23andMe Genetic Health Overview

Prepared for: **JAMES POLLACK**

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What this overview includes

This overview includes brief summaries of your 23andMe results for:

- diseases for which you are at greater than average genetic risk,
- heritable diseases for which you carry one or more genetic variants (carrier status),
- and drugs to which you are likely to have an atypical response based on genetics.

These results are based on your genetic data and any sex and ancestry information you have provided along with population-level risk data for specified age ranges. They do not take into account non-genetic factors, family history, or additional genetic factors that may influence these conditions. Only results for genetic associations that are scientifically well established are included. This overview does not provide details regarding diseases for which you are at typical or lower than average genetic risk, heritable diseases for which you aren't known to carry a variant, or drugs to which you are likely to have a typical response. If you would like more information on any of your 23andMe results, please go to that topic's individual report page on our website at https://www.23andme.com/you/health/.

Overview of Genetic Health

James Pollack
Year of Birth: 1986
Eastern European

Disease risk results are included in this overview only if your risk based on genetics is greater than 1%. Note that certain conditions may have genetic information applicable only to specific populations.

Components of this test were performed in a clinical laboratory regulated under the Clinical Laboratory Improvement Amendments of 1988 (CLIA) to perform high-complexity testing. The data provided are intended for informational and educational use and are not for diagnostic use.

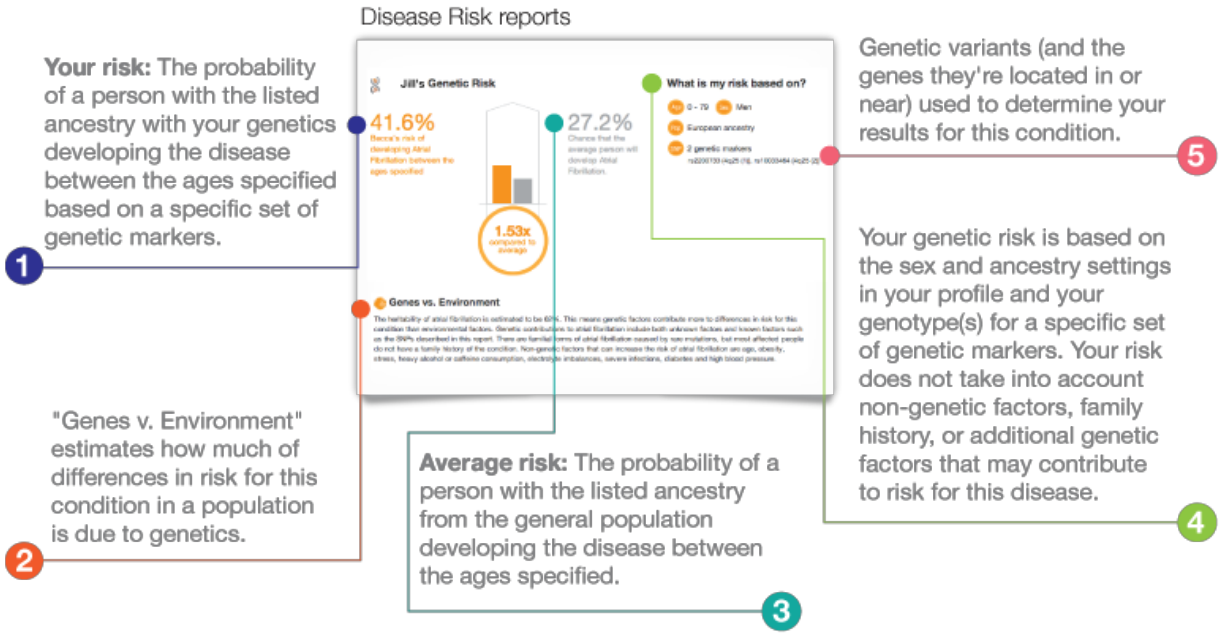
**All conditions tested are listed at the end of the report. You may not have data for every report.*

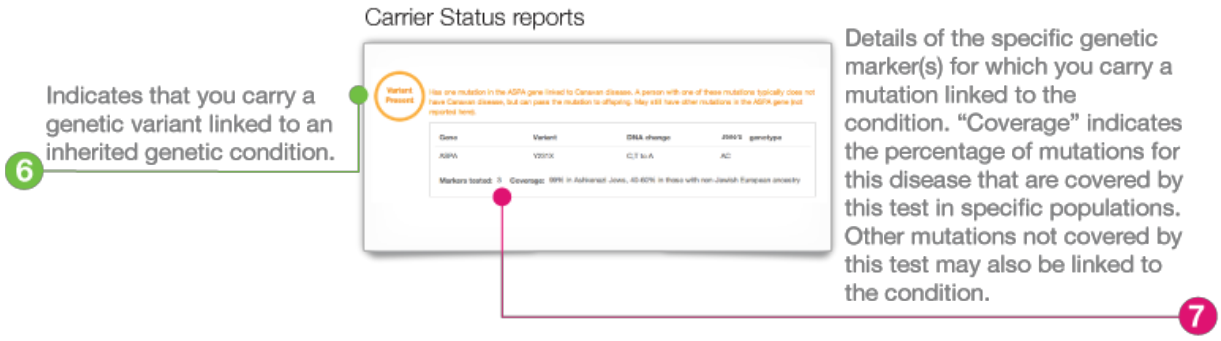
Disease risk	Your risk	Average risk
Alzheimer's Disease	14.2%	7.2%
Restless Legs Syndrome	2.5%	2.0%
Exfoliation Glaucoma	2.2%	0.7%
27 conditions*	Typical or decreased risk	

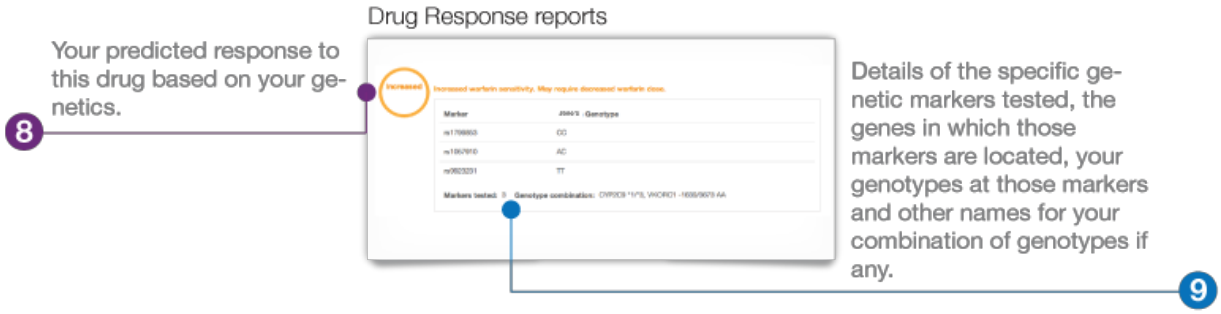
Carrier status	Status
Alpha-1 Antitrypsin Deficiency	Variant Present
46 heritable conditions*	Variant Absent

Drug response	Response
Warfarin (Coumadin®) Sensitivity	Increased
7 other drugs*	Typical Response

How to read your reports





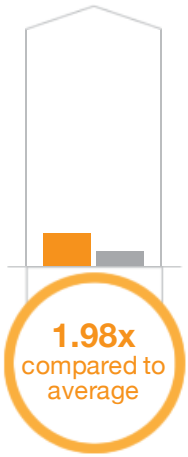


Alzheimer's Disease

Alzheimer's disease (AD) is a neurodegenerative condition characterized by decline in thinking and reasoning skills. Eventually, people with AD are unable to perform the basic activities of daily life. The most common cause of dementia in people over 65, AD currently affects about five million people in the United States. As the population ages, many more people are expected to develop AD; some estimate 14 million Americans will have the disease by the year 2050. There is currently no cure for AD, but scientists and physicians are working to understand how the disease develops, to improve the management of its symptoms and ultimately to develop ways of slowing or stopping its progression.

James's Genetic Risk

14.2%
James's risk of developing Alzheimer's Disease between the ages specified



7.2%
Chance that the average person will develop Alzheimer's Disease

What is my risk based on?

Age 50 - 79
Sex Men
Pop European ancestry
SNP 1 genetic markers
APOE ε2/ε3/ε4 (APOE)

Genes vs. Environment

The heritability of AD is estimated to be 60-80%. This means that genetic factors contribute more to individual differences in risk for AD than environmental factors do. Genetic contributions to AD risk include known factors, such as the APOE gene variants we describe in this report. There are also rare mutations in other genes that cause early-onset (before age 65) forms of AD that run in families; this report does not currently include information on these mutations, or for additional genetic factors that have relatively weaker effects on AD risk. Non-genetic risk factors for AD include high blood pressure, high cholesterol, obesity, poorly controlled diabetes, and history of head trauma.

Additional Information

Other Genetic Factors

Your result is based on your genotype at the APOE gene, the gene with the strongest effect on genetic risk for late-onset Alzheimer's disease. 23andMe does not currently report on other common genetic variants that have weaker effects on AD risk or rarer variants associated with early-onset Alzheimer's disease.

Lifestyle Factors

- **Stay active:** Regular exercise is associated with decreased risk of AD. And don't forget to exercise your mind too -- some studies suggest that staying mentally active throughout life can also reduce your risk.
- **Eat healthy:** A balanced, low-fat diet can not only help you maintain healthy weight and cholesterol, but can also help reduce your risk of AD.

Other Medical Conditions

Studies suggest that risk factors for heart disease (such as lack of exercise, smoking, high blood pressure, high cholesterol, and poorly-controlled diabetes) may also increase risk for dementia. Your health care provider can work with you to help manage your heart health.

View the full report online for links to resources, references, and more detailed genetic results and information.

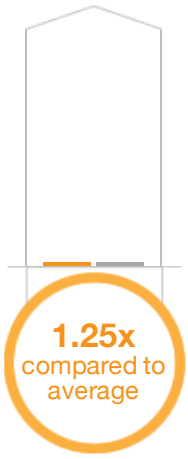
Restless Legs Syndrome

Imagine what it would be like to crawl into bed every night, ready to catch some much-needed Zs, only to be struck by an irrepressible urge to move your legs as soon as you began to relax. No matter how tired you were, instead of drifting off peacefully, you would be compelled to get up and move around. It may sound crazy, but this is exactly the situation people with restless legs syndrome (RLS) experience. Though the symptoms in many people are milder, it is estimated that about 4% of the U.S. population suffers from this puzzling disorder.

James's Genetic Risk

2.5%

James's risk of developing Restless Legs Syndrome between the ages specified



2.0%

Chance that the average person will develop Restless Legs Syndrome

What is my risk based on?

Age 0 - 79 Sex Men

Pop European ancestry

SNP 1 genetic markers
rs3923809 (BTBD9)

Genes vs. Environment

The heritability of restless legs syndrome is estimated to be 54%. This means that genetic and environmental factors contribute nearly equally to differences in risk for this condition. Genetic factors that play a role in restless legs syndrome include both unknown factors and known factors such as the SNPs we describe here. Environmental factors include pregnancy. Low iron levels, dialysis for end-stage renal disease, and damage to the nerves of the hands and feet tend to worsen the condition.

Additional Information

Other Medical Conditions

Chronic diseases such as kidney failure, diabetes, Parkinson's, and peripheral neuropathy can exacerbate symptoms of RLS. If you have RLS, your health care provider may work with you to manage these conditions to reduce your symptoms. Pregnancy can sometimes trigger symptoms of RLS. If this happens, the symptoms will usually disappear once the pregnancy is completed.

Lifestyle Factors

- **Limit caffeine, alcohol, and tobacco use:** Caffeine, alcohol, and tobacco intake can trigger or aggravate symptoms in predisposed individuals.
- **Get enough iron:** Insufficient iron levels can also trigger or aggravate symptoms.

Medications and Treatment

Taking certain drugs can sometimes cause symptoms of RLS. These symptoms usually disappear once the drug regimen is stopped. Your health care provider can work with you to manage drug regimens that may be triggering RLS.

View the full report online for links to resources, references, and more detailed genetic results and information.

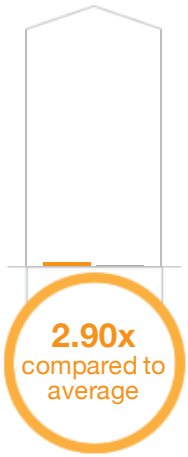
Exfoliation Glaucoma

Glaucoma is one of the most common causes of blindness in the United States and globally, accounting for about 12% of the world's cases. It is caused by a buildup of fluid pressure inside the eye, which eventually damages the optic nerve and causes sight to deteriorate. Exfoliation glaucoma (sometimes called pseudo-exfoliation glaucoma) is a subtype of the disease that often results from exfoliation syndrome, a disorder which causes an accumulation of flaky, white material inside the eye that blocks fluid drainage. Exfoliation syndrome affects about 10% of the population over 50, though some populations — especially Scandinavians — have much higher rates of the condition.

James's Genetic Risk

2.2%

James's risk of developing Exfoliation Glaucoma between the ages specified



0.7%

Chance that the average person will develop Exfoliation Glaucoma

What is my risk based on?

- Age 40 - 79
- Sex Men
- Pop European ancestry
- SNP 1 genetic markers
  rs2165241 (LOXL1)

Genes vs. Environment

The heritability of exfoliation glaucoma has not been studied. However, the heritability of open-angle glaucoma, a broad category of the disease that includes many cases of exfoliation glaucoma, has been estimated to be 13%. This means that environmental factors contribute more to differences in risk for this condition than genetic factors. Environmental factors that may increase the risk for glaucoma include diabetes, high blood pressure, heart disease, eye injury or disease and prolonged corticosteroid use.

Additional Information

Screening and Risk Assessment

See the Glaucoma Research Foundation recommendations for screening. Anyone at high risk for glaucoma should be tested every year or two after the age of 35.

Family History

Having an immediate family member with glaucoma increases your risk substantially. Use 23andMe's Family Health History tool to collect this important information.

View the full report online for links to resources, references, and more detailed genetic results and information.

Carrier status: Alpha-1 Antitrypsin Deficiency

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.

James's Genetic Results

Variant Present

MZ: Has one M and one Z form of the SERPINA1 gene. People with this combination may be at increased risk for liver disease, and may experience decreased lung function if they smoke.

Gene	Variant	DNA change	James's genotype
SERPINA1	rs28929474	C to T	CT

Markers tested: 2 **Coverage:** > 95%

What does this test cover?

More than 20 mutations in the SERPINA1 gene causing AAT deficiency have been documented. 23andMe reports data for two mutations that cause the PI*S and PI*Z forms of the SERPINA1 gene which account for more than 95% of the disease.

How is Alpha-1 Antitrypsin Deficiency inherited?

AAT deficiency is inherited in a recessive manner, meaning that only a child who receives two mutated copies of the SERPINA1 gene (one from each parent) will develop the disease.

How common is this condition?

Approximately one out of every 5,000-7,000 North Americans and one out of every 1,500-3,000 people from Scandinavia has AAT deficiency.

Additional Information

Other Medical Conditions

AAT deficiency typically results in chronic obstructive pulmonary disease (COPD) and liver disease. Other common complications include bronchial asthma and chronic bronchitis.

Other Risk Factors

Individuals with AAT deficiency should avoid cigarette smoke (including secondhand smoke), exposure to environmental pollutants, and alcohol use, and take steps to prevent lung infections, all of which accelerate lung or liver disease.

View the full report online for links to resources, references, and more detailed genetic results and information.

Drug response: Warfarin (Coumadin®) Sensitivity

Each time a doctor writes a prescription for warfarin (Coumadin ®), a blood thinner given to about two million people each year in the United States, it's a guessing game. There is no "right" dose of the drug. Everyone is different and it can take weeks of adjustment to find a patient's optimal amount of the medication. Too much puts the patient at risk for bleeding. Too little can lead to clots and in turn, heart attack, stroke or even death. A patient's optimal dose depends not only on age, size, other medications and even diet, but also to a large extent on genetics.

James's Genetic Results

Increased

Slightly increased warfarin sensitivity. May require decreased warfarin dose.

Marker	James's Genotype
rs1799853	CC
rs1057910	AA
rs9923231	CT

Markers tested: 3 **Genotype combination:** CYP2C9 *1/*1, VKORC1 -1639/3673 AG

What does this test cover?

Several genes involved in warfarin metabolism play prominent roles in the variable response to warfarin. 23andMe tests for two variants in the CYP2C9 gene (*2, defined using rs1799853, and *3, defined using rs1057910) that are associated with reduced ability to break down warfarin. 23andMe also tests for a variant near the VKORC1 gene (rs9923231) that is associated with increased sensitivity to the drug. Read more about the genetics.

Additional Information

Other Risk Factors

Many other clinical and demographic factors affect the optimal warfarin dose for an individual, including age, sex, weight, alcohol consumption, smoking status, ethnicity, vitamin K intake, and other medications. Other genetic variations in other genes (not reported here) can also impact a person's response to warfarin. Only a medical professional can determine the optimal dose for an individual.

Medications and Treatment

Warfarin can interact with other medications, including some antibiotics, non-steroidal anti-inflammatory drugs, some antidepressants, cholesterol medications, and chemotherapy drugs. If you are taking one of these drugs, your health care provider can help devise appropriate treatment plans.

View the full report online for links to resources, references, and more detailed genetic results and information.

James Pollack's results for all conditions tested by 23andMe

Conditions and diseases tested by 23andMe: This list is continually expanding as new genetic associations are discovered and reported. Please visit our website at https://www.23andme.com/health/all/ to view the most up-to-date list of conditions tested by 23andMe.

About Risk Estimates:
23andMe reports results as genotype-specific incidence, which is an estimate of how many individuals in a population composed of people with a customer's genotype are expected to be diagnosed with a condition given a specified ancestry and age range. These estimates are based on well-established genetic associations reported in the biomedical literature and do not account for non-genetic factors, family history, or additional genetic factors that may modify a customer's risk. The genotype-specific incidence estimate combines the odds for a condition for a customer's genotypes at a set of SNPs with data about disease incidence. For more information on how 23andMe calculates these estimates, please see our technical papers available at https://www.23andme.com/howitworks/.

Disease risk (30)	Your risk	Average risk
Alzheimer's Disease	14.2%	7.2%
Restless Legs Syndrome	2.5%	2.0%
Exfoliation Glaucoma	2.2%	0.7%
Esophageal Squamous Cell Carcinoma (ESCC)	0.4%	0.4%
Celiac Disease	0.4%	0.1%
Stomach Cancer (Gastric Cardia Adenocarcinoma)	0.3%	0.2%
Scleroderma (Limited Cutaneous Type)	0.08%	0.07%
Atrial Fibrillation	Typical risk	
Bipolar Disorder	Typical risk	
Breast Cancer	Typical risk	
Chronic Kidney Disease	Typical risk	
Colorectal Cancer	Typical risk	
Coronary Heart Disease	Typical risk	
Gallstones	Typical risk	
Lung Cancer	Typical risk	
Lupus (Systemic Lupus Erythematosus)	Typical risk	
Obesity	Typical risk	
Primary Biliary Cirrhosis	Typical risk	
Prostate Cancer	Typical risk	
Type 2 Diabetes	Typical risk	
Venous Thromboembolism	Typical risk	

Age-related Macular Degeneration	Decreased risk
Crohn's Disease	Decreased risk
Melanoma	Decreased risk
Multiple Sclerosis	Decreased risk
Parkinson's Disease	Decreased risk
Psoriasis	Decreased risk
Rheumatoid Arthritis	Decreased risk
Type 1 Diabetes	Decreased risk
Ulcerative Colitis	Decreased risk

About Carrier Status:
23andMe tests for specific genetic variants that are strongly linked to a number of inherited genetic conditions. These variants are typically the most common ones linked to the condition. Certain variants may be more common in certain populations than others. The absence of specific variants does not rule out the possibility that a customer may carry another variant linked to the condition.

Carrier status (47)	**Status**
Alpha-1 Antitrypsin Deficiency	Variant Present
ARSACS	Variant Absent
Agenesis of the Corpus Callosum with Peripheral Neuropathy (ACCPN)	Variant Absent
Autosomal Recessive Polycystic Kidney Disease	Variant Absent
BRCA Cancer Mutations (Selected)	Variant Absent
Beta Thalassemia	Variant Absent
Bloom's Syndrome	Variant Absent
Canavan Disease	Variant Absent
Congenital Disorder of Glycosylation Type 1a (PMM2-CDG)	Variant Absent
Connexin 26-Related Sensorineural Hearing Loss	Variant Absent
Cystic Fibrosis	Variant Absent
D-Bifunctional Protein Deficiency	Variant Absent
DPD Deficiency	Variant Absent
Dihydrolipoamide Dehydrogenase Deficiency	Variant Absent
Factor XI Deficiency	Variant Absent
Familial Dysautonomia	Variant Absent
Familial Hypercholesterolemia Type B	Variant Absent
Familial Hyperinsulinism (ABCC8-related)	Variant Absent
Familial Mediterranean Fever	Variant Absent

Fanconi Anemia (FANCC-related)	Variant Absent
G6PD Deficiency	Variant Absent
GRACILE Syndrome	Variant Absent
Gaucher Disease	Variant Absent
Glycogen Storage Disease Type 1a	Variant Absent
Glycogen Storage Disease Type 1b	Variant Absent
Hemochromatosis	Variant Absent
Hypertrophic Cardiomyopathy (MYBPC3 25bp-deletion)	Variant Absent
LAMB3-related Junctional Epidermolysis Bullosa	Variant Absent
Leigh Syndrome, French Canadian Type (LSFC)	Variant Absent
Limb-girdle Muscular Dystrophy	Variant Absent
Maple Syrup Urine Disease Type 1B	Variant Absent
Medium-Chain Acyl-CoA Dehydrogenase (MCAD) Deficiency	Variant Absent
Mucolipidosis IV	Variant Absent
Neuronal Ceroid Lipofuscinosis (CLN5-related)	Variant Absent
Neuronal Ceroid Lipofuscinosis (PPT1-related)	Variant Absent
Niemann-Pick Disease Type A	Variant Absent
Nijmegen Breakage Syndrome	Variant Absent
Pendred Syndrome	Variant Absent
Phenylketonuria	Variant Absent
Primary Hyperoxaluria Type 2 (PH2)	Variant Absent
Rhizomelic Chondrodysplasia Punctata Type 1 (RCDP1)	Variant Absent
Salla Disease	Variant Absent
Sickle Cell Anemia & Malaria Resistance	Variant Absent
Tay-Sachs Disease	Variant Absent
Torsion Dystonia	Variant Absent
Tyrosinemia Type I	Variant Absent
Zellweger Syndrome Spectrum	Variant Absent

About Drug Response:
23andMe displays your likely response to a number of drugs

Drug response (8)	**Response**

based on genetic variants associated with differences in response. These may be differences in sensitivity, in the likelihood or severity of side effects, or differences in disease risk tied to use of a drug. Only a medical professional can determine whether a drug is right for a particular patient. The information contained in this report should not be used to independently establish a drug regimen, or abolish or adjust an existing course of treatment.

Warfarin (Coumadin®) Sensitivity	Increased
Abacavir Hypersensitivity	Typical
Alcohol Consumption, Smoking and Risk of Esophageal Cancer	Typical
Clopidogrel (Plavix®) Efficacy	Typical
Fluorouracil Toxicity	Typical
Oral Contraceptives, Hormone Replacement Therapy and Risk of Venous Thromboembolism	Not Applicable
Pseudocholinesterase Deficiency	Typical
Response to Hepatitis C Treatment	Typical

References

Alzheimer's Disease

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Warfarin (Coumadin®) Sensitivity

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About the 23andMe Personal Genome Service®

23andMe's Personal Genome Service provides customers with data on nearly 1,000,000 single nucleotide polymorphisms (SNPs) in their genome using a microarray-based genotyping assay. Customers provide saliva samples, which are analyzed by a CLIA-certified laboratory. Results are viewable on the 23andMe website at https://www.23andme.com/you/ where reports are considered Established or Preliminary Research reports depending on the amount of evidence supporting the associations reported. We currently provide more than 60 Established Research reports on various disease risk, drug response, and carrier status topics, as well as Preliminary Research reports on more than 150 conditions and traits.