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We are back for the third and final day of Huntington's disease therapeutics conference 2026! This morning's session focuses on new and breakthrough science both in and out of the HD field that is driving therapeutic development.

Taekjip Ha – Watching DNA repair and CAG “bubble-outs” one molecule at a time

First up is Taekjip Ha from Boston Children's Hospital and Harvard Medical School. He zooms waaaaay inside cells to look at single molecules so that he can track what's going on in somatic expansion in HD.

The first question he's asking is what DNA repair machines bind to in terms of the part of the DNA letter code and its shape, and what they seek to fix. These repair machines are trying to help but instead they can cause expansion of the CAG repeat.

Our DNA is supposed to line up like a zipper, but when the CAG repeat becomes expanded, it can “bubble out.” Taekjip (TJ) has made a collection of different size CAG bubble-outs in order to study which proteins bind to them.

TJ uses specialized light tags to track single molecular machines. He can see how they move along different types of DNA, and where they stop off to make repairs. This allows his lab to compare how well different machines work on different repair jobs in cells.

Taekjip's team also looks at how these machines might bend or contort the DNA. They ask how the bending might be altered with CAG DNA bubbles of different shapes and sizes, and when different repair machines work to fix them.



We have known that small changes in the genes encoding these DNA bending machines can influence when people may begin to show symptoms of HD. These nitty gritty details give us a better understanding of why subtle differences in DNA bending could influence onset.

Now TJ is showing us some cool movies of the bending DNA "dancing" around. Although we often think of these molecules as static, they are actually in constant motion, and the movement of DNA can impact how it is repaired.

The bubble-outs don't stay in place either; they can move along the DNA strand. TJ and his team are modelling this as well to get the full picture of how repair is accomplished at the molecular level.

TJ's team have also invented a new way to "paint" different parts of the genetic material in the nucleus. This allows researchers to see how close or far apart specific DNA codes might be. This technology could be used to track the DNA bubble-outs and better understand their role in HD.

TJ also describes a new technology that is a better way to read the DNA sequence around the CAG repeat in the huntingtin gene. Those CAG repeats can be pesky to measure!

A lot of the current methods to measure CAG repeat number provide only an estimate, but TJ's method can give a much more precise readout. It's early days for this technology, but these tools could be super helpful to

better understand somatic expansion in more detail.

Alice Stanton – Building vascularized mini-brains to model HD

In the next talk Alice Stanton of Harvard/MGH will talk about modeling the human brain in a dish – a way to simulate a whole organ by growing complex layers of cells in a dish. This is a way to study HD in a way that more closely resembles the human condition.

Alice kicks off by reminding us that a lot of the brain is not actually neurons – there are support cells, blood vessels and other types of cells which are just as important for a healthy functional brain.

Alice specialises in making engineered tissues which form 3D shapes and try to mimic real life organs in the body. These are called organised or mini brains and can be helpful to study brain disease and how treatments might help.

One advantage that Alice's system has that many other "mini brain" models don't is a network of blood vessels (vasculature), better mimicking conditions in the human body. This requires specialized engineering in the lab so that different cell types can assemble and form biological structures.

Alice's team starts with stem cells, coaxes them into many different cell types, and then brings them together in a special scaffold that mimics what happens in the brain, including layers of cells resembling a blood-brain barrier.

To check that these 3D cell clumps actually mimic the human brain, Alice and her team visualize all of the different cell types in globe-shaped maps to see exactly where they all are in the structure and how well they are all working together.



Alice talked about the "mini brains" her team can make to help study different diseases.

They can test how well connected the neurons are by adding different signalling molecules and measuring how efficiently electrical and chemical signals are transmitted through the mini brains.

Glial cells are an important non-neuron cell type which support a good immune response for a healthy brain. They seem to be doing their job in Alice's minibrains, so she thinks this system could be helpful to study diseases like HD, where glia play an important role when the brain starts to get sick.

They also checked on the formation of blood vessels in these mini brains, comparing them with blood vessels in real full-size human brains. The shape, structure and organization looks very similar, reinforcing that these mini brains could be a useful tool to study HD in a dish.

Including blood vessels as part of laboratory brain modeling approaches is particularly important for understanding how different types of molecules, including drugs, can cross from the blood stream into different parts of the brain.

Because mini brains are formed from stem cells, Alice's team can design them using stem cells from people with different brain diseases. She shows examples from models of different types of dementia, and explains how each mini brain system mimics features of each disease.

It has been a challenge for HD researchers to recapitulate brain-like features in a dish, especially vasculature, in a way that is resource-efficient, so there's some excitement about having a sharper tool in our belts.

Vincent Dion – Using CRISPR nickases to shrink expanded CAG repeats

Our next speaker is Alvaro Murillo Bartolome, presenting work from the lab of Vincent Dion at Cardiff University. They're trying to understand if they can genetically correct the CAG repeat that causes HD, using a CRISPR-based system.

Their approach uses a molecule that causes little nicks in one strand of the DNA at the CAG repeat. In theory, this will cause the DNA to contract, because DNA, like spaghetti, doesn't want to be broken.

Messing around with DNA like this carries lots of risks – potentially expanding repeats, introducing errors elsewhere or other side effects. We have to be absolutely sure that CRISPR and other DNA editing tools are not making things worse, so every experiment must be carefully controlled.

To reinforce this point, Alvaro showed (many!) papers from other groups where experimental evidence revealed safety concerns. Those approaches caused nicks on both strands of the DNA, whereas their approach just targets one strand.

In brain cells created from stem cells, Alvaro's data suggests that their compound is indeed able to cause contractions of the CAG repeat. Exciting potential! But the real magic would come from knowing if they can induce these contractions in animals. So they moved on to mice that model HD.

They delivered their DNA-nicking molecule to the brains of these HD mice using a harmless virus. Normally, we would expect to see the CAG repeat get bigger over time as the mice get older. And this is exactly what they see in the mice that did not receive the drug.

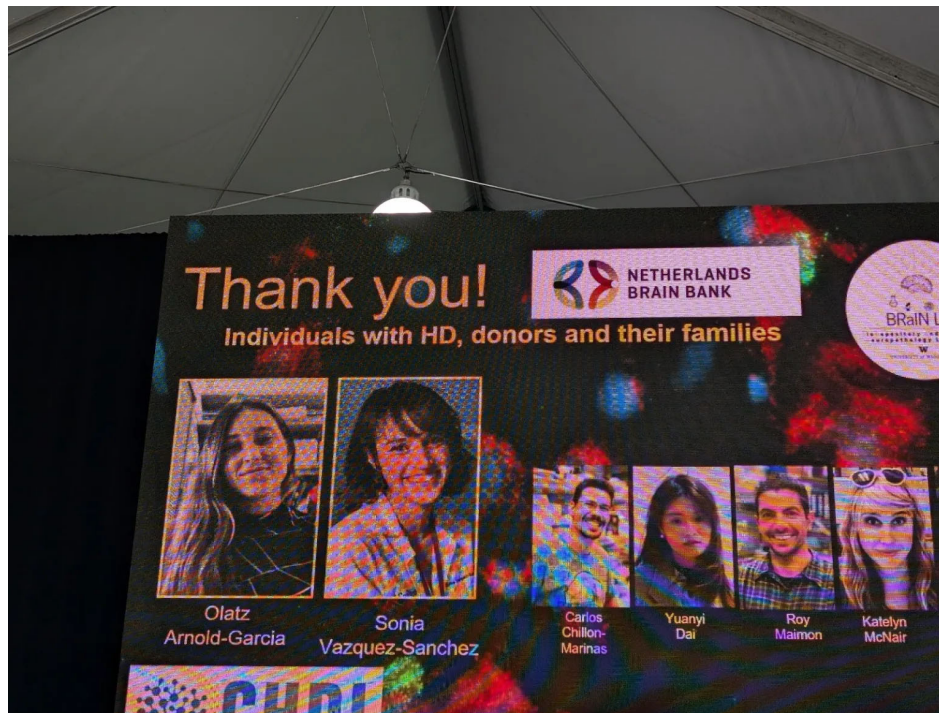
In the animals treated with their compound (called a nickase), they still see some expansions, but they also see contractions down to CAG numbers that do not cause disease. Exciting potential! But it's worth highlighting that there were still some expansions.

That experiment was done where the nickase drug was delivered before the HD mice showed symptoms. They next added their nickase after the animals were symptomatic. In this case there were still some expansions and some contractions.

They also looked at how the drug affected molecular aspects of HD in mice. In the striatum, the brain region most affected by HD, the number of HTT protein clumps went way down in animals treated with their DNA-nicking molecule.

We know that HD affects the "transcriptome," the entire library of RNA messages made within an organism, essentially which genes are turned on and off. In HD, this balance becomes more and more disrupted over time. Animals treated with their DNA nicking drug had fewer disrupted genetic

messages.



Many talks, like this one, make a point to thank Huntington's disease families, without whom the progress discussed in this meeting wouldn't be possible. THANK YOU!

They then moved on to behavior experiments: seeing if their DNA-nicking drug could improve coordination problems in mice that model HD. The mice treated with the nickase performed more similarly to animals without the gene for HD, suggesting that it does have potential for behavior improvements.

Alvaro noted a major challenge of moving this technology into humans: it would require adding 2 separate viruses, rather than the 1 they've been able to use in animal studies. The concern is that increased amounts of viruses, even harmless ones, could induce an immune response.

To overcome this potential safety concern, they will test a 2-virus approach in animal models. The room is buzzing about this promising therapeutic avenue, and you can be sure that a roomful of 450 HD researchers will take on the challenge!

Gregory L. Verdine – Drugging the “undruggable” by learning from nature

After a quick coffee break, our next speaker is Gregory Verdine from Harvard. He's hoping to open our eyes to a new avenue of drug discovery. A lofty goal!

He's starting by defining where drug targets are located – either inside or outside the cell. Targets outside the cell are the most “druggable,” but only make up about 10% of targets, whereas 90% of what we want to hit exists

inside the cell. Largely these targets have been considered “undruggable.”

Gregory asks, how can we drug the undruggable? He notes that many of the most successful drug innovations have mimicked things that have evolved naturally, like antibodies and viruses. These successful drugs have been reversed engineered from nature, then forward engineered to treat disease.

To answer this question, Gregory looked at what’s done in nature. Inside the cell, there are proteins that span the cell’s membrane. Interestingly they all have the same shape – a spiral looking thing called an alpha helix.

To create his own alpha helices, Gregory has molecularly “stapled” little pieces of genetic material together. As proof-of-concept (to show this approach could work), he first went after a gene that regulates various types of cancers.

Thus far, a lead candidate discovered in this way is having “extraordinary clinical responses.” It just received fast track approval for a wide array of cancers driven by a particular gene. This could be available by the FDA as early as 2027 – encouraging!



Researchers from around the world come together at this meeting, to discuss the latest breakthroughs and try and find answers to the big questions in HD. Image credit: Anastasia Khvorova.

Gregory is going into deep detail about a particular class of molecules considered undruggable because they're all very flat. Turning to nature, there are still compounds that will bind to these ultra flat molecules. And Gregory is teasing all of that out.

Biological molecules can be "undruggable" for different reasons, like "flatness," difficulty of two molecules bonding, or energy. Gregory's group has overcome many of these challenges by turning to nature.

Gregory is even doing cross-species experiments in his role at the company LifeMine, where they're de-coding the genetics of fungi to find advancements for "undruggable" targets.

Now he's moving onto the good stuff – Huntington's disease! When we add ASOs (a type of genetic drug) to cells, they bind to an RNA message, and

are recognized by a suite of molecules, ultimately leading to the lowering of HTT protein levels.

Gregory is interested in the multiple interactions of the ASO package and how it's bound at the molecular level in intricate detail, to improve drug design.

His approaches have the potential to control which HTT gene these ASOs target – like the expanded copy of HTT only. He's going into some detail about the Wave Life Science trial, that is using this approach to lower only the expanded copy of HTT.

To summarize, Gregory says all of this is possible by reverse engineering what has already been made by nature.

Aseem Z. Ansari – Small molecules that target disease-causing DNA repeats

Our speaker now is Aseem Ansari from St. Jude Children's Research Hospital, sharing his work on targeting repeat expansions. He also turns to nature to figure out better ways to develop drugs for diseases.

Most current disease treatments are designed to target protein molecules and change how they work. Aseem is interested in targeting drugs to genetic material (DNA and RNA) instead.

Normally scientists design drugs that target genetic material using OTHER man-made pieces of genetic material like ASOs. This is effective, but a real pain for delivery to where it's needed, especially within the brain, so these drugs are often given by spinal tap or other invasive procedures.

Small molecule drugs are an attractive approach instead. Being small means they can get around the body to where they need to work much more easily, and can be given to people as pills or other more accessible formats.

Aseem is working to make small molecule drugs for repeat diseases like HD. His team have been studying another repeat disease called Friedrichs Ataxia, which happens due to a lengthening of GAA repeats.

They hope to make small molecules that bind genetic material and machinery to change which genes are switched on or off. Their drugs are actually two molecules joined together so it can do 2 jobs – move to the right place, and bring in a protein called BRD4 which regulates genetic on/off switches.

Aseem's molecules can move into liquid-y structures (droplets) in cells where gene regulation happens, which is good news. Each half of the 2-part molecule can't do this alone, but together it can get to the right place, bring the BRD4 along for the ride, and get to work.

They have tested their drug in different types of cells in a dish, including

cells from people with Friedreich's Ataxia. The drug seems to be working as expected and changing gene expression. Now they are interested in applying this technology and approach to target other repeat diseases.

They are working on targeting the CTG repeats in a disease called Fuchs dystrophy which affects people's eyes. In different laboratory model systems, their molecules are able to change how genes are switched on or off and help to treat features of this disease.

The strand of DNA opposite to a CTG repeat stretch actually contains a CAG repeat. Aseem is interested to see whether his platform might be used to target the CAGs in the HTT gene, specifically the expanded HTT which has the longer CAG stretch.

Beatriz Osuna – RNA trans-splicing to repair the HTT message

The final talk before we break for lunch is from Beatriz Osuna from Tacit Therapeutics. This company has built an RNA editing technology and has recently applied it to HD. They think this could be a cool way to "correct" the expanded CAG run in the HTT gene in people with HD.

Their RNA technology is packaged into a harmless virus and alters the way RNA is processed by a type of cell machinery called splicing. They use an AI based platform to screen a huge number of molecules to figure out which RNA may be best for a given application or disease.

In the case of HD, they are looking for RNAs which would work to "edit" the HTT RNA message molecule, to bring down the CAG number, which can then be used to make the regular HTT protein without such a big expansion.

From tens of thousands of possible RNAs, the Tacit team narrowed down potential drug candidates in cell experiments to a smaller pool of just three, which they went on to test in mouse models of HD.



Inside the conference idea tent, a hub for bold hypotheses, lively debate, and the next generation of HD discoveries. Image credit Emilie Vocaj Wan.

They assessed how well these molecules altered the CAG repeat number in both cells and mice using custom genetic readouts in the lab. One of their molecular designs showed good editing, reducing CAGs.

They looked throughout the brains of the mice they treated, and importantly saw good levels of RNA repair in the striatum, a deep part of the brain which is one of the most affected regions in HD.

Not only did they see that RNA messages were repaired to contain fewer CAGs, but they also saw that the HTT protein made from these message templates was repaired, based on a special molecular decoration added during the repair process.

The drug seems to be safe and not harming the mouse brains. It also does not drastically alter which genes are switched on or off, which Tacit thinks is a good sign that their lead molecule design has limited off-target effects.

However, there were some off-target effects (when a drug alters other biological pathways), particularly around how other RNA message molecules are processed. Tacit is not too worried about this as they are minor and not thought to change how proteins are made so are predicted to be safe.

Tacit are busy in the lab testing their molecules in different HD models as well as doing more robust studies to assess how well these potential drug molecules are tolerated and turned over. We'll be keeping our eyes peeled for future updates!

That's all for this morning. We are going to lunch and to cool off before we

get back with updates on the final session of talks this afternoon. Stay tuned.

Jim Gusella – New insights from genetic modifiers of HD onset

Today's (final) afternoon session of will focus on human genetics and biomarkers. Speakers will talk about the genetics behind symptom onset, and things we can measure to track HD and understand whether drugs are working.

Our first speaker is Jim Gusella of Harvard Medical School/MGH. He played a major role in the discovery of the HD gene, and notes that none of this work would be possible without the many people from HD families who have donated blood and brain tissue.

Jim reminds us of the genome wide association studies (GWAS) that helped to identify tiny genetic changes that can speed up or slow down when HD symptoms begin. About half of those genes are related to DNA repair, and the other half have totally different functions.

GWAS studies and other discoveries related to the length of the CAG repeat and how it may drive disease led to current theories about how somatic instability plays a role in HD. Those discoveries have directly led to ideas that are advancing toward clinical trials today.

We heard yesterday that 150 CAGs may be a threshold for loss of neurons in HD, but Jim posits that the loss of function within circuits, prior to cell loss, is also very important to study and address with therapeutics.

He notes that many of the genetic modifiers uncovered by his group and others could have effects on the rate of CAG repeat expansion, the threshold for toxicity, the harm that occurs within cells, or the harm that occurs within circuits when cells are sick or lost.

He is sharing several vignettes from his lab – experimental stories that he hopes will lead to answers around questions in the HD field.

One question Jim wants to ask is about the timing of genetic modifiers. When we analyze humans and human tissue, we are looking at a snapshot of one period of time of a person's HD trajectory. It can be particularly hard to compare people statistically who are of different ages and stages of HD.

Jim's lab has developed novel statistics that help to make the most of the human data we have, aligning groups and ages in a way that can identify new genetic modifiers that are more relevant at earlier or later stages of disease, or within specific populations of brain cells.

A second question his lab is asking is how tiny variations within the huntingtin gene itself can change the age at which HD symptoms begin. Applying his advanced analyses, they find rare genetic mutations that drive changes in particular symptoms, like with movement or thinking.

Drawing on a wealth of human genetic data, they can identify subpopulations of people with HD (for example, people of European ancestry) who have specific modifiers that either drive changes in motor changes or cognitive processing. Very cool for drug development that seeks to target specific aspects of HD.

A third question Jim's lab is asking is whether one genetic modifier can affect another. Combinations of two tiny genetic changes can have multiplied effects on the age when symptoms occur, or completely different consequences from each tiny change alone.



Conversations continued past the talk tent into the poster sessions, where breaking research was shared by HD researchers. Image credit: May Meltzer.

Jim talks about the need to work through the combinations of various modifiers since researchers can't infer the function of combinations based on what each one does alone. Quite a puzzle to tease apart!

Another question he can ask is whether we can identify novel genetic modifiers in people who HAVE a modifier that's expected to change their age of onset, but it doesn't. Looking at "outliers" – people who don't fit the statistical curves as expected – can pull out new potential pathways and drug targets.

For example, we know that mutations in a gene called PMS1 usually cause much earlier onset of HD – but not always. In those individuals, more statistical methods can be applied to understand why, and trace this back to additional genes to explore – one of them (BRCA2) also pops up in breast cancer!

Jim emphasizes that a lot of this data is very new and needs to be expanded upon. We love it when scientists share work in progress so that others can jump in and do more experiments and modeling to confirm!

Qingqin Serena Li & Sahar Gelfman – Mining whole-genome data for DNA repair clues

Next, a double-header: Qingqin Serena Li from CHDI and Sahar Gelfman from Regeneron Genetics Center will talk about whole-genome sequencing analysis – reading all the letters within the DNA code – including the genetic changes in DNA repair genes.

CHDI runs the global [Enroll-HD](#) study that follows people with HD over time and also takes samples of blood and spinal fluid (in the HDClarity sub-study).

These valuable studies have collected a huge amount of data that now allows scientists from across the globe to closely compare detailed genetic information with detailed clinical information.

Researchers can now ask questions about how very specific “milestones” like reaching a certain level of movement impairment or cognitive symptoms are related to specific genetic changes.

The Regeneron Genetics Center has worked on confirming GWAS data and diving even deeper. In particular they look at data from very rare genetic variations – those that only occur in a tiny percentage of the population – to understand their effect on the age when HD symptoms show up.

This has historically been difficult to do, especially in a disease that is already rare, but a combination of statistics, computing power, clues from previous HD studies, and a wealth of willing HD participants can allow them to look at how particular genetic changes are likely to influence earlier and later stages of HD.

They are also studying “exosomes,” which you can think of as bubbles that carry genetic information. These are created and released by different types of cells, and are increasingly being explored as potential biomarkers.

The takeaway from these complex statistics and genetics is that we have increasingly powerful tools allowing HD scientists to dive deeper into the relationship between genes and symptoms observed in the clinic.

These talks have also been an important reminder of the contributions that the HD community makes to advancing HD research. Without HD families contributing to GWAS studies, and trials like Enroll-HD and HD-Clarity, what we know from the last few talks wouldn’t have been possible. THANK YOU!

Evan Eichler – Structural variation and hidden genetic risk

The next talk is from Evan Eichler from the University of Washington. He is not an HD researcher, but studies structural variation in genes – big changes like deletions and replacements of many letters – as opposed to the single-letter changes other speakers have been referring to.

These big (50+ letter) changes are very rare. Evan points out that a lot of the technologies HD researchers use to capture one-letter changes miss really big ones. Good news though – better sequencing technologies have been developed to do a better job of this.

Evan's lab played a role in filling in the gaps in the long-standing human genome project. We are now at a point where, if you have enough time and resources, you can parse 99.9% of the genome – and this continues to grow.

His group has recently worked out a CGG triplet repeat mutation which causes a rare disease called Barak-Oren syndrome. This is just one example of how his technology can be applied to do individualized analysis of human genomes for the purpose of ultra-rare diagnostics.

They also look at evolutionary history using genetic material from non-human primates like gorillas, gibbons, and macaques. Their overall goal is to use diverse animal and human genetic material to better understand the richness of human variation and how it drives disease.

They can use this wealth of data to zoom out and look at structural variants (big genetic changes) and how they differ by geography and racial background. A huge percentage of genetic changes are big ones like deletions and expansions (like we see with HD).

He can use detailed sequencing data to work backwards and identify genetic repeats that represent risk factors for common diseases like heart failure, which can then offer biological explanations and new therapeutic targets.

One source of his data is an US-based research program called All of Us, which attempted to recruit an extremely diverse group of Americans for inclusive genetics and medical records research. The researchers sought out diversity in racial, socioeconomic, geographic, and rare disease groups.



At the heart of HD research are families. Representatives from HD organisations help connect scientists, clinicians, and the community, ensuring research stays grounded in the needs of those most affected.

Evan's analyses revealed that there are some people who have HD-length CAG repeats (40+), but they do not have symptoms and do not have a family history of HD. It might be interesting to study why these folks are so resilient to symptoms!

He can also use these new methods of genome sequencing to discover new types of triplet repeats, and to infer new structural variants – previously unknown, large genetic changes – that could be causing both rare or common diseases in specific populations.

This is all possible because new genetic sequencing methods can pick up things that were previously missed!

Paolo Beuzer – An assay to track somatic expansion in living cells

Up next is Paolo Beuzer from CHDI. He'll be sharing work he's been doing with several collaborators on a new assay that can help with studying somatic expansion in HD.

While somatic expansion has been a hot topic in HD research, Paolo points out that there are a few bottlenecks in advancing this finding toward drug development.

A lot of our understanding of somatic expansion comes from post-mortem brain samples. But we can't take brain samples in a clinical trial to test if a drug can alter somatic expansion! We need ways to monitor somatic instability in living things, ideally not too invasively.

Mismatches, the bubble-outs in the DNA that often occur with expanded CAGs, are repaired with new DNA letters to fill in the gaps they create. Paolo suggests that we can infer somatic expansion by looking out for this newly synthesised DNA in the cell.

In a proof-of-concept experiment, the scientists feed a special type of DNA letter to the neurons, called EdU (pronounced E-D-U), which they can measure as it is used to fill in these gaps in the expansion bubbles in the HTT gene.

This experiment was a success! They could see the EdU but only in the HTT gene of cells which model HD, and not in healthy cells. The signal became more obvious in the HD cell models with longer and longer CAG numbers.

They also looked to see where else EdU was added into the genome during repair. They found good signal in repair hot spots, suggesting this new approach was working well. Paolo and his team are now confident they can see where repair is occurring, and therefore infer where expansion has happened.

Next they looked to see how this EdU signal might change if different DNA repair machinery components were blocked, like MSH2, MSH3, MSH6 and PMS1 (all known to affect age of HD symptom onset. The HTT gene repair signal was impacted by blocking these genes, but the other repair hot spots were not affected.

CHDI is not providing or is providing financial support to researchers who have work described in this article. Tam Maiuri works for the Huntington's Disease Society of America, which has relationships with many of the companies who attended this meeting or were mentioned in articles related to this conference. Leora Fox works with non profit and pharmaceutical entities in the HD space who attended this meeting.

For more information about our disclosure policy see our FAQ. They also worked in partnership with Veronica Brito's lab, who gave a talk earlier this conference. This system worked in her lab's models too – they

SUPPORT HD BUZZ blood cells. This suggests that blood samples from

people with HD could be used to track somatic expansion.

Claudia Langenberg – Multi-omics and AI for precision health in HD

The next talk is from Claudia Landenberg, who has positions in both London and Berlin. Her work uses large-scale data on both the molecular and clinical level to understand the effects of genetics on cells and organs

ent diseases.

- reinforces the fact that new tools are allowing us to connect with human health measures and symptoms on a large scale and is therefore. This can help us diagnose diseases and predict their

for the last ~10 years has been in its “big data” phase, which science calls “omics”. We have tons of data which clever scientists like Claudia are trawling through with cool computing tools to look for trends and patterns which can be used to better understand diseases, and maybe develop new drugs.

One of the biggest and best databases to study the gene-health connection is the UK Biobank. Big pharmaceutical companies and academic scientists have been analysing data to try and find links. The problem is that biology is blooming complex so the trends are not easily found.

Claudia shows us a huge dataset where most diseases do not have signals in their protein signatures that track robustly with disease – this is disappointing, but there are other datasets to consider.

Instead of considering proteins in isolation, they are looking at all kinds of readouts – genetics, proteomics, and other data, all in combination. This is called a multi-omics approach. Using AI tools, they can pull out trends and patterns too complicated to spot with regular statistical approaches.

Claudia and her powerhouse team have applied their analysis to see how well they are able to predict different diseases based on data signatures alone. They have had success with multiple myeloma, a type of blood cancer, and are expanding their approach to other diseases, including HD.



From all the whole polyQ posse, thanks for following along with our coverage!

This kind of analysis is only made possible because of the tens of thousands of folks who participate in data collection efforts. However, these databases are dominated by people of European ancestry, which means these trends might not hold true for people from other backgrounds.

To overcome this challenge, there are ongoing efforts to diversify these databases with participation from folks who come from all over the world. Hopefully this means that moving forward, gene-health connection research can be more inclusive.

Claudia has applied a bottom-up approach to connect very rare symptomatic observations with new genetic causes. She is also applying meta-analyses that combine findings from multiple studies for an even richer collection of "big data."

And that wraps the meeting! As always, we're floored by the novelty and diversity of ideas moving us closer to treatments for HD. Summary articles will follow, so stay tuned!

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