding RNAs that control epigenetic silencing.

“While these non-coding RNAs do not produce proteins themselves, many of them do serve to fine-tune how much protein is produced by their target genes in the cell,” said IGSP Investigator Ashley Chi. “For instance, microRNAs can bind the sequences next to a target gene to keep them from being translated into protein.”

Chi specifically studies the composition of microRNAs in red blood cells from patients with sickle cell disease. He has found that microRNA levels change dramatically between healthy people and those with the illness. In particular, certain microRNAs are linked to more severe forms of sickle cell disease by making the cells less able to defend against oxidative stress. On the flip side, increased levels of microRNAs may make patients more resistant to malaria.

Over a decade ago, no one had even heard of non-coding RNAs. Today, scientists are finding that they can have a significant impact not only on sickle cell disease but also on other illnesses like cancer, autism and Alzheimer's disease. Clearly, much of the “other 98 percent” of our genome is doing something – whether by directing when and where genes are active or by packaging chromatin in ways that ensure the proper regulation of gene expression and the proper mechanics of chromosomes. And only time will tell what researchers will uncover next as they continue to decode the information stored in our genome. ▶



Once ignored, centromeres are now being recognized as critical to genome function.

Genomes by Design

design

IGSP Researchers Move Synthetic Biology Forward Through Fundamental and Technological Advances

For IGSP Investigator and Engineering faculty member Lingchong You, synthetic biology is a lot like computer programming. New gene circuits are loaded like software into bacteria or other cells in an attempt to re-wire their behavior in useful or otherwise interesting ways. But there is a very big catch.

"In the case of computers, we know exactly how they are constructed," he says. "We've put the components together and have a good understanding of how a computer behaves. In synthetic biology, we may know a lot but our understanding of the biology is far from complete."

That's true even for the simplest and most thoroughly studied organisms like *E. coli*, You's subject of choice. "*E. coli* has about 4,000 genes and we know the entire sequence. We don't know exactly how they all function even for *E. coli*, let alone human cells. We are dealing with a 'computer' we don't yet fully understand."

That means that genetic 'software' loaded into bacterial cells may interact with other genes or proteins in unexpected ways, and cells might not always respond to external commands in exactly the same manner.

You Zig, I'll Zag

A report published by You's team in *Nature Chemical Biology* in October demonstrates just such a case, in which individual, reprogrammed *E. coli* cells do behave unpredictably. Individual cells turn either 'on' or 'off' when stimulated by an external cue, in that case a chemical compound. Those differences in outcome result from the fact that gene circuits influence the physiology of the cells that house them and vice versa.

"The expression of the genetic alteration can drastically impact the cell, and therefore the circuit," said graduate student Cheemeng Tan, who is the first author of the study. "We now know that when the circuit is activated, it affects the cell, which in turn acts as an additional feedback loop influencing the circuit."

That kind of interplay between cells native machinery and their synthetic circuits hadn't been demonstrated experimentally before, and the very idea has often been discounted in the field.

"In the past, synthetic biologists have assumed that the components of a circuit would act in a predictable fashion every time and that the cells carrying the circuit would just serve as a passive

reactor," You said. "In essence, they have taken a circuit-centric view for the design and optimization process."

Setting Standards

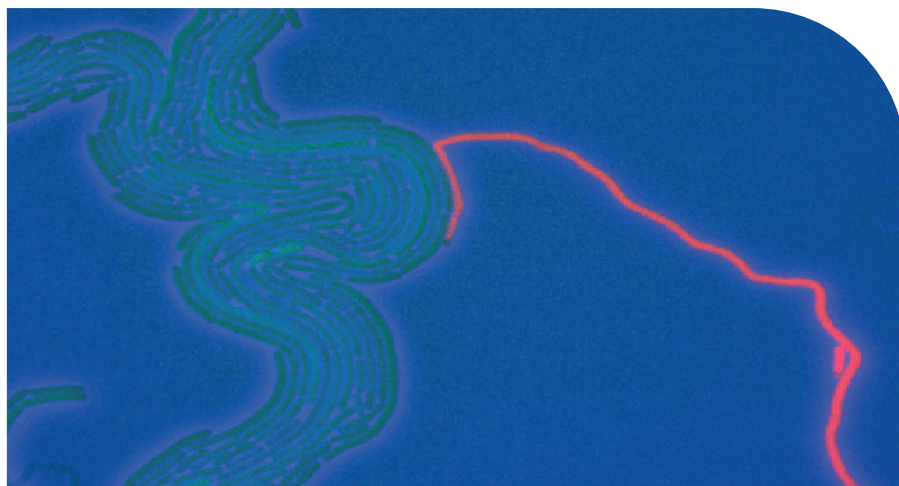
That circuit-centric view is evident in efforts made by researchers in the field to standardize synthetic biological parts – so-called BioBricks™. At an online registry, synthetic biologists can peruse a catalog of about 3,200 genetic parts "that can be mixed and matched to build synthetic biology devices and systems." They can also submit new ones.

Although You and Tan's new study shows that even well-characterized parts can yield some surprises, in their particular case they were able to predict how many cells would switch to the 'on' or 'off' state. "It turns out the consequence is quite predictable and easily accounted for," You said.

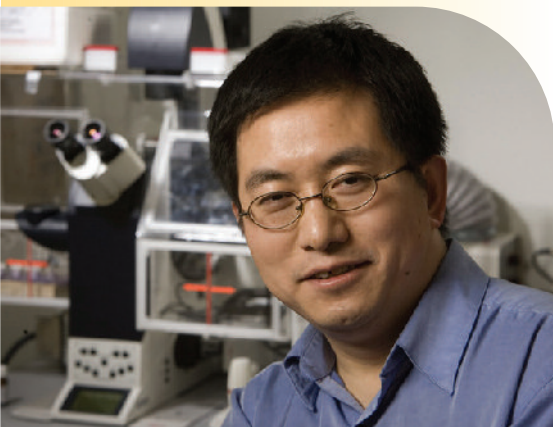
Even if it weren't, synthetic biology is advanced each time cells are programmed with gene circuits. "We get better every time," You said. "If we get a surprising result, you look back at why the circuits failed to behave as anticipated. The next round, you do a better job."

Despite the gaps in scientists' understanding of the genome and how cells will respond to DNA tinkering, the promise of synthetic biology for turning bacteria into tiny, living factories for everything from drugs to biofuels is already being realized in some corners. You points to the poster child of synthetic biology – yeast reconfigured by scientists at UC Berkeley to produce artemisinic acid, a precursor to the potent anti-malarial drug artemisinin that is otherwise in short supply.

Others have shown that bacteria can be made to invade tumors – a proof of concept that engineered bacteria might one day recognize and kill cancerous cells while leaving healthy cells unscathed. You has interest in such practical applications too, but his approach to reaching



Lingchong You's lab created two strains of *E. coli* bacteria that can control each other's population.



Lingchong You

those ultimate goals is to focus first at a much more fundamental level.

"I want to push the limits," he says. "To find out to what extent we can program cell behavior."

Predator and Prey

That was clearly the impetus for an effort published in *Molecular Systems Biology* last year that You still considers one of his favorites: a synthetic predator-prey ecosystem made up of two distinct populations of *E. coli*, each with its own specific set of programming and the ability to affect the existence of the other.

"The key to the success of this kind of circuit is the ability of the two populations to communicate with each other," You said. "We created bacteria representing the predators and the prey, with each having the ability to secrete chemicals into their shared ecosystem that can protect or kill."

Central to the operation of this circuit are the numbers of predator and prey cells relative to each other in their controlled environment.

However, as the population of prey increases, those bacteria secrete a chemical that, when it achieves a high enough concentration, activates an 'antidote' gene in the predator. This leads to an increase in predators, which in turn causes the predator to produce another chemical, which enters the prey cell and activates a 'killer' gene, causing the prey to die.

"This system is much like the natural world, where one species – the prey – suffers from

growth of another species – the predator," You said. "Likewise, the predator benefits from the growth of the prey."

While the circuit is not an exact representation of the predator-prey relationship in nature – the two actually compete with one another for food – You nevertheless believes that the circuit and others like it can be useful for exploring basic ecological principles. In this regard, he says, synthetic biology falls somewhere between traditional experimental biology and computer simulation.

"This system provides clear mapping between genetics and the dynamics of population change, which will help in future studies of how molecular interactions can influence population changes, a central theme of ecology," You said. "There are literally unlimited ways to change variables in this system to examine in detail the interplay between environment, gene regulation and population dynamics."

In their latest study, which just appeared in another *Nature Chemical Biology* report in November, You and his lab members took their synthetic predator-prey system a step further, showing that it can lend insight into the population dynamics that maintain or erode biodiversity in microbial ecosystems.

Built from Scratch

In his belief that synthetic biology can be useful for elucidating 'real' biology, You and new IGSP Investigator Nick Buchler are kindred spirits. Buchler considers himself part of the synthetic biology camp that seeks to better understand how genetic circuits in nature work by reconstructing them to the best of his ability in the dish. The basic idea is that scientists might understand how biological networks – say, those that drive circadian clocks – function not by taking them apart, but rather by attempting to build new ones out of similar parts.

"I take a synthetic approach to get at the biology," Buchler says.

In the journal *Systems and Synthetic Biology*, Buchler and his colleagues reported the design of a new genetic circuit for conditional memory. The circuitry is theorized to work similarly to a molecular

'on-off' switch with an added layer of control, such that the switch-flipping signal can be conditionally remembered or ignored. (It's similar to the Hold switch on an iPod, which can prevent other functions from being turned on or off when buttons get pressed accidentally.)

He says they aren't yet sure whether molecular networks like the one they've designed actually exist in nature and they haven't built it yet, but their findings do suggest that such sequential logic elements, familiar in electronic circuitry, may be readily implemented in cells by combining protein-protein interactions with simple transcriptional regulation.

All or Nothing

More recently, Buchler and Fred Cross at The Rockefeller University have shown in yeast cells how simple interactions between two proteins – one that acts as an inhibitor of the other – can generate 'ultrasensitive' all-or-none responses. The position of those sharp thresholds can be adjusted up or down by simply adjusting the concentration of the inhibitor.

In natural systems, proteins are commonly sequestered by inhibitors similar in function to those used in the study, but it hadn't been entirely clear whether their interaction could generate the kind of ultrasensitivity that is important for generating circadian clocks and other biological networks, Buchler explained. "Putting a synthetic gene circuit into yeast is about the clearest way to test whether an activator-inhibitor pair actually generates ultrasensitivity," he says.

He now suspects that this kind of simple interaction between proteins may play an unexpectedly large role in the evolution of genetic circuits in nature. After all, he says, protein inhibitors can arise rather easily when genes get duplicated in the genome, which is a relatively frequent occurrence. The extra (and extraneous) copy can pick up additional mutations that turn it into a competitive inhibitor of the first, or vice versa. Buchler explains it simply: "Your worst competitor is your twin gone bad."

He plans to conduct synthetic evolution experiments in the yeast to find out whether this underlying mechanism can, via artificial selection in the lab,

generate more complex dynamics like those observed in natural systems. "We will be running evolution forward at an accelerated rate," he said. "The question is: can you generate sharp threshold behaviors in this way and how difficult is it to do? How many mutations does it take – one, three, ten?"

You is hatching his next round of experiments as well, in which he hopes to realize a division of labor among bacteria. The idea is to select some metabolic pathway and separately engineer the circuitry for each of its steps into bugs that will then work together toward a larger end goal.

The effort presents significant design challenges, but could ultimately lead to an efficient microbial factory complete with its own assembly line. "Each population will be dedicated to a subtask. You will need coordination between the populations so that the overall performance of the consortium is optimal." Step one, he says, is to find the right task.

DNA Writer

As in You's and Buchler's labs, the focus of much of synthetic biology today is at the level of gene networks.

"The question is how can we take advantage of available components with diverse functions – transcription factors, promoters, binding sites – in our design?" You explained. "How can we assemble those components to generate predictable or useful functions?"

Ultimately, synthetic biologists would like to be able to design or optimize new genes, even whole new genomes, from scratch. For that, the field still faces a very big hurdle, says Jingdong Tian, an Engineering faculty member and IGSP investigator. While genome technologies for reading DNA have taken off in recent years – a whole genome can be sequenced (or 'read') for a few thousand dollars and in a few days – it turns out the state-of-the-art for writing DNA is much more rudimentary.

"Currently, DNA synthesis is the bottleneck for the field," Tian says.

"Until recently, DNA manipulations were done in a 'copy, cut and paste' manner," You adds. "While this enzymatic approach has produced a wealth of scientific advances, implementing a

complicated biological design by these means is the literary equivalent of writing a paper using a photocopier, scissors and a stick of glue."

Today, synthetic biologists order short stretches of DNA known as oligos – 100 base pairs or so long – routinely. But the longer sequences needed to encode whole genes, on the order of 1,000 to 10,000 base pairs, remain prohibitively expensive for most labs, and they can take weeks to get, Tian says.

Craig Venter and colleagues set a record last year when they reported the creation of the largest man-made DNA structure by synthesizing and assembling the more than 500,000 base pair genome of the parasitic bacteria known as *Mycoplasma genitalium*, notable for having one of the smallest known non-viral genomes. But it was a massive undertaking that had to be done manually, and "it took tens of millions of dollars to make," Tian said.

For the last several years, Tian and colleagues have been developing a large-scale DNA synthesis on a chip technology that promises to change all that. "We are trying to build an automated microfluidic machine so everyone can do DNA synthesis right on their lab bench, just like PCR. You would simply type in a sequence and the instrument would synthesize it without the user even having to know how." He thinks such a machine could also be used on the cheap, slashing the cost of DNA synthesis from the 50 cents per base it is now to less than one cent per base.

Visitors to Tian's lab can already see a working prototype of the machine he envisions, which is based on inkjets like those found in your run-of-the-mill printer. (They literally built the prototype with an inkjet repurposed from one of those printers.)

"Instead of printing ink, it prints the chemicals for DNA synthesis on a chip," he says. Whereas a color printer would have different color inks, the DNA printer has the four bases that make up DNA and two chemicals to link them together. Instead of printing on paper, it prints DNA in 100 base pair stretches on a slide.

"It's like printing a microarray on a plastic slide, then you release the DNA and assemble it [into longer stretches]," he explains. Right now, they have the initial synthesis step down and are working on the assembly of long sequences.

In the meantime, Tian's lab is also demonstrating applications for the technology he hopes can become available to all in about five year's time. He is collaborating with IGSP member Bruce Donald in an effort to create a new and improved DNA-cutting restriction enzyme. His lab is also devising a metabolic pathway consisting of four genes that together convert *E. coli* into manufacturers of biodegradable plastic.

"Instead of using petroleum to make the normal plastics, you can have *E. coli* make biodegradable plastic for you," Tian says. ▶



Graduate student Ali Saaem tinkers with the prototype inkjet DNA synthesizer.

Food for Thought

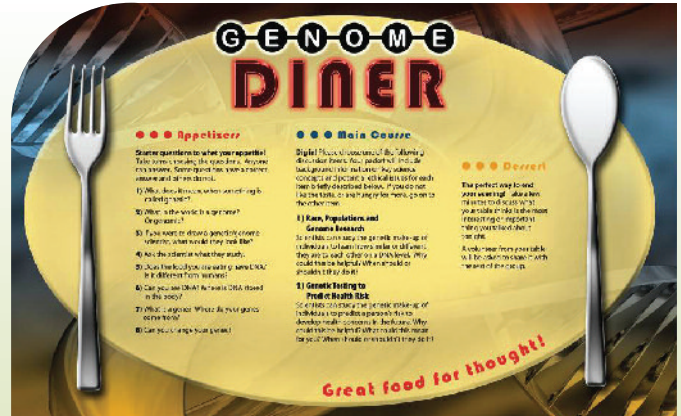
Digging in at the Genome Diner

When members of the community sit down for some hearty conversation with genome researchers, perspectives can change on both sides. In fact, the most dramatic change may be an improvement in researchers' perceptions of the public in terms of their knowledge and their attitudes toward genetic and genomic research. That's according to a preliminary analysis of the Genome Diner pilot project conducted last year with funding from the NIH Partners in Research program. The effort resulted from a partnership between the IGSP and the Museum of Life & Science that engaged students from Rogers-Herr Middle School and their parents.

"We were surprised at the immediate impact [on researchers] of interacting with the public," said Julianne O'Daniel, an associate in research at the IGSP and the project lead. "We need to find ways to sustain that."

The pilot followed a unique strategy designed to bridge formal schooling and informal science learning by engaging 7th- and 8th-graders who had just been introduced to genetics concepts in the classroom, together with their parents, in an open exchange with practicing researchers.

The discussion agenda followed a set "menu" facilitated by museum volunteers, with a series of starter questions to whet the appetite, followed by a main course of deeper discussion and sharing for dessert. In small groups, participants were provided with some basic background information on key science concepts and potential ethical issues related to studying the genetic make-up of individuals or populations and the use of genetic and genomic testing in predicting health risks.



O'Daniel said that students and their parents showed an increase in interest in genetic and genomic research based on their experience, but they also became more skeptical in some regards.

"They took a more complex view as they began to understand the shades of gray," O'Daniel said. The interest of students and parents rose overall as did their willingness to participate in research. But, she said, they also began to think there might be some questions genome researchers simply should not ask.

About 100 community participants took part in last year's event, and O'Daniel said they plan to increase that number to 150 or even 200 this year. Afterwards, the team plans to make the materials they have developed available for others to use.

If you are a genetic or genomic researcher who is interested in participating in this year's Genome Diner, contact Julianne at julianne.odaniel@duke.edu. ▶

Summer Fellow Returns



Aisha Hilliard, former IGSP Summer Fellow, transferred to Duke this fall.

As an IGSP Summer Fellow two summers ago, Aisha Hilliard, then a chemistry major at Winston-Salem State, got an early taste of what it's like to be a researcher and an undergraduate at Duke. Now, she's back for more of both. Inspired by the support and encouragement of the students working in Philip Benfey's lab, where she spent that first summer, Hilliard officially enrolled as a Duke transfer student this fall.

While Hilliard is still getting used to the idea of her new life on campus, it's clear she fits right in. "I went from a small, campus school to a

large, diverse school," she says. "But the rigor of the education, the culture here, I love it."

She is tackling a hefty course load including Physical Chemistry, Physics, Gender Studies and German and is back at work in Benfey's lab, this time focusing on an *Arabidopsis* Phenome Project aimed at connecting changes to the model plant at the genomic level to their observable, phenotypic consequences. If all goes according to schedule, she intends to complete her chemistry degree and graduate from Duke in 2011. ▶



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Genome Research & Education Society

Undergraduates can now get involved in genome research and education at Duke in a whole new way. Sophomore students Brandon Ruderman and Jason Klein have founded a new student club – the Genome Research & Education Society (GRES) – dedicated to promoting scholarship and research at Duke and educating others about the genome sciences and their implications.

"We felt like there wasn't enough opportunity outside of the classroom for undergraduates to learn about genome sciences and the research opportunities on campus," said Ruderman, a former IGSP Summer Fellow and Focus student who continues to conduct independent research in Hunt Willard's lab. "The growing Durham community is also not that informed."

"We want to work with the community and get more students at Duke involved," added Jason Klein, who was also a Summer Fellow and Focus student and is now a member of David McClay's lab.

They say the group will reach out to the community through Science Saturdays at the North Carolina Museum of Life & Science and a Science Policy, Ethics & Law course at the North Carolina School of Science and Mathematics. Klein and Ruderman are also approved to teach the house course "From Science to Society: Decoding Your Genome" at Duke in Spring 2010. In addition, they plan to sponsor guest speakers on campus and to hold casual dinners where faculty members and students can talk about genome sciences



Brandon Ruderman and Jason Klein

research and opportunities "more personally."

The club is off to a strong start. Already, more than 100 people have signed up for the GRES listserv and about 40 students attended their first meeting. We encourage you to get involved in the new group by attending the next meeting, subscribing to the listserv and/or becoming a facebook fan. ▶