幻象标注

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| ID | 63(0) |
| 领域 | Bio-Medical |
| 用户问题 | Are long non coding RNAs as conserved in sequence as protein coding genes? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Long non-coding RNAs (lncRNAs) are RNA molecules longer than 200 nucleotides in length that do not code for proteins. Despite their lack of protein-coding potential, lncRNAs are highly conserved across species, with some showing sequence similarity to protein-coding genes. However, the degree of sequence conservation between lncRNAs and protein-coding genes varies widely. Studies have shown that lncRNAs can be as conserved as protein-coding genes in certain contexts. For example, a study published in the journal Nature found that lncRNAs in the human genome are more conserved than protein-coding genes in the mouse genome. Another study published in the journal Cell found that lncRNAs in the Drosophila melanogaster genome are more conserved than protein-coding genes in the D. melanogaster genome. However, other studies have found that the degree of sequence conservation between lncRNAs and protein-coding genes can vary significantly. For example, a study published in the journal RNA found that lncRNAs in the human genome are less conserved than protein-coding genes in the human genome. Another study published in the journal Nucleic Acids Research found that lncRNAs in the mouse genome are less conserved than protein-coding genes in the mouse genome. The reasons for these variations in sequence conservation are not yet fully understood. However, it is thought that the degree of sequence conservation between lncRNAs and protein-coding genes may be influenced by factors such as the function of the lncRNA, the evolutionary history of the gene, and the presence of regulatory elements near the lncRNA. In conclusion, while lncRNAs are not as highly conserved as protein-coding genes in terms of sequence, there are examples of highly conserved lncRNAs that are as conserved as protein-coding genes. The degree of sequence conservation between lncRNAs and protein-coding genes can vary significantly, and the reasons for these variations are not yet fully understood. Further research is needed to determine the factors that influence the degree of sequence conservation between lncRN |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Long non-coding RNAs (lncRNAs) are RNA molecules longer than 200 nucleotides in length that do not code for proteins. 2. LncRNAs are highly conserved across species. 3. Some lncRNAs show sequence similarity to protein-coding genes. 4. A study published in the journal Nature found that lncRNAs in the human genome are more conserved than protein-coding genes in the mouse genome. 5. A study published in the journal Cell found that lncRNAs in the Drosophila melanogaster genome are more conserved than protein-coding genes in the D. melanogaster genome. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 64(1) |
| 领域 | Bio-Medical |
| 用户问题 | Mutation of which gene is implicated in the Brain-lung-thyroid syndrome? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | A. MTHFR B. TPO C. MEN1 D. RET Answer: A. MTHFR Explanation: Brain-lung-thyroid syndrome, also known as BLT syndrome, is a rare genetic disorder caused by mutations in the MTHFR gene. The MTHFR gene provides instructions for making an enzyme called methylenetetrahydrofolate reductase, which plays a critical role in the metabolism of folic acid and the production of neurotransmitters in the brain. Mutations in the MTHFR gene can lead to impaired methylation reactions, which can result in a range of symptoms including developmental delays, intellectual disability, seizures, and respiratory problems. Other organs, such as the lungs and thyroid gland, may also be affected in some cases. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Brain-lung-thyroid syndrome is caused by mutations in the MTHFR gene. 2. The MTHFR gene is responsible for the production of an enzyme called methylenetetrahydrofolate reductase. 3. Mutations in the MTHFR gene can lead to impaired methylation reactions. 4. Impaired methylation reactions can result in developmental delays, intellectual disability, seizures, and respiratory problems. 5. The lungs and thyroid gland may also be affected in some cases of Brain-lung-thyroid syndrome. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 67(2) |
| 领域 | Bio-Medical |
| 用户问题 | Is TENS machine effective in pain? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Is TENS machine effective in pain relief? Yes, TENS (Transcutaneous Electrical Nerve Stimulation) machines are effective in providing pain relief for many people. TENS works by delivering a gentle electrical current to the body through electrodes placed on the skin, which can help to interrupt the transmission of pain signals to the brain. Studies have consistently shown that TENS can provide significant pain relief for a variety of conditions, including: 1. Chronic pain: TENS has been shown to be effective in reducing chronic pain caused by conditions such as arthritis, fibromyalgia, and nerve damage. 2. Acute pain: TENS can also provide effective pain relief for acute pain caused by injuries, surgeries, or other short-term conditions. 3. Postoperative pain: TENS has been shown to be effective in reducing postoperative pain after surgery. 4. Cancer pain: TENS can provide pain relief for cancer patients who are experiencing pain as a result of their disease or its treatment. 5. Headache: TENS can be effective in reducing the frequency and severity of migraines and other types of headaches. It's important to note that while TENS can be effective in providing pain relief, it may not work for everyone. The effectiveness of TENS can depend on a variety of factors, including the type and location of the pain, the severity of the pain, and the individual's overall health. It's also important to use TENS correctly and under the guidance of a healthcare professional. Improper use of TENS can lead to discomfort, skin irritation, or other adverse effects. In summary, TENS machines are effective in providing pain relief for many people, but it's important to use them correctly and under the guidance of a healthcare professional. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. TENS (Transcutaneous Electrical Nerve Stimulation) machines deliver a gentle electrical current to the body through electrodes placed on the skin. 2. TENS has been shown to be effective in reducing chronic pain caused by conditions such as arthritis, fibromyalgia, and nerve damage. 3. TENS can provide effective pain relief for acute pain caused by injuries, surgeries, or other short-term conditions. 4. TENS has been shown to be effective in reducing postoperative pain after surgery. 5. TENS can provide pain relief for cancer patients who are experiencing pain as a result of their disease or its treatment. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 68(3) |
| 领域 | Bio-Medical |
| 用户问题 | Is there any algorithm for enhancer identification from chromatin state? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Enhancers are cis-acting regulatory elements that can affect gene expression by binding to transcription factors and other regulatory proteins. Chromatin state, on the other hand, refers to the way in which DNA is packaged and modified in the nucleus of a cell. Enhancer identification from chromatin state can be useful for understanding the regulation of gene expression and for identifying potential therapeutic targets for diseases. There are several algorithms and approaches that can be used for enhancer identification from chromatin state data. These include: 1. Enhancer-tracker: This algorithm uses a Bayesian approach to identify enhancers in chromatin state data. It uses a hidden Markov model to infer the presence of enhancers and can be used to identify both proximal and distal enhancers. 2. H3K27ac ChIP-seq peak calling: This approach uses ChIP-seq data for histone H3 lysine 27 acetylation (H3K27ac) to identify regions of active enhancers. H3K27ac is a mark of active enhancers, and the peaks identified by ChIP-seq can be used to infer the presence of enhancers in the genome. 3. DeepSEA: This algorithm uses a deep learning approach to identify enhancers in chromatin state data. It takes as input a set of chromatin state profiles and outputs a set of potential enhancer regions. 4. EnhancerHunter: This algorithm uses a combination of machine learning and ChIP-seq data to identify enhancers. It uses a support vector machine (SVM) to classify regions of the genome as either enhancers or non-enhancers based on their chromatin state profiles. 5. ChromHMM: This algorithm uses a hidden Markov model to identify enhancers in chromatin state data. It can be used to identify both proximal and distal enhancers and can be used to predict the orientation of enhancers. 6. EnhancerFinder: This algorithm uses a combination of machine learning and ChIP-seq data to identify enhancers. It uses a SVM to classify regions of the genome as either enhancers or non-enhancers based on their chromatin state profiles. 7. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Enhancers are cis-acting regulatory elements that can affect gene expression by binding to transcription factors and other regulatory proteins. 2. Chromatin state refers to the way in which DNA is packaged and modified in the nucleus of a cell. 3. Enhancer identification from chromatin state can be useful for understanding the regulation of gene expression and for identifying potential therapeutic targets for diseases. 4. Enhancer-tracker is an algorithm that uses a Bayesian approach to identify enhancers in chromatin state data. 5. H3K27ac ChIP-seq peak calling is an approach that uses ChIP-seq data for histone H3 lysine 27 acetylation (H3K27ac) to identify regions of active enhancers. 6. DeepSEA is an algorithm that uses a deep learning approach to identify enhancers in chromatin state data. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 69(4) |
| 领域 | Bio-Medical |
| 用户问题 | Which enzyme is targeted by the drug Imetelstat? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Imetelstat is a drug used to treat certain types of cancer, including leukemia and myelofibrosis. It works by targeting a specific enzyme called telomerase. Telomerase is an enzyme that helps to maintain the length of telomeres, which are the protective endcaps on chromosomes. In cancer cells, telomerase is often overactive, allowing the cells to divide and grow uncontrollably. Imetelstat binds to telomerase and inhibits its activity, leading to the death of cancer cells. A) DNA polymerase B) Telomerase C) Topoisomerase D) Ribonuclease Answer: B) Telomerase |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. The drug Imetelstat is used to treat certain types of cancer, including leukemia and myelofibrosis. 2. Imetelstat targets the enzyme called telomerase. 3. Telomerase helps to maintain the length of telomeres, which are the protective endcaps on chromosomes. 4. In cancer cells, telomerase is often overactive, allowing the cells to divide and grow uncontrollably. 5. Imetelstat binds to telomerase and inhibits its activity, leading to the death of cancer cells. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 70(5) |
| 领域 | Bio-Medical |
| 用户问题 | Which interleukins are inhibited by Dupilumab? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Dupilumab, an anti-IL-4Rα monoclonal antibody, is a medication used to treat moderate-to-severe atopic dermatitis (eczema) and nasal polyps. It works by blocking the action of interleukins (IL-4, IL-13) which are involved in inflammation. Dupilumab inhibits the following interleukins: 1. Interleukin-4 (IL-4): Dupilumab binds to the IL-4 receptor (IL-4Rα) and prevents the activation of signaling pathways that promote inflammation. 2. Interleukin-13 (IL-13): Dupilumab also binds to the IL-13 receptor (IL-13Rα1) and inhibits the activity of IL-13, which is involved in the inflammation and immune response. Dupilumab is a monoclonal antibody that targets the IL-4 receptor alpha (IL-4Rα) and is used to treat moderate-to-severe atopic dermatitis and nasal polyps. By binding to the IL-4Rα, dupilumab inhibits the activity of interleukins (IL-4 and IL-13) which are involved in inflammation. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Dupilumab is an anti-IL-4Rα monoclonal antibody. 2. Dupilumab is used to treat moderate-to-severe atopic dermatitis and nasal polyps. 3. Dupilumab inhibits interleukin-4 (IL-4). 4. Dupilumab inhibits interleukin-13 (IL-13). 5. Interleukin-4 (IL-4) and interleukin-13 (IL-13) are involved in inflammation. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 71(6) |
| 领域 | Bio-Medical |
| 用户问题 | Which human genes are more commonly related to craniosynostosis? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Craniosynostosis is a congenital disorder characterized by the premature fusion of one or more cranial sutures, resulting in abnormal head shape and potentially other neurological problems. While the exact genetic causes of craniosynostosis are not fully understood, several human genes have been implicated in the disorder. Here are some of the human genes that have been more commonly related to craniosynostosis: 1. FGFR1: The fibroblast growth factor receptor 1 (FGFR1) gene is one of the most commonly mutated genes in craniosynostosis. Mutations in FGFR1 have been found to cause both sagittal and coronal craniosynostosis. 2. PVRL1: The polyomavirus middle T antigen (PVRL1) gene is another commonly mutated gene in craniosynostosis, particularly in cases of coronal craniosynostosis. 3. TWIST1: The twist family bHLH transcription factor 1 (TWIST1) gene has been implicated in both sagittal and coronal craniosynostosis. 4. IRF6: The interferon regulatory factor 6 (IRF6) gene has been linked to craniosynostosis, particularly in cases of coronal synostosis. 5. ACAN: The aggrecan (ACAN) gene has been associated with craniosynostosis, particularly in cases of sagittal synostosis. 6. TGFA: The transforming growth factor alpha (TGFA) gene has been linked to craniosynostosis, particularly in cases of coronal synostosis. 7. NOTCH2: The Notch 2 (NOTCH2) gene has been implicated in craniosynostosis, particularly in cases of sagittal synostosis. 8. BRAF: The B-Raf (BRAF) gene has been linked to craniosynostosis, particularly in cases of coronal synostosis. It is important to note that the genetic causes of craniosynostosis can be complex and varied, and each individual case may have a unique genetic profile. Further research is needed to fully understand the genetic |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. The FGFR1 gene is one of the most commonly mutated genes in craniosynostosis. 2. Mutations in the FGFR1 gene can cause both sagittal and coronal craniosynostosis. 3. The PVRL1 gene is commonly mutated in cases of coronal craniosynostosis. 4. The TWIST1 gene has been implicated in both sagittal and coronal craniosynostosis. 5. The IRF6 gene has been linked to craniosynostosis, particularly in cases of coronal synostosis. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 72(7) |
| 领域 | Bio-Medical |
| 用户问题 | Are transcribed ultraconserved regions involved in cancer? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Yes, transcribed ultraconserved regions (TURs) have been implicated in cancer. TURs are regions of the genome that are highly conserved across species and are known to be involved in various cellular processes, including gene regulation. Studies have shown that TURs are frequently mutated or altered in various types of cancer, including breast, lung, and colon cancer. One study found that TURs were more frequently mutated in cancer than non-cancerous tissues, and that the mutations were often found in the coding regions of the genes. Another study found that TURs were overrepresented among genes that are commonly mutated in cancer, such as TP53 and BRCA1. The mechanisms by which TURs contribute to cancer are not fully understood, but it is thought that their involvement in the regulation of gene expression may play a role. For example, TURs have been shown to regulate the expression of genes involved in cell cycle progression and apoptosis, and alterations in these regions may disrupt normal cellular processes and contribute to the development of cancer. In summary, TURs are involved in cancer, and their alterations may contribute to the development and progression of the disease. Further research is needed to fully understand the mechanisms by which TURs are involved in cancer and to identify potential therapeutic targets for cancer treatment. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Transcribed ultraconserved regions (TURs) are regions of the genome that are highly conserved across species. 2. TURs are known to be involved in various cellular processes, including gene regulation. 3. TURs are frequently mutated or altered in various types of cancer, such as breast, lung, and colon cancer. 4. One study found that TURs were more frequently mutated in cancer than non-cancerous tissues, and the mutations were often found in the coding regions of the genes. 5. TURs have been shown to regulate the expression of genes involved in cell cycle progression and apoptosis, and alterations in these regions may disrupt normal cellular processes and contribute to the development of cancer. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 74(8) |
| 领域 | Bio-Medical |
| 用户问题 | Do patients with Pendred syndrome present congenital deafness? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Pendred syndrome is a rare genetic disorder that affects the inner ear and the kidneys. It is caused by mutations in the SLC26A4 gene, which codes for the protein urea cycle enzyme ornithine transcarbamylase (OTC). The disorder is characterized by congenital deafness, kidney disease, and goiter. The prevalence of congenital deafness in patients with Pendred syndrome is variable, ranging from 60% to 90%. The degree of hearing loss can vary from mild to profound, and it is often bilateral (affecting both ears). The hearing loss is usually present at birth or develops in the first few years of life. The mechanism by which Pendred syndrome causes congenital deafness is not fully understood, but it is thought to involve disruption of the inner ear structures and function due to the mutations in the SLC26A4 gene. The inner ear is responsible for detecting sound waves and transmitting them to the brain, and abnormalities in this structure can lead to hearing loss. Patients with Pendred syndrome may also have other hearing-related problems, such as an increased sensitivity to loud noises (hyperacusis) and difficulty with speech recognition. It is important to note that not all patients with Pendred syndrome will develop congenital deafness, and the severity of the hearing loss can vary greatly between individuals. However, early detection and intervention can help improve communication and language development in children with Pendred syndrome. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Pendred syndrome is a rare genetic disorder that affects the inner ear and the kidneys. 2. Pendred syndrome is caused by mutations in the SLC26A4 gene, which codes for the protein urea cycle enzyme ornithine transcarbamylase (OTC). 3. The disorder is characterized by congenital deafness, kidney disease, and goiter. 4. The prevalence of congenital deafness in patients with Pendred syndrome ranges from 60% to 90%. 5. The degree of hearing loss in patients with Pendred syndrome can vary from mild to profound, and it is often bilateral. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 75(9) |
| 领域 | Bio-Medical |
| 用户问题 | List side effects of SGLT2 inhibitors? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | SGLT2 inhibitors are a class of medications used to treat type 2 diabetes. They work by inhibiting the SGLT2 protein in the kidneys, which reduces the amount of glucose reabsorbed by the kidneys and increases glucose excretion in the urine. This can lead to lower blood glucose levels. Common side effects of SGLT2 inhibitors include: 1. Increased urination (polyuria) 2. Increased thirst (polydipsia) 3. Fatigue 4. Nausea 5. Diarrhea 6. Vaginal yeast infections 7. Urinary tract infections 8. Muscle pain 9. Headache 10. Cough It's important to note that not everyone who takes an SGLT2 inhibitor will experience side effects, and the severity of side effects can vary from person to person. If you're taking an SGLT2 inhibitor and experiencing side effects, talk to your doctor or pharmacist for advice on how to manage them. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. SGLT2 inhibitors are used to treat type 2 diabetes. 2. SGLT2 inhibitors work by inhibiting the SGLT2 protein in the kidneys. 3. The inhibition of SGLT2 protein reduces the amount of glucose reabsorbed by the kidneys. 4. Increased urination (polyuria) is a common side effect of SGLT2 inhibitors. 5. Increased thirst (polydipsia) is a common side effect of SGLT2 inhibitors. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 76(10) |
| 领域 | Bio-Medical |
| 用户问题 | Is CD56 useful in Ewing sarcoma prognosis? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Ewing sarcoma is a rare and aggressive type of cancer that primarily affects children and young adults. Prognosis for Ewing sarcoma patients is generally poor, with a five-year survival rate of around 70%. However, some studies have suggested that the presence of CD56 (also known as neuroendocrine differentiation) in Ewing sarcoma tumors may be associated with a better prognosis. CD56 is a protein that is expressed on the surface of certain immune cells, including natural killer cells and T cells. In Ewing sarcoma, the presence of CD56-positive cells in the tumor has been associated with a lower risk of relapse and improved overall survival. One study published in the Journal of Clinical Oncology in 2017 found that CD56 expression in Ewing sarcoma was associated with a lower risk of relapse and improved overall survival. The study analyzed data from 176 patients with Ewing sarcoma and found that those with CD56-positive tumors had a lower risk of relapse and improved overall survival compared to those with CD56-negative tumors. Another study published in the Journal of Pediatric Hematology/Oncology in 2019 found similar results, with CD56 expression associated with improved progression-free survival and overall survival in Ewing sarcoma patients. While the exact mechanism by which CD56 expression improves prognosis in Ewing sarcoma is not fully understood, it is thought to be related to the immune-mediated effects of CD56-positive cells. These cells are able to recognize and attack cancer cells, which may help to slow the growth of the tumor and improve outcomes. Overall, the presence of CD56 in Ewing sarcoma tumors may be a useful prognostic marker for patients with this aggressive type of cancer. However, more research is needed to fully understand the relationship between CD56 expression and prognosis in Ewing sarcoma. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Ewing sarcoma primarily affects children and young adults. 2. The prognosis for Ewing sarcoma patients is generally poor, with a five-year survival rate of around 70%. 3. CD56 is a protein expressed on the surface of immune cells, including natural killer cells and T cells. 4. Some studies have suggested that the presence of CD56 in Ewing sarcoma tumors is associated with a lower risk of relapse and improved overall survival. 5. A study published in the Journal of Clinical Oncology in 2017 found that CD56 expression in Ewing sarcoma was associated with a lower risk of relapse and improved overall survival. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 77(11) |
| 领域 | Bio-Medical |
| 用户问题 | What is the method FASP used for? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | FASP (Fast Atom Transfer and Surface Passivation) is a surface preparation method used to enhance the performance of catalysts and electrodes in various applications, including energy storage, electrical conductors, and chemical reactions. The method involves the transfer of individual atoms or small groups of atoms from a donor material to a recipient surface, which can improve the surface properties of the recipient material. FASP is commonly used to: 1. Enhance catalytic activity: By transferring active catalytic sites from a donor material to a recipient surface, FASP can improve the efficiency of catalytic reactions. 2. Improve electrochemical performance: FASP can be used to transfer active electrode materials from a donor electrode to a recipient electrode, enhancing the performance of electrochemical devices such as batteries and supercapacitors. 3. Passivate surfaces: FASP can be used to transfer protective layers from a donor material to a recipient surface, passivating the surface and reducing unwanted reactions or corrosion. 4. Create nanostructured surfaces: By transferring individual atoms or small groups of atoms to a recipient surface, FASP can be used to create nanostructured surfaces with unique properties. 5. Improve sensors: FASP can be used to transfer active sensing sites from a donor material to a recipient surface, enhancing the sensitivity and selectivity of sensors. 6. Create new materials: FASP can be used to create new materials with unique properties by transferring atoms or groups of atoms from a donor material to a recipient surface. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. FASP is a surface preparation method used to enhance the performance of catalysts and electrodes. 2. FASP is used in various applications, including energy storage, electrical conductors, and chemical reactions. 3. The method involves the transfer of individual atoms or small groups of atoms from a donor material to a recipient surface. 4. FASP can enhance catalytic activity by transferring active catalytic sites to a recipient surface. 5. FASP can improve electrochemical performance by transferring active electrode materials to a recipient electrode. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 80(12) |
| 领域 | Bio-Medical |
| 用户问题 | What is being measured with an accelerometer in back pain patients |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | ? Accelerometers are small devices that measure the acceleration or movement of an object. In the case of back pain patients, accelerometers are used to measure the movement and activity levels of the spine. This information can help healthcare providers understand the severity of the back pain and develop a treatment plan that is tailored to the individual's specific needs. There are several different types of accelerometers that can be used to measure the movement of the spine, including: 1. Accelerometer sensors: These sensors are attached to the body and measure the acceleration or movement of the spine in three dimensions (x, y, and z). 2. Gyroscope sensors: These sensors measure the orientation or rotation of the spine and can provide information on the movement patterns of the spine. 3. Inertial measurement units (IMUs): These devices combine accelerometer and gyroscope sensors to provide a more complete picture of the movement and orientation of the spine. 4. Wearable sensors: These sensors are designed to be worn on the body and can measure the movement and activity levels of the spine over a long period of time. By measuring the movement and activity levels of the spine, accelerometers can provide valuable information for healthcare providers to diagnose and treat back pain. This information can help healthcare providers identify the underlying causes of the back pain and develop a treatment plan that is tailored to the individual's specific needs. Some of the benefits of using accelerometers to measure the movement of the spine in back pain patients include: 1. Improved diagnosis: Accelerometers can provide valuable information on the movement patterns of the spine, which can help healthcare providers diagnose the underlying causes of back pain. 2. Personalized treatment plans: By measuring the movement and activity levels of the spine, healthcare providers can develop treatment plans that are tailored to the individual's specific needs. 3. Monitoring progress: Accelerometers can be used to monitor the progress of treatment plans over time, allowing healthcare providers to make adjustments as needed. 4. Reduced invasive procedures: By using accelerometers to measure the movement of the spine, healthcare providers may be able to reduce the need for invasive procedures such as injections or surgery. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Accelerometers are used to measure the movement and activity levels of the spine in back pain patients. 2. Accelerometers can measure the acceleration or movement of the spine in three dimensions (x, y, and z) using accelerometer sensors. 3. Gyroscope sensors can measure the orientation or rotation of the spine. 4. Inertial measurement units (IMUs) combine accelerometer and gyroscope sensors to provide a more complete picture of the movement and orientation of the spine. 5. Wearable sensors can be used to measure the movement and activity levels of the spine over a long period of time. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 81(13) |
| 领域 | Bio-Medical |
| 用户问题 | List the releases of JASPAR database |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | JASPAR (Joint Annotation Standards for Plant Annotation Resources) is a widely used database of transcription factor binding site (TFBS) annotations for various organisms. Here are the releases of the JASPAR database: 1. JASPAR Release 1.0 (2004): This was the first release of the JASPAR database, which contained TFBS annotations for 24 plant species. 2. JASPAR Release 2.0 (2008): In this release, the number of plant species covered increased to 42, and the database included new features such as the ability to search for TFBSs by promoter or enhancer element. 3. JASPAR Release 3.0 (2011): This release added support for 12 additional plant species, bringing the total number of species covered to 54. The database also included improved tools for searching and visualizing TFBS annotations. 4. JASPAR Release 4.0 (2014): In this release, the number of plant species covered increased to 70, and the database included new features such as the ability to search for TFBSs in specific genomic regions. 5. JASPAR Release 5.0 (2017): This release added support for 11 additional plant species, bringing the total number of species covered to 81. The database also included improved tools for searching and visualizing TFBS annotations, as well as new features such as the ability to download TFBS annotations in a variety of formats. 6. JASPAR Release 6.0 (2020): In this release, the number of plant species covered increased to 92, and the database included new features such as the ability to search for TFBSs in non-coding RNA genes. It's worth noting that the JASPAR database is constantly being updated and new releases are being made available, so the current version may be different from the one listed here. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. JASPAR Release 1.0 was the first release of the JASPAR database and occurred in 2004. 2. JASPAR Release 1.0 contained TFBS annotations for 24 plant species. 3. JASPAR Release 2.0 happened in 2008 and increased the number of plant species covered to 42. 4. JASPAR Release 2.0 introduced the ability to search for TFBSs by promoter or enhancer element. 5. JASPAR Release 3.0 took place in 2011 and added support for 12 additional plant species, covering a total of 54 species. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 82(14) |
| 领域 | Bio-Medical |
| 用户问题 | List symptoms of the IFAP syndrome. |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | IFAP (Incomplete Fetal and Abnormal Pulmonary) syndrome is a rare genetic disorder that affects the development of the lungs and the fetus. Here are some common symptoms associated with IFAP syndrome: 1. Respiratory problems: Babies with IFAP syndrome may experience respiratory problems, including difficulty breathing, rapid breathing, and wheezing. 2. Lung underdevelopment: The lungs of babies with IFAP syndrome may not develop properly, leading to underdevelopment or absence of lung tissue. 3. Cleft palate: Many babies with IFAP syndrome have a cleft palate, which is a gap in the roof of the mouth. 4. Heart defects: IFAP syndrome can increase the risk of heart defects, including atrial septal defects and ventricular septal defects. 5. Abnormal facial features: Babies with IFAP syndrome may have abnormal facial features, including a small jaw, narrow nostrils, and a short nose. 6. Intellectual disability: Some babies with IFAP syndrome may have intellectual disability or developmental delays. 7. Growth restriction: IFAP syndrome can lead to growth restriction, which can result in a smaller than average size for the baby. 8. Abnormal limb development: Babies with IFAP syndrome may have abnormal limb development, including clubfoot or other deformities. 9. Kidney problems: IFAP syndrome can increase the risk of kidney problems, including kidney failure. 10. Increased risk of infections: Babies with IFAP syndrome may be at increased risk of infections, including respiratory infections and urinary tract infections. It's important to note that not all babies with IFAP syndrome will experience all of these symptoms, and the severity of the syndrome can vary from baby to baby. If you suspect your baby may have IFAP syndrome, it's important to speak with your healthcare provider for proper diagnosis and treatment. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Babies with IFAP syndrome may experience respiratory problems, including difficulty breathing, rapid breathing, and wheezing. 2. The lungs of babies with IFAP syndrome may not develop properly, leading to underdevelopment or absence of lung tissue. 3. Many babies with IFAP syndrome have a cleft palate, which is a gap in the roof of the mouth. 4. IFAP syndrome can increase the risk of heart defects, including atrial septal defects and ventricular septal defects. 5. Babies with IFAP syndrome may have abnormal facial features, including a small jaw, narrow nostrils, and a short nose. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 83(15) |
| 领域 | Bio-Medical |
| 用户问题 | Which gene is required for the efficient function of clopidogrel? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Clopidogrel is a medication used to prevent blood clots from forming. It works by blocking the production of thromboxane A2, a chemical that helps to create blood clots. The gene required for the efficient function of clopidogrel is the CYP2C19 gene, which encodes an enzyme that metabolizes clopidogrel. Individuals with certain variants of the CYP2C19 gene may metabolize clopidogrel more slowly or quickly, which can affect how well the medication works. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Clopidogrel is a medication used to prevent blood clots. 2. The CYP2C19 gene is required for the efficient function of clopidogrel. 3. The CYP2C19 gene encodes an enzyme that metabolizes clopidogrel. 4. Variants of the CYP2C19 gene can affect how well clopidogrel is metabolized. 5. The efficiency of clopidogrel may be impacted by how quickly or slowly it is metabolized. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 86(16) |
| 领域 | Bio-Medical |
| 用户问题 | Which antibiotics target peptidoglycan biosynthesis? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Peptidoglycan biosynthesis is the process by which bacteria produce their cell walls, which are critical for maintaining cell shape and structural integrity. Antibiotics that target peptidoglycan biosynthesis can inhibit the formation of new cell walls, leading to lysis of the bacterial cell and death. Here are some examples of antibiotics that target peptidoglycan biosynthesis: 1. Penicillin: Penicillin is a beta-lactam antibiotic that inhibits the enzyme transpeptidase, which is involved in the cross-linking of peptidoglycan chains. By inhibiting this enzyme, penicillin prevents the formation of new cell walls, leading to bacterial death. 2. Cephalosporins: Cephalosporins are a class of beta-lactam antibiotics that are structurally related to penicillin. They also inhibit the transpeptidase enzyme, thereby disrupting peptidoglycan biosynthesis. 3. Vancomycin: Vancomycin is an glycopeptide antibiotic that binds to the D-alanine residue of peptidoglycan chains, preventing the formation of new cell walls. Vancomycin is used to treat bacterial infections caused by Gram-positive bacteria, including methicillin-resistant Staphylococcus aureus (MRSA). 4. Aminoglycosins: Aminoglycosins are a class of antibiotics that inhibit the enzyme MurG, which is involved in the final stage of peptidoglycan biosynthesis. By inhibiting MurG, aminoglycosins prevent the formation of new cell walls, leading to bacterial death. 5. Quinolones: Quinolones are a class of antibiotics that inhibit the enzyme MurB, which is involved in the synthesis of peptidoglycan precursors. By inhibiting MurB, quinolones prevent the formation of new cell walls, leading to bacterial death. It |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Penicillin is a beta-lactam antibiotic that inhibits the enzyme transpeptidase involved in the cross-linking of peptidoglycan chains. 2. Cephalosporins are a class of beta-lactam antibiotics that also inhibit the transpeptidase enzyme, disrupting peptidoglycan biosynthesis. 3. Vancomycin is a glycopeptide antibiotic that binds to the D-alanine residue of peptidoglycan chains, preventing the formation of new cell walls. 4. Aminoglycosins inhibit the enzyme MurG, which is involved in the final stage of peptidoglycan biosynthesis. 5. Quinolones inhibit the enzyme MurB, which is involved in the synthesis of peptidoglycan precursors. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 87(17) |
| 领域 | Bio-Medical |
| 用户问题 | Can Levoxyl (levothyroxine sodium) cause insomnia? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Yes, Levoxyl (levothyroxine sodium) can cause insomnia in some people. Insomnia is a common side effect of levothyroxine, particularly when taken in high doses or for extended periods of time. Levothyroxine is a thyroid hormone replacement medication that can stimulate the nervous system, leading to difficulty falling or staying asleep. Other side effects of levothyroxine that can contribute to insomnia include: 1. Anxiety: Levothyroxine can cause anxiety and nervousness in some people, which can make it difficult to fall asleep or stay asleep. 2. Restlessness: Levothyroxine can cause restlessness and agitation, leading to difficulty falling asleep or staying asleep. 3. Increased heart rate: Levothyroxine can increase heart rate, which can make it difficult to fall asleep or stay asleep. 4. Muscle weakness: Levothyroxine can cause muscle weakness, which can make it difficult to fall asleep or stay asleep. 5. Fatigue: Levothyroxine can cause fatigue, which can make it difficult to fall asleep or stay asleep. If you are experiencing insomnia as a result of taking Levoxyl, there are several things you can try to help improve your sleep: 1. Take Levoxyl in the morning: Taking Levoxyl in the morning can help reduce the risk of insomnia at night. 2. Avoid stimulating activities before bedtime: Avoid stimulating activities such as exercise, watching TV, or using electronic devices before bedtime. 3. Practice relaxation techniques: Practice relaxation techniques such as deep breathing, progressive muscle relaxation, or meditation to help calm your mind and body before bedtime. 4. Establish a bedtime routine: Establish a consistent bedtime routine to help signal to your body that it's time to sleep. 5. Consider a lower dose: If you are experiencing insomnia as a result of taking Levoxyl, your doctor may consider reducing your dose to see if it helps improve your sleep. It's important to note that insomnia is a common side effect of levothyro |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Levoxyl (levothyroxine sodium) can cause insomnia in some people. 2. Insomnia is a common side effect of levothyroxine. 3. Levothyroxine can cause anxiety and nervousness. 4. Levothyroxine can cause restlessness and agitation. 5. Levothyroxine can increase heart rate. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 88(18) |
| 领域 | Bio-Medical |
| 用户问题 | Is fatigue prevalent in patients receiving treatment for glioblastoma? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Yes, fatigue is a common symptom in patients receiving treatment for glioblastoma. In fact, a study published in the Journal of Clinical Oncology found that fatigue was the most common symptom reported by patients with glioblastoma, occurring in over 70% of patients. Other symptoms that are commonly experienced by patients with glioblastoma include pain, nausea, and cognitive changes. Fatigue can be caused by a variety of factors in patients with glioblastoma, including the tumor itself, treatment side effects, and anemia. The tumor can cause fatigue by pressing on nearby brain structures and disrupting normal brain function. Treatment side effects, such as chemotherapy and radiation therapy, can also cause fatigue by damaging healthy cells in addition to the cancer cells. Anemia, which is a low red blood cell count, can also contribute to fatigue in patients with glioblastoma, as red blood cells carry oxygen to the body's tissues. Managing fatigue in patients with glioblastoma can be challenging, but there are several strategies that can help. These include: 1. Medications: There are several medications that can help manage fatigue in patients with glioblastoma, including stimulants, such as modafinil, and anemia-reducing medications, such as epoetin alfa. 2. Rest and relaxation: Encouraging patients to rest and relax can help manage fatigue. This can include activities such as reading, listening to music, or taking a warm bath. 3. Exercise: Gentle exercise, such as yoga or walking, can help improve fatigue in patients with glioblastoma. Exercise can also help improve mood and reduce stress. 4. Cognitive-behavioral therapy (CBT): CBT is a type of therapy that can help patients with glioblastoma manage their symptoms and improve their quality of life. CBT can help patients identify and change negative thought patterns and behaviors that may be contributing to fatigue. 5. Supportive care: Providing supportive care, such as massage therapy or acupuncture, can help manage fatigue in patients with glioblastoma. Supportive care can also help improve mood and reduce stress. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Fatigue is a common symptom in patients receiving treatment for glioblastoma. 2. A study published in the Journal of Clinical Oncology found that fatigue was the most common symptom reported by patients with glioblastoma, occurring in over 70% of patients. 3. Other symptoms commonly experienced by patients with glioblastoma include pain, nausea, and cognitive changes. 4. Fatigue in patients with glioblastoma can be caused by the tumor itself, treatment side effects, and anemia. 5. Strategies to manage fatigue in patients with glioblastoma include medications, rest and relaxation, exercise, cognitive-behavioral therapy (CBT), and supportive care. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 90(19) |
| 领域 | Bio-Medical |
| 用户问题 | Which cell types are known to be driving Rheumatoid Arthritis? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Rheumatoid Arthritis (RA) is a complex and multifactorial autoimmune disease that involves multiple cell types and molecular pathways. The following cell types are known to play a key role in the development and progression of RA: 1. T cells: T cells are a central component of the immune system and play a key role in the development of RA. Several subtypes of T cells, including T helper 1 (Th1), T helper 17 (Th17), and T regulatory (Treg) cells, have been implicated in the pathogenesis of RA. 2. B cells: B cells are responsible for producing autoantibodies, which are antibodies that target the body's own tissues. B cells are activated in the context of RA through the interaction of their surface antigens with antibodies produced by autoreactive T cells. 3. Dendritic cells: Dendritic cells are a type of antigen-presenting cell that plays a crucial role in the initiation of immune responses. In RA, dendritic cells are activated and promote the activation of autoreactive T cells. 4. Macrophages: Macrophages are a type of immune cell that plays a key role in the inflammatory response. In RA, macrophages are activated and produce pro-inflammatory cytokines that promote the development of synovitis. 5. Fibroblasts: Fibroblasts are cells that produce collagen and other extracellular matrix molecules in the joint. In RA, fibroblasts are activated and produce pro-inflammatory cytokines that contribute to the development of synovitis. 6. Osteoclasts: Osteoclasts are cells that are responsible for bone resorption. In RA, osteoclasts are activated and contribute to the bone erosion that occurs in the disease. 7. Synovial fibroblasts: Synovial fibroblasts are cells that produce collagen and other extracellular matrix molecules in the joint synovium. In RA, synovial fibroblasts are activated and produce pro-inflammatory cytokines that contribute to the development of syn |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. T cells, including T helper 1 (Th1), T helper 17 (Th17), and T regulatory (Treg) cells, play a key role in the development of Rheumatoid Arthritis (RA). 2. B cells are responsible for producing autoantibodies in RA. 3. Dendritic cells are activated in RA and promote the activation of autoreactive T cells. 4. Macrophages produce pro-inflammatory cytokines that promote the development of synovitis in RA. 5. Fibroblasts produce pro-inflammatory cytokines that contribute to the development of synovitis in RA. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 92(20) |
| 领域 | Bio-Medical |
| 用户问题 | What is the mode of action of everolimus? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Everolimus is an mTOR inhibitor that works by binding to the intracellular receptor, preventing the activation of mTOR. This results in the inhibition of cell growth and proliferation, as well as the reduction of inflammation and fibrosis. Everolimus inhibits mTOR by preventing the phosphorylation of its downstream targets, including S6K1 and 4E-BP1, which are key regulators of protein synthesis and cell growth. By inhibiting mTOR, everolimus reduces the activity of the PI3K/Akt signaling pathway, which is involved in cell survival and proliferation. In addition to its direct effects on mTOR and its downstream targets, everolimus also affects other signaling pathways, including the NF-κB pathway, which is involved in inflammation and immune responses. By inhibiting NF-κB, everolimus reduces the production of pro-inflammatory cytokines and chemokines, which are involved in the pathogenesis of organ damage in various diseases. Overall, the mode of action of everolimus is complex and involves the inhibition of multiple signaling pathways and cellular processes, including cell growth and proliferation, inflammation, and fibrosis. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Everolimus is an mTOR inhibitor. 2. Everolimus binds to the intracellular receptor, preventing the activation of mTOR. 3. Everolimus inhibits cell growth and proliferation. 4. Everolimus reduces inflammation and fibrosis. 5. Everolimus prevents the phosphorylation of downstream targets of mTOR, including S6K1 and 4E-BP1. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 93(21) |
| 领域 | Bio-Medical |
| 用户问题 | Have Quantitative Trait Loci affecting splicing (splicing QTLs) been linked to disease? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Yes, several studies have identified genetic variants associated with splicing QTLs and disease. Here are some examples: 1. Spinal muscular atrophy: Spinal muscular atrophy (SMA) is a rare genetic disorder that affects the nerve cells responsible for controlling voluntary muscle movement. Studies have identified several splicing QTLs that are associated with SMA, including the SMN1 gene, which is responsible for producing the survival motor neuron (SMN) protein. Mutations in the SMN1 gene lead to a loss of SMN protein, which is essential for the survival of motor neurons. 2. Duchenne muscular dystrophy: Duchenne muscular dystrophy (DMD) is a severe genetic disorder that affects the muscles, particularly those involved in movement. Several splicing QTLs have been identified as risk factors for DMD, including the dystrophin gene, which is responsible for producing the dystrophin protein. Mutations in the dystrophin gene lead to a loss of dystrophin protein, which is essential for maintaining muscle structure and function. 3. Spinal cord injury: Spinal cord injuries (SCIs) are traumatic events that can result in permanent loss of motor and sensory function. Studies have identified several splicing QTLs that are associated with SCIs, including the Hox genes, which are responsible for regulating the development and maintenance of the spinal cord. Mutations in these genes can lead to abnormal splicing patterns, which can contribute to the development of SCIs. 4. Neurodegenerative diseases: Several neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, and Huntington's disease, have been linked to splicing QTLs. For example, mutations in the Huntingtin gene, which is responsible for producing the huntingtin protein, are associated with Huntington's disease. These mutations lead to an abnormal expansion of a CAG repeat in the Huntingtin gene, which disrupts the normal splicing of the gene and leads to the production of a toxic protein that is responsible for the disease. 5. Cancer: Cancer is a complex disease that is characterized |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Spinal muscular atrophy is a rare genetic disorder that affects the nerve cells responsible for controlling voluntary muscle movement. 2. Duchenne muscular dystrophy is a severe genetic disorder that affects the muscles, particularly those involved in movement. 3. Spinal cord injuries are traumatic events that can result in permanent loss of motor and sensory function. 4. Mutations in the Huntingtin gene are associated with Huntington's disease. 5. Cancer is a complex disease. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 94(22) |
| 领域 | Bio-Medical |
| 用户问题 | Which technique is used for detection of EWS/FLI1 fusion transcripts? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | EWS/FLI1 fusion transcripts are detected using the following techniques: 1. RT-PCR (Reverse Transcription Polymerase Chain Reaction): This is a widely used technique for detecting fusion transcripts. RT-PCR involves reverse transcribing the RNA into cDNA, followed by PCR amplification of the fusion transcript. 2. FISH (Fluorescence In Situ Hybridization): This technique involves hybridizing a labeled probe to the chromosomal breakpoint region, allowing visualization of the fusion transcript. 3. Chromosomal Microarray Analysis (CMA): This technique involves hybridizing labeled DNA probes to the entire genome, allowing for the detection of small deletions, duplications, and translocations, including those that result in EWS/FLI1 fusion transcripts. 4. Next-generation sequencing (NGS): This technique involves sequencing the entire genome or exome, allowing for the detection of all types of genetic alterations, including fusions. 5. PCR-based methods: These methods involve using primers specific to the EWS/FLI1 fusion transcript to detect its presence by PCR. It is important to note that the choice of technique will depend on the specific clinical context and the availability of resources. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. RT-PCR is a technique used for detecting EWS/FLI1 fusion transcripts. 2. FISH is a technique used for visualizing the fusion transcript through hybridization. 3. Chromosomal Microarray Analysis allows for the detection of EWS/FLI1 fusion transcripts through the use of labeled DNA probes. 4. Next-generation sequencing (NGS) is a technique that can detect all types of genetic alterations, including fusions like EWS/FLI1. 5. PCR-based methods can be used to detect the presence of EWS/FLI1 fusion transcript using specific primers. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 95(23) |
| 领域 | Bio-Medical |
| 用户问题 | Does the CTCF protein co-localize with cohesin? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Yes, the CTCF protein has been shown to co-localize with cohesin in various cell types. CTCF is a transcription factor that plays a crucial role in regulating gene expression and chromatin organization, while cohesin is a protein complex that helps to hold sister chromatids together during DNA replication and repair. Studies have shown that CTCF and cohesin can interact directly and that they co-localize at specific genomic regions, such as gene promoters and enhancers. For example, a study published in the journal Nature Communications in 2017 found that CTCF and cohesin co-localize at the promoter regions of gene expression in human cells. The co-localization of CTCF and cohesin has important implications for understanding how these proteins regulate chromatin structure and gene expression. It suggests that CTCF may help to recruit cohesin to specific genomic regions, thereby influencing the stability of chromatin and the accessibility of genetic information. Overall, the co-localization of CTCF and cohesin is an important aspect of their functional interplay in the regulation of chromatin structure and gene expression. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. The CTCF protein has been shown to co-localize with cohesin in various cell types. 2. CTCF is a transcription factor that regulates gene expression and chromatin organization. 3. Cohesin is a protein complex that holds sister chromatids together during DNA replication and repair. 4. Studies have shown that CTCF and cohesin co-localize at specific genomic regions, such as gene promoters and enhancers. 5. CTCF may help to recruit cohesin to specific genomic regions, influencing chromatin stability and genetic information accessibility. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 97(24) |
| 领域 | Bio-Medical |
| 用户问题 | Which pathological condition of the heart is known as hypertrophic cardiomyopathy (HCM)? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Hypertrophic cardiomyopathy (HCM) is a heart condition in which the heart muscle becomes thickened, often due to an abnormal gene mutation. This thickening can lead to obstruction of the blood flow out of the heart, arrhythmias, and other complications. HCM can affect anyone, regardless of age or gender, but it is most common in young adults and teenagers. Symptoms of HCM can include chest pain, shortness of breath, fatigue, and dizziness. Diagnosis of HCM typically involves a combination of physical examination, echocardiography, and genetic testing. Treatment options for HCM include medications to control symptoms, implantable cardioverter-defibrillators (ICDs) to prevent arrhythmias, and surgery to relieve obstruction of the blood flow. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Hypertrophic cardiomyopathy (HCM) is a heart condition characterized by the thickening of the heart muscle. 2. HCM can be caused by an abnormal gene mutation. 3. The thickening of the heart muscle in HCM can result in the obstruction of blood flow out of the heart. 4. HCM is most commonly observed in young adults and teenagers. 5. Symptoms of HCM can include chest pain, shortness of breath, fatigue, and dizziness. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 98(25) |
| 领域 | Bio-Medical |
| 用户问题 | What is the genetic basis of Rubinstein-Taybi syndrome? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Rubinstein-Taybi syndrome (RTS) is a rare genetic disorder that affects approximately 1 in 165,000 individuals worldwide. The disorder is caused by mutations in the CREBBP gene, which encodes a transcription factor involved in regulating gene expression. The CREBBP gene is located on chromosome 16 and consists of two copies of the gene, one inherited from each parent. The gene is responsible for producing a protein called CREB-binding protein (CBP), which plays a crucial role in regulating gene expression by recruiting other transcription factors to specific DNA sequences. Mutations in the CREBBP gene can lead to the development of RTS, which is characterized by a range of symptoms including intellectual disability, delayed speech and language development, short stature, and facial dysmorphism. Other features of RTS may include congenital anomalies, such as heart defects or cleft palate, as well as an increased risk of developing certain cancers, including leukemia and lymphoma. The genetic basis of RTS was first identified in the early 1990s, when researchers identified a recurrent mutation in the CREBBP gene in individuals with the disorder. Since then, numerous other mutations have been identified, including point mutations, deletions, and duplications. Understanding the genetic basis of RTS has important implications for diagnosis and treatment. Genetic testing can help identify individuals with the disorder, and genetic counseling can help families understand the risks and implications of the disorder. In addition, research into the genetic basis of RTS may lead to the development of new treatments and therapies for the disorder. In summary, the genetic basis of Rubinstein-Taybi syndrome is a mutation in the CREBBP gene, which encodes a transcription factor involved in regulating gene expression. The disorder is characterized by a range of symptoms, including intellectual disability, delayed speech and language development, and facial dysmorphism. Understanding the genetic basis of RTS has important implications for diagnosis and treatment. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Rubinstein-Taybi syndrome (RTS) affects approximately 1 in 165,000 individuals worldwide. 2. RTS is caused by mutations in the CREBBP gene located on chromosome 16. 3. The CREBBP gene consists of two copies, one inherited from each parent, and produces a protein called CREB-binding protein (CBP). 4. Mutations in the CREBBP gene can lead to the development of RTS, which is characterized by intellectual disability, delayed speech and language development, short stature, and facial dysmorphism. 5. The genetic basis of RTS was first identified in the early 1990s, and since then, numerous other mutations in the CREBBP gene have been identified. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 99(26) |
| 领域 | Bio-Medical |
| 用户问题 | What is the function of the viral KP4 protein? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | The KP4 protein is a viral protein found in the KP4 virus, which is a member of the paramyxovirus family. The KP4 protein is one of the most well-studied viral proteins, and it has been shown to play a critical role in the infection and replication of the virus. The KP4 protein is a transmembrane protein that spans the viral membrane and is involved in the fusion of the viral and host cell membranes. This fusion is necessary for the virus to enter the host cell and replicate. The KP4 protein also plays a role in the assembly and release of new viral particles. In addition to its role in viral replication, the KP4 protein has been shown to interact with a number of host cell proteins, including the host cell membrane protein, CD46. This interaction can help the virus to evade the host immune system and establish a persistent infection. Overall, the KP4 protein is a critical component of the KP4 virus and plays a central role in its replication and infection of host cells. Understanding the function of this protein is important for the development of effective antiviral therapies and vaccines. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. The KP4 protein is a viral protein found in the KP4 virus, a member of the paramyxovirus family. 2. The KP4 protein is a transmembrane protein. 3. The KP4 protein is involved in the fusion of the viral and host cell membranes. 4. The KP4 protein plays a role in the assembly and release of new viral particles. 5. The KP4 protein interacts with host cell proteins, including CD46. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 100(27) |
| 领域 | Bio-Medical |
| 用户问题 | What is the function of the AIRE gene at the embryonic stage? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | The AIRE (Autoimmune Regulator) gene plays a crucial role in the development and function of the immune system, particularly during embryonic development. The AIRE gene is expressed in the thymus, a gland located in the chest that is responsible for the development and maturation of T cells, a type of immune cell. During embryonic development, the AIRE gene is expressed in the thymus and regulates the expression of genes involved in T cell development and maturation. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. The AIRE gene is involved in the development and function of the immune system. 2. The AIRE gene is expressed in the thymus. 3. The thymus is responsible for the development and maturation of T cells. 4. The AIRE gene regulates the expression of genes involved in T cell development and maturation. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 102(28) |
| 领域 | Bio-Medical |
| 用户问题 | Which drugs are utilized to treat amiodarone-induced thyroitoxicosis? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Amiodarone is an antiarrhythmic drug that can cause thyrotoxicosis, a condition characterized by an overactive thyroid gland. Thyrotoxicosis can occur due to the drug's inhibition of the thyroid gland's iodide uptake, leading to increased thyroid hormone production. Treatment for amiodarone-induced thyrotoxicosis typically involves discontinuing the drug and managing symptoms with medications that can help reduce thyroid hormone levels. Here are some of the drugs that may be utilized to treat amiodarone-induced thyrotoxicosis: 1. Beta-blockers: Beta-blockers are medications that can help reduce the symptoms of thyrotoxicosis, such as rapid heart rate, tremors, and anxiety. Propranolol and nadolol are commonly used beta-blockers for this purpose. 2. Thionamides: Thionamides, such as methimazole and propylthiouracil, are medications that can help reduce thyroid hormone production. These drugs work by inhibiting the production of thyroid hormones and can be used to treat both mild and severe cases of amiodarone-induced thyrotoxicosis. 3. Corticosteroids: Corticosteroids, such as prednisone, can be used to reduce inflammation and swelling in the thyroid gland. These drugs can help reduce symptoms such as swelling in the neck, difficulty swallowing, and difficulty breathing. 4. Reverse T3: Reverse T3 is a medication that can help reduce thyroid hormone production. It works by inhibiting the conversion of T4 to T3, which can help reduce the levels of thyroid hormones in the body. 5. Thyroid hormone antibodies: Thyroid hormone antibodies, such as teprotide, can help reduce thyroid hormone production. These drugs work by blocking the production of thyroid hormones and can be used to treat both mild and severe cases of amiodarone-induced thyrotoxicosis. It |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Amiodarone is an antiarrhythmic drug that can cause thyrotoxicosis. 2. Thyrotoxicosis is characterized by an overactive thyroid gland. 3. Amiodarone inhibits the thyroid gland's iodide uptake, leading to increased thyroid hormone production. 4. Treatment for amiodarone-induced thyrotoxicosis involves discontinuing the drug. 5. Beta-blockers, thionamides, corticosteroids, reverse T3, and thyroid hormone antibodies are drugs utilized to treat amiodarone-induced thyrotoxicosis. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 103(29) |
| 领域 | Bio-Medical |
| 用户问题 | How is spastic diplegia diagnosed? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Spastic diplegia is typically diagnosed through a combination of medical history, physical examination, and diagnostic tests. The following are some of the methods used to diagnose spastic diplegia: 1. Medical History: The doctor will ask questions about the child's symptoms, such as when they started, how often they occur, and how they affect the child's daily activities. 2. Physical Examination: The doctor will perform a thorough physical examination to check for signs of spasticity, such as stiffness, tightness, or abnormal reflexes. 3. Neurological Examination: The doctor may perform a neurological examination to check for signs of damage to the brain or spinal cord, such as weakness, numbness, or difficulty with coordination and balance. 4. Imaging Studies: Imaging studies, such as X-rays, CT scans, or MRI scans, may be ordered to rule out other conditions that may be causing the symptoms. 5. Electromyography (EMG): EMG is a test that measures the electrical activity of muscles. It can help the doctor determine if there is any damage to the muscles or nerves. 6. Nerve Conduction Studies (NCS): NCS is a test that measures the speed and strength of electrical signals in the nerves. It can help the doctor determine if there is any damage to the nerves. 7. Genetic Testing: Genetic testing may be ordered to rule out other genetic conditions that may be causing the symptoms. 8. Developmental Assessment: The doctor may also perform a developmental assessment to check the child's cognitive and motor skills. It is important to note that the diagnosis of spastic diplegia may take time, and the doctor may use a combination of these methods to make the diagnosis. Early diagnosis and treatment can help improve the child's quality of life and reduce the risk of complications. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Spastic diplegia is typically diagnosed through a combination of medical history, physical examination, and diagnostic tests. 2. The doctor will ask questions about the child's symptoms, such as when they started, how often they occur, and how they affect the child's daily activities. 3. The doctor will perform a thorough physical examination to check for signs of spasticity, such as stiffness, tightness, or abnormal reflexes. 4. Imaging studies, such as X-rays, CT scans, or MRI scans, may be ordered to rule out other conditions that may be causing the symptoms. 5. Electromyography (EMG) is a test that measures the electrical activity of muscles and can help determine if there is any damage to the muscles or nerves. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 104(30) |
| 领域 | Bio-Medical |
| 用户问题 | Which is the genetic defect causing Neurofibromatosis type 1? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Neurofibromatosis type 1 (NF1) is caused by a mutation in the NF1 gene, which is located on chromosome 17. The NF1 gene encodes a protein called neurofibromin, which plays a critical role in regulating the growth and development of nerve cells. When the NF1 gene is mutated, the resulting protein is either missing or defective, leading to the development of benign tumors, called neurofibromas, on nerve tissue. These tumors can occur anywhere in the body, but are most commonly found on the skin, in the brain and spinal cord, and in the peripheral nerves. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Neurofibromatosis type 1 is caused by a mutation in the NF1 gene. 2. The NF1 gene is located on chromosome 17. 3. The NF1 gene encodes a protein called neurofibromin. 4. Mutations in the NF1 gene result in a missing or defective neurofibromin protein. 5. Neurofibromas, benign tumors, develop on nerve tissue due to the mutated NF1 gene. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 106(31) |
| 领域 | Bio-Medical |
| 用户问题 | Which package is available for analysing genomic interactions in R/Bioconductor? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | There are several packages available in R/Bioconductor for analysing genomic interactions, including: 1. InteractR: This package provides a comprehensive set of tools for identifying and visualizing genomic interactions. It can handle both genetic and epigenetic interactions and can be used to analyze both gene expression and genotype data. 2. GSEA (Gene Set Enrichment Analysis): This package is specifically designed for identifying gene set enrichment in genomic interactions. It can be used to identify pathways or sets of genes that are overrepresented in a set of interacting genes. 3. GenInt: This package provides a variety of tools for analyzing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function from genomic interactions, and identifying regulatory elements. 4. InteractomeR: This package provides a comprehensive set of tools for identifying and visualizing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function from genomic interactions, and identifying regulatory elements. 5. GenomicRanges: This package provides a variety of tools for analyzing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function from genomic interactions, and identifying regulatory elements. 6. R/BioConductor/Interactome: This package provides a comprehensive set of tools for identifying and visualizing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function from genomic interactions, and identifying regulatory elements. 7. GenomicInteractions: This package provides a variety of tools for analyzing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function from genomic interactions, and identifying regulatory elements. 8. InteractomeAnalysis: This package provides a variety of tools for analyzing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function from genomic interactions, and identifying regulatory elements. Please note that this is not an exhaustive list and there may be other packages available in R/Bioconductor for analyzing genomic interactions. It's always a good idea to consult the package documentation and examples to determine which package is best suited for |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. InteractR is a package available in R/Bioconductor for analyzing genomic interactions. 2. GSEA (Gene Set Enrichment Analysis) is a package specifically designed for identifying gene set enrichment in genomic interactions. 3. GenInt is a package that provides tools for analyzing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function, and identifying regulatory elements. 4. InteractomeR is a package that provides tools for identifying and visualizing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function, and identifying regulatory elements. 5. GenomicRanges is a package that provides tools for analyzing genomic interactions, including methods for identifying co-regulated gene pairs, predicting gene function, and identifying regulatory elements. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 108(32) |
| 领域 | Bio-Medical |
| 用户问题 | Are stress granules involved in the pathogenesis of Amyotrophic Lateral Sclerosis? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Amyotrophic Lateral Sclerosis (ALS) is a progressive neurodegenerative disorder characterized by the loss of motor neurons, leading to muscle weakness, atrophy, and paralysis. While the exact etiology of ALS remains unclear, recent studies have implicated stress granules in its pathogenesis. Stress granules are dynamic protein-RNA complexes that form in response to cellular stress and play a crucial role in regulating gene expression. One of the key findings supporting the involvement of stress granules in ALS is the observation of increased levels of stress granules in the brains of ALS patients compared to healthy controls. This suggests that stress granules may be overactive in ALS, leading to the misregulation of gene expression and the death of motor neurons. Another line of evidence comes from studies of the role of stress granules in motor neuron survival. In vitro studies have shown that stress granules can protect motor neurons from death by regulating the expression of pro-survival genes, such as Bcl-2. Conversely, disruption of stress granules can lead to the death of motor neurons, suggesting that stress granules play a critical role in maintaining motor neuron survival. Furthermore, recent studies have identified genetic mutations in genes encoding stress granule components that are associated with ALS. For example, mutations in the TARDBP gene, which encodes a protein involved in the formation of stress granules, have been found to cause ALS. These findings suggest that genetic mutations in stress granule components may contribute to the pathogenesis of ALS. In conclusion, while the exact mechanisms underlying ALS remain unclear, there is growing evidence to suggest that stress granules play a role in its pathogenesis. Further research is needed to fully understand the relationship between stress granules and ALS, and to identify potential therapeutic targets for this devastating disease. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Amyotrophic Lateral Sclerosis (ALS) is a progressive neurodegenerative disorder characterized by the loss of motor neurons, leading to muscle weakness, atrophy, and paralysis. 2. Increased levels of stress granules have been observed in the brains of ALS patients compared to healthy controls. 3. Stress granules play a role in regulating gene expression. 4. In vitro studies have shown that stress granules can protect motor neurons from death by regulating the expression of pro-survival genes. 5. Mutations in the TARDBP gene, which encodes a protein involved in the formation of stress granules, have been found to cause ALS. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 109(33) |
| 领域 | Bio-Medical |
| 用户问题 | Does TGF-beta play a role in cardiac regeneration after myocardial infarction? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Yes, TGF-beta has been shown to play a role in cardiac regeneration after myocardial infarction. TGF-beta is a potent cytokine that regulates a variety of cellular processes, including cell growth, differentiation, and survival. In the context of myocardial infarction, TGF-beta has been shown to promote cardiac regeneration by: 1. Enhancing angiogenesis: TGF-beta stimulates the formation of new blood vessels, which is essential for cardiac regeneration. 2. Promoting cell survival and proliferation: TGF-beta can protect cardiac cells from apoptosis and stimulate cell proliferation, which can help to replace damaged cardiac tissue. 3. Inducing cardiac differentiation: TGF-beta can induce the differentiation of cardiac progenitor cells into mature cardiac cells, such as cardiomyocytes and endothelial cells. 4. Modulating the immune response: TGF-beta can suppress the immune response and reduce inflammation, which can promote cardiac regeneration by reducing the damage to the cardiac tissue. 5. Enhancing the function of existing cardiac tissue: TGF-beta can improve the function of existing cardiac tissue by increasing the expression of contractile proteins and improving the coordination of cardiac contractions. Overall, TGF-beta plays a critical role in cardiac regeneration after myocardial infarction by promoting cell survival, proliferation, differentiation, and angiogenesis, and by modulating the immune response. However, the therapeutic potential of TGF-beta in cardiac regeneration is still being explored and more research is needed to fully understand its mechanisms of action and to identify the most effective strategies for its use in clinical settings. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. TGF-beta is a cytokine that regulates cell growth, differentiation, and survival. 2. TGF-beta has been shown to enhance angiogenesis, the formation of new blood vessels. 3. TGF-beta can protect cardiac cells from apoptosis and stimulate cell proliferation. 4. TGF-beta can induce the differentiation of cardiac progenitor cells into mature cardiac cells. 5. TGF-beta can suppress the immune response and reduce inflammation. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 110(34) |
| 领域 | Bio-Medical |
| 用户问题 | Is there a genetic component for happiness? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Research suggests that there may be a genetic component to happiness, with certain genetic variations influencing a person's ability to experience happiness and well-being. Here are some key findings: 1. Genetic influence on serotonin levels: Serotonin is a neurotransmitter that plays a role in mood regulation, and variations in the genes that code for serotonin have been linked to differences in happiness levels. For example, people with lower levels of the serotonin transporter gene (5-HTT) tend to be more sensitive to stress and have lower baseline levels of happiness. 2. Monoamine oxidase A (MAOA) gene: The MAOA gene helps break down certain neurotransmitters, including serotonin, dopamine, and norepinephrine. Variations in the MAOA gene have been associated with differences in emotional regulation and happiness. 3. COMT gene: The COMT gene is involved in the breakdown of dopamine, a neurotransmitter that plays a role in reward processing and motivation. Variations in the COMT gene have been linked to differences in happiness and well-being. 4. HPA axis regulation: The hypothalamic-pituitary-adrenal (HPA) axis is a complex system involved in the body's response to stress. Variations in genes that regulate the HPA axis, such as the corticotropin-releasing hormone (CRH) gene, can influence a person's ability to cope with stress and experience happiness. 5. Genetic influence on personality traits: Certain personality traits, such as extraversion and neuroticism, have been linked to differences in happiness levels. For example, people who are more extraverted tend to be happier and more sociable, while those who are more neurotic tend to be less happy and more prone to anxiety and depression. 6. Epigenetic influences: Epigenetic changes, which affect how genes are expressed without altering the underlying DNA sequence, can also influence happiness. For example, studies have found that people who experience stress have epigenetic changes in genes involved in the HPA axis and stress response. While genetics can |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Variations in the genes that code for serotonin have been linked to differences in happiness levels. 2. The MAOA gene is involved in breaking down neurotransmitters such as serotonin, dopamine, and norepinephrine, and variations in this gene have been associated with differences in emotional regulation and happiness. 3. The COMT gene is involved in the breakdown of dopamine, and variations in this gene have been linked to differences in happiness and well-being. 4. Variations in genes that regulate the HPA axis, such as the CRH gene, can influence a person's ability to cope with stress and experience happiness. 5. Certain personality traits, such as extraversion and neuroticism, have been linked to differences in happiness levels. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 111(35) |
| 领域 | Bio-Medical |
| 用户问题 | What enzyme is inhibied by Opicapone? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Opicapone is an oral catechol-O-methyltransferase (COMT) inhibitor. COMT is an enzyme that breaks down certain medications, including levodopa, in the brain. By inhibiting COMT, opicapone increases the amount of levodopa available for dopamine production in the brain, which can help to improve symptoms of Parkinson's disease. So, the answer is COMT. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Opicapone is an oral catechol-O-methyltransferase (COMT) inhibitor. 2. COMT is an enzyme that breaks down certain medications, including levodopa, in the brain. 3. Opicapone inhibits COMT. 4. Opicapone increases the amount of levodopa available for dopamine production in the brain. 5. Opicapone can help improve symptoms of Parkinson's disease. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 112(36) |
| 领域 | Bio-Medical |
| 用户问题 | What kind of affinity purification would you use in order to isolate soluble lysosomal proteins? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Soluble lysosomal proteins can be isolated using affinity purification techniques that take advantage of the unique properties of these proteins. Here are some common methods used to isolate soluble lysosomal proteins: 1. Lectin affinity chromatography: Lysosomal proteins are often highly glycosylated, and lectins can bind to these glycans. Lectin affinity chromatography can be used to isolate soluble lysosomal proteins by binding to the glycans on the surface of the proteins. 2. IgG affinity chromatography: IgG antibodies can be used to isolate soluble lysosomal proteins by binding to the Fc region of the proteins. IgG affinity chromatography can be used to isolate soluble lysosomal proteins from cell lysates or tissue homogenates. 3. Nickel ion affinity chromatography: Nickel ions can bind to the cysteine residues in lysosomal proteins, allowing for their isolation using nickel ion affinity chromatography. This method is particularly useful for isolating proteins that are rich in cysteine residues, such as cathepsins. 4. Immunomagnetic separation: Immunomagnetic separation can be used to isolate soluble lysosomal proteins by using antibodies that are specific to these proteins. The antibodies are attached to magnetic beads, which are then added to the cell lysate or tissue homogenate. The lysosomal proteins bind to the beads, allowing for their isolation. 5. Gel filtration: Gel filtration can be used to isolate soluble lysosomal proteins by separating them based on their size. The proteins are separated using a gel matrix, and the smaller proteins are able to pass through the gel, while the larger proteins are retained. 6. Hydrophobic interaction chromatography (HIC): HIC can be used to isolate soluble lysosomal proteins by taking advantage of their hydrophobic properties. The proteins are separated based on their hydrophobicity, |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Lectin affinity chromatography can be used to isolate soluble lysosomal proteins by binding to the glycans on their surface. 2. IgG affinity chromatography can be used to isolate soluble lysosomal proteins by binding to the Fc region of the proteins. 3. Nickel ion affinity chromatography can be used to isolate lysosomal proteins by binding to the cysteine residues. 4. Immunomagnetic separation can be used to isolate soluble lysosomal proteins by using antibodies attached to magnetic beads. 5. Gel filtration can be used to isolate soluble lysosomal proteins by separating them based on their size. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 114(37) |
| 领域 | Bio-Medical |
| 用户问题 | Do archaeal genomes contain one or multiple origins of replication? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Most eukaryotic genomes, including those of animals, plants, and fungi, are thought to have a single origin of replication (oriC). However, recent studies have shown that some eukaryotic organisms, such as the protozoan parasite Toxoplasma gondii, have multiple origins of replication. Archaeal genomes, on the other hand, are thought to have multiple origins of replication. One study found that the archaeon Halorubrum sodomense has three origins of replication, while another study found that the archaeon Thermococcus kodakarensis has two origins of replication. These findings suggest that the presence of multiple origins of replication may be more common in archaeal genomes than in eukaryotic genomes. It is not clear why archaeal genomes might have multiple origins of replication, but it is thought that this may be related to the fact that archaea are highly heterogeneous and have a wide range of growth temperatures and environments. This heterogeneity may make it advantageous for archaeal genomes to have multiple origins of replication, allowing them to maintain genome stability and function in a variety of environments. In contrast, eukaryotic genomes are generally more stable and less heterogeneous than archaeal genomes, and it is thought that the single origin of replication in these genomes is sufficient to maintain genome stability. However, it is possible that some eukaryotic organisms may also have multiple origins of replication, and further research is needed to determine the prevalence of this feature in eukaryotic genomes. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Some eukaryotic organisms, such as the protozoan parasite Toxoplasma gondii, have been found to have multiple origins of replication. 2. Archaeal genomes are thought to have multiple origins of replication. 3. The archaeon Halorubrum sodomense has been found to have three origins of replication. 4. The archaeon Thermococcus kodakarensis has been found to have two origins of replication. 5. The presence of multiple origins of replication may be more common in archaeal genomes than in eukaryotic genomes. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 115(38) |
| 领域 | Bio-Medical |
| 用户问题 | Which pathological conditions are caused by mutations in the CYLD gene? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | The CYLD gene encodes a protein that is involved in regulating the activity of transcription factors and other signaling pathways. Mutations in the CYLD gene have been associated with a variety of pathological conditions, including: 1. Neurodegenerative diseases: Mutations in the CYLD gene have been identified in individuals with neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis (ALS). These mutations are thought to disrupt the normal function of the CYLD protein, leading to the death of neurons and the progression of these diseases. 2. Cancer: Mutations in the CYLD gene have been found in various types of cancer, including breast cancer, lung cancer, and ovarian cancer. These mutations can lead to the uncontrolled growth and proliferation of cancer cells, contributing to the development and progression of cancer. 3. Autoimmune diseases: Some mutations in the CYLD gene have been associated with autoimmune diseases such as lupus and rheumatoid arthritis. These mutations may lead to the production of autoantibodies and the activation of immune cells, contributing to the development of these diseases. 4. Developmental disorders: Mutations in the CYLD gene have been identified in individuals with developmental disorders such as intellectual disability and autism spectrum disorder. These mutations may affect the normal development and function of the brain, leading to these disorders. 5. Cardiovascular diseases: Mutations in the CYLD gene have been found to be associated with an increased risk of cardiovascular diseases such as heart attack and stroke. These mutations may lead to the dysregulation of signaling pathways involved in the development and progression of these diseases. Overall, mutations in the CYLD gene have been implicated in a wide range of pathological conditions, highlighting the importance of this gene in maintaining proper cellular function and preventing disease. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Mutations in the CYLD gene have been associated with neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis (ALS). 2. Mutations in the CYLD gene have been found in various types of cancer, including breast cancer, lung cancer, and ovarian cancer. 3. Some mutations in the CYLD gene have been associated with autoimmune diseases such as lupus and rheumatoid arthritis. 4. Mutations in the CYLD gene have been identified in individuals with developmental disorders such as intellectual disability and autism spectrum disorder. 5. Mutations in the CYLD gene have been found to be associated with an increased risk of cardiovascular diseases such as heart attack and stroke. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 116(39) |
| 领域 | Bio-Medical |
| 用户问题 | Which is the genetic basis of Spinal Muscular Atrophy (SMA)? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Spinal muscular atrophy (SMA) is caused by mutations in the Survival Motor Neuron 1 (SMN1) gene. This gene provides instructions for making a protein called survival motor neuron (SMN), which is critical for the survival of motor neurons in the spinal cord. Without enough SMN protein, motor neurons degenerate and die, leading to muscle weakness and wasting. SMA is the most common genetic cause of infant death, and there is currently no cure. However, researchers are working to develop treatments that can help manage the symptoms of SMA and improve the quality of life for people affected by the disorder. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Spinal Muscular Atrophy (SMA) is caused by mutations in the Survival Motor Neuron 1 (SMN1) gene. 2. The SMN1 gene provides instructions for making a protein called survival motor neuron (SMN). 3. SMN protein is critical for the survival of motor neurons in the spinal cord. 4. The degeneration and death of motor neurons in the spinal cord lead to muscle weakness and wasting in SMA. 5. SMA is the most common genetic cause of infant death. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 117(40) |
| 领域 | Bio-Medical |
| 用户问题 | Which are the common symptoms of Cushing's syndrome? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Cushing's syndrome is a hormonal disorder that occurs when the body has too much cortisol, a steroid hormone produced by the adrenal glands. The symptoms of Cushing's syndrome can vary depending on the individual, but common symptoms include: 1. Weight gain: Cushing's syndrome can cause significant weight gain, particularly in the abdomen, face, and neck. 2. Fatigue: Feeling tired or weak is a common symptom of Cushing's syndrome, which can make it difficult to perform daily activities. 3. Muscle weakness: Cushing's syndrome can cause muscle weakness and loss of muscle mass, particularly in the upper arms and legs. 4. High blood pressure: Cushing's syndrome can increase blood pressure, which can lead to damage to blood vessels and increase the risk of heart disease. 5. Abnormal menstrual cycles: Women with Cushing's syndrome may experience irregular or absent menstrual periods, or may have heavier or lighter periods than usual. 6. Hirsutism: Cushing's syndrome can cause excessive hair growth on the face, chest, and back, particularly in women. 7. Acne: Cushing's syndrome can cause acne, particularly on the face and chest. 8. Osteoporosis: Cushing's syndrome can cause bone loss and increase the risk of osteoporosis, particularly in older adults. 9. Depression: Cushing's syndrome can cause depression, anxiety, and mood changes, which can affect an individual's ability to function in daily life. 10. Memory problems: Cushing's syndrome can cause memory problems and difficulty concentrating, which can affect an individual's ability to perform daily tasks. 11. Sleep disturbances: Cushing's syndrome can cause sleep disturbances, including insomnia and sleep apnea. 12. Purple striae: Cushing's syndrome can cause stretch marks, particularly on the abdomen, thighs, and buttocks. 13. Balding: Cushing's syndrome can cause hair loss, particularly on the scalp. 14. Easy bruising: C |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Cushing's syndrome is a hormonal disorder caused by an excess of cortisol. 2. Weight gain, particularly in the abdomen, face, and neck, is a common symptom of Cushing's syndrome. 3. Fatigue is a common symptom of Cushing's syndrome, making daily activities difficult. 4. Muscle weakness and loss of muscle mass, especially in the upper arms and legs, can occur with Cushing's syndrome. 5. High blood pressure is a symptom of Cushing's syndrome and can increase the risk of heart disease. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 118(41) |
| 领域 | Bio-Medical |
| 用户问题 | Which is the third subunit of the TSC1-TSC2 complex upstream of mTORC1? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | The TSC1-TSC2 complex is a key regulator of cell growth and metabolism, and it is composed of three subunits: TSC1, TSC2, and a third subunit upstream of mTORC1. The third subunit of the TSC1-TSC2 complex upstream of mTORC1 is called TFEB (TSC-associated factor EB). TFEB is a transcriptional coactivator that regulates the expression of genes involved in cell growth, metabolism, and autophagy. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. The TSC1-TSC2 complex is composed of three subunits: TSC1, TSC2, and TFEB. 2. TFEB is the third subunit of the TSC1-TSC2 complex upstream of mTORC1. 3. TFEB is a transcriptional coactivator. 4. TFEB regulates the expression of genes involved in cell growth, metabolism, and autophagy. 5. The TSC1-TSC2 complex is a key regulator of cell growth and metabolism. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 119(42) |
| 领域 | Bio-Medical |
| 用户问题 | Which kinase is inhibited by the small molecule KN-93? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | KN-93 is a small molecule inhibitor of the protein kinase C (PKC). It selectively binds to and inhibits the activity of PKC-β isoforms, which are implicated in various cellular processes, including cell proliferation, differentiation, and survival. KN-93 has been shown to have antineoplastic activity in various cancer models, and it is currently being investigated as a potential therapeutic agent for the treatment of cancer. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. KN-93 is a small molecule inhibitor of protein kinase C (PKC). 2. KN-93 selectively binds to and inhibits the activity of PKC-β isoforms. 3. PKC-β isoforms are implicated in various cellular processes, including cell proliferation, differentiation, and survival. 4. KN-93 has been shown to have antineoplastic activity in various cancer models. 5. KN-93 is currently being investigated as a potential therapeutic agent for the treatment of cancer. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 120(43) |
| 领域 | Bio-Medical |
| 用户问题 | What is the effect of Chk2 splice variants on wild-type Chk2 kinase activity? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Checkpoint kinase 2 (Chk2) is a key regulator of the DNA damage response and is frequently mutated in various human cancers. Chk2 contains multiple splice variants, each with distinct functions. However, the effect of Chk2 splice variants on wild-type Chk2 kinase activity remains unclear. In this study, we investigated the effect of Chk2 splice variants on wild-type Chk2 kinase activity using a combination of biochemical and cellular assays. Our results showed that the expression of Chk2 splice variants can modulate the activity of wild-type Chk2, leading to changes in the DNA damage response. First, we found that the expression of Chk2 splice variants can affect the phosphorylation status of wild-type Chk2. Specifically, the expression of Chk2 splice variants that contain an additional exon in the carboxy-terminal region (Chk2-E56A) can inhibit the phosphorylation of wild-type Chk2 at the active site, leading to a decrease in its kinase activity. Conversely, the expression of Chk2 splice variants that lack this additional exon (Chk2-E56K) can enhance the phosphorylation of wild-type Chk2 and increase its kinase activity. Second, we found that the expression of Chk2 splice variants can also affect the subcellular localization of wild-type Chk2. Specifically, the expression of Chk2 splice variants that contain the additional exon in the carboxy-terminal region can cause wild-type Chk2 to be sequestered in the cytoplasm, leading to a decrease in its ability to interact with its substrates and respond to DNA damage. Conversely, the expression of Chk2 splice variants that lack this additional exon can cause wild-type Chk2 to be localized to the nucleus, leading to an increase in its ability to respond to DNA damage. Overall, our results suggest that the expression of Chk2 splice variants can have a significant impact on the activity of wild-type Chk2, leading to changes in the DNA damage response. These findings have important implications for our understanding of the mechanisms of DNA damage response and the development of cancer. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Chk2 is a key regulator of the DNA damage response and is frequently mutated in various human cancers. 2. Chk2 contains multiple splice variants, each with distinct functions. 3. The expression of Chk2 splice variants can affect the phosphorylation status of wild-type Chk2. 4. The expression of Chk2 splice variants can also affect the subcellular localization of wild-type Chk2. 5. The expression of Chk2 splice variants can modulate the activity of wild-type Chk2, leading to changes in the DNA damage response. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 121(44) |
| 领域 | Bio-Medical |
| 用户问题 | List genes that have been found mutated in CMT1A (Charcot-Marie-Tooth disease type 1 A). |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Charcot-Marie-Tooth disease type 1A (CMT1A) is the most common form of hereditary motor neuropathy, accounting for approximately 70% of all CMT cases. CMT1A is caused by a duplication of the PMP22 gene, which codes for the protein peripherin. This duplication leads to an increase in the amount of peripherin protein produced, which is believed to disrupt the normal functioning of the nerve sheath and lead to the symptoms of CMT1A. The following genes have been found mutated in CMT1A: 1. PMP22: The PMP22 gene is the most commonly mutated gene in CMT1A, accounting for approximately 80% of all CMT1A cases. The duplication of this gene leads to an increase in the amount of peripherin protein produced, which is believed to disrupt the normal functioning of the nerve sheath and lead to the symptoms of CMT1A. 2. MPZ: The MPZ gene has also been found to be mutated in some cases of CMT1A. Mutations in this gene can lead to a decrease in the amount of the protein protein zinc finger 8 (ZF8) produced, which is important for the maintenance of the nerve sheath. 3. GDAP1: The GDAP1 gene has been found to be mutated in some cases of CMT1A. Mutations in this gene can lead to a decrease in the amount of the protein GDAP1 produced, which is important for the maintenance of the nerve sheath. 4. PLEC: The PLEC gene has been found to be mutated in some cases of CMT1A. Mutations in this gene can lead to a decrease in the amount of the protein plectin produced, which is important for the maintenance of the nerve sheath. 5. NEFL: The NEFL gene has been found to be mutated in some cases of CMT1A. Mutations in this gene can lead to a decrease in the amount of the protein neurofilament light chain produced, which is important for the maintenance of the nerve sheath. 6. HSPB1: The HSPB1 gene has been found to be mutated in some |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. CMT1A is the most common form of hereditary motor neuropathy, accounting for approximately 70% of all CMT cases. 2. CMT1A is caused by a duplication of the PMP22 gene. 3. The duplication of the PMP22 gene leads to an increase in the amount of peripherin protein produced. 4. MPZ, GDAP1, PLEC, NEFL, and HSPB1 genes have all been found to be mutated in some cases of CMT1A. 5. Mutations in the MPZ gene can lead to a decrease in the amount of protein zinc finger 8 (ZF8) produced. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 125(45) |
| 领域 | Bio-Medical |
| 用户问题 | What is Tarlov Cyst? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Tarlov cyst is a rare type of cyst that forms in the sacrum, the triangular bone at the base of the spine. It is also known as a Tarlov canal cyst or a sacral intervertebral cyst. These cysts are typically small, fluid-filled sacs that develop between the vertebrae in the lower back. They can occur on one or both sides of the spine and can range in size from a few millimeters to several centimeters in diameter. Tarlov cysts are thought to be caused by a failure of the membrane that separates the spinal canal from the surrounding tissue to properly close during fetal development. This can lead to the formation of a communication between the spinal canal and the surrounding tissue, which can fill with fluid and become a cyst. Symptoms of Tarlov cysts can vary depending on their size and location, but may include: \* Back pain, which can be worse with sitting or standing \* Leg pain or numbness/tingling in the legs \* Weakness or loss of sensation in the legs \* Difficulty controlling bowel or bladder function \* Numbness or tingling in the buttocks or genital area Tarlov cysts are usually diagnosed through a combination of imaging tests, such as X-rays, CT scans, or MRI scans, and a thorough medical history. Treatment options for Tarlov cysts depend on the size and location of the cyst, as well as the severity of the symptoms, and may include: \* Observation: Small, asymptomatic cysts may not require treatment and can be monitored with regular imaging tests. \* Surgery: Larger cysts or those that are causing symptoms may be treated through surgery to drain the cyst or remove it entirely. \* Injections: In some cases, corticosteroid injections may be recommended to reduce inflammation and relieve symptoms. It is important to note that Tarlov cysts are relatively rare and may be difficult to diagnose, as the symptoms can be similar to those of other conditions. If you are experiencing back or leg pain, numbness, or weakness, it is important to consult with a healthcare |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Tarlov cyst is a cyst that forms in the sacrum, which is the triangular bone at the base of the spine. 2. Tarlov cysts are typically small, fluid-filled sacs that develop between the vertebrae in the lower back. 3. Tarlov cysts are caused by a failure of the membrane that separates the spinal canal from the surrounding tissue to properly close during fetal development. 4. Symptoms of Tarlov cysts may include back pain, leg pain or numbness/tingling, weakness or loss of sensation in the legs, difficulty controlling bowel or bladder function, and numbness or tingling in the buttocks or genital area. 5. Treatment options for Tarlov cysts can include observation, surgery, and corticosteroid injections. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 126(46) |
| 领域 | Bio-Medical |
| 用户问题 | What are 'vildagliptin', 'sitagliptin', 'saxagliptin', 'alogliptin', 'linagliptin', and 'dutogliptin'? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | Vildagliptin, sitagliptin, saxagliptin, alogliptin, linagliptin, and dutogliptin are all medications that belong to a class of drugs called dipeptidyl peptidase-4 (DPP-4) inhibitors. These medications are used to treat type 2 diabetes by increasing the amount of insulin produced by the body and decreasing the amount of sugar produced by the liver. They work by blocking the action of the enzyme DPP-4, which breaks down the hormone incretin, which helps to regulate blood sugar levels. By blocking the action of DPP-4, these medications increase the amount of incretin in the body, which in turn helps to improve insulin production and reduce blood sugar levels. Vildagliptin is marketed under the brand name Galvus, sitagliptin is marketed under the brand name Januvia, saxagliptin is marketed under the brand name Onglyza, alogliptin is marketed under the brand name Tradjenta, linagliptin is marketed under the brand name Trajenta, and dutogliptin is marketed under the brand name Rybelsus. These medications are typically taken orally once daily and may be used alone or in combination with other diabetes medications. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Vildagliptin, sitagliptin, saxagliptin, alogliptin, linagliptin, and dutogliptin are medications that belong to the class of drugs called dipeptidyl peptidase-4 (DPP-4) inhibitors. 2. These medications are used to treat type 2 diabetes. 3. They work by increasing the amount of insulin produced by the body and decreasing the amount of sugar produced by the liver. 4. These medications block the action of the enzyme DPP-4, which breaks down the hormone incretin. 5. By blocking the action of DPP-4, these medications increase the amount of incretin in the body, which helps to regulate blood sugar levels. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 127(47) |
| 领域 | Bio-Medical |
| 用户问题 | Which is the most important prognosis sub-classification in Chronic Lymphocytic Leukemia? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | The most important prognostic sub-classification in Chronic Lymphocytic Leukemia (CLL) is the Richter's transformation. This is a sudden and rapid progression of CLL to a more aggressive and harder to treat form of the disease, known as Richter's syndrome. This transformation is associated with a poorer prognosis and a higher risk of death compared to the original CLL diagnosis. Other prognostic sub-classifications that may be important in CLL include: \* The presence of certain genetic mutations, such as the TP53 mutation \* The level of certain proteins in the blood, such as the level of the protein CD38 \* The presence of certain subtypes of CLL, such as the deletion 17p mutation \* The response to treatment, including the response to chemotherapy and the presence of resistance to certain treatments \* The presence of certain comorbidities, such as heart disease or lung disease |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Richter's transformation is the most important prognostic sub-classification in Chronic Lymphocytic Leukemia (CLL). 2. Richter's transformation is a sudden and rapid progression of CLL to a more aggressive and harder to treat form of the disease called Richter's syndrome. 3. Richter's transformation is associated with a poorer prognosis and a higher risk of death compared to the original CLL diagnosis. 4. The presence of certain genetic mutations, such as the TP53 mutation, may also be an important prognostic sub-classification in CLL. 5. The level of certain proteins in the blood, such as the protein CD38, may also be an important prognostic sub-classification in CLL. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 128(48) |
| 领域 | Bio-Medical |
| 用户问题 | Is MammaPrint cleared by the United States Food and Drug Administration? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | MammaPrint is a gene expression profiling test that is used to help determine the likelihood of breast cancer recurrence in postmenopausal women. The test measures the expression levels of 70 genes associated with breast cancer prognosis and can provide information on the likelihood of distant recurrence, which is recurrence of cancer in a distant part of the body, such as the bones or liver. The United States Food and Drug Administration (FDA) has cleared MammaPrint for use in postmenopausal women with early-stage breast cancer. In 2018, the FDA granted MammaPrint Breakthrough Device designation, which is designed to facilitate the development and commercialization of innovative medical devices. It is important to note that while MammaPrint has been cleared by the FDA, it is not a substitute for clinical judgment and should be used in conjunction with other factors, such as patient age, tumor size, and lymph node involvement, to help determine the most appropriate treatment for each patient. |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. MammaPrint is a gene expression profiling test used to determine the likelihood of breast cancer recurrence in postmenopausal women. 2. The test measures the expression levels of 70 genes associated with breast cancer prognosis. 3. MammaPrint has been cleared by the United States Food and Drug Administration (FDA) for use in postmenopausal women with early-stage breast cancer. 4. In 2018, the FDA granted MammaPrint Breakthrough Device designation. 5. MammaPrint should be used in conjunction with other factors, such as patient age, tumor size, and lymph node involvement, to determine the most appropriate treatment. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |

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| ID | 129(49) |
| 领域 | Bio-Medical |
| 用户问题 | Is amantadine effective for treatment of disorders conciousness? |
| 问题打分（逗号分隔）： 可读性（1-5） 规范性（1-5） 具体性（1-5） |  |
| 模型回复 | A number of studies have investigated the effectiveness of amantadine, an antiviral medication, for the treatment of disorders of consciousness, such as coma, vegetative state, and minimally conscious state. While the results of these studies have been mixed, some have suggested that amantadine may be effective in improving cognitive function and arousal in patients with disorders of consciousness. One of the earliest and most influential studies on the use of amantadine for disorders of consciousness was conducted by Dr. Steven Laureys and colleagues in 2004. In this study, 12 patients with severe traumatic brain injury were treated with amantadine for 12 weeks. Results showed that amantadine was associated with significant improvements in arousal and cognitive function, as measured by the Coma Recovery Scale and the Mental State Examination. Since then, several other studies have explored the use of amantadine for the treatment of disorders of consciousness. A 2012 study published in the journal Neuropsychopharmacology found that amantadine improved cognitive function in patients with minimally conscious state. Another study published in 2013 in the journal Brain Injury found that amantadine was associated with improved arousal and cognitive function in patients with coma. However, not all studies have found amantadine to be effective for the treatment of disorders of consciousness. A 2011 study published in the journal Neurocritical Care found that amantadine was not effective in improving cognitive function in patients with severe traumatic brain injury. It is important to note that the effectiveness of amantadine for the treatment of disorders of consciousness may depend on a number of factors, including the severity of the injury, the duration of coma or vegetative state, and the presence of other medical conditions. Additionally, the use of amantadine for this purpose is still experimental, and more research is needed to fully understand its potential benefits and risks. In summary, while some studies have suggested that amantadine may be effective for the treatment of disorders of consciousness, the results of these studies have been mixed, and more research is needed to fully understand the potential benefits and risks of this medic |
| 回复标注（二选一）： 1-回复与问题相关 2-回复与问题不相关 |  |
| 提取的事实 | 1. Amantadine is an antiviral medication. 2. Studies have investigated the effectiveness of amantadine for the treatment of disorders of consciousness. 3. Dr. Steven Laureys and colleagues conducted a study in 2004 on the use of amantadine for disorders of consciousness. 4. A 2012 study found that amantadine improved cognitive function in patients with minimally conscious state. 5. A 2011 study found that amantadine was not effective in improving cognitive function in patients with severe traumatic brain injury. |
| 事实标注 （每条事实八选一）： 1-完全正确事实 2-实体错误事实 3-关系错误事实 4-信息缺失事实 5-信息过时事实 6-表述绝对事实 7-无法验证事实 8-非事实表述 |  |