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A Complex Relationship between Virus Infection and Host Metabolism

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Abstract

To replicate efficiently inside the host, the virus can control or exploit a number of metabolic pathways; however, which route is regulated relies on the conditions that are ideal for the virus's replication or its favorable effect. Viruses not only rely on certain metabolic pathways, but they also alter them when they invade. The virus imposes its own metabolic programme on the host cell, interfering with its metabolic regulation. When a virus infects a cell, it often modifies the host cell's metabolic machinery to meet the bioenergetic and biosynthetic demands of reproducing. The current article focuses on the major metabolic pathways that viruses can affect and change.

Keywords: Virus;, Glucose; Glutaminolysis; Lipid

Introduction

Viruses may infect a wide variety of organisms and have a vast array of genetic material, making them the most abundant and pervasive living entities on Earth. Viruses are able to reproduce and avoid the immune system due to their diverse modes of development. One of these methods involves interfering with essential metabolic pathways (e.g., targeting metabolic master regulator proteins, glucose, lipid, etc..) and nodes in the host organism in order to influence its metabolism [1,2]. Organisms rely on metabolic signalling pathways for essential decision-making and cellular signalling synchronisation, which includes gene transcription and accurate regulation of these pathways. Because of this, viruses have adapted to affect metabolism by targeting specific pathways. Various organisations have investigated and discovered ways that viruses use to elevate cellular levels of macromolecules like glucose and fatty acids for replication throughout the last 30 years [1,2]. This review article examines the modification of metabolic pathways by many kinds of viruses capable of infecting humans or other specific animal species. This article focuses on the studies that have identified the precise method by which viruses affect metabolic signalling. However, it does not include any descriptive studies. Effectively understanding and describing these altered communication channels has the capacity to serve as the crucial factor in the management of various viral diseases.

Alterations to Glucose Metabolism in Cells Infected with Viruses

Glycolysis is a glucose metabolic pathway that takes place in the cytoplasm of the cell and produces energy. The reaction pathway consists of ten steps that turn a single glucose molecule into two pyruvate molecules through a series of intermediary reactions. When oxygen is present, pyruvate enters the tricarboxylic acid (TCA) cycle and undergoes complete oxidation to carbon dioxide (CO2). Under anaerobic conditions, pyruvate is subjected to

homolactic fermentation, resulting in the production of lactic acid [1-3]. Viruses affect the process of glucose metabolism in order to enhance the amount of energy available and facilitate their own replication by adjusting the signalling pathways discussed earlier. Viruses typically achieve this by triggering aerobic glycolysis, which is also referred to as the Warburg effect. The Warburg effect refers to the metabolic phenomenon where pyruvate is converted into lactate utilising lactate dehydrogenase (LDH) at the end of glycolysis, even in the presence of oxygen. Therefore, it regulates the main controlling enzymes of aerobic glycolysis, including glucose transporters (GLUTs), hexokinase (HK), phosphofructokinase (PFK), pyruvate kinase, and LDH. Aerobic glycolysis leads to an accumulation of lactic acid, a decrease in glycolytic intermediates that contribute to the TCA cycle, and a high rate of glucose consumption [1,3-5].

Alterations to Glutaminolysis Process in Cells Infected with Viruses

Glutaminolysis is the conversion of glutamine into intermediates in the TCA cycle when pyruvate is not present. After glutaminase (GLS) converts glutamine into glutamate, glutamate dehydrogenase (GDH) converts glutamate into α-ketoglutarate. This process restores the TCA cycle and enables the generation of additional intermediates, such citrate, which aid in the synthesis of fatty acids. The production of NADPH by the TCA cycle, which transfers electrons to the ETC, means that glutamine can also function as an alternate energy source. Glycolysis is dependent on glutamine because viral life cycles generate aerobic glycolysis, which is disconnected from the TCA cycle [1,6].

Alterations to Lipid Metabolism in Cells Infected with Viruses

As a typical side effect of viral infection, cells often undergo aerobic glycolysis (also known as the Warburg effect) and a shift in lipid metabolism, typically from fatty acid oxidation (FAO) to fatty acid synthesis (FAS). Encased viruses require an increase in FAS even more than other viruses. Infection with certain viruses can downregulate metabolic events, and it can only infect cells that are already actively involved in metabolism. The HIV virus causes this condition because it infects cells more effectively when they are in an active metabolic state. It follows that HIV infects T cells when their metabolic activity is at its peak, which includes increased aerobic glycolysis, tricarboxylic acid cycle activity, oxidative phosphorylation, and glutaminolysis [1,7].

Conclusion

Numerous viral strategies for exploiting the host cell metabolic network have emerged across a wide range of viral types, including DNA and RNA viruses, as well as oncogenic and non-oncogenic viruses. An integral part of viral replication and survival is the viral modulation of the host metabolism. To further our knowledge of viral pathogenesis and pave the way for the development of new antiviral treatments, we need a deeper comprehension of the viral processes that target host metabolism. Further research is required to fill in the gaps in our understanding of the signaling pathways that viruses modify and how they impact the host.

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