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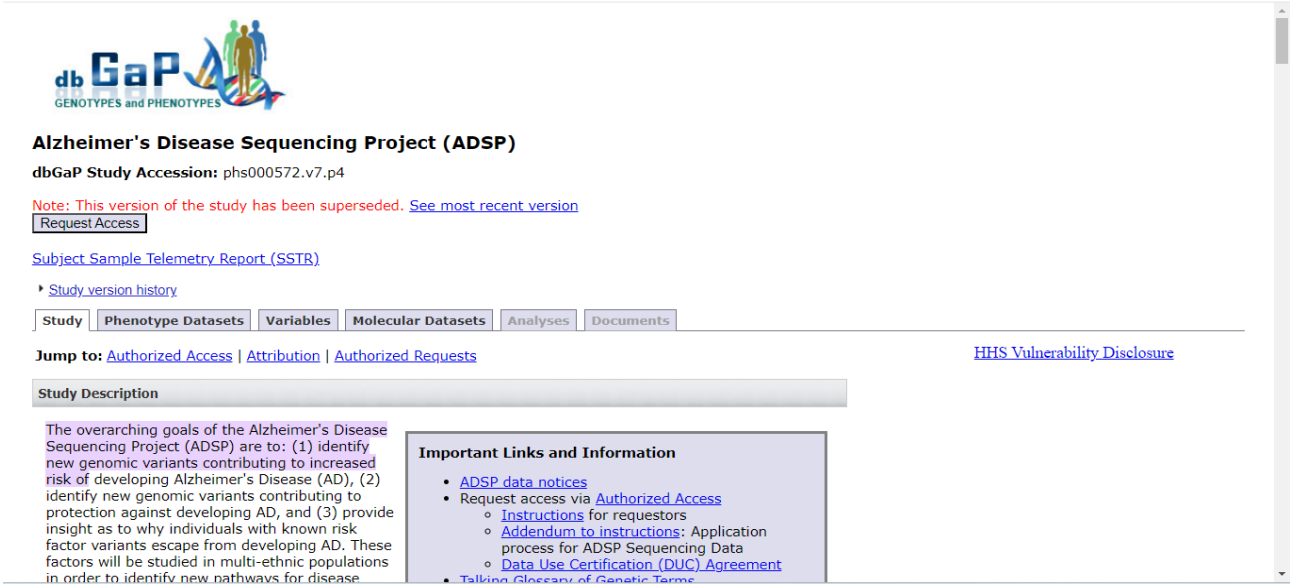
Title: Clinical databases and Medical terminologies

1. Exploring dbGaP.

a. Search the dbGaP database and identify a dataset related to Alzheimer's disease.



ADSP is a valuable resource for researchers investigating the genetic basis of Alzheimer's disease. The Alzheimer's disease Sequencing Project (ADSP) is a large-scale genetic study aiming to identify genes contributing to Alzheimer's Disease (AD).

- Goal: Find new genetic variants that increase or protect against Alzheimer's Disease.
- Subjects: Over 15,000 individuals including patients with AD and healthy controls.
- Data: Whole genome and whole exome sequencing data.
- Access: Requires approval from the National Institutes of Health (NIH).
- Funding: National Human Genome Research Institute (NHGRI) and National Institute on Aging (NIA).

Some key details from the ADSP dbGaP record:

- dbGaP accession number: phs000572.v7.p4 (This is important to cite if you use the data)
- Study design: Case-control (comparing patients with AD to healthy controls)
- Family studies: Also included families with multiple members affected by AD.

- Data types: DNA sequencing data (whole genome and whole exome sequencing)
- Phenotype data: Includes age at onset, diagnosis, ethnicity, and cognitive measures.

b. Describe the types of data available in the dataset, including genomic data, clinical data, and demographic information.

Types of Data in the ADSP Dataset

The Alzheimer's Disease Sequencing Project (ADSP) offers a rich trove of data, including:

Genomic Data

- Whole-genome sequencing (WGS): Complete DNA sequences of individuals, providing a comprehensive view of genetic variation.
- Exome sequencing: Focused sequencing of the protein-coding regions of the genome, which are thought to be more likely to contribute to disease.

Clinical Data

- Diagnostic criteria: Information about the specific criteria used to diagnose Alzheimer's disease in participants.
- Cognitive function: Assessments of cognitive abilities, such as memory, language, and problem-solving.
- Medical history: Records of other medical conditions, medications, and family history.

Demographic Information

- Age: The age of participants at the time of data collection.
- Sex: The gender of participants.
- Ethnicity: The racial or ethnic background of participants.
- Education: The level of education attained by participants.

These types of data are crucial for understanding the genetic and environmental factors that contribute to Alzheimer's disease and for developing new diagnostic tools and therapeutic interventions.

c. Propose three potential research questions that could be addressed using this dataset, considering the available data types.

Given the rich variety of data available in the Alzheimer's Disease Sequencing Project (ADSP), here are three potential research questions:

1. Identifying Novel Genetic Risk Factors

- **Question:** Are there genetic variants beyond those already identified that significantly contribute to the risk of developing Alzheimer's disease?
- **Data:** Whole-genome sequencing (WGS) data, clinical data (diagnostic criteria, cognitive function, medical history), and demographic information.
- **Approach:** Conduct genome-wide association studies (GWAS) to identify single nucleotide polymorphisms (SNPs) associated with Alzheimer's disease risk.

2. Investigating Gene-Environment Interactions

- **Question:** How do genetic factors interact with environmental factors (e.g., lifestyle, diet, education) to influence Alzheimer's disease risk and progression?
- **Data:** WGS data, clinical data, demographic information, and environmental data (if available).
- **Approach:** Use statistical methods to assess gene-environment interactions, such as regression analysis or machine learning techniques.

3. Exploring the Role of Rare Variants

- **Question:** Do rare genetic variants (those with low frequency in the population) contribute to Alzheimer's disease risk, and if so, how?
- **Data:** WGS data, clinical data, and demographic information.
- **Approach:** Use methods specifically designed to identify rare variants, such as burden tests or rare variant association studies.

2. Investigating structural variants with dbVar.

a. Explore the dbVar database and identify a structural variant associated with a particular genetic disorder.

dbVar is a comprehensive database of human genomic structural variation (SV). It provides a platform to search, view, and download data from various studies on SVs.

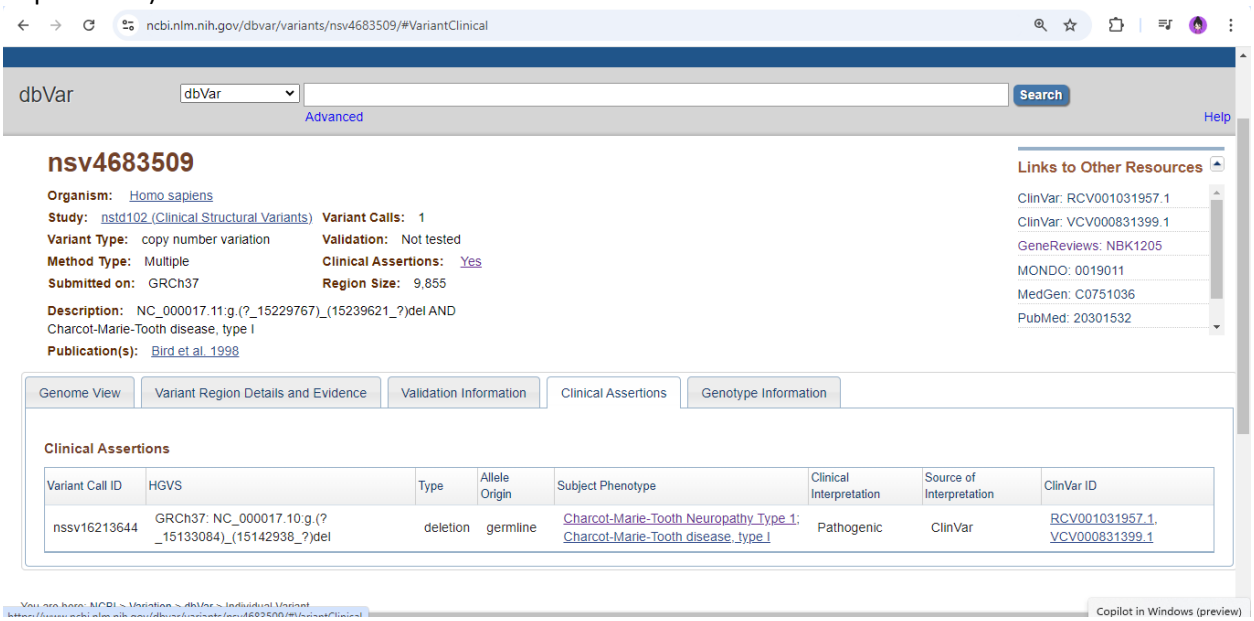
Identifying a Structural Variant Associated with a Genetic Disorder

Charcot-Marie-Tooth (CMT) disease, a hereditary peripheral neuropathy. CMT is often caused by deletions or duplications in the PMP22 gene.

Steps to explore dbVar:

1. Access dbVar: Visit the dbVar website: https://www.ncbi.nlm.nih.gov/dbvar/
2. Search for the gene: In the search bar, enter "PMP22".

3. Filter results: Use the available filters to narrow down the results to structural variants (e.g., deletions, duplications).



4. Examine variants: Review the identified variants, paying attention to their size, location, and frequency in the population.

CMT1 is a hereditary peripheral neuropathy characterized by distal muscle weakness, atrophy, sensory loss, and slow nerve conduction velocity. It is usually slowly progressive and often associated with pes cavus foot deformity and bilateral foot drop.

- Inheritance: Autosomal dominant pattern.
- Symptoms: Distal muscle weakness, atrophy, sensory loss, foot drop, pes cavus.
- Diagnosis: Slow nerve conduction velocity, family history, genetic testing (e.g., PMP22 duplication).
- Management: Symptomatic treatment, physical therapy, assistive devices, orthopedic surgery if needed.
- Genetic counseling: Offspring of affected individuals have a 50% risk of inheriting the condition. Prenatal testing may be possible.

Types of CMT1:

- CMT1A: Most common, caused by a duplication of the PMP22 gene.
- CMT1B: Associated with mutations in the MPZ gene.
- CMT1C: Associated with mutations in the LITAF gene.
- CMT1D: Associated with mutations in the EGR2 gene.
- CMT1E: Associated with mutations in the PMP22 gene.
- CMT2E/1F: Associated with mutations in the NEFL gene.

5. Check clinical significance: Look for information on the clinical significance of the variants, such as whether they have been linked to CMT or other disorders.

   Example:

   You might find a variant like this:

- Variant: A deletion of the PMP22 gene (chr17:19719547-19736633)
- Clinical significance: Associated with CMT type 1A, a common form of the disease.

  Additional Considerations:

- Variant frequency: Consider the frequency of the variant in the general population. A higher frequency might suggest a more common cause of CMT.
- Co-occurrence: Examine if the variant is often found in combination with other genetic factors.
- Functional impact: Assess the potential functional consequences of the variant on the PMP22 protein.

  By exploring dbVar in this manner, researchers can identify specific structural variants that contribute to genetic disorders like CMT. This information can be valuable for understanding disease mechanisms, developing diagnostic tests, and exploring potential therapeutic targets.

b. Provide details about the variant, including its type, location in the genome, and any known clinical implications.

Variant Details for Charcot-Marie-Tooth (CMT) Disease PMP22 Deletion

Variant Type: Deletion

Location in the Genome: Chromosome 17, positions 19719547 to 19736633 (chr17:19719547-19736633)

Gene Affected: PMP22 (Peripheral Myelin Protein 22)

Clinical Implications:

- **Hereditary Motor and Sensory Neuropathy (HMSN):** CMT is a type of HMSN, characterized by progressive muscle weakness and atrophy, primarily in the hands, feet, and lower legs.
- **Dominant Inheritance:** The PMP22 deletion is usually inherited in a dominant pattern, meaning a single copy of the affected gene can cause the disease.
- **Variable Severity:** The severity of CMT symptoms can vary widely among individuals with the PMP22 deletion. Some may experience mild symptoms, while others may have more severe disabilities.
- **Age of Onset:** Symptoms typically begin in childhood or adolescence.
- **Progression:** The disease usually progresses slowly over time.

Additional Information:

- **Duplications:** In addition to deletions, duplications of the PMP22 gene can also cause CMT.
- **Point Mutations:** Rare point mutations within the PMP22 gene can also lead to CMT.
- **Treatment:** There is currently no cure for CMT. Treatment focuses on managing symptoms and improving quality of life.

3. Accessing ClinicalTrials for genomics or for personalized medicine.

a. Access the ClinicalTrials database and find information about an ongoing clinical trial related to pharmacogenomics.

ClinicalTrials.gov is a publicly accessible database maintained by the U.S. National Institutes of Health (NIH). It provides information about clinical trials conducted around the world.

Clinical trial related to pharmacogenomics,

Step 1: Access the ClinicalTrials Database

1. Go to the ClinicalTrials.gov website.

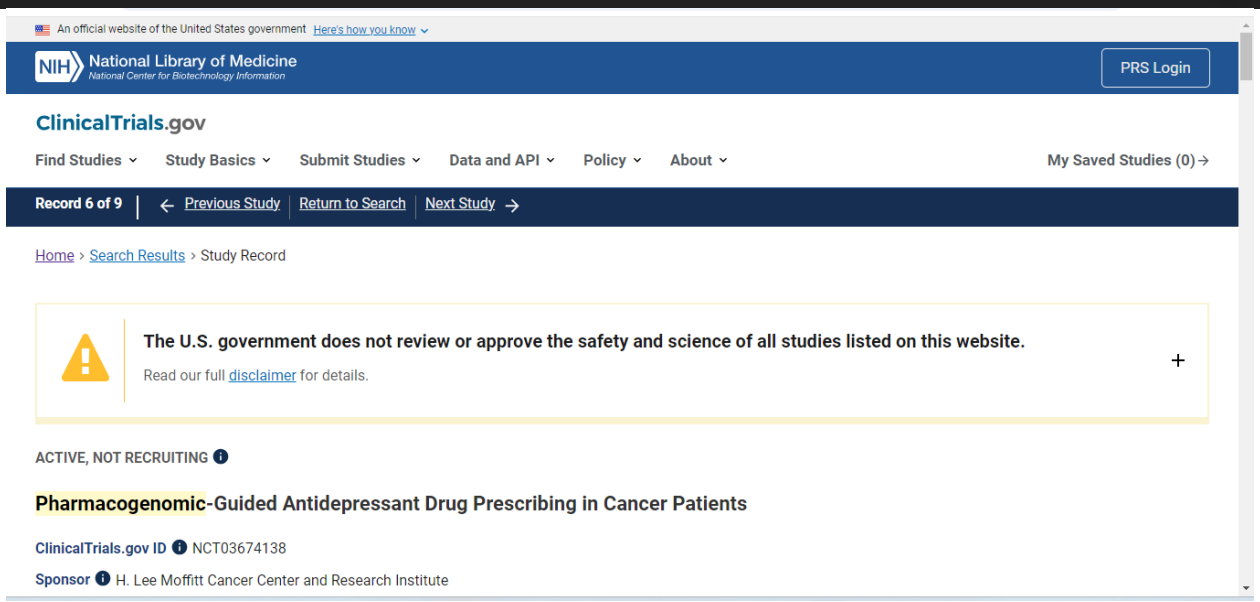
Step 2: Search for Pharmacogenomics-Related Trials

1. In the search bar, type **"pharmacogenomics"** or related terms like "personalized medicine" or "genomics."
2. Click on **Search**.

Step 3: Filter the Results

1. Once you get the results, you can filter by selecting:

    - **Status**: Choose "Recruiting" or "Active, not recruiting" to find ongoing trials.
    - **Condition or Disease**: If you're interested in a specific condition like cancer or cardiovascular diseases, you can narrow down the results using these filters.
    - **Study Type**: Filter for "Interventional" if you are particularly interested in active trials testing interventions.



Step 5: Access Detailed Information

Pharmacogenomic-Guided Antidepressant Drug Prescribing in Cancer Patients

This is a randomized, interventional clinical trial studying the effectiveness of DNA-guided therapy in selecting the best antidepressant medication for patients with depression and anxiety. The study is currently enrolling participants at H. Lee Moffitt Cancer Center and Research Institute in Tampa, Florida.

The study involves collecting a buccal swab for DNA analysis to identify the most suitable antidepressant medication for the participant. Participants will be masked to the DNA results for the first 12 weeks. The primary outcome measures are changes in depressive and anxious symptoms as measured by the Hospital Anxiety and Depression Scale (HADS) at baseline, week 12, and month 12. The secondary outcome measure is quality of life as measured by the Functional Assessment of Cancer Therapy-General (FACT-G) Scale at 12 months.

The study is currently ongoing and is estimated to be completed by October 2nd, 2024.

b. Summarize the trial's objectives, eligibility criteria, and expected outcomes.

Trial Summary: Pharmacogenomic-Guided Antidepressant Drug Prescribing in Cancer Patients

<u>Objectives:</u>

This trial aims to assess the clinical impact of a **preemptive pharmacogenomics strategy** to guide antidepressant therapy in cancer patients. The trial compares **DNA-guided antidepressant prescribing** with traditional **clinical management**. The key objectives are to:

1. Determine if DNA-guided therapy improves treatment outcomes for depression and anxiety in cancer patients.
2. Measure the effects of this approach on **quality of life** and symptom reduction over time.

Eligibility Criteria:

Inclusion Criteria:

- **Cancer diagnosis**
- **Age 18 or older**
- Depression or anxiety score > 5 on a 10-point scale
- Life expectancy greater than **6 months**
- Normal liver and kidney function (serum bilirubin and creatinine < 1.5x upper limit, AST/ALT < 3x upper limit)
- Willingness to provide **informed consent** and complete self-assessment questionnaires

Exclusion Criteria:

- No cancer diagnosis
- Antidepressant management by non-Moffitt psychiatrists
- Known **pregnancy**
- History of **liver** or **stem cell transplant**
- **Cognitive or psychological impairments** (other than depression/anxiety)

Expected Outcomes:

Primary Outcome Measures:

1. **Change in Depressive Symptoms:** Measured by the **Hospital Anxiety and Depression Scale (HADS)** from baseline, at week 12, and month 12.
2. **Change in Anxiety Symptoms:** Measured by **HADS** at the same time intervals.

Secondary Outcome Measures:

1. **Quality of Life:** Assessed using the **Functional Assessment of Cancer Therapy-General (FACT-G)** scale over 12 months, focusing on physical, emotional, social/family, and functional well-being.

Study Design:

- **Randomized, interventional model with crossover assignment**
- **Single (participant) masking:** Participants are blinded to genomic results for 12 weeks.
- Two arms:
    1. **DNA-guided choice of therapy**: Antidepressant choice based on **genotyping** (buccal swab).
    2. **Clinical management**: Traditional clinical approach.

Study Timeline:

- **Start date**: September 12, 2018
- **Primary completion**: September 3, 2021
- **Estimated study completion**: October 2, 2024
- **Enrollment**: 99 participants

The trial evaluates if personalized antidepressant therapy, based on pharmacogenomic data, can lead to better management of depression and anxiety in cancer patients.

c. Discuss the potential impact of the trial on personalized medicine and patient care.

Potential Impact of the Trial on Personalized Medicine and Patient Care

1. Tailored Antidepressant Therapy:

This trial leverages **pharmacogenomics** to personalize antidepressant therapy based on a patient's genetic profile. By guiding drug choice through **DNA genotyping**, the trial could:

- Optimize the efficacy of antidepressants for cancer patients.
- Minimize adverse drug reactions by selecting medications that align with the patient's genetic makeup.
- Improve **treatment adherence** by reducing side effects, enhancing overall patient outcomes.

2. Enhanced Mental Health Support in Cancer Care:

Cancer patients often experience heightened **depression** and **anxiety** due to their diagnosis and treatment. Personalized antidepressant prescribing could improve:

- **Mental health management**, helping patients cope more effectively.
- Their **quality of life**, as the trial also measures improvements in emotional well-being and social functioning using scales like **FACT-G** and **HADS**.

3. Early Adoption of Precision Medicine:

This trial represents a move toward **precision medicine**, where treatments are increasingly based on individual genetic differences. It demonstrates:

- The **feasibility** of integrating pharmacogenomics into routine clinical care.
- The possibility of scaling this approach to other **mental health** conditions and beyond, fostering more **targeted therapies** in broader medical fields.

4. Reduced Trial-and-Error in Prescribing:

Traditional antidepressant prescribing often follows a **trial-and-error** approach, which can lead to prolonged suffering and delayed relief. This trial could reduce the need for:

- Multiple medication changes, which are often necessary when patients do not respond to initial treatments.
- Prolonged symptom management, thus improving **time to recovery** and reducing healthcare costs.

5. Broader Applications in Oncology:

For cancer patients, managing comorbid conditions like **depression** is critical for comprehensive care. Pharmacogenomics may help:

- Customize other treatments (e.g., **pain management** or anti-nausea drugs), enhancing overall **supportive care** in oncology.
- Address the broader impact of mental health on **treatment outcomes** and **survival rates**, since better mental health often correlates with improved **treatment compliance** and overall health outcomes.

6. Foundation for Future Research:

The trial could pave the way for:

- **Further studies** in personalized medicine, expanding to other populations and conditions.
- **Innovative therapies** in mental health management, potentially leading to the development of novel drugs targeting specific genetic markers.

4. Comparing genetic variations with dbSNP and ClinVar.

a. Compare the genetic variations associated with any specific disease or trait in the dbSNP and ClinVar databases.

Both **dbSNP** and **ClinVar** databases are crucial for understanding genetic variations, but they serve slightly different purposes. Here's a comparison based on how each database handles genetic variations associated with diseases or traits:

1. Purpose and Focus:

- **dbSNP (Single Nucleotide Polymorphism Database)**:

- Focuses on **cataloging genetic variants** (e.g., single nucleotide polymorphisms or SNPs, small insertions/deletions) across all populations.
- It is more **comprehensive** for cataloging all types of variations, including those not linked to disease.
- Often used as a resource for **neutral polymorphisms** that might not have clinical significance.

- **ClinVar (Clinical Variation Database)**:

  - Focuses on **clinically relevant variations** associated with diseases and traits.
  - Variants in ClinVar are usually tied to specific **clinical conditions** and are curated based on their **pathogenicity** or **benignity**.
  - Contains expert-curated **clinical significance** labels (e.g., pathogenic, likely benign, uncertain significance), which provide insights into the health impacts of specific variants.

2. Data Types and Content:

- **dbSNP**:

  - Contains a broad spectrum of **neutral and clinically relevant variants**.
  - Variants include **SNPs**, **insertions/deletions**, and **short tandem repeats**.
  - Primarily for researchers looking to explore variations across **human populations** or find **common variants** in different genomic regions.
  - **Lacks detailed clinical interpretation**.

- **ClinVar**:

  - Focuses on **disease-associated variants**, providing detailed **clinical annotations**.
  - Variants include **SNPs**, **copy number variations (CNVs)**, and **structural variants**.
  - Provides **phenotypic and clinical context**, indicating how certain variations impact specific diseases or traits.
  - It offers detailed information on **submissions** from clinical laboratories, researchers, and medical institutions.

3. Example Comparison:

For a specific disease or trait, like **Cystic Fibrosis (CF)**, here's how each database would handle a common genetic variation such as **rs113993960**, associated with the **CFTR gene**:

- **In dbSNP**:

  - The SNP rs113993960 is listed, with information about its location, allele frequency, and population distribution.
  - It provides no definitive information about its clinical relevance but catalogs it as a **variation in CFTR**.
  - dbSNP acts as a variant reference with limited disease associations.

- **In ClinVar**:

  - The same variant, rs113993960, is listed but with **clinical interpretation** indicating that it is **pathogenic** for Cystic Fibrosis.
  - There are **supporting clinical submissions** from various labs, showing that this mutation leads to the disease.
  - ClinVar would also link this variant to **specific clinical reports** and show how it's been evaluated by experts.

4. Clinical Relevance:

- **dbSNP**:

  - Mainly contains variants for **research purposes**, and many entries are not associated with any disease.
  - Data is primarily useful for genome-wide association studies (GWAS), population genetics, and evolutionary studies.

- **ClinVar**:

  - Focused on **medical applications** and **diagnostic purposes**.
  - Directly relevant for clinicians and researchers working on **personalized medicine** or **disease diagnosis**.
  - Variants have annotations about **clinical validity**, including their **evidence** for being pathogenic or benign.

b. Highlight any discrepancies or additional information provided by each database.

Discrepancies or Additional Information in dbSNP and ClinVar

When comparing genetic variations across **dbSNP** and **ClinVar**, you might notice certain discrepancies or differences in how the two databases handle information. Here are some key points that highlight the differences between these databases:

1. Clinical Significance and Interpretation

- **dbSNP**:

  - **No clinical interpretation**: dbSNP primarily catalogues genetic variants without interpreting their potential health impact.
  - Example: A SNP may be listed in dbSNP with basic information such as its location (chromosomal coordinates) and minor allele frequency (MAF) across populations, but

there's **no information** on whether it's pathogenic, benign, or associated with any disease.

- **ClinVar**:
  - **Clinically significant information**: ClinVar provides **clinical interpretations** of variants, indicating if they are pathogenic, likely pathogenic, benign, or of uncertain significance.
  - Example: For the same SNP found in dbSNP, ClinVar might label it as **"pathogenic"** for a particular disorder, based on clinical evidence submitted by researchers or healthcare providers.
  - **Discrepancy**: The same variant could appear in both databases, but only **ClinVar** will provide clinical context or medical relevance, which may not be present in dbSNP.

2. Variant Frequency and Population Data

- **dbSNP**:
  - Focuses heavily on **variant frequencies** across different populations and subpopulations. It provides detailed allele frequency information from datasets such as the 1000 Genomes Project, ExAC, or gnomAD.
  - Example: dbSNP will display the **minor allele frequency (MAF)** of a variant in specific populations, which helps in understanding how common or rare a variant is across different ancestries.

- **ClinVar**:
  - ClinVar often includes **less frequency data** than dbSNP because its focus is more on **clinical relevance** rather than population genetics.
  - **Discrepancy**: In some cases, a variant may have rich frequency data in dbSNP but not much in ClinVar, where its pathogenicity is prioritized over its prevalence.

3. Submission Sources and Evidence Level

- **dbSNP**:
  - Variants in dbSNP are often added through large-scale **genome projects**, databases like **Ensembl**, or **population studies**. The focus is on collecting variants at a **genome-wide scale** rather than clinical submissions.
  - No direct clinical submissions or disease-specific annotations are tied to the variations.

- **ClinVar**:
  - ClinVar heavily relies on **clinical submissions** from laboratories, healthcare professionals, and researchers who provide evidence that a particular variant is linked to a condition.
  - It includes detailed information on the **source** of the data, such as genetic testing labs or curated databases like OMIM or HGMD.

- **Additional Information**: ClinVar provides the **evidence level** for each variant (e.g., reviewed by multiple submitters, expert panel-reviewed), giving insights into the **reliability** of the clinical significance.

4. Number of Variants Cataloged

- **dbSNP**:
  - Contains a far greater number of **neutral variants**. Not all variants in dbSNP have known clinical significance. It is a broader catalog designed to include even the most common polymorphisms found in the general population.
  - **More extensive** in terms of the total number of variants cataloged because it includes both **clinically relevant** and **neutral** variants.

- **ClinVar**:
  - Primarily focuses on variants with **clinical significance**, leading to a smaller subset of genetic variations.
  - Variants without strong clinical evidence or association with diseases may not appear in ClinVar.

5. Variant Naming and Classification

- **dbSNP**:
  - dbSNP assigns **rsIDs** (Reference SNP IDs) to variants, which are identifiers for all types of genetic polymorphisms (including those with unknown effects). These rsIDs are universally used to catalog SNPs.
  - It uses **standardized nomenclature** without specific information about the variant's effect on protein function or phenotype.

- **ClinVar**:
  - ClinVar also includes rsIDs for variants but often provides more information about how the variant affects protein function (e.g., **missense, nonsense**, or **frameshift** mutations).
  - ClinVar might group variants based on **clinical presentations** or different levels of severity, which dbSNP does not do.

6. Functional Annotation

- **dbSNP**:
  - Provides minimal **functional annotation**. Although it might include links to resources like the **UCSC Genome Browser**, the focus is on variant identification rather than functional analysis.

- Example: It tells you where a SNP is located (e.g., intronic, exonic), but does not elaborate on how this affects gene function or phenotype.

- **ClinVar**:

  - **Detailed functional annotation** related to how a variant may affect protein structure, function, or splicing, based on submissions from clinical studies.
  - **Additional Information**: ClinVar may describe how a variant influences the risk of developing a disease or how it alters **drug response** in the context of pharmacogenomics.

5. Utilize SNOMED CT to identify terms related to a specific medical condition.

To identify terms related to a specific medical condition using **SNOMED CT (Systematized Nomenclature of Medicine - Clinical Terms)**, here's a general process that you can follow:

Example Condition: Diabetes Mellitus

Step 1: Access SNOMED CT Database

- One can access SNOMED CT through:

  - The **SNOMED CT Browser** provided by the National Library of Medicine (NLM).
  - **SNOMED International's browser**.
  - **UMLS Terminology Services (UTS)** from the U.S. National Library of Medicine.

Step 2: Search for the Medical Condition

- In the SNOMED CT browser, search for **"Diabetes Mellitus"**.

Step 3: Identify Relevant Terms

Search for **Diabetes Mellitus**, SNOMED CT provides various related terms categorized under specific **concepts**, which may include:

<u>Core Terms:</u>

1. **Diabetes Mellitus (Disorder)**:

   - SNOMED CT Concept ID: **73211009**
   - Definition: A group of metabolic diseases characterized by high blood sugar levels over a prolonged period.

2. **Type 1 Diabetes Mellitus (Disorder)**:

    - SNOMED CT Concept ID: **46635009**
    - Definition: A form of diabetes in which there is a deficiency of insulin production.

3. **Type 2 Diabetes Mellitus (Disorder)**:

    - SNOMED CT Concept ID: **44054006**
    - Definition: A form of diabetes in which the body becomes resistant to insulin or fails to produce enough.

4. **Gestational Diabetes (Disorder)**:

    - SNOMED CT Concept ID: **11687002**
    - Definition: Diabetes diagnosed during pregnancy that is not clearly overt diabetes.

Complications Related to Diabetes:

5. **Diabetic Retinopathy (Disorder)**:

    - SNOMED CT Concept ID: **422034002**
    - Definition: A diabetes-related eye disorder that can cause vision loss and blindness.

6. **Diabetic Nephropathy (Disorder)**:

    - SNOMED CT Concept ID: **90721000119102**
    - Definition: Kidney damage caused by diabetes.

7. **Diabetic Ketoacidosis (Disorder)**:

    - SNOMED CT Concept ID: **420898005**
    - Definition: A life-threatening complication of diabetes that occurs when the body produces high levels of ketones.

Management and Interventions:

8. **Insulin Therapy for Diabetes Mellitus (Procedure)**:

    - SNOMED CT Concept ID: **182893008**
    - Definition: Treatment involving insulin to control blood sugar levels in diabetes.

9. **Blood Glucose Monitoring (Procedure)**:

    - SNOMED CT Concept ID: **312824007**
    - Definition: Regular measurement of blood glucose levels to manage diabetes.

10. **Diabetic Education (Procedure)**:

- SNOMED CT Concept ID: **410211006**
- Definition: Educational support for managing diabetes through diet, exercise, and medication adherence.

Other Related Concepts:

11. **Prediabetes (Finding)**:

- SNOMED CT Concept ID: **15777000**
- Definition: A condition where blood sugar levels are higher than normal but not high enough to be classified as diabetes.

12. **Hyperglycemia (Finding)**:

- SNOMED CT Concept ID: **80394007**
- Definition: An abnormally high level of glucose in the blood, commonly associated with diabetes.

13. **Hypoglycemia (Finding)**:

- SNOMED CT Concept ID: **80394008**
- Definition: Low blood sugar levels, which can be a complication of diabetes treatment.

Step 4: Explore Hierarchical Relationships

SNOMED CT organizes medical conditions and procedures in a hierarchical structure. You can explore **parent concepts** (broader categories) and **child concepts** (specific subtypes). For example, **Diabetes Mellitus** falls under **Endocrine, Nutritional, and Metabolic Disorders**, and you can drill down into specific types of diabetes.

Step 5: Access Synonyms and Definitions

SNOMED CT also provides **synonyms**, making it easier to find related terms that might be used interchangeably in different contexts. For instance, **Insulin-Dependent Diabetes Mellitus (IDDM)** is a synonym for **Type 1 Diabetes**.