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Short Communication

Authentication of Protein Tyrosine Phosphatases on Cell Signaling and Disease

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DESCRIPTION

This action counteracts the activity of protein tyrosine kinases and thereby regulates a wide range of cellular processes including growth, differentiation, and metabolism. Genetic variants in protein tyrosine phosphatases genes have been connected to various hereditary disorders and susceptibilities to diseases highlighting their importance in human health [1-3].

Role of protein tyrosine phosphatases

PTPs are classified into two major families such as receptor like PTPs and non-receptor PTPs. Receptor-like PTPs are membrane-bound and typically act in signaling pathways related to cell-cell interactions and cell adhesion. Non-receptor PTPs, on the other hand, are cytoplasmic and are involved in intracellular signaling pathways that control cell division, survival, and migration. By dephosphorylating tyrosine residues, these enzymes counterbalance the activity of protein tyrosine kinases, which add phosphate groups to tyrosines. This balance is essential for proper cellular function and organismal development [4-6].

Hereditary disorders linked to PTP variants

Mutations and variants in PTP genes can lead to a range of hereditary disorders. These genetic alterations often result in either the loss of PTP function or its aberrant activation, disrupting normal cellular signaling.

Autoimmune disorders: One well-known example is the association between *PTPN22* gene variants and autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus. *PTPN22* encodes the lymphoid tyrosine phosphatase (LYP), which regulates immune cell activation. Variants in this gene can lead to excessive immune activation and subsequent autoimmunity.

Diabetes mellitus: Variants in the *PTPN 11* gene, which encodes the SHP-2 phosphatase, are associated with both Type 1 and Type 2 diabetes mellitus. Mutations in *PTPN 11* can lead to abnormal insulin signaling, which affects glucose metabolism and contributes to the development of diabetes.

Cancer: Aberrant PTP activity is also implicated in various cancers. PTPRK normally acts as a tumour suppressor by dephosphorylating signaling molecules involved in cell proliferation.

Developmental disorders: Genetic variants in the *PTPRD* gene, encoding the receptor-type PTPδ, are linked to developmental disorders such as neurodevelopmental disorders and schizophrenia. *PTPRD* is involved in neuronal development and synaptic function, and its disruption can lead to cognitive and behavioral abnormalities [7-9].

Disease susceptibilities and PTP variants

Beyond specific hereditary disorders, PTP variants can influence susceptibility to a variety of diseases. These variants can affect disease progression, severity, and response to treatment.

Cardiovascular diseases: Variants in *PTPN6*, which encodes the SHP-1 phosphatase, have been linked to cardiovascular diseases. Genetic variants can modulate an individual's risk for heart disease by influencing these pathways.

Neurodegenerative diseases: PTP variants are also implicated in neurodegenerative conditions such as Alzheimer's disease. For instance, *PTPN12*, which encodes the *PTP12* phosphatase, has been associated with altered tau phosphorylation, a key feature of Alzheimer's pathology. Variants in this gene may affect tau phosphorylation and contribute to neurodegeneration.

Infectious diseases: Emerging evidence suggests that PTP variants can influence susceptibility to infectious diseases. For example, certain *PTPN2* variants have been linked to increased susceptibility to tuberculosis. This may be due to their role in modulating immune responses to pathogens.

Metabolic disorders: Variants in PTP genes can also affect susceptibility to metabolic disorders such as obesity and hyperlipidemia variants in *PTP1B* can alter insulin sensitivity and glucose homeostasis.

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Understanding the role of PTP variants in disease susceptibility has significant therapeutic implications. Targeting specific PTPs with small molecules or biologics holds promise for developing novel treatments for diseases associated with PTP dysfunction. For example, *PTP1B* inhibitors are being explored as potential therapies for Type 2 diabetes and obesity. Similarly, targeted therapies for cancers involving aberrant PTP activity are under investigation. Additionally, genetic screening for PTP variants can provide valuable information for personalized medicine approaches. By identifying individuals at higher risk for certain disorders based on their PTP genetic profiles, healthcare providers can modify prevention and treatment strategies to improve patient outcomes. Protein tyrosine phosphatases are critical regulators of cellular signaling, and their genetic variants are linked to a diverse array of hereditary disorders and disease susceptibilities. From autoimmune diseases to cancer, the impact of PTP variants on health is profound. Continued research into the role of PTPs in disease mechanisms and the development of targeted therapies holds the potential to transform the management of these conditions. As our understanding of PTP biology deepens, it determination cover the method for novel diagnostic and therapeutic strategies aimed at improving human health and combating disease [10].

REFERENCES

1. Muntean AG, Hess JL. Epigenetic dysregulation in cancer. Am J Pathol. 2009;175(4):1353-1361.
2. He C. Grand challenge commentary: RNA epigenetics? Nat Chem Biol. 2010;6(12):863-865.
3. He L, Li H, Wu A, Peng Y, Shu G, Yin G. Functions of N6-methyladenosine and its role in cancer. Mol Cancer Res. 2019;18(1):1-5.
4. Roignant JY, Soller M. M6A in mRNA: An ancient mechanism for fine-tuning gene expression. Trends Genet. 2017;33(6):380-390.
5. Engel M, Eggert C, Kaplick PM, Eder M, Röh S, Tietze L, et al. The role of M6A/m-RNA methylation in stress response regulation. Neuron. 2018;99(2):389-403.
6. Beh LY, Debelouchina GT, Clay DM, Thompson RE, Lindblad KA, Hutton ER, et al. Identification of a DNA N6-adenine methyltransferase complex and its impact on chromatin organization. Cell. 2019;177(7):1781-1796.
7. Ji L, Chen S, Gu L, Zhang X. Exploration of potential roles of M6A regulators in colorectal cancer prognosis. Front Oncol. 2020;10:768.
8. Aboukarima A, Zayed M, Minyawi M, Elsoury H, Tarabye H. Image analysis-based system for estimating cotton leaf area. Asian Res J Agric. 2017;5(1):1-8.
9. Ali Z, Maryam H, Saddique MA, Ikram RM. Exploiting genetic diversity in enhancing phenotypic plasticity to develop climate-resilient cotton. Genet Resour Crop Evol. 2023;70(5):1305-1320.
10. Boquet DJ, Clawson EL. Cotton planting date: yield, seedling survival, and plant growth. J Agron. 2009;101(5):1123-1130.