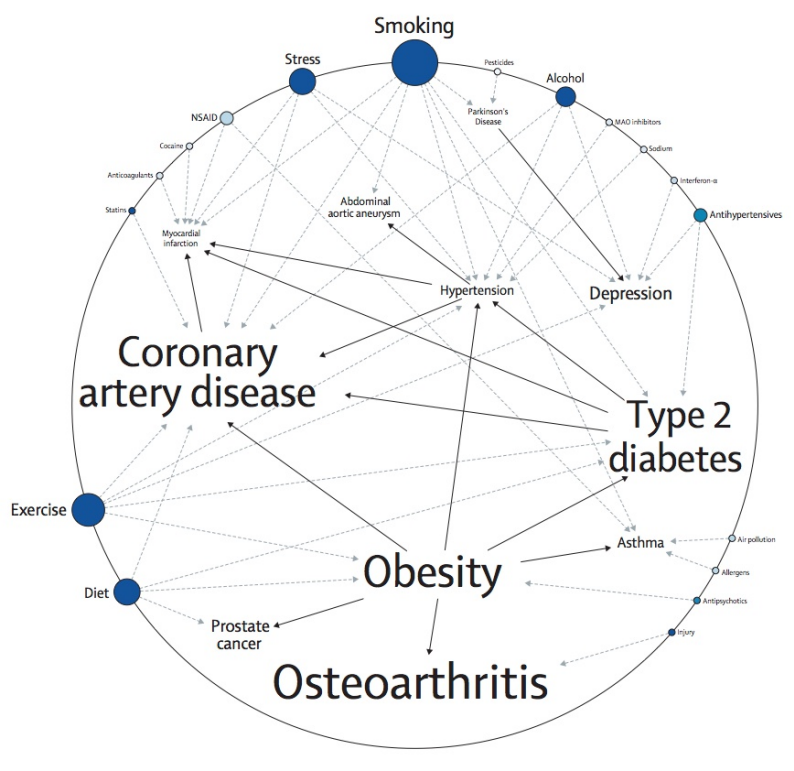
Your Genes and Your Health

http://bio84.stanford.edu/

What We Can Learn from Personal Genomics

http://bio84.stanford.edu/06%20Personal%20Genomics.html

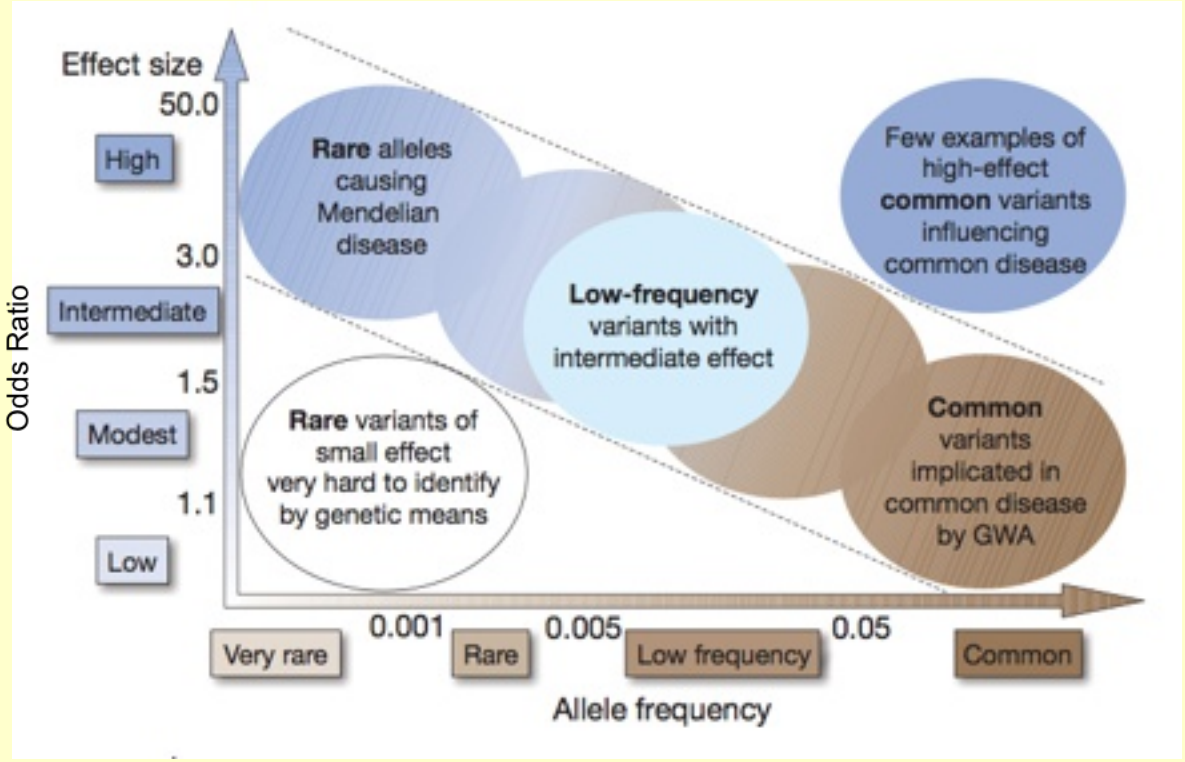


Doug Brutlag, Professor Emeritus of Biochemistry & Medicine (by courtesy)
Stanford University School of Medicine

Genetic Penetrance of Inherited Diseases

- Many inherited diseases are Mendelian and highly penetrant
  - Sickle cell disease
  - Thalassemias
  - Huntington's disease
  - Color blindness
  - Cystic fibrosis
- Most common diseases are complex (multifactorial - caused by multiple genes or multiple pathways as well as multiple environmental factors) and of low penetrance
  - Familial
  - Predisposition to disease
  - Very large environmental and/or behavioral component
    - Type I diabetes and other autoimmune diseases (lupus, rheumatoid arthritis, hyperthyroidism, Crohn's disease, Celiac Sprue, irritable bowel disease etc.)
    - Type 2 diabetes
    - Coronary heart disease (atherosclerosis)
    - Asthma, COPD, pulmonary fibrosis
  - Many complex diseases can be avoided with diet, nutrition, exercise or behavioral modification
  - Many complex diseases can also be monitored by increased vigilance (another behavioral modification)

Common SNPs have Low Odds Ratio and Low Heritability



- Rare High Penetrance Variants Carry High Risk
- Common SNPs Carry Low Risk
- Multiple Variants May Increase Risk Synergistically
- Common SNPs Associated with Genes Containing High Risk Alleles
- Common SNPs Associations can Suggest Regions to Sequence in Cohorts or Trios or Subpopulations

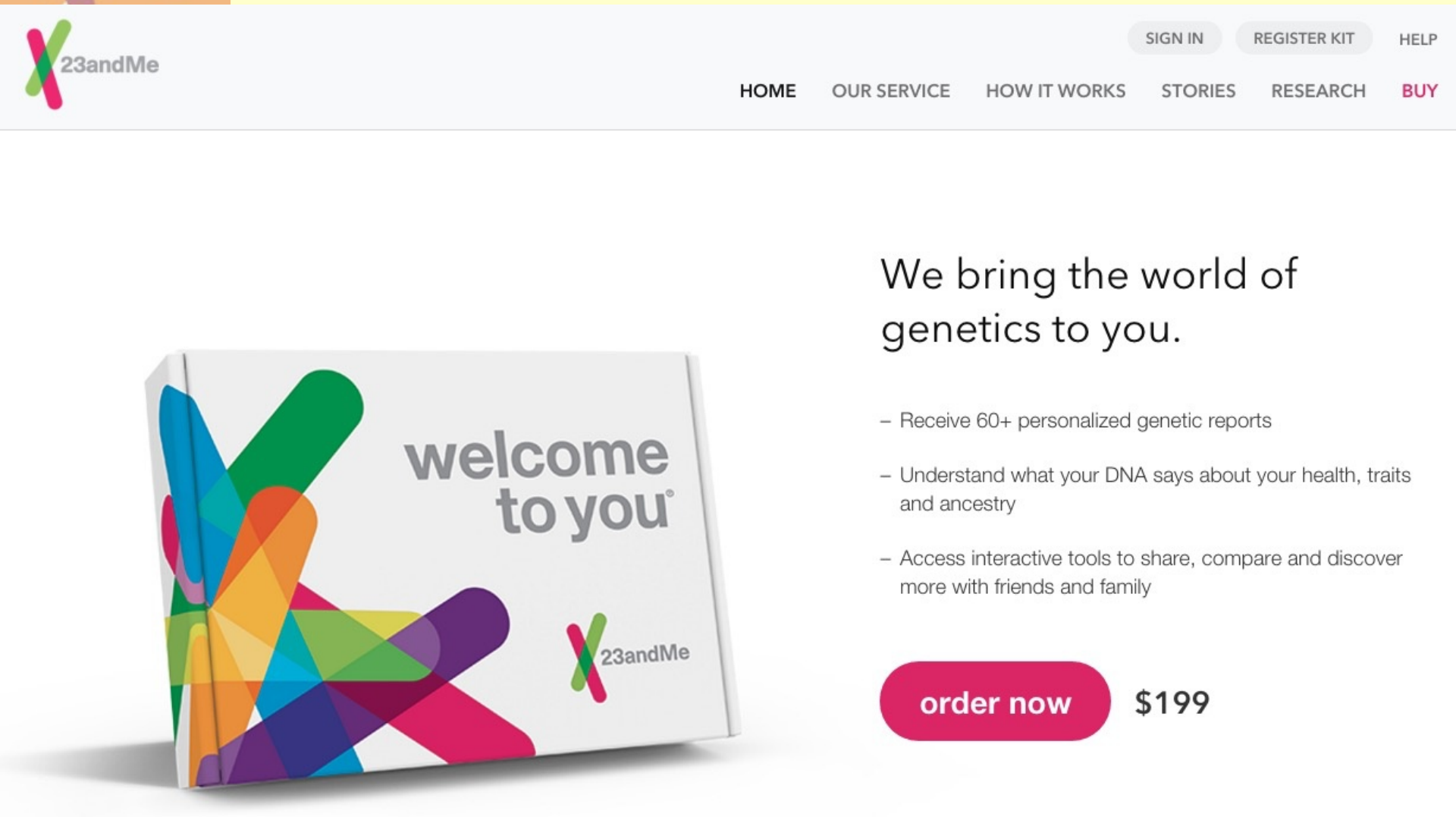
Manolio et al. Nature 461, 747-753 (2009)

So What Can We Learn from Personal Genomics?

- Disease risk for common diseases
  - Genetic predisposition towards a disease (relative risk or odds ratio)
  - Genetic versus environmental contributions to disease (penetrance)
  - How to alter your environment and behavior to avoid the disease
  - How to increase your vigilance for symptoms of specific diseases
- Disease carrier status
  - Premarital genetic counseling
  - Preimplantation genetic diagnosis
  - Neonatal diagnosis
    - Amniocentesis
    - Chorion villus sampling (CVS)
    - Non-Invasive Prenatal Testing tests for fetal DNA in pregnant mothe'rs blood
- Drug susceptibility
  - Efficacy of common drugs
  - Adverse reactions to common drugs
- Ancestry
  - One can follow maternal line using mitochondrial DNA SNPs
  - Males can follow paternal line using Y chromosome SNPs
  - Shared haplotypes with close relatives (up to 5th cousins)
- Familial traits, diseases and relationships
  - Known family diseases (breast cancers, colorectal cancer, lysosome storage diseases, etc.)
  - Paternity (10% of people do not know their true biological father)
  - Maternity (about 1% of people do not know their true biological mother)
  - Inbreeding and incest lead to increased homozygosity and recessive diseases
  - Orphans can find family relations

23andMe

http://23andme.com/



23andMe Spit Kit

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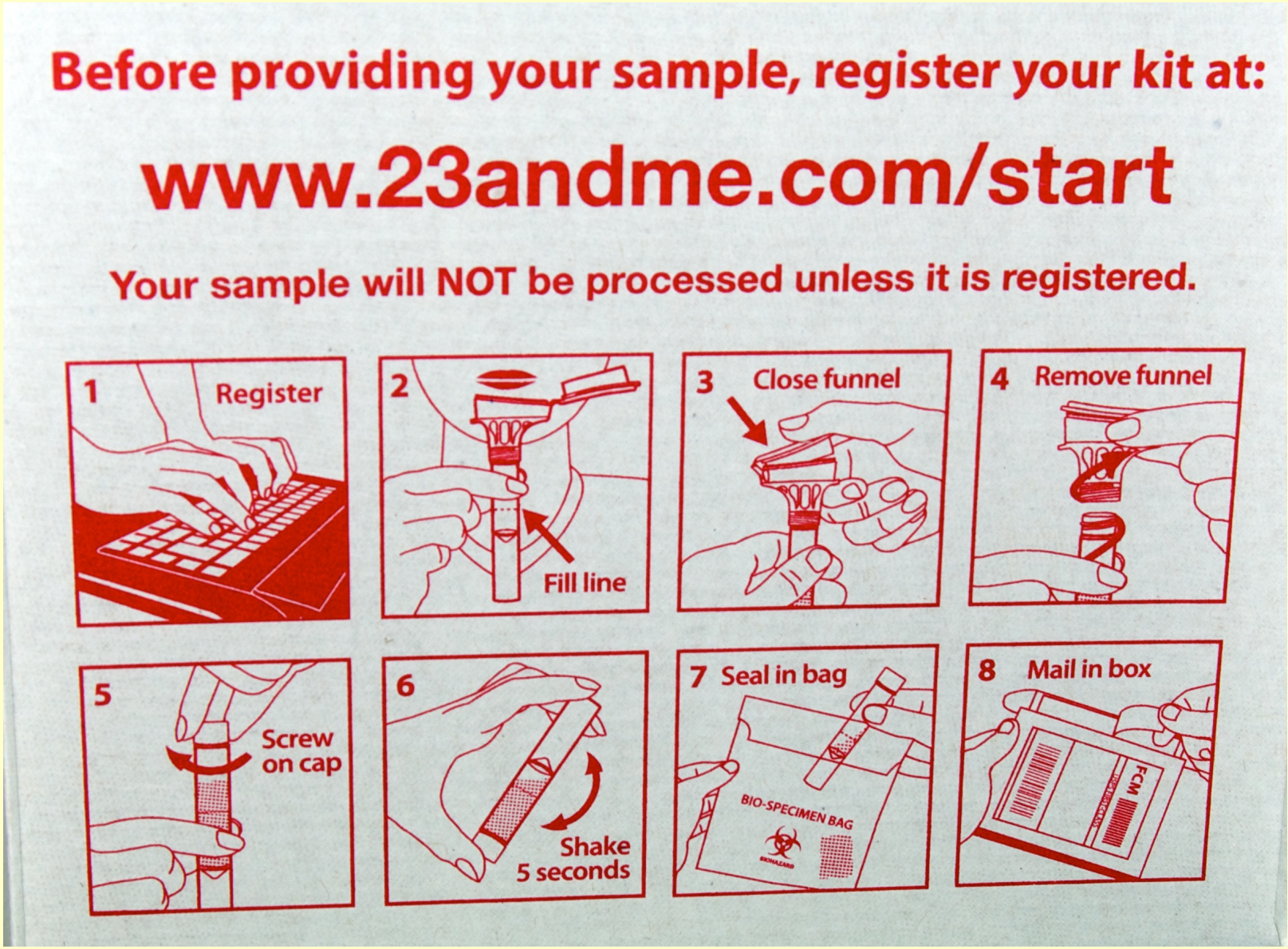

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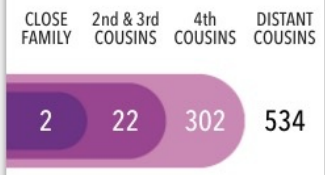
SCANDINAVIAN

ANCESTRY COMPOSITION

HEALTH OVERVIEW



DNA RELATIVES

NEANDERTHAL ANCESTRY

FEATURED CONTENT

Surgical Complications

In the case of a surgical procedure, planned or unplanned, this set of your genetic results and health history information would be important to share with your doctor.

BASED ON YOUR **6** GENETIC REPORTS & **12** SURVEY ANSWERS

23andMe Research

FEATURED SURVEY

ANCESTRY INFORMATION

TAKE SURVEY

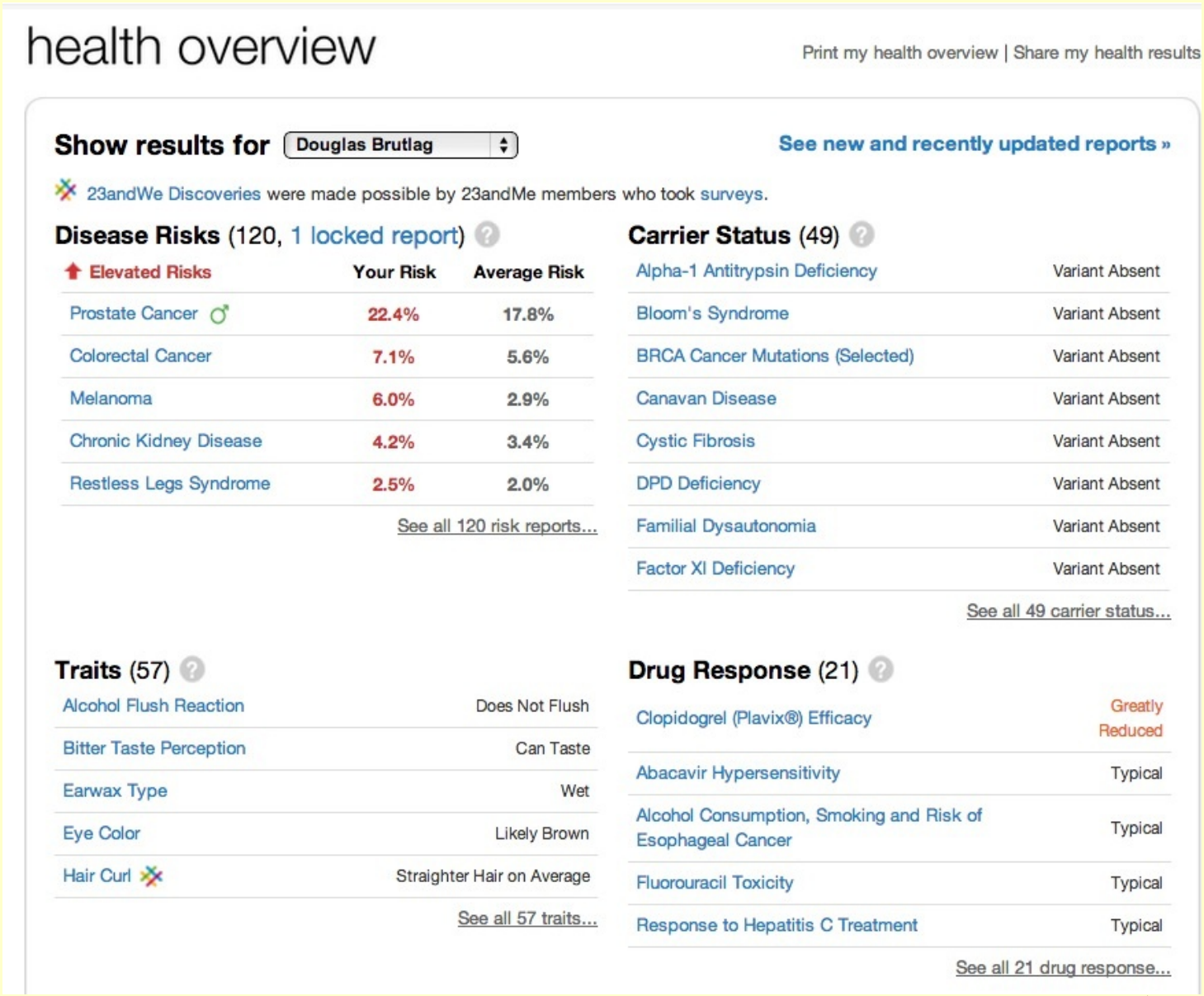
QUICK QUESTIONS

Q:
Have you been diagnosed with rapid eye movement (REM) sleep behavior disorder or been told that you act out your dreams while lying asleep in bed (as opposed to sleepwalking)?

- Yes
- No

23andMe Health Overview

http://23andme.com



health overview

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Show results for Douglas Brutlag

See new and recently updated reports »

23andWe Discoveries were made possible by 23andMe members who took surveys.

Disease Risks (120, 1 locked report)

Elevated Risks	Your Risk	Average Risk
Prostate Cancer ♂	22.4%	17.8%
Colorectal Cancer	7.1%	5.6%
Melanoma	6.0%	2.9%
Chronic Kidney Disease	4.2%	3.4%
Restless Legs Syndrome	2.5%	2.0%

See all 120 risk reports...

Carrier Status (49)

Alpha-1 Antitrypsin Deficiency	Variant Absent
Bloom's Syndrome	Variant Absent
BRCA Cancer Mutations (Selected)	Variant Absent
Canavan Disease	Variant Absent
Cystic Fibrosis	Variant Absent
DPD Deficiency	Variant Absent
Familial Dysautonomia	Variant Absent
Factor XI Deficiency	Variant Absent

See all 49 carrier status...

Traits (57)

Alcohol Flush Reaction	Does Not Flush
Bitter Taste Perception	Can Taste
Earwax Type	Wet
Eye Color	Likely Brown
Hair Curl	Straighter Hair on Average

See all 57 traits...

Drug Response (21)

Clopidogrel (Plavix®) Efficacy	Greatly Reduced
Abacavir Hypersensitivity	Typical
Alcohol Consumption, Smoking and Risk of Esophageal Cancer	Typical
Fluorouracil Toxicity	Typical
Response to Hepatitis C Treatment	Typical

See all 21 drug response...

23andMe Disease Risks

disease risk

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See new and recently updated reports ›

23andWe Discoveries were made possible by 23andMe members who took surveys.

Locked Reports

Name	Confidence	Your Risk	Avg. Risk	Compared to Average
Alzheimer's Disease	★★★★	🔒	🔒	🔒

Elevated Risk

Name	Confidence	Your Risk	Avg. Risk	Compared to Average
Prostate Cancer ♂	★★★★	22.4%	17.8%	1.26x
Colorectal Cancer	★★★★	7.1%	5.6%	1.27x
Melanoma	★★★★	6.0%	2.9%	2.10x
Chronic Kidney Disease	★★★★	4.2%	3.4%	1.22x
Restless Legs Syndrome	★★★★	2.5%	2.0%	1.25x
Exfoliation Glaucoma	★★★★	2.2%	0.7%	2.90x
Abdominal Aortic Aneurysm	★★★			
Ankylosing Spondylitis	★★★			
Asthma	★★★			

23andMe Prostate Cancer Risks

23andMe

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Prostate Cancer
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Prostate Cancer

Printable Version

Prostate cancer is by far the most common cancer affecting men. (Women don't have prostate glands and therefore cannot get prostate cancer, but can pass markers to their children.) About one in six men will develop prostate cancer over their lifetimes, according to the American Cancer Society. Fortunately, most prostate tumors grow slowly, and if detected early, treatment may help control their size. Until recently, the only well-known risk factors for prostate cancer were age, ethnicity, and family history. Although advanced age increases a person's risk for any type of cancer, the involvement of ethnicity and family history suggests that there is a strong genetic component as well.

The following results are based on ★★★★ **Established Research** for 12 reported markers, updated November 4th, 2010.

Learn more about the biology of Prostate Cancer...
Major discoveries in Prostate Cancer...

1 of 3. Prostate cancer affects about 1 in 6 men. (Women don't have prostate glands and therefore cannot get prostate cancer.)

23andMe Prostate Cancer Risks

Your Genetic Data

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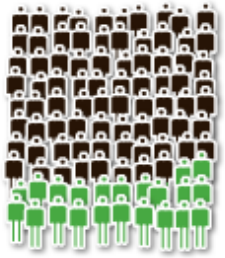
Show information for Douglas Brutlag assuming European ethnicity and an age range of 35–79

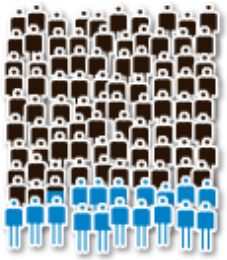
Where's mine?



Douglas Brutlag

22.4 out of 100

men of European ethnicity who share Douglas Brutlag's genotype will develop Prostate Cancer between the ages of 35 and 79.



Average

17.8 out of 100

men of European ethnicity will develop Prostate Cancer between the ages of 35 and 79.

What does the Odds Calculator show me?

Use the ethnicity and age range selectors above to see the estimated incidence of Prostate Cancer due to genetics for men with **Douglas Brutlag**'s genotype. The 23andMe Odds Calculator assumes that a person is free of the condition at the lower age in the range. You can use the name selector above to see the estimated incidence of Prostate Cancer for the genotypes of other people in your account.

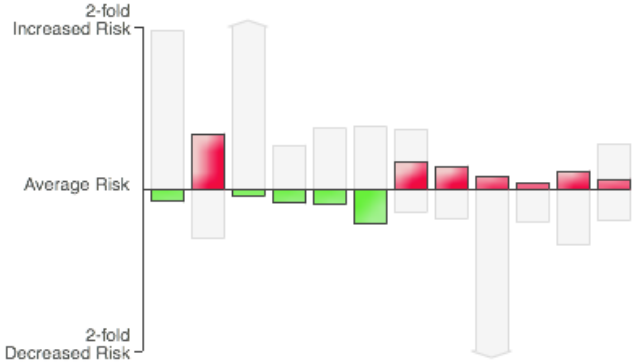
The 23andMe Odds Calculator only takes into account effects of markers with known associations that are also on our genotyping chip. Keep in mind that aside from genetics, environment and lifestyle may also contribute to one's risk for Prostate Cancer.

Genes vs. Environment

42-57 %
Attributable to Genetics

The heritability of prostate cancer is estimated to be 42-57%. This means that genetic and environmental factors contribute nearly equally to differences in risk for this condition. (If you are a woman, you have no chance of getting this type of cancer, but if you have sons, their risk may be affected by what they inherit from you.) Genetic factors that play a role in prostate cancer include both unknown factors and known factors such as the SNPs we describe. Other factors that can increase your risk include being older, having African ancestry, or living in North America, Northwestern Europe, Australia, or the Caribbean islands. The effect of nationality may be tied to diet, as a diet high in red meat and high-fat dairy products, and low in fruits and vegetables, may also put you at increased risk. (sources)

Marker Effects



What does this chart show?

The chart shows the approximate effects of the selected person's genotype at the 12 reported markers. Higher, red bars indicate increased risk from the average, while lower, green bars indicate decreased risk from the average. The light gray bars show the maximum possible effects for the possible genotypes at the marker.

Mouse over individual bars to view additional information about each marker. Click on a bar to view detailed information about that marker below. You can read more about all markers in the technical report.

8q24 (region 1)

Marker: rs1447295

Three SNPs in the same area of the genome have recently been found to be independently associated with prostate cancer risk. This region is called 8q24, because it lies within band 24 on the long arm (named the "q" arm) of chromosome 8. The three SNPs are not close to known genes (although there are others located farther away). But other studies have looked at DNA from prostate tumors and found that in the cancerous cells, this area of the genome often has unusual duplications, or extra copies of DNA.

The duplications might contribute to the progression of prostate cancer (for example, by increasing the number of genes related to cell growth), or they might simply be a side effect of the high mutation rate seen in all types of cancer cells. Similarly, the risk-associated versions of the SNPs in the 8q24 region might directly affect activity levels of genes involved in prostate cancer, or they might somehow make it easier for DNA duplications to occur. (And, they might only be linked to yet-unknown SNPs that are directly involved.)

One study has investigated this association in Japanese Americans. Although the SNP also appears to be associated with prostate cancer risk in this population, evidence suggests that the effect of this SNP on risk may differ between populations. Therefore, the exact association in populations with Asian ancestry still needs to be confirmed.

Citations

Amundadottir et al. (2006) . "A common variant associated with prostate cancer in European and African populations." *Nat Genet* 38(6):652-8.

Freedman et al. (2006) . "Admixture mapping identifies 8q24 as a prostate cancer risk locus in African-American men." *Proc Natl Acad Sci U S A* 103(38):14068-73.

Severi et al. (2007) . "The common variant rs1447295 on chromosome 8q24 and prostate cancer risk: results from an Australian population-based case-control study." *Cancer Epidemiol Biomarkers Prev* 16(3):610-2.

Yeager et al. (2007) . "Genome-wide association study of prostate cancer identifies a second risk locus at 8q24." *Nat Genet* 39(5):645-9.

Gudmundsson et al. (2007) . "Genome-wide association study identifies a second prostate cancer susceptibility variant at 8q24." *Nat Genet* 39(5):631-7.

Wang et al. (2007) . "Two common chromosome 8q24 variants are associated with increased risk for prostate cancer." *Cancer Res* 67(7):2944-50.

Schumacher et al. (2007) . "A common 8q24 variant in prostate and breast cancer from a large nested case-control study." *Cancer Res* 67(7):2951-2956.

Suuriniemi et al. (2007) . "Confirmation of a positive association between prostate cancer risk and a locus at chromosome 8q24." *Cancer Epidemiol Biomarkers Prev* 16(4):809-14.

Cheng et al. (2008) . "8q24 and prostate cancer: association with advanced disease and meta-analysis." *Eur J Hum Genet* 16(4):496-505.

Zheng et al. (2008) . "Cumulative association of five genetic variants with prostate cancer." *N Engl J Med* 358(9):910-9.

23andMe Disease Risks

Decreased Risk

Name	Confidence	Your Risk	Avg. Risk	Compared to Average
Type 2 Diabetes	★★★★	19.2%	25.7%	0.75x
Age-related Macular Degeneration	★★★★	2.9%	6.5%	0.44x
Rheumatoid Arthritis	★★★★	1.2%	2.4%	0.52x
Esophageal Squamous Cell Carcinoma (ESCC)	★★★★	0.29%	0.36%	0.80x
Crohn's Disease	★★★★	0.26%	0.53%	0.50x
Multiple Sclerosis	★★★★	0.20%	0.34%	0.59x
Stomach Cancer (Gastric Cardia Adenocarcinoma)	★★★★	0.18%	0.23%	0.77x
Type 1 Diabetes	★★★★	0.07%	1.02%	0.07x
Primary Biliary Cirrhosis	★★★★	0.05%	0.08%	0.66x
Celiac Disease	★★★★	0.03%	0.12%	0.28x
Atrial Fibrillation: Preliminary Research	★★★			

23andMe Type 2 Diabetes Risks

disease risk

Next ▸
Ulcerative Colitis

Type 2 Diabetes
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Type 2 Diabetes

Printable Version

The most common type of diabetes, type 2 diabetes mellitus occurs when chronically high blood sugar levels cause a breakdown of the body's natural response to eating sweets and starches. Left untreated, type 2 diabetes can result in kidney failure, blindness, and circulatory problems that increase the risk of heart attack or stroke. In the United States, almost 21 million children and adults have diabetes, but the rate of new diagnoses is increasing.

The following results are based on ★★★★ **Established Research** for 11 reported markers, updated March 24th, 2011.

Learn more about the biology of Type 2 Diabetes...
Major discoveries in Type 2 Diabetes...

1 of 3. Smart choices about diet can help delay or prevent type 2 diabetes.

23andMe Type 2 Diabetes Risks

Your Genetic Data

» Share your health results

Show information for Douglas Brutlag assuming European ethnicity

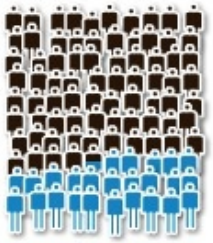
Why are there limited choices of ethnicity in risk reports?

and an age range of 20-79

Douglas Brutlag

19.2 out of 100

men of European ethnicity who share Douglas Brutlag's genotype will develop Type 2 Diabetes between the ages of 20 and 79.



Average

25.7 out of 100

men of European ethnicity will develop Type 2 Diabetes between the ages of 20 and 79.

What does the Odds Calculator show me?

Use the ethnicity and age range selectors above to see the estimated incidence of Type 2 Diabetes due to genetics for men with **Douglas Brutlag**'s genotype. The 23andMe Odds Calculator assumes that a person is free of the condition at the lower age in the range. You can use the name selector above to see the estimated incidence of Type 2 Diabetes for the genotypes of other people in your account.

The 23andMe Odds Calculator only takes into account effects of markers with known associations that are also on our genotyping chip. Keep in mind that aside from genetics, environment and lifestyle may also contribute to one's risk for Type 2 Diabetes.

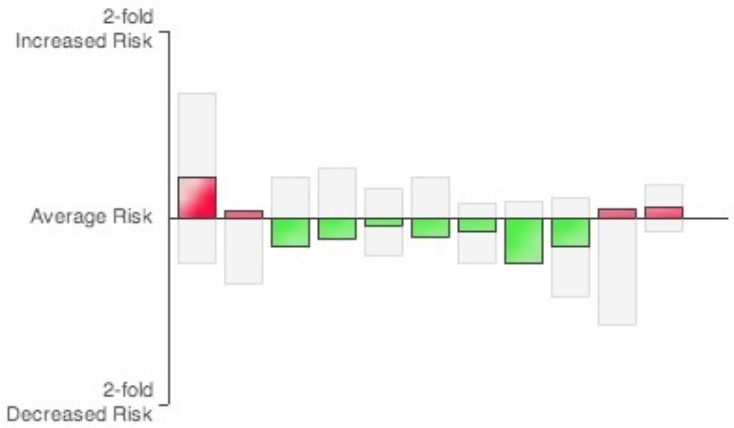
Genes vs. Environment

26 %
Attributable to Genetics

The heritability of type 2 diabetes is estimated to be 26%. This means that environmental factors contribute more to differences in risk for this condition than genetic factors. Genetic factors that play a role in type 2 diabetes include both unknown factors and known factors such as the SNPs we describe here. Environmental factors include obesity, gestational diabetes, giving birth to at least one baby weighing nine pounds or more, high blood pressure, abnormal cholesterol levels, physical inactivity, polycystic ovarian syndrome, other clinical conditions associated with insulin resistance, a history of impaired glucose tolerance or impaired fasting glucose, and a history of cardiovascular disease. (sources)

23andMe Type 2 Diabetes Risks

Marker Effects



What does this chart show?

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Mouse over individual bars to view additional information about each marker. Click on a bar to view detailed information about that marker below. You can read more about all markers in the technical report.

TCF7L2

Marker: rs7903146

This SNP is located in the TCF7L2 gene, which encodes a protein involved in cell signalling. How TCF7L2 affects the development of type 2 diabetes is not completely understood. TCF7L2 has been shown to be involved in the development of pancreatic islets, which contain insulin-producing beta cells. Studies suggest that the T version of this SNP is associated with impaired baseline insulin secretion.

The T version of this SNP is also associated with increased odds of gestational diabetes, a form of diabetes that occurs only during pregnancy. Gestational diabetes can lead to complications for both mother— such as difficult delivery due to unusually large infant size— and baby, such as low blood sugar and breathing problems.

Citations

Grant et al. (2006) . "Variant of transcription factor 7-like 2 (TCF7L2) gene confers risk of type 2 diabetes." *Nat Genet* 38(3):320-3.

Saxena et al. (2006) . "Common single nucleotide polymorphisms in TCF7L2 are reproducibly associated with type 2 diabetes and reduce the insulin response to glucose in nondiabetic individuals." *Diabetes* 55(10):2890-5.

Helgason et al. (2007) . "Refining the impact of TCF7L2 gene variants on type 2 diabetes and adaptive evolution." *Nat Genet* 39(2):218-225.

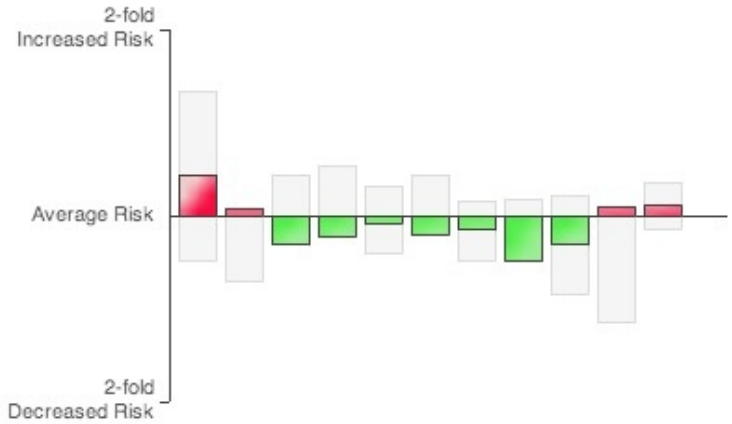
Sladek et al. (2007) . "A genome-wide association study identifies novel risk loci for type 2 diabetes." *Nature* 445(7130):881-5.

Saxena et al. (2007) . "Genome-wide association analysis identifies loci for type 2 diabetes and triglyceride levels." *Science* 316(5829):1331-6.

Zeggini et al. (2007) . "Replication of genome-wide association signals in UK samples reveals risk loci for type 2 diabetes." *Science* 316(5829):1336-41.

23andMe Type 2 Diabetes Risks

Marker Effects



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Mouse over individual bars to view additional information about each marker. Click on a bar to view detailed information about that marker below. You can read more about all markers in the technical report.

MTNR1B

Marker: rs1387153

This SNP is located near the MTNR1B gene, which encodes a pancreatic beta cell protein that interacts with a hormone called melatonin. In healthy individuals, insulin secretion follows a circadian rhythm with peaks during the day and troughs at night. Melatonin levels have the opposite pattern being highest during the night and thus may inhibit insulin secretion, possibly through the MTNR1B protein. Studies have shown that melatonin receptors like MTNR1B are overexpressed in pancreatic islets of individuals with type 2 diabetes compared to non-diabetic individuals.

Multiple studies have confirmed this association in populations with European ancestry. This association has not been studied in Asian or African populations.

Citations

Voight BF et al. (2010) . "Twelve type 2 diabetes susceptibility loci identified through large-scale association analysis." *Nat. Genet.* 42(7):579-89.

Bouatia-Naji N et al. (2009) . "A variant near MTNR1B is associated with increased fasting plasma glucose levels and type 2 diabetes risk." *Nat. Genet.* 41(1):89-94.

Prokopenko I et al. (2009) . "Variants in MTNR1B influence fasting glucose levels." *Nat. Genet.* 41(1):77-81.

Peschke E (2008) . "Melatonin, endocrine pancreas and diabetes." *J. Pineal Res.* 44(1):26-40.

23andMe Carrier Status

carrier status

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23andWe Discoveries were made possible by 23andMe members who took surveys.

Name	Confidence	Confidence
Alpha-1 Antitrypsin Deficiency	★★★★	Variant Absent
Bloom's Syndrome	★★★★	Variant Absent
BRCA Cancer Mutations (Selected)	★★★★	Variant Absent
Canavan Disease	★★★★	Variant Absent
Cystic Fibrosis	★★★★	Variant Absent
DPD Deficiency	★★★★	Variant Absent
Familial Dysautonomia	★★★★	Variant Absent
Factor XI Deficiency	★★★★	Variant Absent
Fanconi Anemia (FANCC-related)	★★★★	Variant Absent
Familial Hypercholesterolemia Type B	★★★★	Variant Absent
Familial Mediterranean Fever	★★★★	Variant Absent
G6PD Deficiency	★★★★	Variant Absent
Gaucher Disease	★★★★	Variant Absent
Glycogen Storage Disease Type 1a	★★★★	Variant Absent
Hemochromatosis (HFE-related)	★★★★	Variant Absent
Limb-girdle Muscular Dystrophy	★★★★	Variant Absent
Maple Syrup Urine Disease Type 1B	★★★★	Variant Absent

23andMe Carrier Status for Alpha-1 Antitrypsin Deficiency

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Alpha-1 Antitrypsin Deficiency
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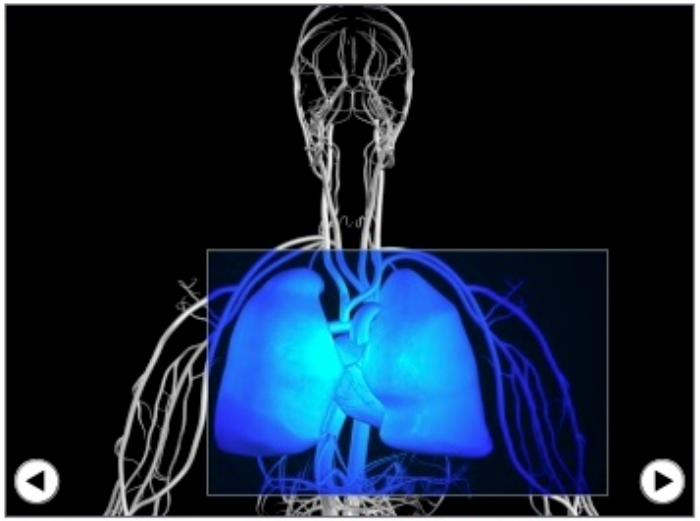
Alpha-1 Antitrypsin Deficiency

Printable Version

The alpha-1 antitrypsin (AAT) protein protects the body, especially fragile lung tissues, from the damaging effects of a powerful enzyme called neutrophil elastase that is released from white blood cells. In AAT deficiency, a genetic mutation reduces levels of the protective protein in the bloodstream. AAT deficiency can lead to chronic obstructive pulmonary disease (COPD), specifically emphysema, and liver disease. Smoking, which can inhibit what little AAT protein an affected person does have, increases the risk of lung disease.

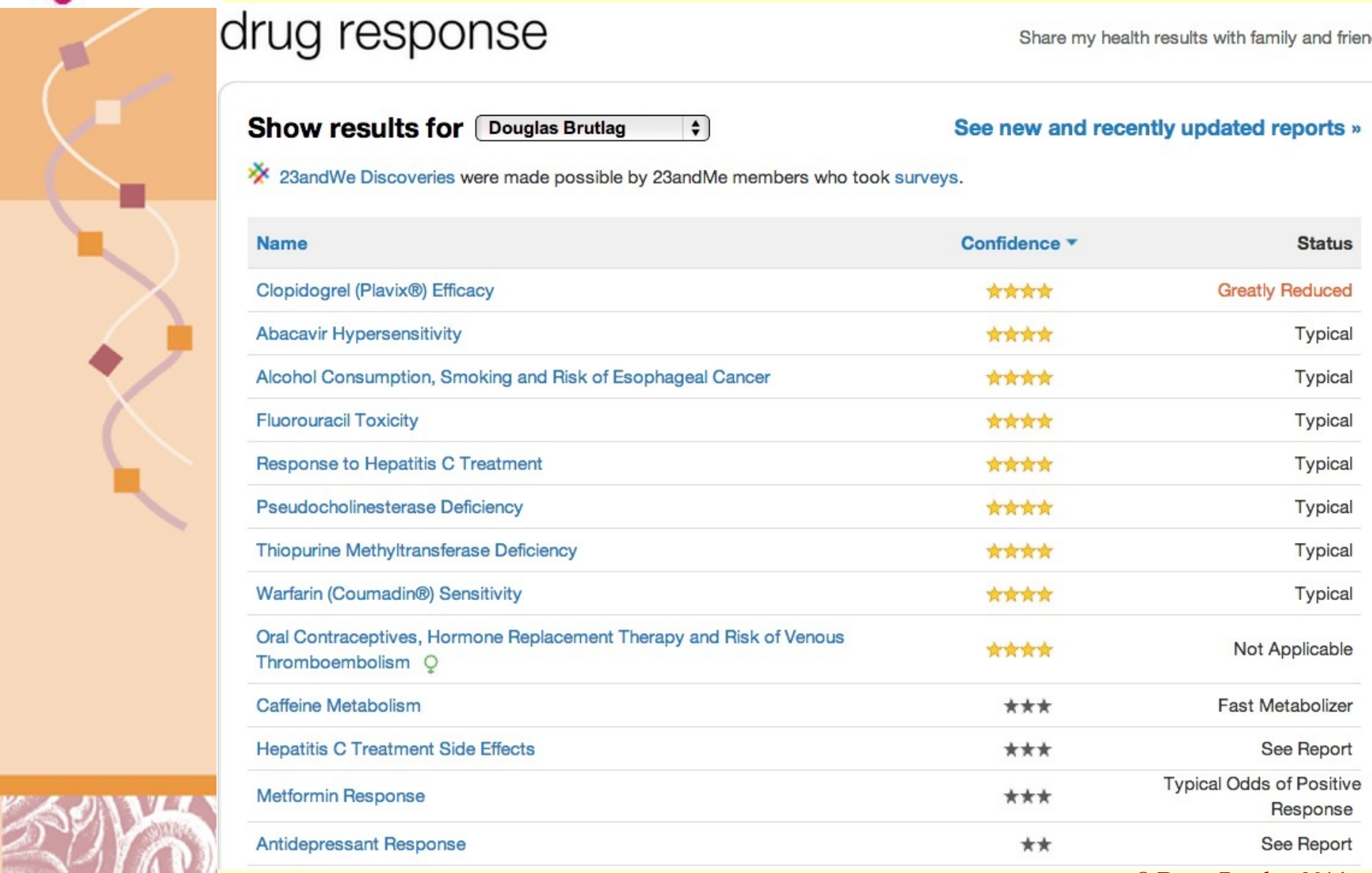
The following results are based on ★★★★ **Established Research** for 2 reported markers.

Learn more about the biology of Alpha-1 Antitrypsin Deficiency...



1 of 3. Low levels of alpha-1 antitrypsin can lead to COPD.

23andMe Drug Responses



drug response

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Show results for Douglas Brutlag

See new and recently updated reports »

23andWe Discoveries were made possible by 23andMe members who took surveys.

Name	Confidence	Status
Clopidogrel (Plavix®) Efficacy	★★★★	Greatly Reduced
Abacavir Hypersensitivity	★★★★	Typical
Alcohol Consumption, Smoking and Risk of Esophageal Cancer	★★★★	Typical
Fluorouracil Toxicity	★★★★	Typical
Response to Hepatitis C Treatment	★★★★	Typical
Pseudocholinesterase Deficiency	★★★★	Typical
Thiopurine Methyltransferase Deficiency	★★★★	Typical
Warfarin (Coumadin®) Sensitivity	★★★★	Typical
Oral Contraceptives, Hormone Replacement Therapy and Risk of Venous Thromboembolism ♀	★★★★	Not Applicable
Caffeine Metabolism	★★★	Fast Metabolizer
Hepatitis C Treatment Side Effects	★★★	See Report
Metformin Response	★★★	Typical Odds of Positive Response
Antidepressant Response	★★	See Report

Clopidogrel (Plavix®) Efficacy

My Home
Inbox (6)

My Health
Disease Risk
Carrier Status
Drug Response
Traits
Health Labs

My Ancestry
Maternal Line
Paternal Line
Relative Finder
Ancestry Painting
Global Similarity
Ancestry Labs

Sharing & Community
Compare Genes
Family Inheritance
23andMe Community
Genome Sharing

23andWe
Research Surveys (24)
Research Snippets

drug response

Clopidogrel (Plavix®) Efficacy ★★★★

share this

Established Research report on 5 reported markers.

Your Data | How It Works | Resources | Technical Report

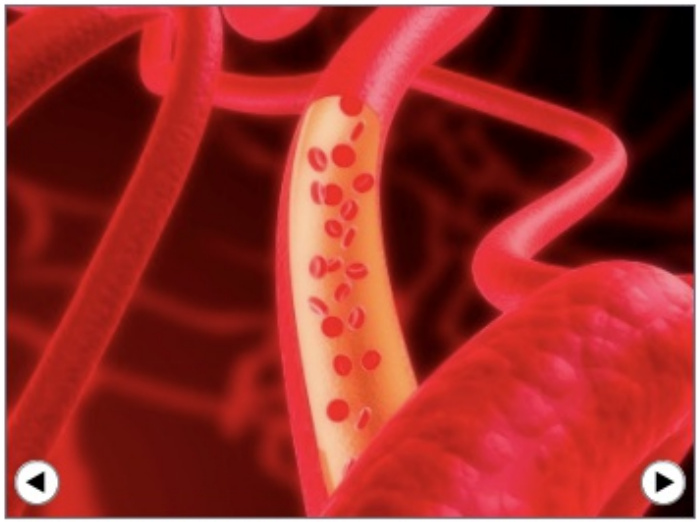
Next ▸ *Floxacillin Toxicity*

Only a medical professional can determine whether clopidogrel is the right medication for a particular patient. The information contained in this report should not be used to independently establish a clopidogrel regimen, or abolish or adjust an existing course of treatment.

About Clopidogrel Efficacy — Printable Version

Clopidogrel (sold under the trade names Plavix®, Iscover®, Clopilet® and Ceruvin®) is a drug commonly prescribed in combination with aspirin to help prevent blood clots that can block blood flow and cause a heart attack or stroke. However, clopidogrel doesn't inhibit clotting to the same extent in everyone. For some people, genetic variations that prevent the drug from being converted into its active form in the body are the cause. Studies have shown that people who are taking clopidogrel who have these genetic variations may have reduced protection from heart attacks, strokes and death from cardiovascular causes.

Learn more about the biology of Clopidogrel Efficacy...



1 of 3. Clopidogrel keeps platelets from sticking together and prevents blood clots.

Plavix Ad with Genetic Requirement

My Home
Inbox (3)

My Health
Disease Risk
Carrier Status
Drug Response
Traits
Health Labs

My Ancestry
Maternal Line
Paternal Line
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23andWe
Research Surveys (21)
Research Snippets
Research Initiatives
Research Discoveries

traits

Share my health results with family and friends

Show results for Douglas Brutlag

See new and recently updated reports »

23andWe Discoveries were made possible by 23andMe members who took surveys.

Name	Confidence ▲	Outcome
Alcohol Flush Reaction	★★★★	Does Not Flush
Bitter Taste Perception	★★★★	Can Taste
Earwax Type	★★★★	Wet
Eye Color	★★★★	Likely Brown
Hair Curl	★★★★	Straighter Hair on Average
Lactose Intolerance	★★★★	Likely Tolerant
Malaria Resistance (Duffy Antigen)	★★★★	Not Resistant
Male Pattern Baldness ♂	★★★★	Decreased Odds
Muscle Performance	★★★★	Likely Sprinter
Non-ABO Blood Groups	★★★★	See Report
Norovirus Resistance	★★★★	Not Resistant
Resistance to HIV/AIDS	★★★★	Not Resistant
Smoking Behavior	★★★★	Typical
Adiponectin Levels new	★★★	See Report
Asparagus Metabolite Detection	★★★	Typical Odds of Detecting
Birth Weight	★★★	See Report
Blood Glucose	★★★	5.18 mmol/L on Average
Breastfeeding and IQ	★★★	See Report
C-reactive Protein Level	★★★	2.09 mg/L on Average

Choice of GWAS Studies

- Common traits of broad interest
  - Prevalence of > 1%
  - Report Mendelian traits when possible
  - Focus on drug responses
- Avoid false discoveries
  - Large case-control studies > 750 cases
  - Highly significant expectation values (<0.01 errors)
  - Published in reputable journals
  - Studies that have been replicated
- May impute highly linked missing SNPs
- Calculate likelihood and odds ratio using customers ethnicity as detected
- Distinguish preliminary studies (non-replicated or smaller sample sizes) from established research.

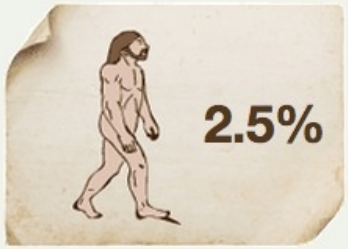
ancestry overview

Your Father's Line

Your father's line was likely in eastern Africa 50,000 years ago. Today that line is still found primarily in Africa.

Neanderthal Ancestry

You have an estimated **2.5%** Neanderthal DNA, which puts you in the **39th** percentile among Northern European 23andMe members.



Your Extended DNA Family

Guess what? If you have a large piece of identical DNA in common with someone, then you're related. You have **505** DNA relatives in 23andMe. Explore their info to learn more about your own ancestry.

Close Family	1st-2nd Cousins	3rd-4th Cousins	Distant Cousins
2	0	60	443

Your Mother's Line

Along your mother's line, you have ancestry in **Europe/the Near East** in the past few hundred years, that traces back to eastern Africa around 50,000 years ago.

From Your Ancestry Expert

It's remarkable what you can discover from a little saliva. On this page are the highlights of what we've learned about your ancestry, based just on your DNA. Enjoy!

Dr. Joanna Mountain, PhD

Joanna Mountain is 23andMe's Senior Director of Research. A former Stanford professor, she has traveled the world studying genetics and human history.

AS SEEN ON

ANDERSON

Ancestry Help

Send Feedback

Top Relative Surnames

Surname	Count	Enrichment
Anderson	5	10
Smith	5	1

23andMe Maternal Inheritance

Maternal Haplogroup: U5b2a

Share

Map | History | Haplogroup Tree | Community

Maternal Haplogroup: U5b2a

U5b2a is a subgroup of U5, which is described below.

Locations of haplogroup U5 circa 500 years ago, before the era of intercontinental travel.

Haplogroup U5 arose among early colonizers of Europe around 40,000 years ago; maternal descendants of those early colonizers persist in the region to this day. After the last Ice Age two subgroups of U5 expanded across Europe and into northern Africa and the Near East. Today, one subgroup, U5b1b, is shared by groups as diverse as the northern African desert-dwelling Berbers and the Scandinavian Arctic-dwelling Saami, also known as the Lapps.

Haplogroup: U5, a subgroup of R

Age: 40,000 years

Region: Europe, Near East, North Africa

Populations: Basques, Saami (Lapps) of northern Scandinavia

Highlight: Though primarily a European haplogroup, U5 was recently found in mitochondrial DNA extracted from the remains of a 6th-century AD Chinese chieftain.

Human Prehistory Videos

Human Prehistory: Prologue

Out of (Eastern) Africa

Your Family and Friends

A2	Samantha Hill
D4e2	Japanese Person
D5a2a'c	Chinese Person
H3	Lilly Mendel (Mom), Erin Mendel (Daughter), Alan Mendel (Son), Ian Mendel (Son), Margo Fisher (Grandma)
H4a1	Ron Fisher (Grandpa)
K1a1b1a	Benjamin Brutlag, Pauline Brutlag, Simone Brutlag
L3e2b2	Nigerian Person
M35b	renu heller

23andMe Paternal Inheritance

Paternal Haplogroup: E1b1b1a2*

Share

Map | History | Haplogroup Tree | Community

Paternal Haplogroup: E1b1b1a2*

E1b1b1a2* is a subgroup of E1b1b1a, which is described below.

Locations of haplogroup E1b1b1a circa 500 years ago, before the era of intercontinental travel.

E1b1b1a is most common in northern Africa and southern Europe. It arose about 23,000 years ago in eastern Africa and spread into the Mediterranean region after the Ice Age. E1b1b1a, a subgroup of E1b1b, expanded out of the Near East 8,000 years ago into northern Africa and southern Europe. Today it is one of the most common haplogroups in those regions.

Haplogroup: E1b1b1a, a subgroup of E1b1b

Age: 23,000 years

Region: Northern Africa, Southern Europe

Populations: Berbers, Iberians, Balkans

Highlight: Two different migrations brought E1b1b1a into Europe.

Your Family and Friends

D2a1b	Japanese Person
E1b1a8a1...	Nigerian Person
E1b1b1a2...	Douglas Brutlag, Benjamin Brutlag
G2a	Brian Becker
I1*	Greg Mendel (Dad), Alan Mendel (Son), Ian Mendel (Son), Fred Mendel (Grandpa)

ancestry overview

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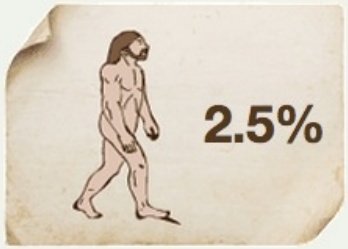

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AS SEEN ON

ANDERSON

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Send Feedback

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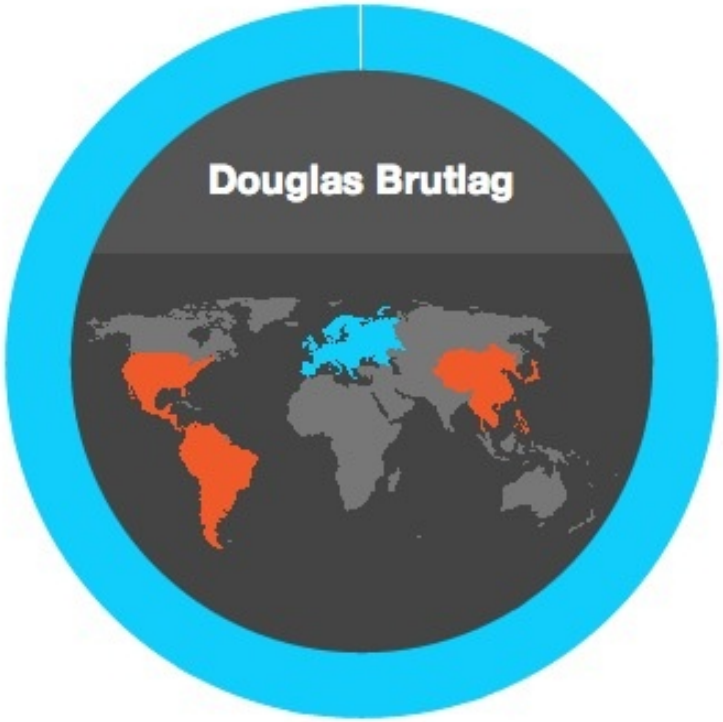
Smith	5	1

Ancestry Composition

ancestry composition

Douglas Brutlag | Standard Estimate

Map View | Global Resolution



Ancestry Composition tells you what percent of your DNA comes from each of 22 populations worldwide. The analysis includes DNA you received from all of your ancestors, on both sides of your family. The results reflect where your ancestors lived 500 years ago, before ocean-crossing ships and airplanes came on the scene.

99.9%	European
< 0.1%	East Asian & Native American
0.1%	Unassigned
100.0%	**Douglas Brutlag**

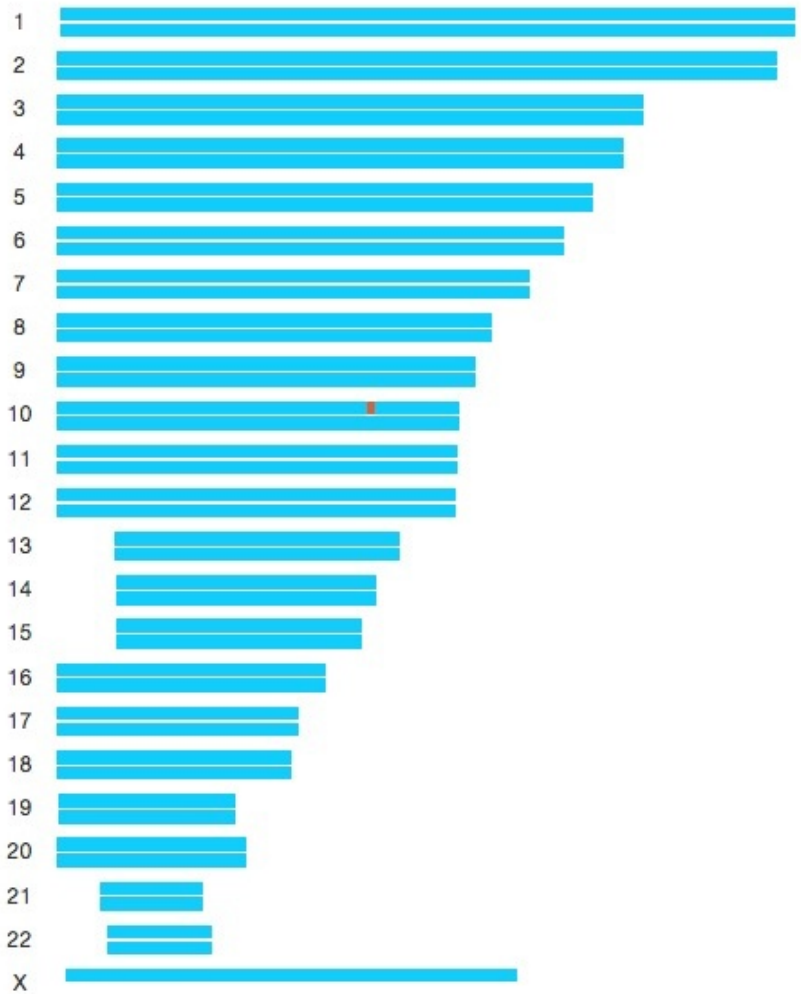
show all populations

Ancestry Composition

ancestry composition

Douglas Brutlag | Speculative Estimate

Chromosome View | Global Resolution



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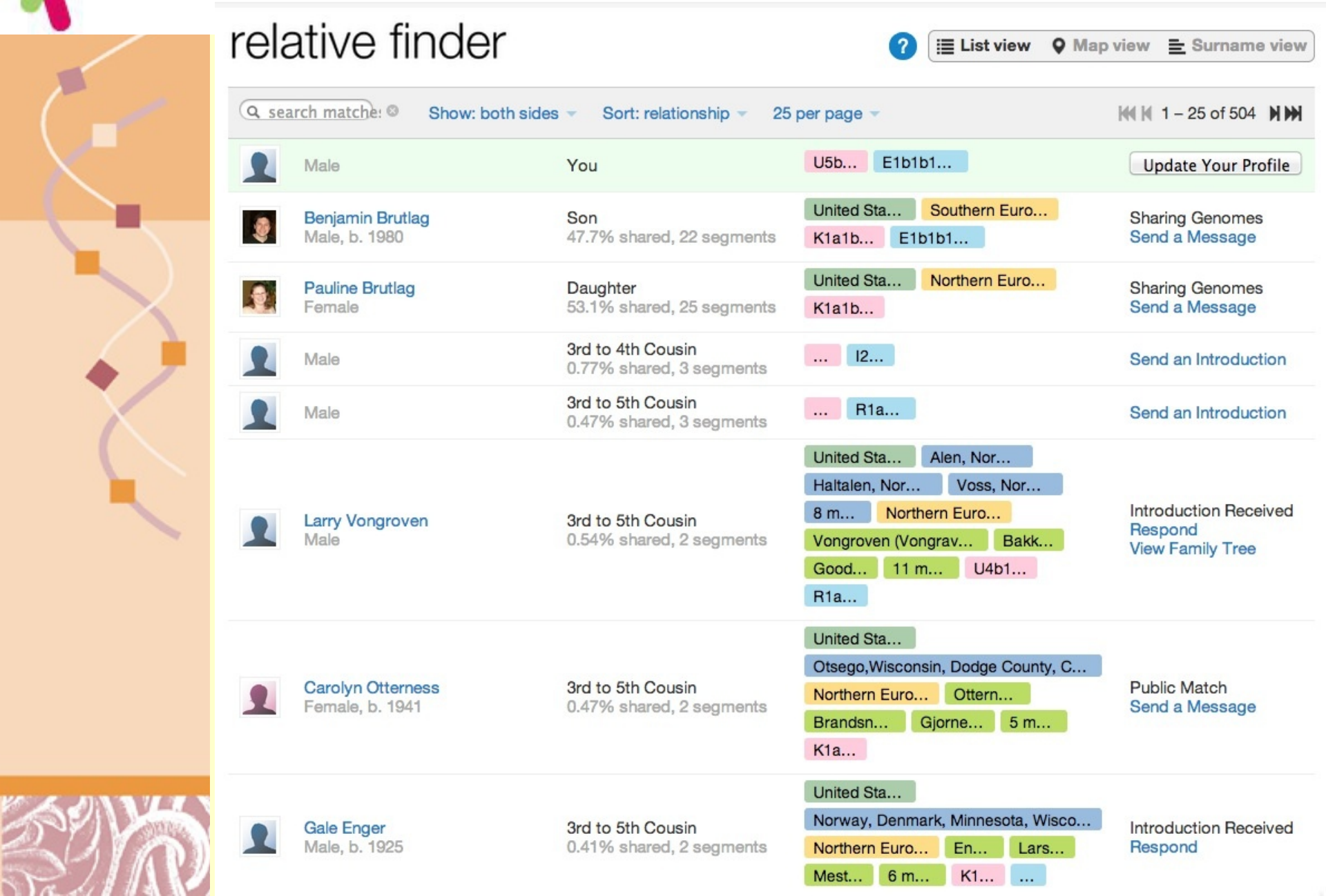
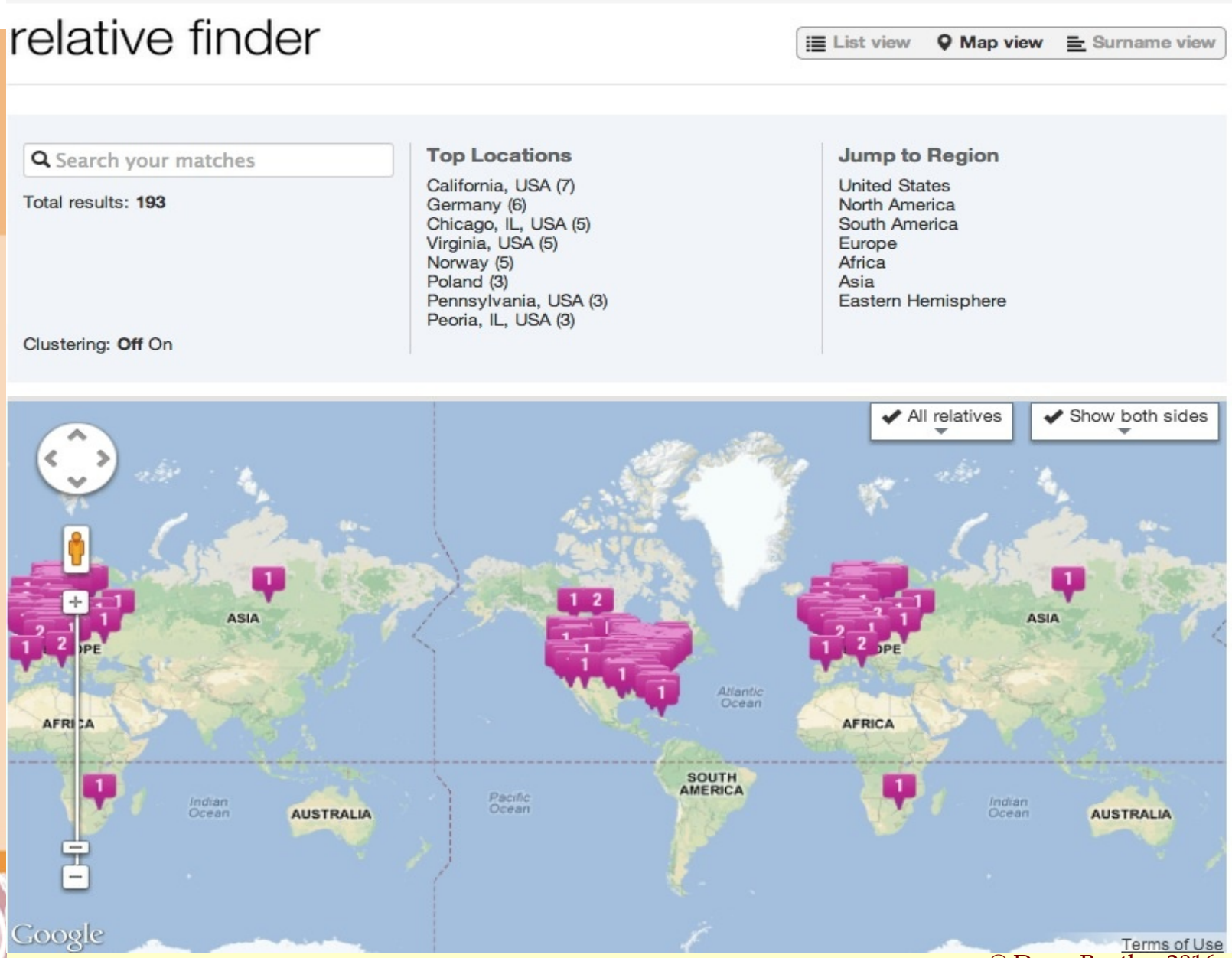
	100.0%	**Douglas Brutlag**

show all populations

23andMe Relative Finder



23andMe Relative Finder Map View



23andMe

Hide Advanced Controls

Number of grandparents from the same country: 1+

Minimum Segment Size: 5cM 10cM 15cM

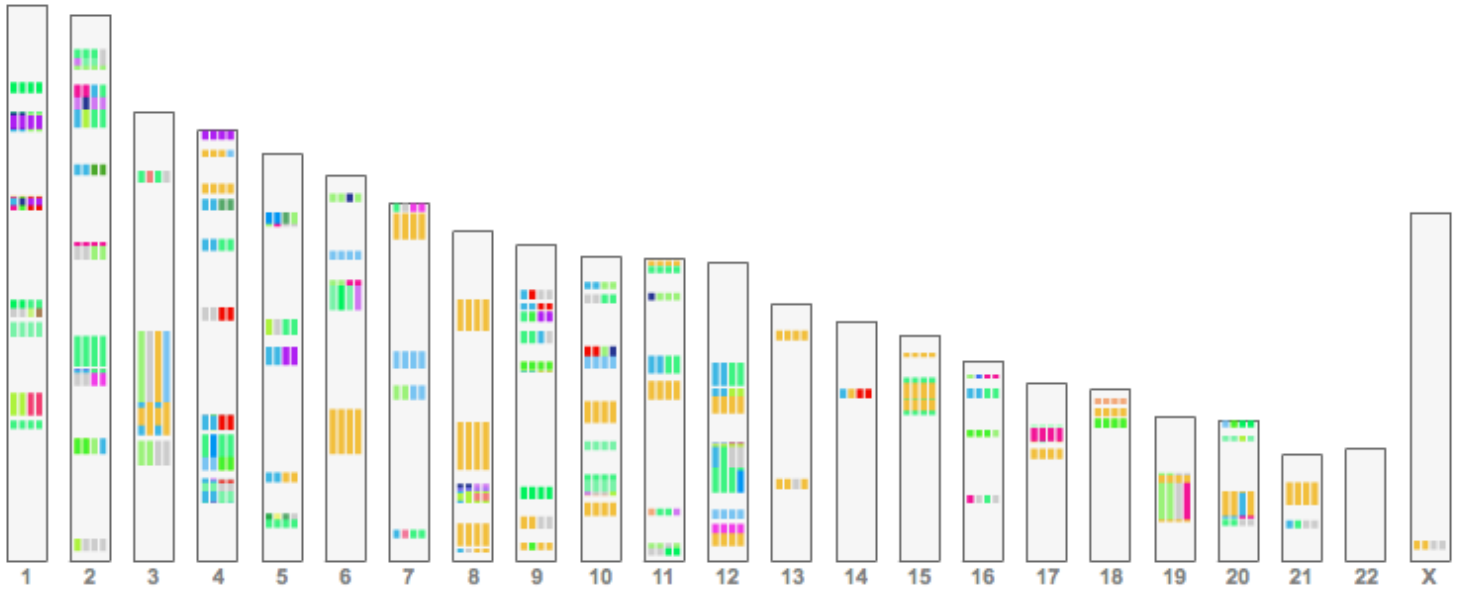
Include matches primarily from US, Canada, Australia, New Zealand & South Africa

Indicate segments declared to be of Ashkenazi Jewish ancestry.

Only show segments belonging to public individuals.

Learn more about Advanced Controls

Country	Color	Percent of Douglas Brutlag's Genome Covered
Norway		3.4% – 5.2%
Germany		1.6% – 6.1%
Ireland		0.7% – 1.5%
Denmark		0.4% – 1.7%
Russia		0.4% – 0.9%
Sweden		0.3% – 1.0%
Netherlands		0.3% – 0.7%
Finland		0.3% – 0.7%



What is a Fifth Cousin?

So You're

23andWe Discoveries

23andWe discoveries

You answer questions.

Other 23andMe members answer questions.

23andMe scientists work their magic.

23andWe discoveries

And make discoveries!

Your contributions

These articles are based on surveys you've taken.

New Genetic Factors for Hypothyroidism

Thanks! You took a survey that fueled this discovery.

view discovery

Ancestry and Disease Risk

Thanks! You took a survey that fueled this discovery.

view discovery

Genes and Geography

Thanks! You took a survey that fueled this discovery.

view discovery

23andMe

research initiatives

A new paradigm for genetic research.

Read our open letter to the science community

23andWe is a new, more efficient way of doing genetic research. Even though new technologies have made it possible to link genes to diseases, traits and conditions more effectively than ever before, collecting the data for this research can be a costly, time-consuming and logistically difficult process. Progress is hindered by the fact that these studies require both genetic and personal information from thousands – sometimes tens of thousands – of people.

23andWe involves our customers in research as collaborators, advisers and contributors by conducting studies that correlate their responses to online surveys with their genetic data. The idea is to enable large studies that would be infeasible using current methods, which typically involve recruiting patients through physicians' practices and other means. We plan to share the results of our research and show you how your contributions are making an impact by posting regular updates on this web site.

Next: How does research work at 23andMe?

Join a research community

Parkinson's Disease

Recent discoveries suggest that genetics plays a greater role in Parkinson's disease than was previously thought. You can advance research into the genetic roots of Parkinson's disease.

Alzheimer's Disease

More than 5 million Americans have Alzheimer's Disease. 23andMe and Genentech have teamed up to find out how genetics might protect against Alzheimer's Disease. This research could lead to new scientific knowledge or possibly a drug that could prevent or slow Alzheimer's Disease.

INFORMED Genetic Counselors

http://informeddna.com/index.php/23andme/schedule-appointment-23.html

INFORMED Genetic Counselors

http://informeddna.com/index.php/23andme/schedule-appointment-23.html

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About InformedDNA

Our nationwide network of board-certified genetic counselors provide genetic expertise to patients, physicians, and organizations across all fifty states in the USA, and are available internationally.

Genetic Expertise

- Cancer Genetics
- Reproductive Genetics
- Cardiac Genetics
- Ocular Genetics
- Neurogenetics
- Adult Genetics

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What other physicians say:

"We believe that Navigenics' preventive genomics service has the potential to be an innovation that could significantly enhance patient care."

-Edward Goldman, M.D.,
Former CEO, MDVIP, Inc.

We care about the science, your patients, and you

Make personalized genomic medicine and pharmacogenomics part of your practice. And provide your patients with a powerful tool for change.

Your patients trust you; you can trust us.

- Founders, practicing physician David Agus, M.D., and geneticist Dietrich Stephan, Ph.D., came together so that they could create a powerful new tool for personalized medicine.
- Focused on prevention, pharmacogenomics, and longitudinal health outcome studies.

We can help answer your questions.

- Medical education programs, resources and board-certified genetic counseling.
- Specifics on our Medications Wallet Card, including background information on each medication result presented on the card.

Partner with the leader in genomic health, just as we partner with the leaders in medicine.

- We collaborate with Mayo Clinic, Scripps Genomic Medicine, Duke, and others.

Resources

Downloads

- Applying Preventive Genomic Medicine in Clinical Practice
- The Science Behind the Navigenics Health Compass Service
- Conditions We Cover

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- Medications Wallet Card
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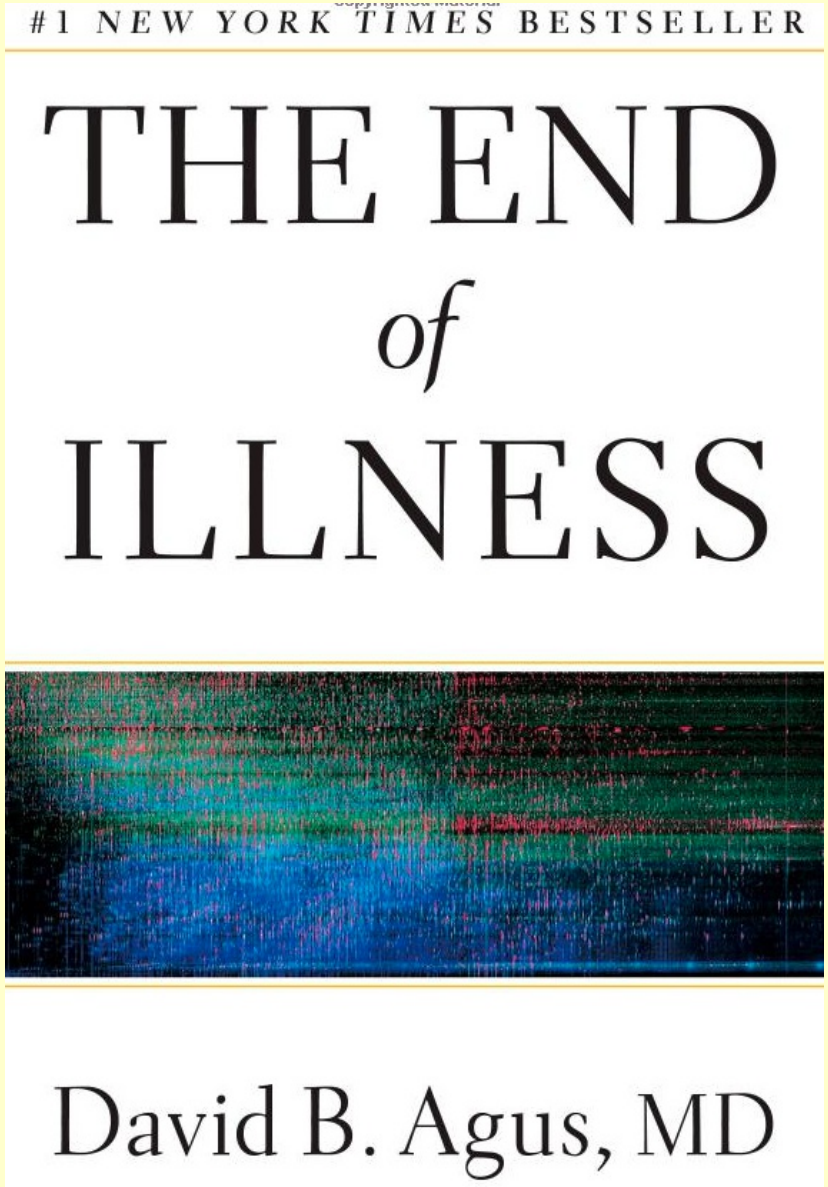
The End of Illness David B. Agus



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- Clinical Testing Programs
- Decision Support Program
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- Genomic Medicine Network

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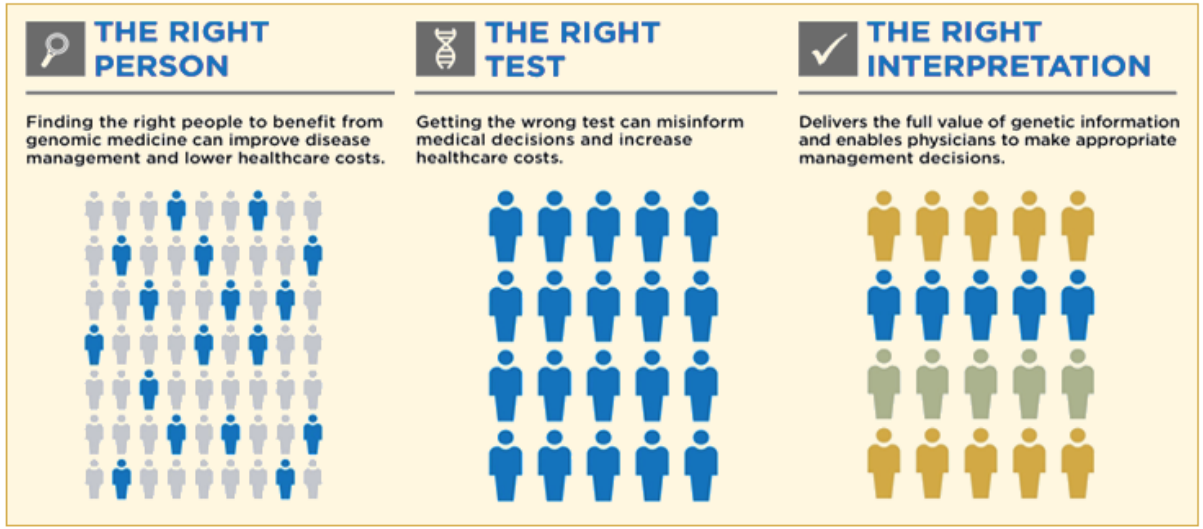
- About Us

DNA Direct brings the power of personalized medicine to payors, providers and patients.



Hospital Plan Webinar

Strategies to Optimize Personalize Medicine: How to Integrate Genomic Services into Your Hospital Community

Dr. Derek Kelly, Vice President, Medical Management at Swedish Covenant Hospital in Chicago discusses integrating genomic services into their clinical care.

Health Plan Webinar

How a Health Plan Successfully Integrated Genomic Services into Its System

Dr. Charles Stemple, Medical Director, Personalized Medicine/Genomics at Humana discusses their genetic guidance program.

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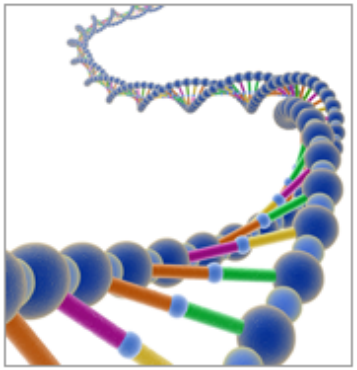


Personalized medicine, also referred to as genomic medicine, is changing the landscape of healthcare. By harnessing the power of genetic testing, physicians can make more informed healthcare decisions and better target treatments and drug therapies. The result is better healthcare outcomes.

Genetic tests are used in all areas of medicine – from prevention and screening to diagnosis and treatment. G2 Intelligence estimated that the market was $14.3B in 2010 and growing rapidly at 16% per year[1] and the Food and Drug Administration (FDA) states that more than 100 medications have pharmacogenomic information included in their drug labels[2]. Research by the Tufts Center for the Study of Drug Development indicates that oncology leads other therapeutic areas in the number of targeted therapies on the market as well as in the pipeline, with the expectation that within the decade all oncology drugs will have a related diagnostic. Other key therapeutic areas in which personalized medicine is impacting clinical decision-making include cardiovascular, neurologic, and immunologic therapies, whereas personalized medicine development is just getting started for metabolic and respiratory therapies, as well as virology[3]. With the advent of all of this new technology and information available to healthcare professionals and consumers, it will be critical to stay abreast of the new developments.

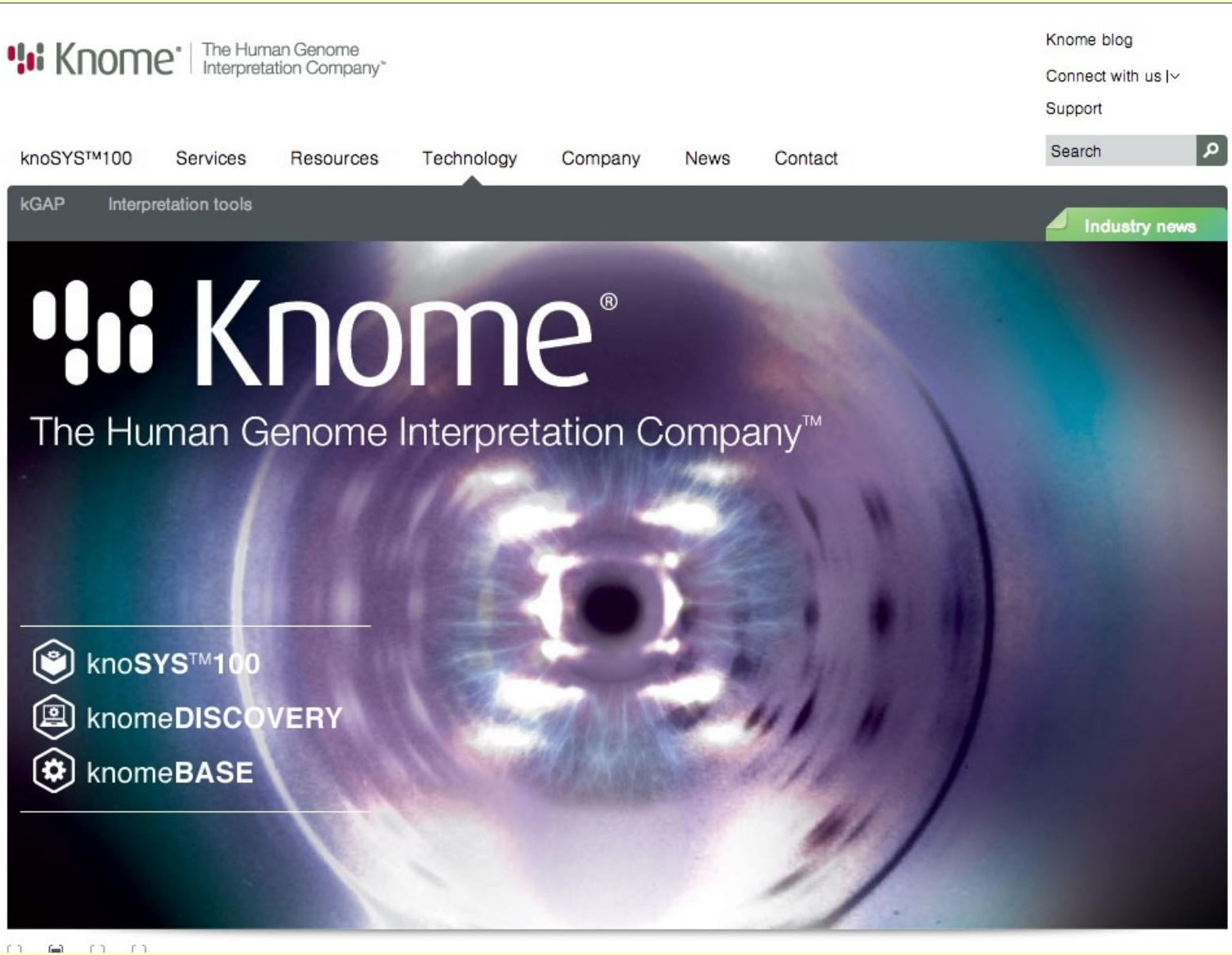
Low-cost whole genome sequencing (WGS) is on the horizon as well, adding a profound new dimension to the personalized medicine arsenal. Healthcare providers and consumers will be challenged with how best to interpret the information available to them.

As advances in personalized medicine continue, patients benefit from the deeper knowledge that genomics brings to healthcare decision making and outcomes.

1. G2 Intelligence: Lab Industry Strategic Outlook 2011: Market Trends & Analysis
2. www.fda.gov/drugs/scienceresearch/researchareas/pharmacogenetics/ucm083378.htm
3. *Personalized Medicine Is Playing A Growing Role In Development Pipelines* November/December 2010 Tufts CSDD Impact Report ; Vol12:6

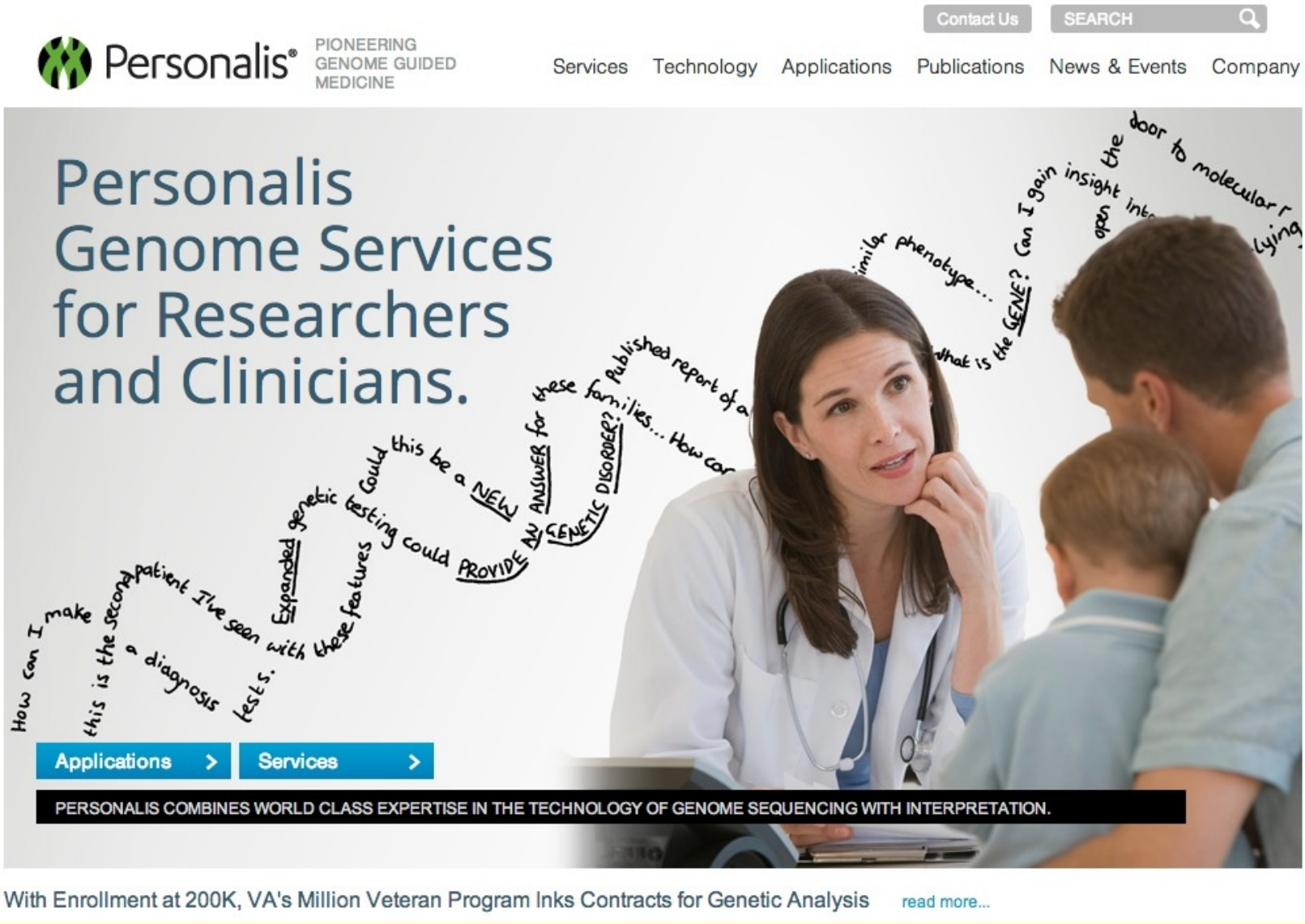
Knome

http://www.knome.com/



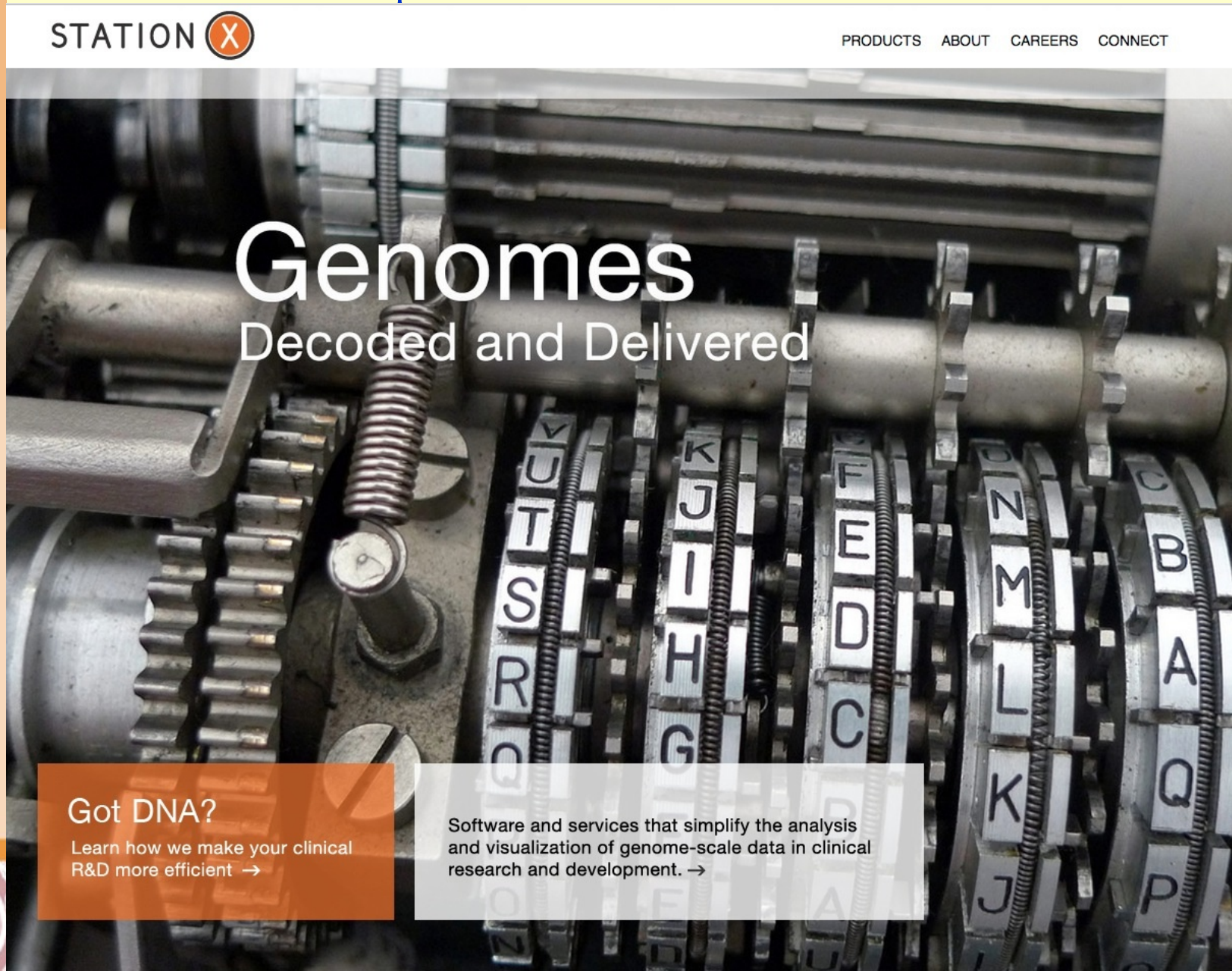
Personalis

http://www.personalis.com/



StationX

http://www.stationxinc.com/



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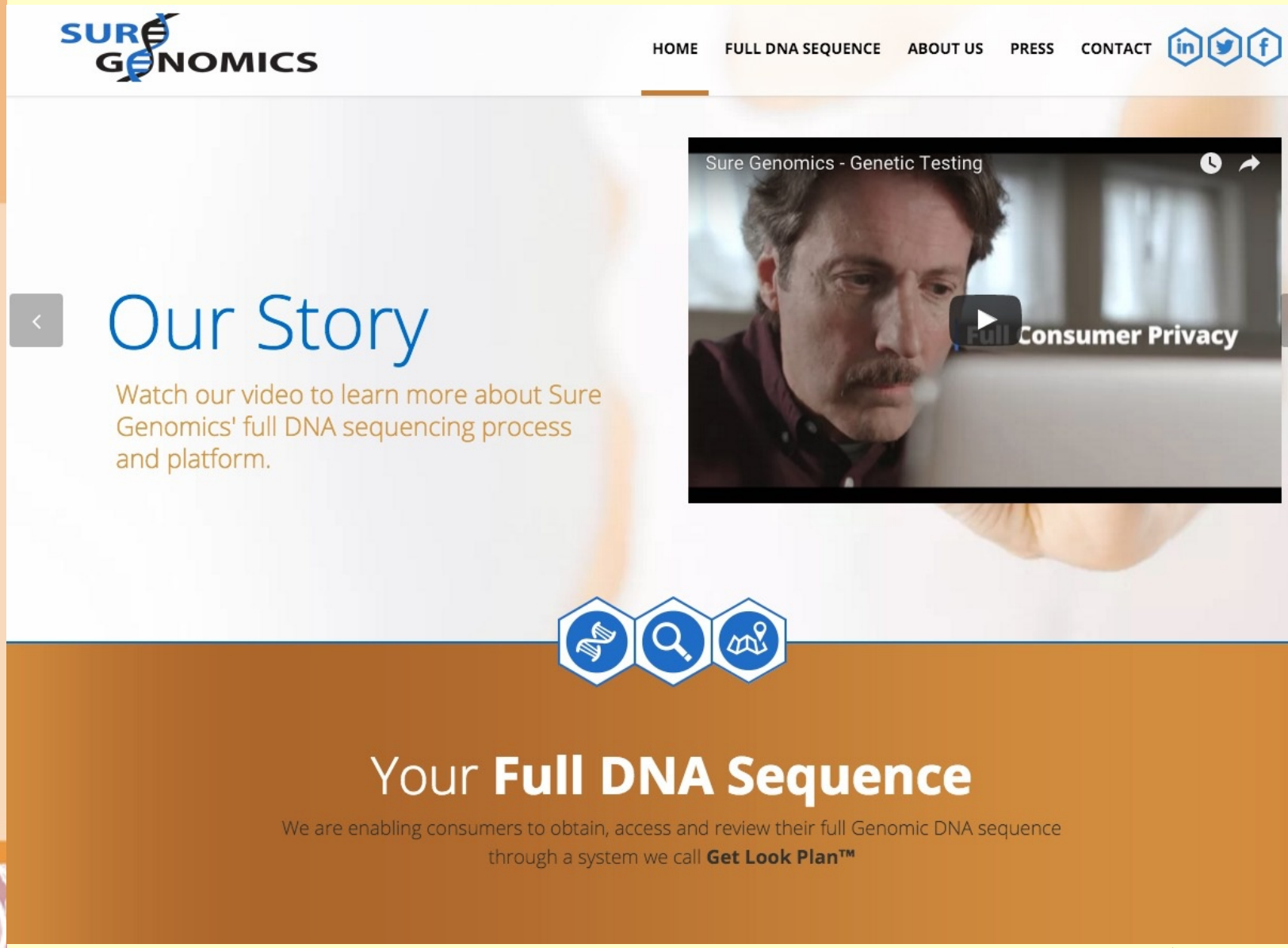

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Accurate Whole Human Genome Sequencing & Analysis

Complete Genomics is a leader in accurate whole human genomic sequencing. Using our proprietary sequencing instruments, chemistry, and software, we have sequenced more than 15,000 whole human genomes for our research customers over the past three years. Our mission is to provide the technology for sequencing one million human genomes, enabling researchers and clinicians to improve human health through the prevention, diagnosis, and treatment of genetic diseases and conditions.

BGI-Shenzhen Completes Acquisition of Complete Genomics

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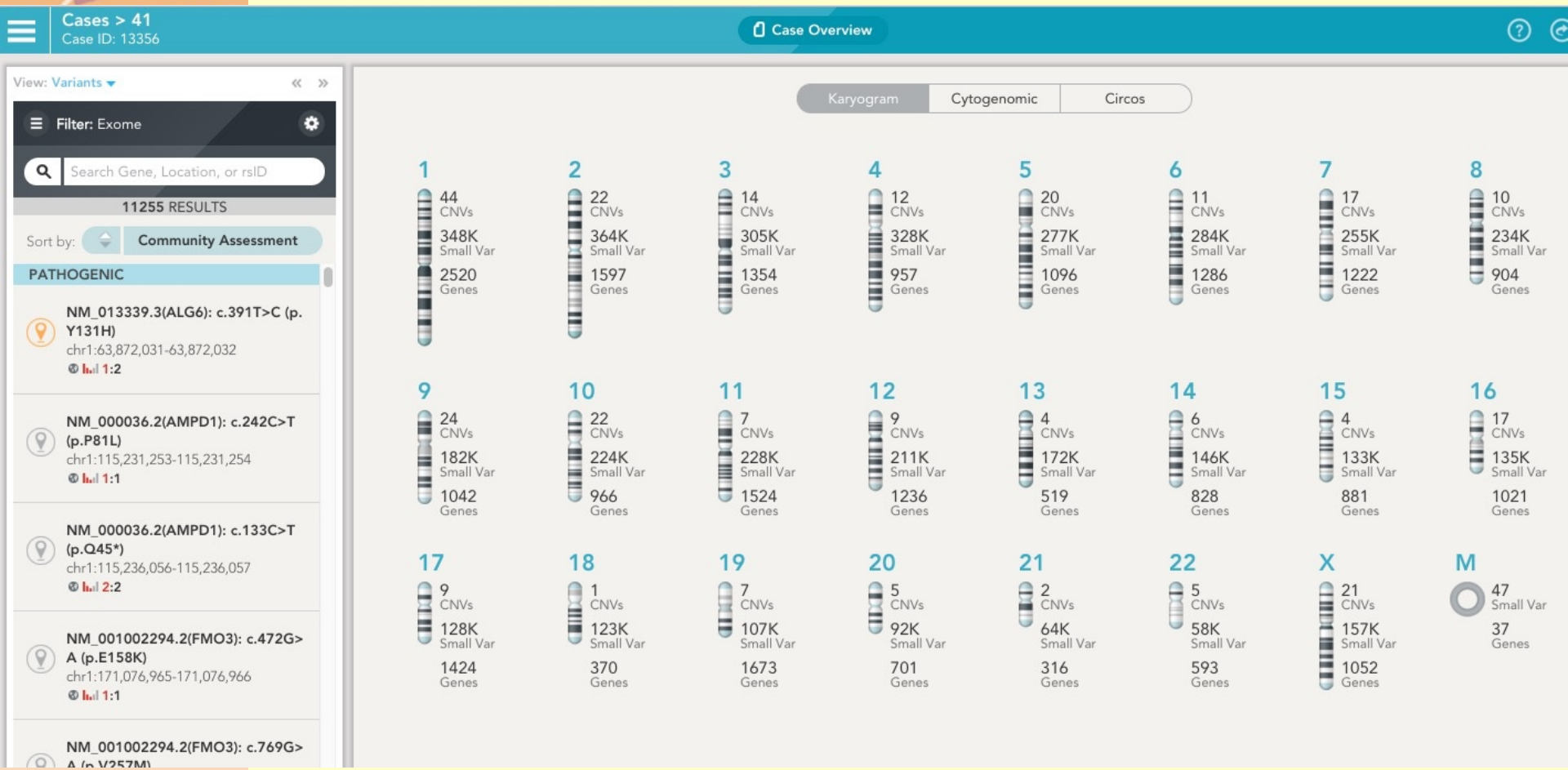
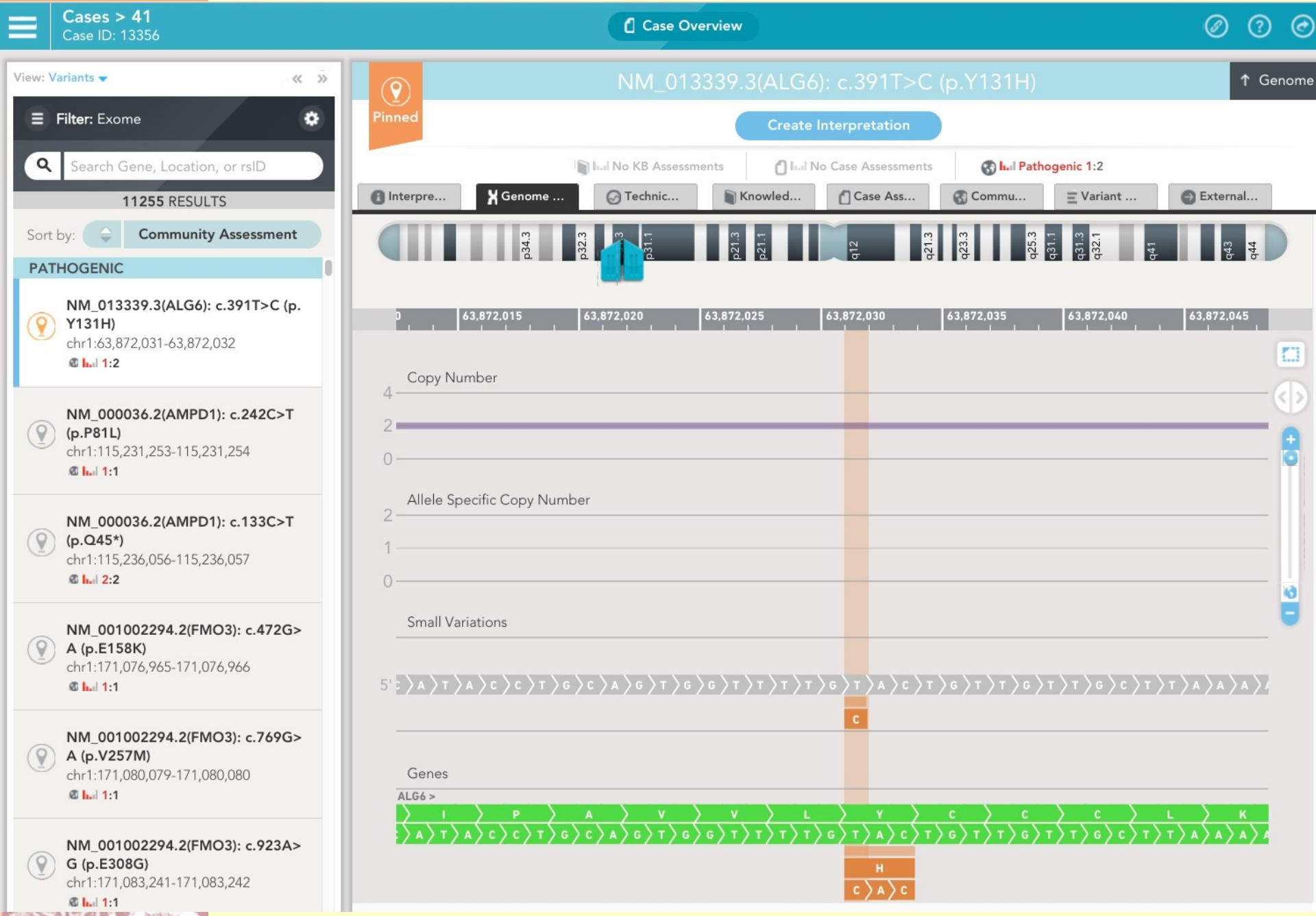
Read the ground breaking Nature paper on LFR Technology

Read More

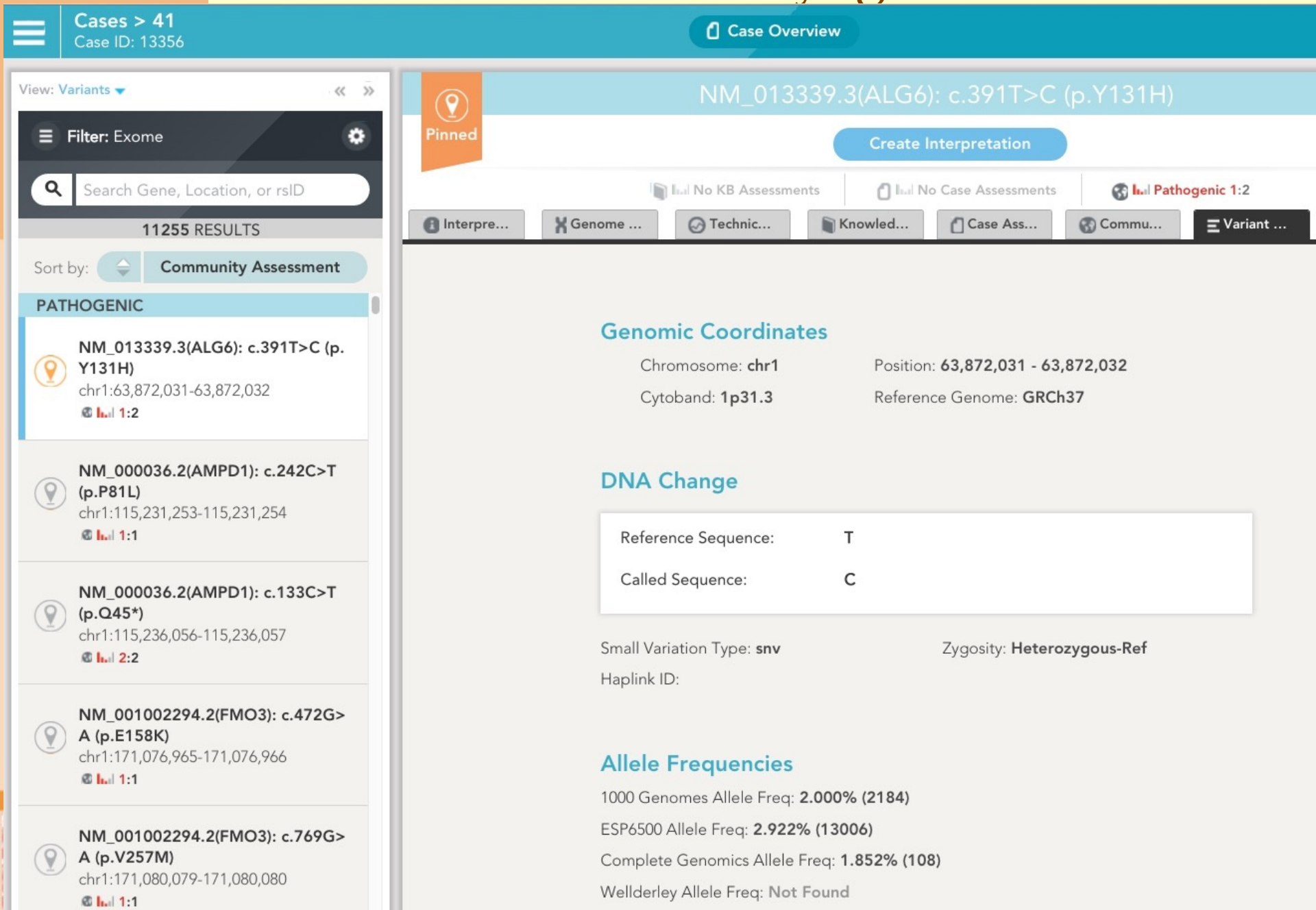
Genome Voyager DX from Genos Research

https://voyager-staging.genosresearch.com/index.html#case/13356/sample/33/workspace





Genome Voyager DX



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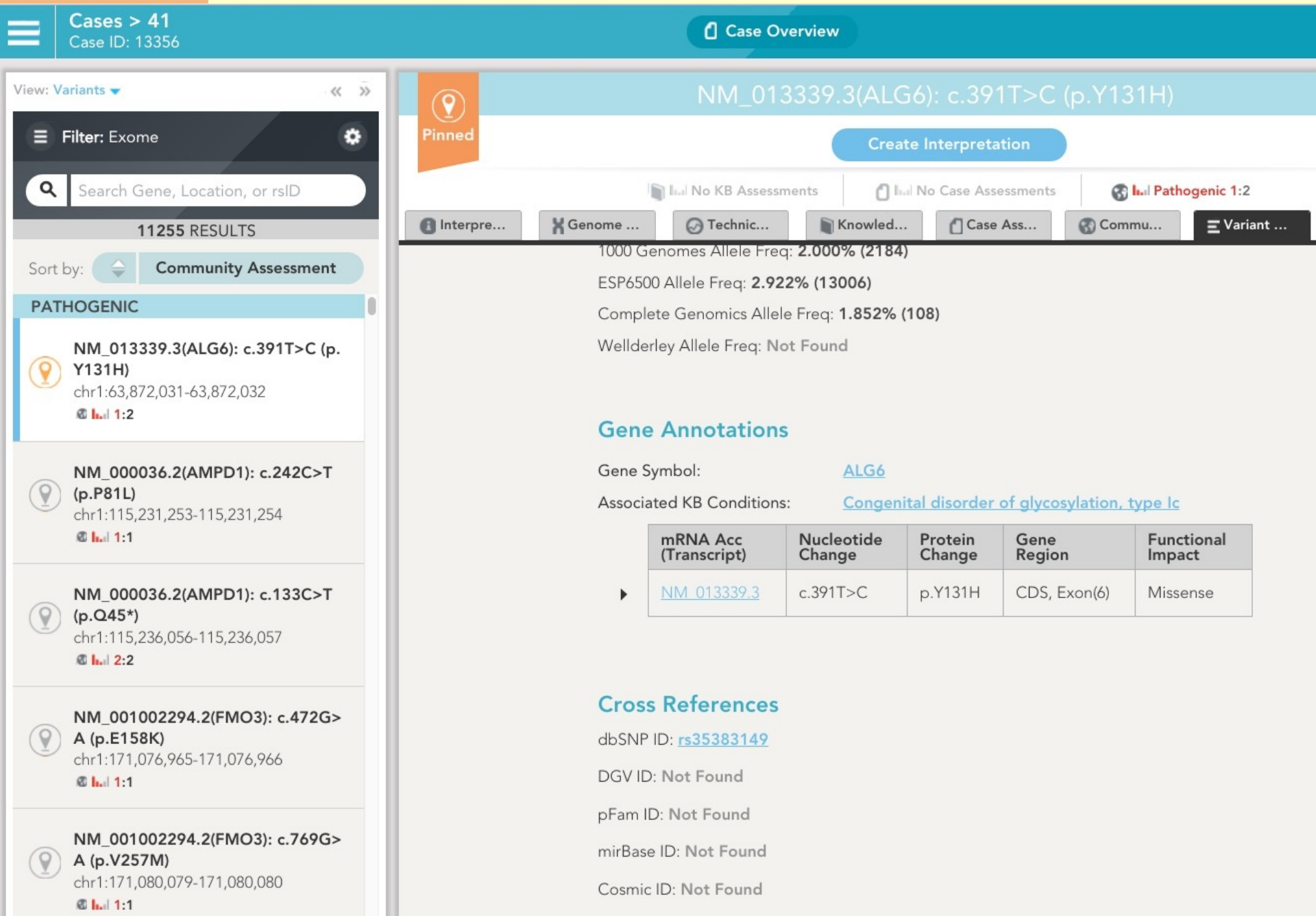

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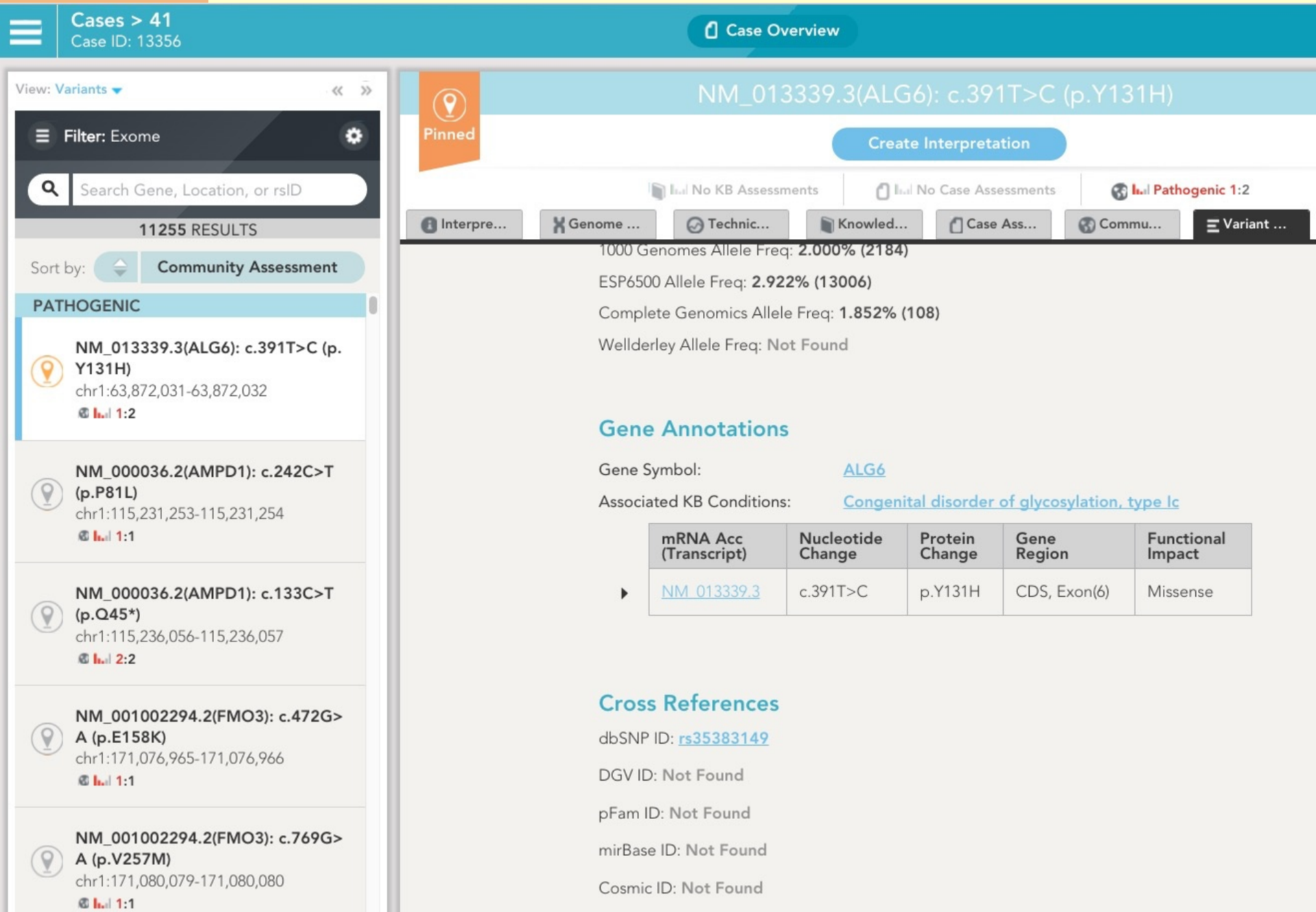
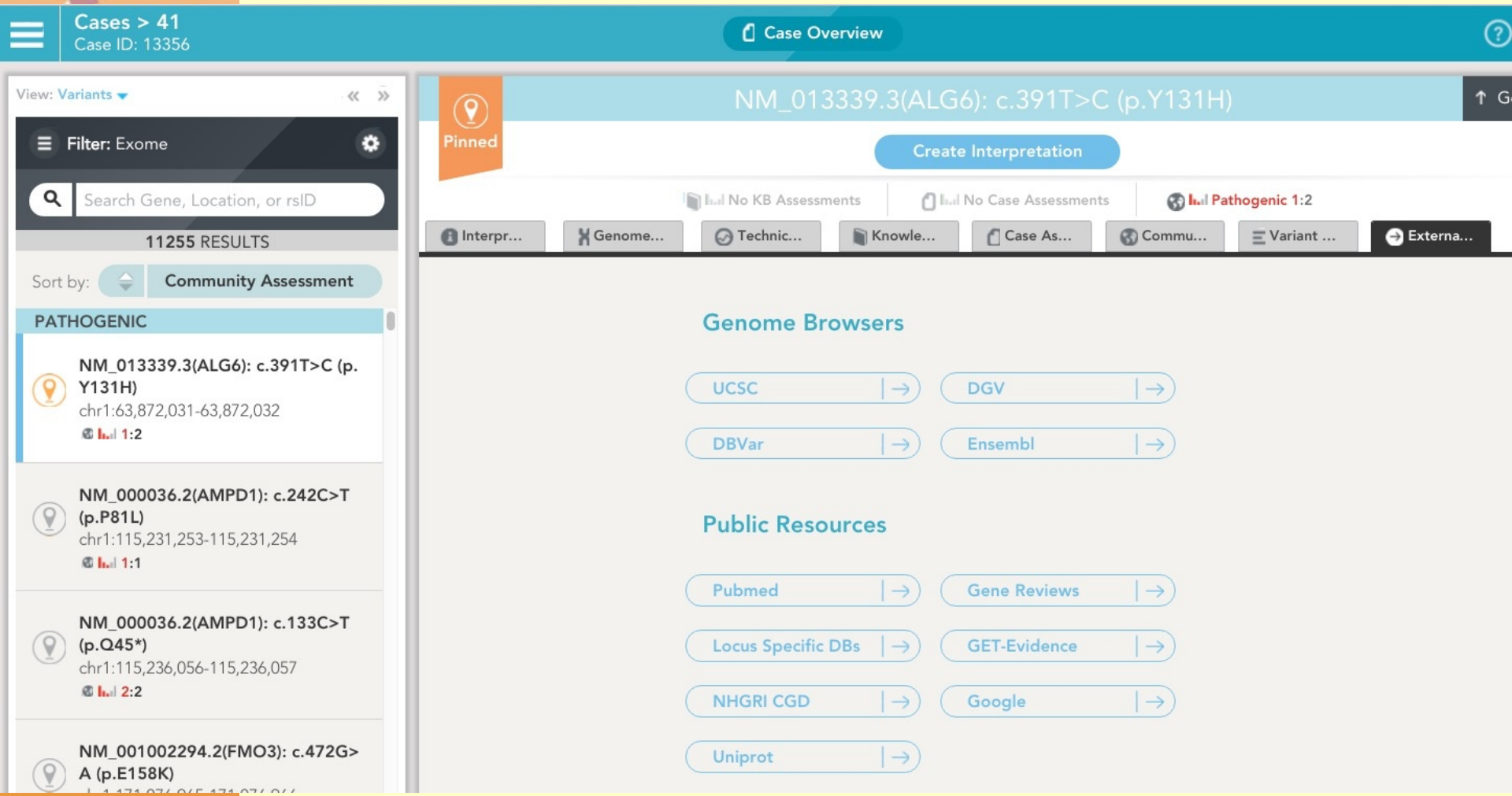


Genome Voyager DX
Genos Research



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