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| ID | 0(0) |
| 领域 | Bio-Medical |
| 用户问题 | Is Hirschsprung disease a mendelian or a multifactorial disorder? |
| 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 | Hirschsprung's disease is a congenital disorder that affects the development of the enteric nervous system (ENS) in the gastrointestinal tract. It is characterized by a failure of ganglion cell production in the distal portion of the intestine, leading to a lack of innervation and abnormal bowel function. Hirschsprung's disease is a complex disorder that is likely to have a multifactorial etiology. Several genetic and environmental factors have been implicated in its pathogenesis, including: 1. Genetic mutations: Mutations in genes encoding proteins involved in ganglion cell development and maintenance, such as the retinoic acid-related orphan receptor (ROBO) gene, have been identified as a cause of Hirschsprung's disease. 2. Retinoid deficiency: Retinoids, such as vitamin A, are essential for ganglion cell development and maintenance. Deficiency in retinoids during fetal development can lead to a failure of ganglion cell production. 3. Maternal smoking: Maternal smoking during pregnancy has been associated with an increased risk of Hirschsprung's disease in offspring. 4. Maternal stress: Maternal stress during pregnancy has been linked to an increased risk of Hirschsprung's disease in offspring. 5. Environmental toxins: Exposure to certain environmental toxins, such as pesticides and heavy metals, has been associated with an increased risk of Hirschsprung's disease. In conclusion, Hirschsprung's disease is a multifactorial disorder that is likely to involve the interplay of genetic and environmental factors. Further research is needed to fully understand the etiology of this complex disorder and to identify potential therapeutic targets for its 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. Hirschsprung's disease is a congenital disorder that affects the development of the enteric nervous system in the gastrointestinal tract. 2. It is characterized by a failure of ganglion cell production in the distal portion of the intestine, leading to a lack of innervation and abnormal bowel function. 3. Mutations in genes encoding proteins involved in ganglion cell development, such as the retinoic acid-related orphan receptor gene, have been identified as a cause of Hirschsprung's disease. 4. Retinoids, such as vitamin A, are essential for ganglion cell development and maintenance. Deficiency in retinoids during fetal development can lead to a failure of ganglion cell production. 5. Exposure to certain environmental toxins, such as pesticides and heavy metals, has been associated with an increased risk of Hirschsprung'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 | 1(1) |
| 领域 | Bio-Medical |
| 用户问题 | List signaling molecules (ligands) that interact with the receptor EGFR? |
| 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 signaling molecules (ligands) that interact with the epidermal growth factor receptor (EGFR) and trigger intracellular signaling pathways. Here are some of the most well-known ligands that interact with EGFR: 1. Epidermal growth factor (EGF): EGF is the most well-studied ligand of EGFR. It is a 6-kDa protein that is produced by many different cell types, including cancer cells. EGF binds to the extracellular domain of EGFR with high affinity and triggers downstream signaling pathways, including the RAS/MAPK and PI3K/AKT pathways. 2. Tumor necrosis factor-alpha (TNF-alpha): TNF-alpha is a cytokine that is involved in inflammation and immune responses. It can also bind to EGFR and trigger downstream signaling pathways, including the NF-κB pathway. 3. Amphiregulin: Amphiregulin is a ligand of EGFR that is produced by many different cell types, including cancer cells. It binds to the extracellular domain of EGFR with high affinity and triggers downstream signaling pathways, including the RAS/MAPK pathway. 4. Epiregulin: Epiregulin is another ligand of EGFR that is produced by many different cell types, including cancer cells. It binds to the extracellular domain of EGFR with high affinity and triggers downstream signaling pathways, including the RAS/MAPK pathway. 5. HB-EGF (heparin-binding EGF): HB-EGF is a ligand of EGFR that is produced by many different cell types, including cancer cells. It binds to the extracellular domain of EGFR with high affinity and triggers downstream signaling pathways, including the RAS/MAPK pathway. 6. Betacellulin: Betacellulin is a ligand of EGFR that is produced by many different cell types, including cancer cells. It binds to the extracellular domain of EGFR with high affinity and triggers |
| 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. Epidermal growth factor (EGF) is a signaling molecule that interacts with EGFR. 2. Tumor necrosis factor-alpha (TNF-alpha) is a signaling molecule that interacts with EGFR. 3. Amphiregulin is a signaling molecule that interacts with EGFR. 4. Epiregulin is a signaling molecule that interacts with EGFR. 5. HB-EGF (heparin-binding EGF) is a signaling molecule that interacts with EGFR. |
| 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 | 2(2) |
| 领域 | Bio-Medical |
| 用户问题 | Is the protein Papilin secreted? |
| 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 | Papilin is a protein that is expressed in the kidney and plays a role in the regulation of ion transport and water balance. It is known to be secreted from the kidney into the urine, where it can interact with other proteins and molecules to modulate its function. |
| 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. Papilin is a protein that is expressed in the kidney. 2. Papilin plays a role in the regulation of ion transport and water balance. 3. Papilin is secreted from the kidney into the urine. 4. Papilin can interact with other proteins and molecules in the urine. 5. The function of Papilin can be modulated by its interaction with other proteins and molecules. |
| 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 | 6(3) |
| 领域 | Bio-Medical |
| 用户问题 | Which miRNAs could be used as potential biomarkers for epithelial ovarian 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 | Epithelial ovarian cancer (EOC) is a highly aggressive and lethal gynecological malignancy. Early detection and accurate diagnosis are crucial for improving patient outcomes, but current diagnostic tools are limited. MicroRNAs (miRNAs) are small non-coding RNAs that have been implicated in the development and progression of EOC. Here, we review the current literature on the potential of miRNAs as biomarkers for EOC. Several miRNAs have been shown to be differentially expressed in EOC tissues compared to normal ovarian tissues. For example, miR-21, miR-145, and miR-200a have been found to be upregulated in EOC, while miR-34a and miR-181a have been found to be downregulated. These miRNAs may serve as potential biomarkers for early detection and monitoring of EOC. In addition, circulating miRNAs in the blood or serum have been shown to be useful as potential biomarkers for EOC. For example, miR-21, miR-145, and miR-200a have been detected in the serum of EOC patients, and their levels have been found to be significantly higher than in healthy controls. Similarly, miR-34a and miR-181a have been detected in the serum of EOC patients, but their levels are significantly lower than in healthy controls. However, it is important to note that the sensitivity and specificity of miRNAs as biomarkers for EOC are still unknown, and more research is needed to validate their use in clinical settings. Moreover, the complexity of the miRNA expression profile in EOC may make it difficult to identify a single miRNA that can accurately diagnose the disease. Therefore, a panel of miRNAs may be needed to improve diagnostic accuracy. In conclusion, miRNAs have shown promise as potential biomarkers for EOC. Further research is needed to validate their use in clinical settings and to identify a panel of miRNAs that can accurately diagnose and monitor EOC. |
| 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. Epithelial ovarian cancer (EOC) is a highly aggressive and lethal gynecological malignancy. 2. MiR-21, miR-145, and miR-200a have been found to be upregulated in EOC tissues. 3. MiR-34a and miR-181a have been found to be downregulated in EOC tissues. 4. MiR-21, miR-145, and miR-200a have been detected in the serum of EOC patients and their levels are significantly higher than in healthy controls. 5. MiR-34a and miR-181a have been detected in the serum of EOC patients, but their levels are significantly lower than in healthy controls. |
| 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 | 7(4) |
| 领域 | Bio-Medical |
| 用户问题 | Which acetylcholinesterase inhibitors are used for treatment of myasthenia gravis? |
| 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 | Myasthenia gravis is a chronic autoimmune disorder that affects the nervous system, particularly the nerve-muscle connection. One of the key symptoms of myasthenia gravis is muscle weakness, which can vary in severity and impact different parts of the body. Acetylcholinesterase inhibitors are a class of drugs that are used to treat myasthenia gravis by increasing the levels of the neurotransmitter acetylcholine in the nervous system. Acetylcholine is an important neurotransmitter that transmits signals between nerve cells and muscle cells, and its levels are often decreased in people with myasthenia gravis. There are several acetylcholinesterase inhibitors that are used to treat myasthenia gravis, including: 1. Pyridostigmine (Mestinon): This is the most commonly used acetylcholinesterase inhibitor for the treatment of myasthenia gravis. It works by increasing the levels of acetylcholine in the nervous system, which can help to improve muscle strength and reduce fatigue. 2. Efferoxamine (Cytotec): This drug is similar to pyridostigmine, but it has a longer duration of action and may be more effective in reducing muscle weakness. 3. Neostigmine (Prostigmin): This drug is similar to pyridostigmine, but it has a shorter duration of action and may be more effective in reducing muscle weakness in the eyes and throat. 4. Tacrine (Cognex): This drug is a less commonly used acetylcholinesterase inhibitor for the treatment of myasthenia gravis. It has a longer duration of action than pyridostigmine and may be more effective in reducing muscle weakness in the legs. It's important to note that acetylcholinesterase inhibitors may have side effects, including gastrointestinal problems, sweating, and headache. It's important to work closely with a healthcare provider to determine the best treatment plan for myasthenia gravis, including the use of acetylcholinesterase inhibitors. In addition to acetylcholin |
| 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. Myasthenia gravis is a chronic autoimmune disorder that affects the nervous system. 2. Acetylcholinesterase inhibitors are used to treat myasthenia gravis by increasing the levels of acetylcholine in the nervous system. 3. Pyridostigmine (Mestinon) is the most commonly used acetylcholinesterase inhibitor for the treatment of myasthenia gravis. 4. Efferoxamine (Cytotec) is a similar acetylcholinesterase inhibitor with a longer duration of action compared to pyridostigmine. 5. Neostigmine (Prostigmin) is a similar acetylcholinesterase inhibitor with a shorter duration of action compared to pyridostigmine. |
| 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 | 8(5) |
| 领域 | Bio-Medical |
| 用户问题 | Has Denosumab (Prolia) been approved by FDA? |
| 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, denosumab (Prolia) was approved by the US Food and Drug Administration (FDA) in 2010 for the treatment of osteoporosis in postmenopausal women and men aged ≥50 years. Denosumab is a monoclonal antibody that targets and inhibits the activity of receptor activator of nuclear factor kappa B ligand (RANKL), a protein that is involved in the regulation of bone metabolism. By inhibiting the activity of RANKL, denosumab helps to reduce bone resorption (the breakdown of bone) and increase bone density, which can help to prevent fractures. Denosumab is also approved for the treatment of bone metastases in patients with solid tumors, and for the treatment of giant cell tumor of the bone. It's important to note that Prolia (denosumab) is a prescription medication and should only be used under the guidance and supervision 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. Denosumab (Prolia) was approved by the US Food and Drug Administration (FDA) in 2010. 2. Denosumab (Prolia) is approved for the treatment of osteoporosis in postmenopausal women and men aged ≥50 years. 3. Denosumab (Prolia) targets and inhibits the activity of receptor activator of nuclear factor kappa B ligand (RANKL). 4. Denosumab (Prolia) is also approved for the treatment of bone metastases in patients with solid tumors. 5. Denosumab (Prolia) is approved for the treatment of giant cell tumor of the bone. |
| 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 | 10(6) |
| 领域 | Bio-Medical |
| 用户问题 | Name synonym of Acrokeratosis paraneoplastica. |
| 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 | Acrokeratosis paraneoplastica is a rare genetic disorder characterized by the development of thickened, scaly skin on the hands and feet, as well as an increased risk of developing certain types of cancer, including melanoma. The condition is also known as acrokeratosis paraneoplastica of the skin or APSS. There are several synonyms for Acrokeratosis paraneoplastica, including: 1. Acrokeratosis paraneoplastica syndrome (APS) 2. Acrokeratosis paraneoplastica of the skin (APS) 3. Familial acrokeratosis paraneoplastica (FAP) 4. Hereditary acrokeratosis paraneoplastica (HAP) 5. Paraneoplastic acrokeratosis (PA) 6. Skin acrokeratosis paraneoplastica (SAP) It is important to note that these synonyms refer to the same condition, and the term Acrokeratosis paraneoplastica is the most commonly used and accepted term. |
| 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. Acrokeratosis paraneoplastica is also known as acrokeratosis paraneoplastica syndrome (APS). 2. Acrokeratosis paraneoplastica is also known as acrokeratosis paraneoplastica of the skin (APS). 3. Acrokeratosis paraneoplastica is also known as familial acrokeratosis paraneoplastica (FAP). 4. Acrokeratosis paraneoplastica is also known as hereditary acrokeratosis paraneoplastica (HAP). 5. Acrokeratosis paraneoplastica is also known as paraneoplastic acrokeratosis (PA). 6. Acrokeratosis paraneoplastica is also known as skin acrokeratosis paraneoplastica (SAP). |
| 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 | 11(7) |
| 领域 | Bio-Medical |
| 用户问题 | Which are the classes of anti-arrhythmic drugs according to Vaughan-Williams classification? |
| 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 | Vaughan-Williams classification is a system used to categorize anti-arrhythmic drugs based on their mechanism of action. The classification system divides anti-arrhythmic drugs into three main classes: Class 1: sodium channel blockers Class 2: beta-adrenergic receptor antagonists Class 3: calcium channel blockers Class 4: drugs that act on the ryanodine receptor Class 5: drugs that act on the sarcoplasmic reticulum Each class has subclasses, and the drugs are further classified based on their specific mechanism of action. For example, sodium channel blockers are further divided into class 1A (such as lidocaine and phenytoin) and class 1B (such as flecainide and propafenone). Beta-adrenergic receptor antagonists are further divided into class 2A (such as propranolol) and class 2B (such as acebutolol). |
| 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. Vaughan-Williams classification categorizes anti-arrhythmic drugs based on their mechanism of action. 2. The classification system divides anti-arrhythmic drugs into three main classes: sodium channel blockers, beta-adrenergic receptor antagonists, and calcium channel blockers. 3. Class 1 of Vaughan-Williams classification includes sodium channel blockers. 4. Class 2 of Vaughan-Williams classification includes beta-adrenergic receptor antagonists. 5. Class 3 of Vaughan-Williams classification includes calcium channel blockers. |
| 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 | 12(8) |
| 领域 | Bio-Medical |
| 用户问题 | Which are the different isoforms of the mammalian Notch receptor? |
| 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 Notch receptor is a transmembrane protein that plays a crucial role in cell-cell communication and differentiation during embryonic development and tissue homeostasis. There are four known isoforms of the mammalian Notch receptor: Notch1, Notch2, Notch3, and Notch4. Each isoform has a distinct tissue expression pattern and function. |
| 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 Notch receptor is a transmembrane protein involved in cell-cell communication and differentiation. 2. The mammalian Notch receptor has four known isoforms: Notch1, Notch2, Notch3, and Notch4. 3. Each isoform of the mammalian Notch receptor has a distinct tissue expression pattern. 4. Each isoform of the mammalian Notch receptor has a distinct function. |
| 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 | 13(9) |
| 领域 | Bio-Medical |
| 用户问题 | Which are the major characteristics of cellular senescence? |
| 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 | Senescence is a state of permanent cell cycle arrest that occurs in response to various forms of cellular stress, such as DNA damage, oxidative stress, or oncogenic signaling. Cells can enter senescence through various mechanisms, including telomere shortening, DNA damage, or exposure to pro-inflammatory cytokines. Once a cell enters senescence, it can no longer divide and will enter a state of permanent cell cycle arrest. Senescent cells can also secrete a variety of factors that can affect the surrounding cells, including pro-inflammatory cytokines, chemokines, and matrix metalloproteinases. Some of the major characteristics of cellular senescence include: 1. Permanent cell cycle arrest: Senescent cells are unable to divide and will remain in the G0 phase of the cell cycle indefinitely. 2. Increased secretion of pro-inflammatory cytokines and chemokines: Senescent cells can secrete a variety of pro-inflammatory cytokines and chemokines, including IL-6, IL-8, and MCP-1, which can promote inflammation and tissue damage. 3. Loss of cellular adhesion and migration: Senescent cells can lose their ability to adhere to extracellular matrix proteins and migrate through tissues, which can contribute to tissue degeneration and dysfunction. 4. Increased oxidative stress: Senescent cells can exhibit increased levels of oxidative stress, which can damage cellular components and contribute to the aging process. 5. Epigenetic alterations: Senescent cells can exhibit epigenetic alterations, including DNA methylation and histone modifications, which can lead to changes in gene expression and contribute to the aging process. 6. Increased production of reactive oxygen species (ROS): Senescent cells can produce high levels of ROS, which can damage cellular components and contribute to the aging process. 7. Decreased ability to respond to stress: Senescent cells can exhibit a decreased ability to respond to stress, which can contribute to the progression of aging and age-related diseases. 8. Increased levels of DNA damage: Senescent |
| 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. Senescence is a state of permanent cell cycle arrest that occurs in response to various forms of cellular stress. 2. Cells can enter senescence through various mechanisms, including telomere shortening, DNA damage, or exposure to pro-inflammatory cytokines. 3. Senescent cells are unable to divide and will remain in the G0 phase of the cell cycle indefinitely. 4. Senescent cells can secrete a variety of pro-inflammatory cytokines and chemokines. 5. Senescent cells can lose their ability to adhere to extracellular matrix proteins and migrate through tissues. |
| 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 | 14(10) |
| 领域 | Bio-Medical |
| 用户问题 | Orteronel was developed for treatment of which 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 | Orteronel is a drug used to treat prostate cancer. It is a non-steroidal anti-inflammatory drug (NSAID) that works by blocking the production of certain enzymes that are involved in the growth and spread of cancer cells. Orteronel is specifically approved for the treatment of metastatic castrate-resistant prostate cancer (mCRPC), which is prostate cancer that has spread to other parts of the body and is no longer responding to hormone therapy. |
| 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. Orteronel is a drug used to treat prostate cancer. 2. Orteronel is a non-steroidal anti-inflammatory drug (NSAID). 3. Orteronel works by blocking the production of certain enzymes involved in the growth and spread of cancer cells. 4. Orteronel is approved for the treatment of metastatic castrate-resistant prostate cancer (mCRPC). 5. Metastatic castrate-resistant prostate cancer is prostate cancer that has spread to other parts of the body and is no longer responding to hormone therapy. |
| 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 | 15(11) |
| 领域 | Bio-Medical |
| 用户问题 | Is the monoclonal antibody Trastuzumab (Herceptin) of potential use in the treatment of prostate 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 | Trastuzumab (Herceptin) is a monoclonal antibody that has been approved for the treatment of breast cancer. However, there is some evidence to suggest that it may also have potential as a treatment for prostate cancer. One study published in the Journal of Clinical Oncology found that trastuzumab was effective in reducing the growth of prostate cancer cells in laboratory experiments. Another study published in the Prostate found that trastuzumab was effective in reducing the growth of prostate cancer cells in mice. There are several potential mechanisms by which trastuzumab may be effective in treating prostate cancer. For example, it may bind to the HER2 protein, which is overexpressed in many types of cancer, including prostate cancer. This binding may help to inhibit the growth of cancer cells. Additionally, trastuzumab may also stimulate the immune system to attack cancer cells. While these findings are promising, it is important to note that more research is needed to fully understand the potential of trastuzumab as a treatment for prostate cancer. For example, larger clinical trials are needed to determine the safety and effectiveness of trastuzumab in humans. Additionally, more research is needed to understand the optimal dosing and administration of trastuzumab for prostate cancer treatment. In summary, while the evidence is still limited, there is some potential for trastuzumab (Herceptin) to be used in the treatment of prostate cancer. Further research is needed to fully understand its potential in this context. |
| 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. Trastuzumab (Herceptin) is a monoclonal antibody approved for the treatment of breast cancer. 2. Trastuzumab has shown potential as a treatment for prostate cancer in laboratory experiments with human cells and mice. 3. One potential mechanism by which trastuzumab may be effective in treating prostate cancer is by binding to the overexpressed HER2 protein. 4. Trastuzumab may also stimulate the immune system to attack cancer cells. 5. More research, including larger clinical trials, is needed to determine the safety, effectiveness, optimal dosing, and administration of trastuzumab in prostate cancer 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 | 16(12) |
| 领域 | Bio-Medical |
| 用户问题 | Which are the Yamanaka factors? |
| 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 Yamanaka factors are a set of transcription factors that were first identified in 2006 as the key regulators of induced pluripotent stem cell (iPSC) reprogramming. These factors are: 1. Oct4 (also known as POU5F1): a transcription factor that regulates the expression of genes involved in pluripotency and self-renewal. 2. Sox2: a transcription factor that regulates the expression of genes involved in stem cell self-renewal and differentiation. 3. Klf4: a transcription factor that regulates the expression of genes involved in cellular differentiation and development. 4. c-Myc: a transcription factor that regulates the expression of genes involved in cellular proliferation and differentiation. Together, these factors are able to reprogram adult cells into a pluripotent state, mimicking the process of embryonic stem cell development. The discovery of the Yamanaka factors has revolutionized the field of stem cell biology and has opened up new avenues for the study of cellular differentiation and disease modeling. |
| 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 Yamanaka factors were first identified in 2006 as the key regulators of induced pluripotent stem cell (iPSC) reprogramming. 2. Oct4 (POU5F1) is a transcription factor that regulates the expression of genes involved in pluripotency and self-renewal. 3. Sox2 is a transcription factor that regulates the expression of genes involved in stem cell self-renewal and differentiation. 4. Klf4 is a transcription factor that regulates the expression of genes involved in cellular differentiation and development. 5. c-Myc is a transcription factor that regulates the expression of genes involved in cellular proliferation and differentiation. |
| 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 | 17(13) |
| 领域 | Bio-Medical |
| 用户问题 | What is the aim of the Human Chromosome-centric Proteome Project (C-HPP)? |
| 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 Human Chromosome-centric Proteome Project (C-HPP) is a collaborative research effort that aims to generate a comprehensive and integrated understanding of the human proteome, focusing on the chromosomal locations and functional interactions of proteins. The project's ultimate goal is to provide a detailed map of the human proteome, including its structure, function, and interactions, and to use this knowledge to improve our understanding of human health and disease. The C-HPP is a multi-disciplinary effort that brings together researchers from around the world, including geneticists, biochemists, cell biologists, and computational biologists. The project is using a range of cutting-edge technologies, including mass spectrometry, chromosome conformation capture (3C), and RNA sequencing, to generate high-resolution maps of protein localization, protein-protein interactions, and gene expression across the human genome. Some of the specific goals of the C-HPP include: 1. Generating a comprehensive map of the human proteome, including its structure, function, and interactions. 2. Identifying the chromosomal locations and functional interactions of proteins in different cell types and tissues. 3. Investigating the role of protein misfolding and aggregation in human disease, including neurodegenerative disorders such as Alzheimer's and Parkinson's. 4. Developing new computational methods for analyzing large-scale proteomics data sets and integrating them with other types of genomic data. 5. Exploring the potential of the human proteome as a source of new therapeutic targets and biomarkers for disease diagnosis and monitoring. By achieving these goals, the C-HPP aims to provide a fundamental understanding of the human proteome and its role in health and disease, and to use this knowledge to improve human health and quality of life. |
| 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 Human Chromosome-centric Proteome Project (C-HPP) aims to generate a comprehensive and integrated understanding of the human proteome. 2. The project aims to provide a detailed map of the human proteome, including its structure, function, and interactions. 3. The C-HPP brings together researchers from different disciplines, including geneticists, biochemists, cell biologists, and computational biologists. 4. The project uses cutting-edge technologies such as mass spectrometry, chromosome conformation capture (3C), and RNA sequencing to generate high-resolution maps of protein localization, protein-protein interactions, and gene expression across the human genome. 5. The goals of the C-HPP include investigating the role of protein misfolding and aggregation in human disease, developing new computational methods for analyzing proteomics data, and exploring the potential of the human proteome for therapeutic targets and biomarkers. |
| 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 | 18(14) |
| 领域 | Bio-Medical |
| 用户问题 | Where is the protein Pannexin1 located? |
| 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 | Pannexin1 is a protein that is located in the plasma membrane of various cell types, including neurons, astrocytes, and endothelial cells. It is particularly abundant in the brain, where it plays a critical role in the regulation of the blood-brain barrier (BBB) and the clearance of neurotransmitters. 1. What is the function of Pannexin1? Pannexin1 is a transmembrane protein that regulates the exchange of ions and small molecules across the plasma membrane. It is involved in a variety of cellular processes, including the formation of the blood-brain barrier (BBB), the clearance of neurotransmitters, and the regulation of cellular signaling pathways. |
| 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. Pannexin1 is a protein located in the plasma membrane of various cell types. 2. Pannexin1 is particularly abundant in the brain. 3. Pannexin1 plays a critical role in the regulation of the blood-brain barrier (BBB). 4. Pannexin1 is involved in the clearance of neurotransmitters. 5. Pannexin1 regulates the exchange of ions and small molecules across the plasma membrane. |
| 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 | 19(15) |
| 领域 | Bio-Medical |
| 用户问题 | Which currently known mitochondrial diseases have been attributed to POLG mutations? |
| 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 | POLG mutations have been implicated in a wide range of mitochondrial diseases, including: 1. MELAS (Mitochondrial Encephalomyopathy, Lactic Acidosis, and Stroke-like episodes) syndrome: This is the most common mitochondrial disease caused by POLG mutations. MELAS is characterized by recurrent stroke-like episodes, seizures, and developmental delay. 2. Myopathy, encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS): This is a rare genetic disorder that affects the brain and muscles. POLG mutations are found in about 70% of MELAS cases. 3. Kearns-Sayre syndrome: This is a rare genetic disorder that affects the nervous system, particularly the brain and spinal cord. POLG mutations are found in about 20% of Kearns-Sayre syndrome cases. 4. Chronic progressive external ophthalmoplegia (CPEO): This is a rare genetic disorder that affects the muscles around the eyes. POLG mutations are found in about 20% of CPEO cases. 5. Mitochondrial myopathies: POLG mutations have been identified in some cases of mitochondrial myopathies, which are muscle disorders caused by defects in mitochondrial function. 6. Neurodegenerative diseases: POLG mutations have been identified in some cases of neurodegenerative diseases, such as Alzheimer's disease and Parkinson's disease. 7. Other mitochondrial disorders: POLG mutations have also been identified in other mitochondrial disorders, such as mitochondrial DNA depletion syndrome and mitochondrial encephalopathy with lactic acidosis and stroke-like episodes (MELAS-like). It's worth noting that the full extent of POLG-related mitochondrial diseases is still being discovered and characterized, and this list may not be exhaustive. |
| 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. POLG mutations have been associated with MELAS (Mitochondrial Encephalomyopathy, Lactic Acidosis, and Stroke-like episodes) syndrome. 2. POLG mutations are found in about 70% of MELAS cases. 3. POLG mutations have been associated with Kearns-Sayre syndrome. 4. POLG mutations are found in about 20% of Kearns-Sayre syndrome cases. 5. POLG mutations have been associated with Chronic progressive external ophthalmoplegia (CPEO). 6. te: Due to the lack of clear independent facts and the repetition of information, there are no more factual statements that can be extracted. |
| 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 | 20(16) |
| 领域 | Bio-Medical |
| 用户问题 | What is the effect of ivabradine in heart failure 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 | Ivabradine is a selective sinus node inhibitor that slows the heart rate and reduces the force of the heart's contractions. It is used to treat heart failure after myocardial infarction (heart attack) in patients who have a normal ejection fraction (the percentage of blood that is pumped out of the heart with each beat). Studies have shown that ivabradine can improve symptoms and reduce hospitalization rates in patients with heart failure after myocardial infarction. It can also reduce the risk of death from cardiovascular causes. Ivabradine works by slowing the heart rate, which reduces the demand for oxygen and reduces the workload on the heart. This can help to improve the heart's function and reduce symptoms such as shortness of breath, fatigue, and swelling in the legs. It is important to note that ivabradine is not a cure for heart failure, and it may not be effective in all patients. It is usually used in combination with other medications, such as angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), to treat heart failure. Overall, ivabradine can be an effective treatment option for heart failure after myocardial infarction, particularly in patients with a normal ejection fraction. However, it is important to discuss the potential benefits and risks of ivabradine with a healthcare provider to determine if it is the right treatment option for you. |
| 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. Ivabradine is a selective sinus node inhibitor that slows the heart rate and reduces the force of the heart's contractions. 2. Ivabradine is used to treat heart failure after myocardial infarction in patients who have a normal ejection fraction. 3. Studies have shown that ivabradine can improve symptoms and reduce hospitalization rates in patients with heart failure after myocardial infarction. 4. Ivabradine works by slowing the heart rate, reducing the demand for oxygen, and reducing the workload on the heart. 5. Ivabradine is usually used in combination with other medications, such as ACE inhibitors or ARBs, to treat heart failure after myocardial infarction. |
| 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 | 22(17) |
| 领域 | Bio-Medical |
| 用户问题 | Are transcription and splicing connected? |
| 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 | Transcription and splicing are two important post-transcriptional processes that play critical roles in the regulation of gene expression. While they are distinct processes, they are connected in several ways. 1. Overlapping functions: Both transcription and splicing are involved in the processing of RNA molecules. Transcription converts DNA sequences into RNA molecules, while splicing removes introns and joins exons to form mature RNA. 2. Regulation of splicing: Transcription factors can regulate splicing by binding to specific DNA sequences near exon-intron boundaries and altering the splicing patterns. Conversely, splicing can also affect transcription by removing or modifying transcriptional activators or repressors. 3. Splicing of transcriptional activators: Some transcriptional activators are spliced to regulate their activity. For example, the activator protein c-Myc is spliced to produce different isoforms with varying transcriptional activity. 4. Splicing of repressor proteins: Similarly, repressor proteins can also be spliced to regulate their function. For instance, the repressor protein p53 is spliced to produce different isoforms with distinct functions in cell cycle regulation and apoptosis. 5. Co-regulation of transcription and splicing: Many transcription factors can also regulate splicing, and vice versa. For example, the transcription factor NF-κB can activate the splicing of the gene encoding the cytokine tumor necrosis factor-α (TNF-α). 6. Common signaling pathways: Transcription and splicing are connected through common signaling pathways, such as the PI3K/Akt pathway, which regulates both processes. 7. Regulation by RNA-binding proteins: RNA-binding proteins (RBPs) can regulate both transcription and splicing by binding to specific RNA sequences and recruiting other proteins to modulate gene expression. 8. Overlapping expression patterns: Many genes are co-regulated by both transcription and splicing, and their expression patterns can overlap. For example, the genes encoding the pro-inflammatory cytokines interleukin |
| 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. Transcription converts DNA sequences into RNA molecules. 2. Splicing removes introns and joins exons to form mature RNA. 3. Transcription factors can regulate splicing by binding to specific DNA sequences near exon-intron boundaries and altering the splicing patterns. 4. Some transcriptional activators are spliced to regulate their activity. 5. Repressor proteins can be spliced to regulate their function. |
| 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 | 23(18) |
| 领域 | Bio-Medical |
| 用户问题 | What is the mode of inheritance of Facioscapulohumeral muscular dystrophy (FSHD)? |
| 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 | Facioscapulohumeral muscular dystrophy (FSHD) is a genetic disorder that affects the muscles of the face, shoulder, and upper arm. It is caused by a mutation in the D4Z4 repeat on chromosome 4, which leads to a deficiency of the protein dystrophin. The mutation is inherited in an autosomal dominant manner, meaning that a single copy of the mutated gene is enough to cause the condition. This means that each affected parent has a 50% chance of passing the mutated gene to each of their children. |
| 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. Facioscapulohumeral muscular dystrophy (FSHD) affects the muscles of the face, shoulder, and upper arm. 2. FSHD is caused by a mutation in the D4Z4 repeat on chromosome 4. 3. The mutation in FSHD leads to a deficiency of the protein dystrophin. 4. FSHD is inherited in an autosomal dominant manner. 5. Each affected parent has a 50% chance of passing the mutated gene to each of their children. |
| 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 | 24(19) |
| 领域 | Bio-Medical |
| 用户问题 | Is Alu hypomethylation associated with breast 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 | A growing body of evidence suggests that Alu hypomethylation is associated with breast cancer. Here are some key findings: 1. Alu hypomethylation is common in breast cancer: Studies have shown that Alu hypomethylation is frequent in breast cancer, occurring in approximately 60-70% of cases. 2. Alu hypomethylation is associated with aggressive breast cancer: Alu hypomethylation has been linked to more aggressive breast cancer subtypes, such as basal-like and HER2-enriched tumors. 3. Alu hypomethylation promotes breast cancer cell proliferation: Research has shown that Alu hypomethylation can lead to the upregulation of oncogenic genes, such as c-Myc, and promote breast cancer cell proliferation. 4. Alu hypomethylation is associated with poor prognosis: Studies have found that Alu hypomethylation is associated with poorer prognosis in breast cancer patients, including reduced overall survival and increased risk of recurrence. 5. Alu hypomethylation may be a potential therapeutic target: Given the association between Alu hypomethylation and aggressive breast cancer subtypes, targeting this epigenetic alteration may be a potential therapeutic strategy for improving breast cancer treatment outcomes. In summary, Alu hypomethylation is a common epigenetic alteration in breast cancer, associated with aggressive tumor subtypes and poor prognosis. Further research is needed to fully understand the role of Alu hypomethylation in breast cancer and to explore its potential as a therapeutic target. |
| 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. Alu hypomethylation is frequent in approximately 60-70% of breast cancer cases. 2. Alu hypomethylation is associated with more aggressive breast cancer subtypes, such as basal-like and HER2-enriched tumors. 3. Alu hypomethylation can lead to the upregulation of oncogenic genes, such as c-Myc, promoting breast cancer cell proliferation. 4. Alu hypomethylation is associated with poorer prognosis in breast cancer patients, including reduced overall survival and increased risk of recurrence. 5. Targeting Alu hypomethylation may be a potential therapeutic strategy to improve breast cancer treatment outcomes. |
| 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 | 25(20) |
| 领域 | Bio-Medical |
| 用户问题 | Which proteins participate in the formation of the ryanodine receptor quaternary macromolecular complex? |
| 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 ryanodine receptor (RyR) is a Ca2+ release channel found in the endoplasmic reticulum (ER) of mammalian cells. The RyR complex is composed of four subunits (RyR1, RyR2, RyR3, and RyR4) that form a tetrameric structure. The quaternary structure of the RyR complex is crucial for its function as a Ca2+ release channel. Several proteins have been identified that participate in the formation of the RyR quaternary complex. These include: 1. RyR subunits: RyR1, RyR2, RyR3, and RyR4 are the subunits that make up the core of the RyR complex. These subunits are highly homologous and share a common structure, with four transmembrane domains (TMDs) and a large cytoplasmic domain. 2. Calmodulin (CaM): CaM is a small calcium-binding protein that binds to the RyR complex and regulates its activity. CaM binds to specific sites on the RyR subunits and modulates their interactions with each other and with other proteins. 3. Inositol 1,4,5-trisphosphate receptor (IP3R): IP3R is a Ca2+ release channel that is also located in the ER. IP3R and RyR form a complex that regulates Ca2+ release from the ER. The IP3R subunits are also highly homologous to RyR subunits and share similar structural features. 4. Calcium-binding protein (CBP): CBP is a protein that binds to the RyR complex and regulates its activity. CBP is a calcium-binding protein that is highly homologous to RyR subunits and shares similar structural features. 5. Membrane-associated guanylate kinase (MAGUK): MAGUK is a family of proteins that bind to the RyR complex and regulate its activity. MAGUK proteins are highly homologous to RyR subunits and share similar structural features. These proteins play a crucial role in the formation of the RyR quaternary complex and its |
| 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 ryanodine receptor complex is composed of four subunits: RyR1, RyR2, RyR3, and RyR4. 2. Calmodulin (CaM) is a protein that binds to the RyR complex and regulates its activity. 3. Inositol 1,4,5-trisphosphate receptor (IP3R) forms a complex with RyR and regulates Ca2+ release from the endoplasmic reticulum (ER). 4. Calcium-binding protein (CBP) binds to the RyR complex and regulates its activity. 5. Membrane-associated guanylate kinase (MAGUK) proteins bind to the RyR complex and regulate its activity. |
| 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 | 26(21) |
| 领域 | Bio-Medical |
| 用户问题 | What kind of chromatography is HILIC? |
| 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 | High-performance liquid chromatography (HPLC) is a popular analytical technique used to separate, identify, and quantify the components of a mixture. Within HPLC, there are several types of chromatography, including hydrophilic interaction liquid chromatography (HILIC). HILIC is a type of chromatography that utilizes a hydrophilic column, which is capable of retaining and separating compounds based on their hydrophilic interactions with the stationary phase. HILIC is particularly useful for separating polar compounds, such as sugars, amino acids, and nucleotides, as well as for analyzing complex biological samples, such as plasma and urine. In HILIC, the stationary phase is typically a hydrophilic polymer, such as poly(vinylpyrrolidone) (PVDF) or poly(ethylene oxide) (PEO), which is bonded to a silica or silica-based support. The mobile phase is typically a water-based solution, such as water with a small amount of organic solvent, such as methanol or acetonitrile. The hydrophilic interaction between the stationary phase and the mobile phase allows for the separation of compounds based on their hydrophilic properties. Some of the key applications of HILIC include: 1. Separation and analysis of polar compounds: HILIC is particularly useful for separating and analyzing polar compounds, such as sugars, amino acids, and nucleotides. 2. Analysis of complex biological samples: HILIC can be used to analyze complex biological samples, such as plasma and urine, by separating and identifying the various components present. 3. Method development and optimization: HILIC can be used to develop and optimize methods for the analysis of a wide range of compounds, including those that are difficult to separate using other types of chromatography. 4. Identification and quantification of unknown compounds: HILIC can be used to identify and quantify unknown compounds in a mixture by comparing their retention times and peak shapes to those of known compounds. 5. Drug metabolism and pharmacokinetics: HILIC can be used to study the metabolism and pharmacokinet |
| 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. HILIC stands for hydrophilic interaction liquid chromatography. 2. HILIC utilizes a hydrophilic column for separating compounds based on their hydrophilic interactions with the stationary phase. 3. HILIC is particularly useful for separating polar compounds, such as sugars, amino acids, and nucleotides. 4. The stationary phase in HILIC is typically a hydrophilic polymer bonded to a silica or silica-based support. 5. The mobile phase in HILIC is typically a water-based solution with a small amount of organic solvent. |
| 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 | 28(22) |
| 领域 | Bio-Medical |
| 用户问题 | Proteomic analyses need prior knowledge of the organism complete genome. Is the complete genome of the bacteria of the genus Arthrobacter available? |
| 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 | Arthrobacter is a genus of bacteria that includes several species, such as A. globiformis, A. crystalloclasticus, and A. nitrofigilis. These bacteria are gram-positive, rod-shaped, and can be found in a variety of environments, including soil, sediments, and the rhizosphere of plants. The complete genome of an organism refers to the entire set of genetic material, including all of the genes and non-coding regions, that make up the organism's DNA. The availability of a complete genome for an organism can be an important factor in determining the feasibility of certain types of analyses, such as proteomic studies. As of 2023, there are several complete genomes available for species within the genus Arthrobacter, including A. globiformis, A. crystalloclasticus, and A. nitrofigilis. These genomes were sequenced using a variety of techniques, including whole-genome shotgun sequencing and single-cell genomics. The availability of these genomes has allowed researchers to study the genomics of Arthrobacter species in greater detail, including the identification of genes involved in various metabolic processes, such as the degradation of aromatic compounds and the biosynthesis of antibiotics. These studies have also provided insights into the evolutionary relationships between different species within the genus Arthrobacter. In summary, the complete genome of an organism is its entire set of genetic material, and the availability of a complete genome can be an important factor in determining the feasibility of certain types of analyses, such as proteomic studies. Several complete genomes are available for species within the genus Arthrobacter, which has allowed researchers to study the genomics of these organisms in greater detail. |
| 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. Arthrobacter is a genus of bacteria that includes several species such as A. globiformis, A. crystalloclasticus, and A. nitrofigilis. 2. Arthrobacter bacteria are gram-positive and rod-shaped. 3. Arthrobacter bacteria can be found in soil, sediments, and the rhizosphere of plants. 4. As of 2023, there are several complete genomes available for species within the genus Arthrobacter, including A. globiformis, A. crystalloclasticus, and A. nitrofigilis. 5. The availability of these genomes has allowed researchers to study the genomics of Arthrobacter species in greater detail, including the identification of genes involved in various metabolic processes and the evolutionary relationships between different species within the genus. |
| 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 | 30(23) |
| 领域 | Bio-Medical |
| 用户问题 | List the endoscopic diagnoses that have been reported in children with autism |
| 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 | spectrum disorder (ASD) Endoscopic diagnoses that have been reported in children with autism spectrum disorder (ASD) include: 1. Gastroesophageal reflux disease (GERD): Endoscopy can help diagnose GERD in children with ASD by visualizing the esophageal mucosa and detecting erosions or ulcers. 2. Aspiration: Endoscopy can help diagnose aspiration in children with ASD by visualizing the upper airway and detecting aspirated material. 3. Airway abnormalities: Endoscopy can help diagnose airway abnormalities in children with ASD, such as tracheomalacia or subglottic stenosis. 4. Inflammatory bowel disease (IBD): Endoscopy can help diagnose IBD in children with ASD by visualizing the mucosa and detecting inflammation or ulcers. 5. Food impaction: Endoscopy can help diagnose food impaction in children with ASD by visualizing the esophagus and detecting any blockages or impactions. 6. Allergic bronchopulmonary aspergillosis (ABPA): Endoscopy can help diagnose ABPA in children with ASD by visualizing the airways and detecting any inflammation or infection. 7. Asthma: Endoscopy can help diagnose asthma in children with ASD by visualizing the airways and detecting any inflammation or narrowing. 8. Nasal cavity abnormalities: Endoscopy can help diagnose nasal cavity abnormalities in children with ASD, such as nasal polyps or deviated septum. 9. Sinusitis: Endoscopy can help diagnose sinusitis in children with ASD by visualizing the sinuses and detecting any inflammation or infection. 10. Cervical lymphadenopathy: Endoscopy can help diagnose cervical lymphadenopathy in children with ASD by visualizing the lymph nodes in the neck and detecting any abnormalities. It is important to note that endoscopy is just one tool that healthcare providers use to diagnose and manage ASD, and it may not |
| 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. Endoscopy can be used to diagnose gastroesophageal reflux disease (GERD) in children with autism spectrum disorder (ASD). 2. Endoscopy can be used to diagnose aspiration in children with ASD by visualizing the upper airway and detecting aspirated material. 3. Endoscopy can be used to diagnose airway abnormalities, such as tracheomalacia or subglottic stenosis, in children with ASD. 4. Endoscopy can be used to diagnose inflammatory bowel disease (IBD) in children with ASD by visualizing the mucosa and detecting inflammation or ulcers. 5. Endoscopy can be used to diagnose food impaction in children with ASD by visualizing the esophagus and detecting any blockages or impactions. |
| 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 | 31(24) |
| 领域 | Bio-Medical |
| 用户问题 | What are the outcomes of Renal sympathetic denervation? |
| 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 | Renal sympathetic denervation is a minimally invasive procedure that has been shown to improve blood pressure control in patients with resistant hypertension. The outcomes of renal sympathetic denervation have been studied in several clinical trials, and the results have been promising. Here are some of the key outcomes that have been reported: 1. Sustained blood pressure reduction: Renal sympathetic denervation has been shown to result in sustained blood pressure reduction, with a mean reduction of 15-20 mmHg in systolic blood pressure and 10-15 mmHg in diastolic blood pressure, both at 6 months and 12 months after the procedure. 2. Improved quality of life: Renal sympathetic denervation has also been shown to improve quality of life in patients with resistant hypertension. Patients have reported improved physical function, reduced fatigue, and improved mental health. 3. Reduced medication use: Many patients who undergo renal sympathetic denervation are able to reduce or eliminate their use of blood pressure medications, which can lead to improved medication adherence and a lower risk of medication side effects. 4. Reduced risk of cardiovascular events: Renal sympathetic denervation has been shown to reduce the risk of cardiovascular events, such as heart attacks and strokes, in patients with resistant hypertension. 5. No significant changes in renal function: Renal sympathetic denervation has been shown to have no significant changes in renal function, including creatinine clearance and estimated glomerular filtration rate (eGFR), in patients with normal baseline renal function. 6. No significant changes in electrocardiogram (ECG) parameters: Renal sympathetic denervation has been shown to have no significant changes in ECG parameters, including heart rate, QTc interval, and T wave amplitude, in patients with normal baseline ECG parameters. 7. No significant changes in blood test results: Renal sympathetic denervation has been shown to have no significant changes in blood test results, including liver function tests, renal function tests, and thyroid function tests, in patients with normal baseline test results. 8. Good |
| 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. Renal sympathetic denervation is a minimally invasive procedure that has been shown to improve blood pressure control in patients with resistant hypertension. 2. Renal sympathetic denervation results in a mean reduction of 15-20 mmHg in systolic blood pressure and 10-15 mmHg in diastolic blood pressure at 6 and 12 months after the procedure. 3. Renal sympathetic denervation improves quality of life in patients with resistant hypertension, including improved physical function, reduced fatigue, and improved mental health. 4. Many patients who undergo renal sympathetic denervation are able to reduce or eliminate their use of blood pressure medications. 5. Renal sympathetic denervation reduces the risk of cardiovascular events, such as heart attacks and strokes, in patients with resistant hypertension. 6. Renal sympathetic denervation has no significant changes in renal function in patients with normal baseline renal function. 7. Renal sympathetic denervation has no significant changes in ECG parameters in patients with normal baseline ECG parameters. 8. Renal sympathetic denervation has no significant changes in blood test results in patients with normal baseline test results. |
| 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 | 32(25) |
| 领域 | Bio-Medical |
| 用户问题 | Which MAP kinase phosphorylates the transcription factor c-jun? |
| 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 mitogen-activated protein kinase (MAPK) pathway is a signaling cascade that plays a crucial role in regulating various cellular processes, including gene expression. One of the key transcription factors that are regulated by the MAPK pathway is c-Jun, a transcription factor that plays a crucial role in the regulation of gene expression in response to various stimuli. There are several MAPKs that can phosphorylate and activate c-Jun, including: 1. ERK1/2 (extracellular signal-regulated kinase 1/2): ERK1/2 is a MAPK that is activated in response to various growth factors and hormones, and it phosphorylates and activates c-Jun. 2. JNK (c-Jun N-terminal kinase): JNK is a MAPK that is activated in response to UV radiation, osmotic stress, and other stimuli, and it phosphorylates and activates c-Jun. 3. p38 MAPK (MAPK14): p38 MAPK is activated in response to bacterial pathogens, inflammatory cytokines, and other stimuli, and it phosphorylates and activates c-Jun. The phosphorylation and activation of c-Jun by MAPKs leads to the activation of various transcriptional targets, including the c-Jun-dependent promoter and enhancer elements. This results in the upregulation of genes involved in cell proliferation, differentiation, and survival. In summary, the MAPK pathway plays a crucial role in regulating the expression of c-Jun and its downstream targets, which are involved in a wide range of cellular processes. Understanding the regulation of c-Jun by MAPKs is important for understanding the molecular mechanisms underlying various diseases, including cancer, inflammation, and neurodegenerative disorders. |
| 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 MAP kinase pathway regulates gene expression. 2. c-Jun is a transcription factor that is regulated by the MAP kinase pathway. 3. ERK1/2 is a MAP kinase that phosphorylates and activates c-Jun. 4. JNK is a MAP kinase that phosphorylates and activates c-Jun. 5. p38 MAPK is a MAP kinase that phosphorylates and activates c-Jun. |
| 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 | 33(26) |
| 领域 | Bio-Medical |
| 用户问题 | What is the meaning of the acronym "TAILS" used in protein N-terminomics? |
| 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 | TAILS stands for "Terminal Analysis of Incomplete Lysine Specificity" and is a method used in protein N-terminomics to analyze the N-terminus of proteins. The acronym TAILS stands for "Terminal Analysis of Incomplete Lysine Specificity." The method involves using a combination of chemical labeling and mass spectrometry to identify and quantify the N-terminal peptides of proteins. In protein N-terminomics, the N-terminus of a protein is the portion of the protein that is located at the amino terminus, or the end of the protein chain. This region is important for protein function and structure, and can be modified post-translationally. TAILS is a method that allows researchers to identify and characterize the N-terminal peptides of proteins, which can provide information about protein function, modification, and localization. The TAILS method involves labeling the N-terminus of proteins with a specific chemical group, such as a fluorescent or radioactive label. The labeled proteins are then separated from other proteins in a sample using mass spectrometry, and the N-terminal peptides are identified and quantified based on their mass-to-charge ratio. Overall, TAILS is a powerful tool for analyzing the N-terminus of proteins, and can provide valuable insights into protein function and modification. |
| 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. TAILS stands for "Terminal Analysis of Incomplete Lysine Specificity." 2. TAILS is a method used in protein N-terminomics to analyze the N-terminus of proteins. 3. The N-terminus of a protein is the portion located at the amino terminus or the end of the protein chain. 4. TAILS involves using a combination of chemical labeling and mass spectrometry to identify and quantify the N-terminal peptides of proteins. 5. TAILS can provide valuable insights into protein function and modification. |
| 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 | 34(27) |
| 领域 | Bio-Medical |
| 用户问题 | Do mutations of AKT1 occur in meningiomas? |
| 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, mutations in the AKT1 gene have been identified in meningiomas. Meningiomas are benign tumors that arise from the meninges, the protective coverings of the brain and spinal cord. AKT1 is a key signaling protein that plays a critical role in cell survival and proliferation, and mutations in this gene have been found to be associated with increased tumor aggressiveness and poorer prognosis in various types of cancer, including meningiomas. Studies have shown that approximately 10-20% of meningiomas have mutations in the AKT1 gene, with the majority of these mutations occurring in the serine/threonine kinase domain of the protein. These mutations can lead to constitutive activation of the AKT1 signaling pathway, promoting cell proliferation and survival, and contributing to the development and progression of meningiomas. The specific mutations identified in AKT1 in meningiomas vary, but some of the most common include: 1. Point mutations in the ATP-binding site: These mutations result in the loss of ATP binding and inhibition of the AKT1 enzyme, leading to constitutive activation of the AKT1 signaling pathway. 2. Deletions or duplications in the kinase domain: These mutations result in the loss or gain of function of the AKT1 protein, leading to constitutive activation of the AKT1 signaling pathway. 3. Mutations in the regulatory regions of the AKT1 gene: These mutations can affect the expression and activity of the AKT1 protein, leading to changes in the AKT1 signaling pathway. The prognostic and therapeutic implications of AKT1 mutations in meningiomas are still being studied, but these mutations may be a potential target for future therapies. For example, drugs that target the AKT1 signaling pathway, such as PI3K inhibitors, have shown promise in preclinical studies as a potential treatment for meningiomas. However, further research is needed to confirm these findings and to determine the optimal therapeutic approach for meningiom |
| 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 AKT1 gene have been identified in meningiomas. 2. Approximately 10-20% of meningiomas have mutations in the AKT1 gene. 3. The majority of AKT1 mutations in meningiomas occur in the serine/threonine kinase domain of the protein. 4. Some of the common mutations in AKT1 in meningiomas include point mutations in the ATP-binding site, deletions or duplications in the kinase domain, and mutations in the regulatory regions of the gene. 5. AKT1 mutations in meningiomas are associated with increased tumor aggressiveness and poorer prognosis. |
| 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 | 35(28) |
| 领域 | Bio-Medical |
| 用户问题 | What are the main indications of lacosamide? |
| 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 | Lacosamide is an anticonvulsant medication that is used to treat partial onset seizures in adults and children with epilepsy. It is also used to treat trigeminal neuralgia, a chronic pain condition characterized by facial pain. The main indications for lacosamide are: 1. Partial onset seizures: Lacosamide is used to treat partial onset seizures in adults and children with epilepsy. 2. Trigeminal neuralgia: Lacosamide is used to treat trigeminal neuralgia, a chronic pain condition characterized by facial pain. It is important to note that lacosamide is not recommended for other types of seizures or conditions, such as generalized seizures, status epilepticus, or migraines. |
| 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. Lacosamide is an anticonvulsant medication. 2. Lacosamide is used to treat partial onset seizures in adults and children with epilepsy. 3. Lacosamide is also used to treat trigeminal neuralgia, a chronic pain condition characterized by facial pain. 4. Lacosamide is not recommended for other types of seizures or conditions, such as generalized seizures, status epilepticus, or migraines. |
| 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 | 36(29) |
| 领域 | Bio-Medical |
| 用户问题 | Which fusion protein is involved in the development of Ewing sarcoma? |
| 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 typically affects children, teenagers, and young adults. It is characterized by the presence of a specific genetic abnormality, known as a translocation t(11;22)(q24;q12), which involves the fusion of two genes, EWS and FLI1. The resulting chimeric protein, EWS-FLI1, is overexpressed in Ewing sarcoma cells and plays a crucial role in the development and progression of the 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. Ewing sarcoma is a rare and aggressive type of cancer that primarily affects children, teenagers, and young adults. 2. Ewing sarcoma is characterized by a specific genetic abnormality called a translocation t(11;22)(q24;q12). 3. The translocation involves the fusion of two genes, EWS and FLI1. 4. The fusion of EWS and FLI1 genes results in the formation of a chimeric protein called EWS-FLI1. 5. The overexpression of the EWS-FLI1 protein is observed in Ewing sarcoma cells and has a significant role in the development and progression of the 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 | 37(30) |
| 领域 | Bio-Medical |
| 用户问题 | List Hemolytic Uremic Syndrome Triad. |
| 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 | Hemolytic uremic syndrome (HUS) is a rare but potentially life-threatening disorder that is caused by the combination of hemolytic anemia, thrombocytopenia, and kidney failure. The triad of HUS is composed of the following three components: 1. Hemolytic anemia: This is the destruction of red blood cells, which can lead to anemia and a decrease in the amount of oxygen being carried to the body's tissues. 2. Thrombocytopenia: This is a decrease in the number of platelets in the blood, which can increase the risk of bleeding. 3. Kidney failure (acute kidney injury): This is the most severe and potentially life-threatening complication of HUS, as it can lead to the failure of the kidneys and the accumulation of toxins in the body. The triad of HUS is often caused by a genetic mutation in the factor H gene, which is a protein that plays a critical role in the immune system. The mutation can lead to the production of a defective form of factor H, which can cause the immune system to attack the body's own red blood cells and platelets, leading to the development of HUS. The triad of HUS can be caused by a variety of factors, including: 1. Infections: Certain infections, such as E. coli O157:H7, can cause HUS by triggering an immune response that leads to the destruction of red blood cells and platelets. 2. Medications: Certain medications, such as penicillamine, can cause HUS by triggering an immune response. 3. Genetic disorders: Certain genetic disorders, such as Alport syndrome, can increase the risk of developing HUS. 4. Other medical conditions: Certain medical conditions, such as lupus, can increase the risk of developing HUS. The triad of HUS can have a significant impact on a person's quality of life, as it can lead to anemia, fatigue, weakness, and a decreased ability to perform daily activities. In severe cases, HUS can lead to kidney failure, which can require dialysis and a kidney |
| 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. Hemolytic Uremic Syndrome (HUS) is a disorder characterized by the triad of hemolytic anemia, thrombocytopenia, and kidney failure. 2. Hemolytic anemia is the destruction of red blood cells, leading to anemia and decreased oxygen delivery to the body's tissues. 3. Thrombocytopenia is a decrease in the number of platelets in the blood, increasing the risk of bleeding. 4. Kidney failure is the most severe and life-threatening complication of HUS, resulting in kidney failure and toxin buildup in the body. 5. HUS can be caused by various factors, including infections, medications, genetic disorders, and other medical conditions. |
| 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 | 38(31) |
| 领域 | Bio-Medical |
| 用户问题 | Does physical activity influence gut hormones? |
| 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: Yes, physical activity has been shown to influence gut hormones in several ways. 1. Increased gut hormone secretion: Exercise has been shown to increase the secretion of several gut hormones, including ghrelin, cholecystokinin (CCK), and peptide YY (PYY). These hormones play important roles in regulating appetite, satiety, and glucose metabolism. 2. Modulation of gut motility: Exercise can also modulate gut motility, which can affect the absorption and secretion of gut hormones. For example, aerobic exercise has been shown to increase gut motility and reduce inflammation in the gut, which can improve insulin sensitivity and glucose metabolism. 3. Effects on the gut microbiome: Physical activity has also been shown to affect the gut microbiome, which can influence the production and secretion of gut hormones. Exercise has been shown to increase the abundance of certain beneficial bacteria in the gut, such as Akkermansia muciniphila, which has been linked to improved glucose metabolism and reduced body weight. 4. Increased gut-brain communication: Exercise has been shown to increase the communication between the gut and the brain, which can also influence gut hormone secretion. For example, exercise has been shown to increase the release of neurotransmitters such as serotonin and dopamine, which can affect appetite and satiety. Overall, the effects of physical activity on gut hormones are complex and multifaceted, and more research is needed to fully understand the mechanisms involved. However, it is clear that regular exercise can have a positive impact on gut health and hormone regulation, which can in turn influence overall health and well-being. |
| 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. Exercise has been shown to increase the secretion of gut hormones, such as ghrelin, cholecystokinin (CCK), and peptide YY (PYY). 2. Exercise can modulate gut motility, which can affect the absorption and secretion of gut hormones. 3. Physical activity can affect the gut microbiome by increasing the abundance of certain beneficial bacteria like Akkermansia muciniphila. 4. Exercise increases the communication between the gut and the brain, leading to the release of neurotransmitters like serotonin and dopamine. 5. Regular exercise can have a positive impact on gut health and hormone regulation, influencing overall health and well-being. |
| 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 | 39(32) |
| 领域 | Bio-Medical |
| 用户问题 | What are the effects of depleting protein km23-1 (DYNLRB1) in a cell? |
| 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 | Depleting protein km23-1 (DYNLRB1) in a cell can have a variety of effects, including: 1. Disruption of endoplasmic reticulum (ER) function: Km23-1 is an ER-resident protein that plays a critical role in regulating ER structure and function. Depletion of Km23-1 can lead to ER stress and disruption of ER-associated degradation (ERAD) pathways, which can result in accumulation of misfolded proteins and impaired protein quality control. 2. Alterations in protein synthesis and folding: Km23-1 is involved in the folding and transport of secretory and membrane proteins from the ER to the Golgi apparatus. Depletion of Km23-1 can impair the proper folding and transport of these proteins, leading to their accumulation in the ER and impaired cellular function. 3. Changes in cellular signaling pathways: Km23-1 has been shown to interact with a variety of signaling proteins, including the mTOR complex, and play a role in regulating cellular signaling pathways. Depletion of Km23-1 can disrupt these pathways, leading to changes in cellular behavior and function. 4. Increased sensitivity to oxidative stress: Km23-1 has been shown to play a role in protecting cells against oxidative stress by regulating the activity of antioxidant enzymes. Depletion of Km23-1 can increase cellular sensitivity to oxidative stress, leading to DNA damage and impaired cellular function. 5. Impaired cellular migration and invasion: Km23-1 has been shown to regulate cellular migration and invasion by controlling the activity of proteins involved in these processes. Depletion of Km23-1 can impair cellular migration and invasion, leading to changes in tissue architecture and impaired cellular function. 6. Changes in cellular metabolism: Km23-1 has been shown to regulate cellular metabolism by controlling the activity of enzymes involved in glycolysis and gluconeogenesis. Depletion |
| 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. Depleting protein km23-1 (DYNLRB1) in a cell can disrupt endoplasmic reticulum (ER) function. 2. Depletion of Km23-1 can lead to ER stress and disruption of ER-associated degradation (ERAD) pathways. 3. Km23-1 is involved in the folding and transport of secretory and membrane proteins from the ER to the Golgi apparatus. 4. Depletion of Km23-1 can impair the proper folding and transport of these proteins. 5. Km23-1 has been shown to interact with a variety of signaling proteins, including the mTOR complex. 6. ote: The original answer provided six statements, but only five independent statements could be extracted) |
| 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 | 40(33) |
| 领域 | Bio-Medical |
| 用户问题 | Treatment of which disease was investigated in the MR CLEAN study? |
| 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. Alzheimer's disease B. Parkinson's disease C. Chronic obstructive pulmonary disease (COPD) D. Major depressive disorder (MDD) Correct answer: A. Alzheimer's disease. The MR CLEAN study (MRI Cognition and Lifestyle in Alzheimer's Disease) was a research study that investigated the use of magnetic resonance imaging (MRI) to identify early changes in the brain associated with Alzheimer's disease. The study aimed to determine whether MRI can detect changes in the brain up to 10 years before clinical symptoms of Alzheimer's disease appear, and to investigate the relationship between these changes and lifestyle factors such as physical activity, cognitive stimulation, and APOE genotype. The study did not investigate any other 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. The MR CLEAN study investigated the use of magnetic resonance imaging (MRI) to identify early changes in the brain associated with Alzheimer's disease. 2. The study aimed to determine whether MRI can detect changes in the brain up to 10 years before clinical symptoms of Alzheimer's disease appear. 3. The MR CLEAN study investigated the relationship between changes in the brain and lifestyle factors such as physical activity, cognitive stimulation, and APOE genotype. 4. The MR CLEAN study did not investigate Parkinson's disease, Chronic obstructive pulmonary disease (COPD), or Major depressive disorder (MDD). |
| 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 | 41(34) |
| 领域 | Bio-Medical |
| 用户问题 | Which factors activate zygotic gene expression during the maternal-to-zygotic transition in zebrafish? |
| 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 maternal-to-zygotic transition (MZT) is a critical process in the development of most vertebrates, including zebrafish. During the MZT, the embryo transitions from a state of dependency on the maternal environment to one of self-sufficiency, as the zygote begins to express its own genetic program. While the mechanisms underlying the MZT are not fully understood, several factors have been identified that contribute to its activation. 1. Nuclear migration: During the MZT, the nucleus of the zygote migrates from the periphery of the embryo to its center, where it begins to express its own genetic program. This process is mediated by the actin cytoskeleton and the Rho-associated protein kinase (ROCK). 2. Transcription factors: Transcription factors are proteins that regulate gene expression by binding to specific DNA sequences. During the MZT, several transcription factors are activated, including T-box proteins, GATA factors, and Nanos. These factors play a crucial role in regulating the expression of genes involved in the MZT and the development of the embryo. 3. Hormonal signals: Hormones, such as activin and inhibin, play a crucial role in regulating the MZT. These hormones signal through their receptors to activate signaling pathways that promote the transition from the maternal to the zygotic genetic program. 4. Epigenetic modifications: Epigenetic modifications, such as DNA methylation and histone modification, play a crucial role in regulating the expression of genes involved in the MZT. These modifications can be inherited from the mother or generated during the MZT itself, and they are essential for the proper activation of zygotic gene expression. 5. Meiosis: Meiosis is the process by which the diploid zygote becomes haploid. During the MZT, meiosis is initiated, and the zygote begins to divide mitotically to form the embryo. Meiosis is essential for the proper activation of zygotic gene expression, as it allows the zygote to segregate its genetic material and establish the proper gene dosage. In |
| 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. During the maternal-to-zygotic transition (MZT) in zebrafish, the nucleus of the zygote migrates from the periphery of the embryo to its center. 2. Transcription factors, including T-box proteins, GATA factors, and Nanos, are activated during the MZT in zebrafish. 3. Hormones like activin and inhibin play a role in regulating the MZT in zebrafish. 4. Epigenetic modifications, such as DNA methylation and histone modification, are involved in regulating gene expression during the MZT in zebrafish. 5. Meiosis is initiated during the MZT in zebrafish, allowing the zygote to divide mitotically and form the embryo. |
| 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 | 43(35) |
| 领域 | Bio-Medical |
| 用户问题 | What is evaluated using the EORTC QLQ – INFO25 questionnaire? |
| 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 EORTC QLQ – INFO25 questionnaire is used to evaluate the quality of life (QoL) of patients with cancer. It assesses 25 different aspects of QoL, including: 1. Physical functioning 2. Role functioning 3. Cognitive functioning 4. Emotional functioning 5. Social functioning 6. Personal relationships 7. Global health status 8. Financial difficulties 9. Employment status 10. Education level 11. Living situation 12. Marital status 13. Sexual functioning 14. Pain 15. Fatigue 16. Nausea and vomiting 17. Dyspnea 18. Insomnia 19. Appetite loss 20. Financial difficulties 21. Transportation difficulties 22. Housing difficulties 23. Legal problems 24. Perceived health 25. Global QoL Each item is rated on a 7-point Likert scale, with higher scores indicating better QoL. The EORTC QLQ – INFO25 questionnaire is widely used in clinical trials and research studies to evaluate the impact of cancer and its treatment on patients’ QoL. |
| 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 EORTC QLQ – INFO25 questionnaire is used to evaluate the quality of life (QoL) of patients with cancer. 2. The questionnaire assesses 25 different aspects of QoL. 3. Physical functioning is one of the aspects evaluated by the questionnaire. 4. Role functioning is one of the aspects evaluated by the questionnaire. 5. Cognitive functioning is one of the aspects evaluated by the questionnaire. |
| 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 | 44(36) |
| 领域 | Bio-Medical |
| 用户问题 | Does BNP increase after intensive exercise in athletes? |
| 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, BNP (B-type natriuretic peptide) levels can increase after intensive exercise in athletes. BNP is a hormone produced by the heart in response to stress, and exercise can cause an increase in BNP levels as the heart works harder to meet the increased metabolic demands of the body. Studies have shown that BNP levels can increase significantly after intense exercise in athletes, particularly in endurance athletes who engage in activities such as distance running, cycling, or swimming. For example, one study found that BNP levels increased by an average of 27% after a 30-minute all-out cycling test in trained cyclists. The increase in BNP levels after exercise is thought to be related to the increased cardiac output and myocardial oxygen demand during intense exercise. The heart must work harder to pump blood to the muscles and deliver oxygen and nutrients to meet the increased metabolic demands of the body, leading to an increase in BNP production. While an increase in BNP levels after exercise is generally considered normal and harmless, it can be a useful marker for monitoring athletes' cardiovascular health and detecting potential cardiovascular problems. However, it is important to interpret BNP levels in the context of other markers of cardiovascular health and to consult with a healthcare professional to determine the appropriate course of action. |
| 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. BNP (B-type natriuretic peptide) levels can increase after intensive exercise in athletes. 2. BNP is a hormone produced by the heart in response to stress. 3. Studies have shown that BNP levels can increase significantly after intense exercise, particularly in endurance athletes. 4. One study found that BNP levels increased by an average of 27% after a 30-minute all-out cycling test in trained cyclists. 5. The increase in BNP levels after exercise is related to increased cardiac output and myocardial oxygen demand during intense exercise. |
| 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 | 46(37) |
| 领域 | Bio-Medical |
| 用户问题 | Are there web based self management strategies for chronic 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 | Aug 18, 2018 | Chronic Pain, Health, Self Management, Technology Aug 18, 2018 | Chronic Pain, Health, Self Management, Technology Chronic pain affects millions of people worldwide, and it can be challenging to manage. Fortunately, there are several web-based self-management strategies that can help individuals with chronic pain manage their symptoms and improve their quality of life. Here are some of the most effective web-based self-management strategies for chronic pain: 1. Online Pain Management Programs: Many hospitals and healthcare organizations offer online pain management programs that provide a structured approach to managing chronic pain. These programs typically include educational resources, exercise programs, and cognitive-behavioral therapy. 2. Pain Diaries: Keeping a pain diary can help individuals track their pain levels, identify patterns, and monitor the effectiveness of treatments. There are many online tools available that allow individuals to log their pain levels, track their medication use, and set goals. 3. Mindfulness and Meditation: Mindfulness and meditation are two powerful tools for managing chronic pain. There are many online resources available that provide guided meditations and mindfulness exercises that can be practiced from the comfort of home. 4. Online Support Groups: Joining an online support group can provide individuals with chronic pain with a sense of community and connection with others who are going through similar experiences. These groups can be a valuable source of emotional support and practical advice. 5. Telemedicine: Telemedicine allows individuals with chronic pain to connect with healthcare providers remotely. This can be especially helpful for individuals who live in rural areas or have difficulty traveling to appointments. 6. Mobile Apps: There are many mobile apps available that provide a range of tools for managing chronic pain, including pain tracking, medication reminders, and guided relaxation exercises. 7. Online Education: There are many online resources available that provide educational information about chronic pain, including its causes, treatment options, and self-management strategies. 8. Cognitive Behavioral Therapy (CBT): CBT is a type of therapy that can help individuals with chronic pain manage their sympt |
| 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. Online pain management programs are offered by many hospitals and healthcare organizations. 2. Keeping a pain diary can help individuals track their pain levels, identify patterns, and monitor the effectiveness of treatments. 3. Mindfulness and meditation can be used as tools for managing chronic pain and there are online resources available for practicing them. 4. Online support groups can provide individuals with chronic pain a sense of community and emotional support. 5. Telemedicine allows individuals with chronic pain to connect with healthcare providers remotely. |
| 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 | 47(38) |
| 领域 | Bio-Medical |
| 用户问题 | Is Weaver syndrome similar to Sotos? |
| 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 | Weaver syndrome and Sotos syndrome are both rare genetic disorders that affect the brain and nervous system. While they share some similarities, they are distinct conditions with different genetic causes and clinical presentations. Here are some key differences and similarities between the two conditions: Similarities: 1. Both Weaver and Sotos syndrome are caused by mutations in genes involved in brain development and function. 2. Both disorders are characterized by intellectual disability, developmental delays, and seizures. 3. Both conditions can lead to problems with speech, language, and social interactions. 4. Both disorders can result in motor skill delays and coordination problems. 5. Both conditions can cause vision problems, including strabismus (crossed eyes) and cataracts. Differences: 1. Genetic cause: Weaver syndrome is caused by mutations in the SLC16A2 gene, while Sotos syndrome is caused by mutations in the USP9X gene. 2. Age of onset: Weaver syndrome typically becomes apparent in the first year of life, while Sotos syndrome can be diagnosed at any age but is usually apparent by the second year of life. 3. Severity: Sotos syndrome is generally more severe than Weaver syndrome, with more pronounced intellectual disability and developmental delays. 4. Growth and development: Children with Sotos syndrome tend to be shorter and have slower growth rates than those with Weaver syndrome. 5. Facial features: While both conditions can cause distinctive facial features, such as a small head, narrow eyes, and a flat nose bridge, the facial features of Sotos syndrome are generally more pronounced than those of Weaver syndrome. 6. Brain structure: Sotos syndrome is associated with enlargement of the brain, particularly in the frontal and parietal lobes, while Weaver syndrome is not typically associated with brain abnormalities. 7. Other features: Sotos syndrome can also cause heart defects, feeding difficulties, and a higher risk of infections, while Weaver syndrome may be associated with hearing loss, kidney problems, and a higher risk of seizures. In summary, while Weaver |
| 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. Both Weaver syndrome and Sotos syndrome are rare genetic disorders that affect the brain and nervous system. 2. Both disorders are caused by mutations in genes involved in brain development and function. 3. Both Weaver syndrome and Sotos syndrome are characterized by intellectual disability, developmental delays, and seizures. 4. Both conditions can result in problems with speech, language, social interactions, motor skills, and vision. 5. Weaver syndrome is caused by mutations in the SLC16A2 gene, while Sotos syndrome is caused by mutations in the USP9X gene. 6. Weaver syndrome typically becomes apparent in the first year of life, while Sotos syndrome can be diagnosed at any age but is usually apparent by the second year of life. 7. Sotos syndrome is generally more severe than Weaver syndrome, with more pronounced intellectual disability and developmental delays. 8. Children with Sotos syndrome tend to be shorter and have slower growth rates than those with Weaver syndrome. 9. The facial features of Sotos syndrome are generally more pronounced than those of Weaver syndrome. 10. . Sotos syndrome is associated with enlargement of the brain, particularly in the frontal and parietal lobes. 11. . Weaver syndrome may be associated with hearing loss, kidney problems, and a higher risk of seizures. |
| 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 | 49(39) |
| 领域 | Bio-Medical |
| 用户问题 | Are ultraconserved elements often transcribed? |
| 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 | Ultraconserved elements (UCEs) are regions of DNA that are highly conserved across different species. While the degree of conservation can vary, UCEs are generally more conserved than other regions of the genome. However, the degree of transcriptional activity of UCEs can vary. Some UCEs are not transcribed at all, while others are highly transcribed. There are several reasons why UCEs may be transcribed at different levels: 1. Functional importance: UCEs are often associated with functional elements such as promoters, enhancers, or silencers. These elements can influence the transcription of nearby genes, and their conservation may reflect their functional importance. 2. Evolutionary history: UCEs may have been conserved due to their evolutionary history. For example, some UCEs may have been inherited from a common ancestor, and their conservation may reflect their functional importance in that ancestor. 3. Regulatory elements: UCEs can act as regulatory elements, controlling the expression of nearby genes. The degree of transcriptional activity of these elements can vary, depending on the specific context. 4. Cis-regulatory elements: UCEs can also act as cis-regulatory elements, controlling the expression of distant genes. The degree of transcriptional activity of these elements can vary, depending on the specific context. 5. Chromatin structure: The chromatin structure of UCEs can also influence their transcriptional activity. For example, UCEs that are located in regions of open chromatin may be more accessible to the RNA polymerase complex and therefore more transcribed. 6. Epigenetic modifications: Epigenetic modifications, such as DNA methylation or histone modifications, can also influence the transcriptional activity of UCEs. For example, UCEs that are located in regions of hypomethylated DNA may be more transcribed than those located in regions of hypermethylated DNA. 7. Non-coding RNA: Some UCEs may be transcribed into non-coding RNA molecules, such as microRNAs or long non-coding RNAs. These molecules can regulate gene expression by binding to messenger RNA (mRNA) molecules or by acting as decoys |
| 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. Ultraconserved elements (UCEs) are regions of DNA that are highly conserved across different species. 2. UCEs are generally more conserved than other regions of the genome. 3. Some UCEs are not transcribed at all, while others are highly transcribed. 4. UCEs are often associated with functional elements such as promoters, enhancers, or silencers. 5. UCEs may have been conserved due to their evolutionary history. |
| 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 | 50(40) |
| 领域 | Bio-Medical |
| 用户问题 | What is the methyl donor of DNA (cytosine-5)-methyltransferases? |
| 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 methyl donor of DNA (cytosine-5)-methyltransferases is S-adenosylmethionine (SAM). DNA (cytosine-5)-methyltransferases are enzymes that add a methyl group to the cytosine residue of CpG dinucleotides in DNA. This process is known as 5-methylcytosine (5-mC) modification and plays a crucial role in the regulation of gene expression, particularly in the silencing of gene transcription. SAM is the methyl donor for these enzymes, providing the methyl group that is transferred to the cytosine residue in the DNA molecule. The transfer of the methyl group from SAM to cytosine is facilitated by a covalent complex between the enzyme and the DNA, and the reaction is reversible, allowing the methylation state of the DNA to be dynamically regulated. |
| 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. DNA (cytosine-5)-methyltransferases add a methyl group to the cytosine residue of CpG dinucleotides in DNA. 2. S-adenosylmethionine (SAM) is the methyl donor for DNA (cytosine-5)-methyltransferases. 3. The methyl group from SAM is transferred to the cytosine residue in the DNA molecule. 4. The transfer of the methyl group from SAM to cytosine is facilitated by a covalent complex between the enzyme and the DNA. 5. The reaction of adding a methyl group to cytosine in DNA is reversible, allowing the methylation state of the DNA to be dynamically regulated. |
| 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 | 52(41) |
| 领域 | Bio-Medical |
| 用户问题 | Which signaling pathway does sonidegib inhibit? |
| 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 | Sonidegib is a small molecule inhibitor of the smoothened (SMO) signaling pathway. SMO is a G-protein-coupled receptor that is activated by the hedgehog (Hh) signaling pathway. When activated, SMO triggers a cascade of downstream signaling events that regulate various cellular processes, including cell proliferation, differentiation, and survival. Sonidegib binds to SMO and prevents it from activating the downstream signaling pathway, leading to the inhibition of Hh-dependent cellular processes. |
| 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. Sonidegib is a small molecule inhibitor. 2. Sonidegib specifically inhibits the smoothened (SMO) signaling pathway. 3. SMO is a G-protein-coupled receptor that is activated by the hedgehog (Hh) signaling pathway. 4. The activation of SMO triggers downstream signaling events that regulate cellular processes such as cell proliferation, differentiation, and survival. 5. Sonidegib binds to SMO and prevents its activation, leading to the inhibition of Hh-dependent cellular processes. |
| 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 | 53(42) |
| 领域 | Bio-Medical |
| 用户问题 | In which phase of the cell cycle arrest is impaired in Fanconi anemia? |
| 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) G1 phase B) G2 phase C) M phase D) S phase Correct answer: B) G2 phase Explanation: Fanconi anemia is a rare genetic disorder caused by mutations in any of the 16 FANCA, FANCB, FANCD1, FANCD2, FANDE, FANDF, FANDI, or FANDR genes. These genes are involved in the repair of DNA interstrand cross-links (ICLs) and other DNA damage. Impaired DNA repair in Fanconi anemia leads to genetic instability and an increased risk of cancer. One of the key features of Fanconi anemia is impaired cell cycle arrest in G2 phase, which can result in the release of unreplicated DNA into the cytoplasm, leading to apoptosis or the formation of micronuclei. Therefore, the answer is B) G2 phase. |
| 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. Fanconi anemia is a genetic disorder caused by mutations in certain genes. 2. The genes involved in Fanconi anemia are FANCA, FANCB, FANCD1, FANCD2, FANDE, FANDF, FANDI, and FANDR. 3. Impaired DNA repair in Fanconi anemia can lead to genetic instability and an increased risk of cancer. 4. One of the key features of Fanconi anemia is impaired cell cycle arrest in the G2 phase. 5. Impaired cell cycle arrest in the G2 phase of Fanconi anemia can result in the release of unreplicated DNA into the cytoplasm, leading to apoptosis or the formation of micronuclei. |
| 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 | 54(43) |
| 领域 | Bio-Medical |
| 用户问题 | Which DNA sequences are more prone for the formation of R-loops? |
| 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 | R-loops are a type of non-canonical DNA structure that forms when a DNA molecule is wrapped around itself in a circular manner, creating a loop. R-loops are formed through the interaction of two complementary DNA strands, and they play important roles in regulating gene expression, DNA replication, and repair. Several factors can influence the formation of R-loops, including the sequence composition of the DNA molecule. In general, R-loops are more likely to form in regions of the DNA molecule that have a high GC content, as the GC bases are more prone to hydrogen bonding and therefore more likely to form stable interactions with each other. Additionally, R-loops are more likely to form in regions of the DNA molecule that have a high degree of secondary structure, such as stem-loops or G-quartets. Some specific DNA sequences that are known to be more prone to R-loop formation include: 1. Rich in GC bases: Regions of the DNA molecule that are rich in GC bases (such as G-C rich sequences) are more likely to form R-loops due to the increased stability of GC base pairs. 2. High secondary structure: Regions of the DNA molecule with high levels of secondary structure, such as stem-loops or G-quartets, are more likely to form R-loops. 3. A-T rich regions: Regions of the DNA molecule that are rich in A-T bases (such as A-T rich sequences) are also more likely to form R-loops. 4. Repeats: Regions of the DNA molecule with repetitive sequences, such as microsatellites or minisatellites, are more likely to form R-loops due to the increased stability of the repeat sequences. 5. Chromatin-associated regions: Regions of the DNA molecule that are associated with chromatin, such as heterochromatin or euchromatin, are more likely to form R-loops due to the higher degree of compaction and higher levels of DNA-binding proteins in these regions. 6. Regions with high DNA supercoiling: Regions of the DNA molecule with high levels of DNA supercoiling are more likely to form R-loops due to the |
| 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. R-loops are a type of non-canonical DNA structure that forms when a DNA molecule is wrapped around itself in a circular manner, creating a loop. 2. R-loops are formed through the interaction of two complementary DNA strands. 3. R-loops play important roles in regulating gene expression, DNA replication, and repair. 4. R-loops are more likely to form in regions of the DNA molecule that have a high GC content. 5. R-loops are more likely to form in regions of the DNA molecule that have a high degree of secondary structure, such as stem-loops or G-quartets. |
| 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 | 55(44) |
| 领域 | Bio-Medical |
| 用户问题 | Mutation of which gene is implicated in the familial isolated pituitary adenoma? |
| 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. PIT1 B. AIP C. HESX1 D. PROP1 Correct answer: B. AIP. Familial isolated pituitary adenoma (FIPA) is a rare genetic disorder characterized by the development of pituitary adenomas in the absence of other endocrine tumors or syndromes. Mutations in the AIP (Adenoma-Associated Protein) gene are implicated in the development of FIPA. The AIP gene is located on chromosome 17q23 and encodes a protein that plays a critical role in the regulation of pituitary cell proliferation and differentiation. Mutations in the AIP gene have been identified in approximately 70% of families with FIPA. |
| 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 AIP (Adenoma-Associated Protein) gene are implicated in the development of familial isolated pituitary adenoma (FIPA). 2. The AIP gene is located on chromosome 17q23. 3. The AIP gene encodes a protein that regulates pituitary cell proliferation and differentiation. 4. Mutations in the AIP gene have been identified in approximately 70% of families with FIPA. |
| 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 | 56(45) |
| 领域 | Bio-Medical |
| 用户问题 | which mutations of troponin C gene have been found to cause hypertrophic cardiomyopathy? |
| 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 genetic disorder that affects the heart muscle and is caused by mutations in several genes, including the troponin C gene (TNNC1). Mutations in the TNNC1 gene can lead to an increase in the thickness of the heart muscle (hypertrophy), which can obstruct the flow of blood through the heart and lead to symptoms such as shortness of breath, fatigue, and chest pain. Several specific mutations in the TNNC1 gene have been identified as causing HCM. These include: 1. R145G: This mutation substitutes an arginine residue for a glycine residue at position 145 of the TNNC1 protein. This mutation has been found to be a common cause of HCM and can lead to abnormal calcium handling and contractile function in the heart muscle. 2. R92Q: This mutation substitutes a glutamine residue for an arginine residue at position 92 of the TNNC1 protein. This mutation has been associated with HCM and can lead to impaired calcium handling and contractile function in the heart muscle. 3. D143E: This mutation substitutes a glutamic acid residue for an aspartic acid residue at position 143 of the TNNC1 protein. This mutation has been found to cause HCM and can lead to abnormal calcium handling and contractile function in the heart muscle. 4. D185E: This mutation substitutes a glutamic acid residue for an aspartic acid residue at position 185 of the TNNC1 protein. This mutation has been associated with HCM and can lead to impaired calcium handling and contractile function in the heart muscle. 5. H146Y: This mutation substitutes a tyrosine residue for a histidine residue at position 146 of the TNNC1 protein. This mutation has been found to cause HCM and can lead to abnormal calcium handling and contractile function in the heart muscle. 6. Y158C: |
| 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 genetic disorder that affects the heart muscle. 2. HCM is caused by mutations in several genes, including the troponin C gene (TNNC1). 3. Specific mutations in the TNNC1 gene have been identified as causing HCM, including R145G, R92Q, D143E, D185E, and H146Y. 4. The R145G mutation substitutes an arginine residue for a glycine residue at position 145 of the TNNC1 protein. 5. The R92Q mutation substitutes a glutamine residue for an arginine residue at position 92 of the TNNC1 protein. |
| 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 | 57(46) |
| 领域 | Bio-Medical |
| 用户问题 | What is known about the effect of acupuncture in smoking cessation ? |
| 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 | Acupuncture is a form of traditional Chinese medicine that involves the insertion of thin needles into specific points on the body to stimulate the body's natural healing processes. While the evidence for acupuncture's effectiveness in smoking cessation is mixed, some studies suggest that it may be a useful adjunctive treatment for smokers who are trying to quit. One study published in the Journal of the American Medical Association found that acupuncture was associated with a significantly higher quit rate among smokers compared to those who received standard smoking cessation treatment. Another study published in the Journal of Acupuncture and Meridian Studies found that acupuncture was associated with a significant reduction in withdrawal symptoms and cravings among smokers. The exact mechanisms by which acupuncture may help smokers quit are not fully understood, but it is thought that it may help to reduce stress and anxiety, increase feelings of relaxation and well-being, and alter brain chemistry in ways that make it easier to resist the urge to smoke. Additionally, acupuncture may help to reduce the physical symptoms of nicotine withdrawal, such as irritability, headaches, and nausea, which can make it easier for smokers to stick to their quit plan. It is important to note that acupuncture is not a magic cure for smoking cessation, and it is unlikely to work for everyone. However, it may be a useful addition to other quit smoking strategies, such as nicotine replacement therapy, counseling, and medication. If you are interested in trying acupuncture to help you quit smoking, it is important to find a qualified acupuncturist who has experience treating smokers and to discuss the potential risks and benefits with your healthcare provider. |
| 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. Acupuncture is a form of traditional Chinese medicine that involves the insertion of thin needles into specific points on the body. 2. Some studies suggest that acupuncture may be a useful adjunctive treatment for smokers trying to quit. 3. One study published in the Journal of the American Medical Association found that acupuncture was associated with a significantly higher quit rate among smokers. 4. Another study published in the Journal of Acupuncture and Meridian Studies found that acupuncture was associated with a significant reduction in withdrawal symptoms and cravings among smokers. 5. Acupuncture may help to reduce stress and anxiety, increase feelings of relaxation and well-being, and alter brain chemistry in ways that make it easier to resist the urge to smoke. |
| 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 | 58(47) |
| 领域 | Bio-Medical |
| 用户问题 | Which post-translational histone modifications are characteristic of constitutive heterochromatin? |
| 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 | Constitutive heterochromatin is a type of chromatin that is characterized by the presence of specific post-translational histone modifications. These modifications include: 1. Tri-methylation of lysine 9 on histone H3 (H3K9me3): This modification is a hallmark of constitutive heterochromatin and is associated with the silencing of gene expression. 2. Di-methylation of lysine 27 on histone H3 (H3K27me2): This modification is also a characteristic of constitutive heterochromatin and is involved in the silencing of gene expression. 3. Mono-methylation of lysine 4 on histone H3 (H3K4me1): This modification is found in constitutive heterochromatin and is associated with the activation of gene expression. 4. Acetylation of histone H3 and H4: These modifications are found in constitutive heterochromatin and are involved in the regulation of gene expression. 5. Methylation of histone H3 at lysine 79 (H3K79me): This modification is found in constitutive heterochromatin and is involved in the regulation of gene expression. 6. Methylation of histone H4 at lysine 20 (H4K20me): This modification is found in constitutive heterochromatin and is involved in the regulation of gene expression. These modifications are found in constitutive heterochromatin and play a crucial role in regulating gene expression and chromatin structure. |
| 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. Tri-methylation of lysine 9 on histone H3 (H3K9me3) is a post-translational histone modification characteristic of constitutive heterochromatin. 2. Di-methylation of lysine 27 on histone H3 (H3K27me2) is a post-translational histone modification characteristic of constitutive heterochromatin. 3. Mono-methylation of lysine 4 on histone H3 (H3K4me1) is a post-translational histone modification found in constitutive heterochromatin. 4. Acetylation of histone H3 and H4 is a post-translational histone modification found in constitutive heterochromatin. 5. Methylation of histone H3 at lysine 79 (H3K79me) is a post-translational histone modification found in constitutive heterochromatin. |
| 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 | 59(48) |
| 领域 | Bio-Medical |
| 用户问题 | GV1001 vaccine targets which enzyme? |
| 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 | GV1001 is a vaccine that targets the enzyme non-structural protein 5 (NS5). NS5 is a key component of the hepatitis C virus (HCV) and plays a critical role in the replication of the virus. GV1001 is an investigational vaccine that is being developed to prevent HCV infection and to treat chronic HCV infection. |
| 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. GV1001 is a vaccine. 2. GV1001 targets the enzyme non-structural protein 5 (NS5). 3. NS5 is a key component of the hepatitis C virus (HCV). 4. NS5 plays a critical role in the replication of the HCV. 5. GV1001 is being developed to prevent HCV infection and to treat chronic HCV infection. |
| 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 | 62(49) |
| 领域 | Bio-Medical |
| 用户问题 | Is pregnancy an additional risk during during H1N1 infection? |
| 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 | Pregnant women are at higher risk for severe illness and complications from H1N1 influenza, including pneumonia and hospitalization. Pregnant women are more susceptible to H1N1 infection and are at higher risk for severe illness and complications from H1N1 influenza, including pneumonia and hospitalization, according to the Centers for Disease Control and Prevention (CDC). The CDC advises pregnant women to take precautions to protect themselves and their unborn babies from H1N1 infection, such as: Getting vaccinated against H1N1: Pregnant women should get vaccinated against H1N1 as soon as the vaccine becomes available. Practicing good hygiene: Pregnant women should wash their hands frequently with soap and water, especially after coughing or sneezing, and before touching their faces or mouths. Avoiding close contact with people who are sick: Pregnant women should avoid close contact with people who are sick with H1N1, especially if they have severe symptoms. Staying home when sick: Pregnant women who are sick with H1N1 should stay home from work or school to avoid spreading the virus to others. Seeking medical attention: Pregnant women who are sick with H1N1 should seek medical attention right away, especially if they have severe symptoms or if they are experiencing difficulty breathing. It is important to note that H1N1 infection during pregnancy can also lead to preterm labor and low birth weight, which can increase the risk of health problems for the baby. Therefore, pregnant women should consult their healthcare provider for proper evaluation and management of H1N1 infection. |
| 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. Pregnant women are at higher risk for severe illness and complications from H1N1 influenza, including pneumonia and hospitalization. 2. The Centers for Disease Control and Prevention (CDC) advises pregnant women to get vaccinated against H1N1 as soon as the vaccine becomes available. 3. Pregnant women should wash their hands frequently with soap and water, especially after coughing or sneezing, and before touching their faces or mouths. 4. Pregnant women should avoid close contact with people who are sick with H1N1, especially if they have severe symptoms. 5. Pregnant women who are sick with H1N1 should stay home from work or school to avoid spreading the virus to others. |
| 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 |  |