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A review of the literature on viral infections and the development of chronic metabolic diseases

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ABSTRACT

Introduction: Viruses often trigger chronic conditions such as diabetes, obesity, and NAFLD by disrupting metabolism. The present review examines recent evidence on how viral pathogens alter the body's energy processing mechanisms and the subsequent impact on host metabolism. *Results*: These changes help the virus to replicate and disrupt metabolic finding a balance can be good for your health, but it can also cause health problems. It is clear that viruses such SARS-CoV-2 and hepatitis C show how glucose (sugar) and fat can be processed by the body. It should be regulated in the same way as other things. Viral infections like HIV can make the keep the immune system active for a healthy state. Concurrently, treatment-related adverse effects may also be present. Evidently, there is a bidirectional relationship between viral infection and metabolic disease. This means that people with pre-existing metabolic conditions are more at risk of severe illness from viral infections, and these illnesses can be more aggressive due to underlying metabolic problems. Understanding the immune and metabolic systems is vital for understanding chronic metabolic diseases. Early recognition and treatment are pivotal. This could have favourable outcomes.

Keywords: Viral infections, Metabolic diseases; Metabolic reprogramming; Insulin resistance; Chronic inflammation; SARS-CoV-2; Hepatitis C virus; Immune dysregulation; Viral mimicry, Glucose metabolism

1. INTRODUCTION

Viral infections have always been a major problem for human health. These factors have the capacity to influence the development of society and the outcomes of medical treatment on a global scale (Perakakis et al., 2023). Viruses can cause more than tissue damage. Furthermore, they have the capacity to modify the metabolic processes within the body. Viruses such as those responsible for diseases like the flu and coronaviruses, and chronic infections like hepatitis and HIV, are just a few examples. Evidence suggests a growing interest in viral infections and chronic metabolic diseases. People with metabolic conditions are more likely to contract infectious diseases and suffer more severely from them (Palmer, 2022).

In recent decades, the number of people worldwide who have these diseases, which are not passed from person to person, has increased a lot. These diseases include obesity, type 2 diabetes and liver disease caused by fat. The diseases we talked about earlier have been shown to disrupt metabolic equilibrium. This has been identified as a significant contributing factor to both poor health outcomes and mortality, frequently resulting in complications related to heart and kidney function (Perakakis et al., 2023). It is clear that viral infections can cause or make worse metabolic conditions. At the same time, people with pre-existing metabolic issues are more likely to get very sick from a virus. The current findings show a two-way relationship (Perakakis et al., 2023; Palmer, 2022).

The fact that NAFLD is a common liver problem that is linked to obesity and T2DM shows this rising epidemic. It is estimated that NAFLD affects between 23% and 25% of people worldwide, with up to 30% to 32% of people in some regions, such as the Middle East and South America, affected. Trends in urbanisation, lifestyle changes and diet mean that the number of people with NAFLD is expected to rise a lot. In some regions, it is predicted that more than 55% of people will have NAFLD by 2040. This problem is made worse by other health problems, such as insulin resistance, high cholesterol and high blood pressure. These can all make liver inflammation, scarring and heart disease worse.

T2DM, which is closely linked to insulin resistance and excess fat, has also increased around the world as obesity rates have risen. The number of adults with diabetes around the world has almost doubled in the last few decades. It is also becoming more common among younger people, mainly because they lead sedentary lifestyles and eat a lot of high-calorie food. NAFLD is much more common in people with T2DM, with almost 37% showing the more severe non-alcoholic steatohepatitis (NASH) form and 17% displaying advanced fibrosis, which increases the risk of cirrhosis and liver-related death.

Viral infections may also contribute to metabolic dysregulation, making this problem worse. Viruses can make insulin resistance worse, change how your body processes fats, and cause inflammation, which might speed up metabolic disease (Perakakis et al., 2023). Pre-existing metabolic abnormalities impair antiviral immune defenses and escalate the severity of infections such asCOVID-19, influenza, and hepatitis viral diseases, highlighting the bidirectional impact (Palmer, 2022). We urgently need to find ways to manage metabolic health and the risk of infectious disease.

Regional disparities exist for metabolic diseases. Rich countries see the biggest rises in people with NAFLD and diabetes due to urbanisation and changes in behaviour. In low-income regions, underdiagnosis and lack of access to healthcare lead to an underestimation of the global picture. We need to develop public health strategies taking into account social and economic issues. These strategies should raise awareness of chronic metabolic diseases and control infections to prevent the health and financial problems they cause.

The reasons for this are complex and include changes to the body caused by the virus, communication between the immune system and metabolism, molecular mimicry, and ongoing inflammation (Girdhar et al., 2021; Palmer, 2022). Viruses change how their host cells use glucose, lipids and amino acids to make copies of themselves, often causing a change in how those cells use glucose called the Warburg effect (Girdhar et al., 2021). These changes to how the body uses energy help the virus to survive, but they can also cause problems with how the body controls blood sugar, which can lead to problems with the cells that make insulin (Girdhar et al., 2021; Perakakis et al., 2023).

It is important to note that some viruses have been found to be linked to certain metabolic diseases. There is a well-known link between certain viruses and type 1 diabetes (T1D). These viruses can damage the cells in the pancreas that make insulin (beta cells) and can cause the body to attack itself (autoimmune reactions). These viruses are very common and most people catch them when they are young. Studies of outbreaks show a link between the timing of enterovirus infection and the development of islet autoantibodies (P7, P9). In simple terms, enteroviruses like coxsackievirus B (especially types B1 and B4) infect the cells in the pancreas that produce insulin (pancreatic beta cells). These viruses cause the cells to die in a number of ways, which leads to a reaction in the immune system that causes the body to attack the cells. The virus can stay in the pancreatic tissue, which can lead to ongoing swelling and the slowdown of the cells that make insulin (P10). However, the development of T1D only affects a certain group of infected people. This suggests that there are many things that can cause it, including a person's genes and when they got infected (P9).

The hepatitis C virus (HCV) can lead to insulin resistance and type 2 diabetes (T2DM), as well as liver fat accumulation. HCV stops liver cells from using glucose properly and makes it hard for insulin to work, by breaking down some of the proteins involved. The virus also causes problems with the mitochondria (the cell's energy production units) and increases oxidative stress (the damage caused by reactive oxygen species), which makes insulin resistance worse. Genotype 3 of the hepatitis C virus (HCV) is particularly linked to liver fat, as the virus changes how the body processes fat to make more fat and stops the body from using fat for energy (P11). If you have both diabetes and hepatitis C, you are more likely to develop liver fibrosis and cancer. Importantly, if you take direct-acting

antivirals to treat the hepatitis C, it can improve how your body uses insulin, which suggests that the effect on your metabolism from the hepatitis C may be reversible (P12).

HIV infection and its treatment can cause big changes to the body's fat levels and how it processes fat. This can lead to metabolic syndrome and non-alcoholic fatty liver disease (NAFLD). HIV damages the cells that control fat in the body and causes long-lasting swelling throughout the body, which makes it hard for the body to use insulin and affects the levels of different fats in the blood. Antiretroviral therapy (ART), especially protease inhibitors, makes these metabolic effects worse by interfering with insulin signals, promoting fat redistribution that causes lipodystrophy, and increasing fat accumulation in the liver. HIV-infected fat tissue also helps the virus to survive, which causes ongoing inflammation and further metabolic problems. We can treat these problems by changing the way the body processes energy (P13).

The recent SARS-CoV-2 pandemic has shown how viral infections and metabolic disorders can be connected. People with pre-existing metabolic diseases such as obesity, T2DM, and NAFLD are much more likely to have a severe case of the disease and to die if they catch it. In addition, people who have had SARS-CoV-2 infection are more likely to develop new diabetes, problems controlling blood sugar and damage to the cells in the pancreas that produce insulin. The virus infects cells in the pancreas that regulate hormone production and changes how the body uses energy. It does this by causing inflammation in fat tissue, creating more of the hormone glucagon in the liver, and reducing insulin production. These changes to the body's energy production may continue even after the initial infection has gone, which can lead to long-term symptoms. The mechanisms are complex and involve direct viral effects, systemic inflammation, cytokine dysregulation, and modulation of metabolic signaling pathways such as phosphoinositide 3-kinase/Akt (PI3K/Akt) and HIF-1alpha (P14, P15).

All of these examples show that viruses can cause problems with the body's normal functions in different ways. These include directly infecting parts of the body that control metabolism, changing how the host's body uses energy, causing long-term immune system problems that lead to the body's tissues becoming inflamed, and the virus copying the body's own hormones. What's more, metabolic diseases have an effect on how the body responds to infection and how bad the infection can become. This creates a connection that goes both ways, affecting how bad the disease is and how it affects the body in the long term. Learning about how viruses and the body work together could lead to new ways to treat viral infections and metabolic diseases.

The immune system is vital for the body's metabolism. The immune system fights infections, which hinders insulin usage and digestion (Palmer, 2022). Metabolic diseases stop the immune system from working properly. This causes changes in the activity of macrophages, a type of white blood cell. It can also impair T-cell responses and lead to a stronger, more challenging reaction to viruses. This can lead to increased inflammation and lower energy levels due to the presence of the virus (Perakakis et al., 2023; Palmer, 2022).

What's more, scientists have recently discovered that viruses can now copy insulin and other proteins that help growth. This is a new way for viruses to infect and spread people, and it involves changing the signals that tell the body how to process energy. Viral insulin-like peptides (VILPs) may change how the body uses glucose, how cells grow and die. This could be a link between viral infections and the development of metabolic diseases and cancer (Girdhar et al., 2021).

There are many issues to consider, so it's really important to do as much research as we can into how viruses change our metabolism, which parts of our health are affected, and how it could be use, for example treatments that target how our bodies process energy to make people better when they have a viral infection or another long-term health problem related to their metabolism (Perakakis et al., 2023). This review looks at how the body processes glucose (sugar) and lipids (fats), the connection between the immune system (our body's defense system) and metabolism (the process by which our body uses food to produce energy), how viruses can mimic hormones made by the body, and what this means for diseases like diabetes and NAFLD (non-alcoholic fatty liver disease). This is important, as it helps to find ways to deal with the growing number of metabolic disorders and the growing number of viruses around the world.

2. REVIEW METHODS

This review was done in a step-by-step way to collect current scientific information about viral infections and their role in the development of long-term metabolic diseases. There were searched many important academic databases, like PubMed, Scopus, Web of Science, and Google Scholar, were searched to find relevant articles that other scientists had checked. The search looked for original research studies, reviews of research studies, and reviews of other reviews. It also looked for studies on how viruses cause diseases, how the body changes when it has a disease, and how viruses are linked to other diseases.

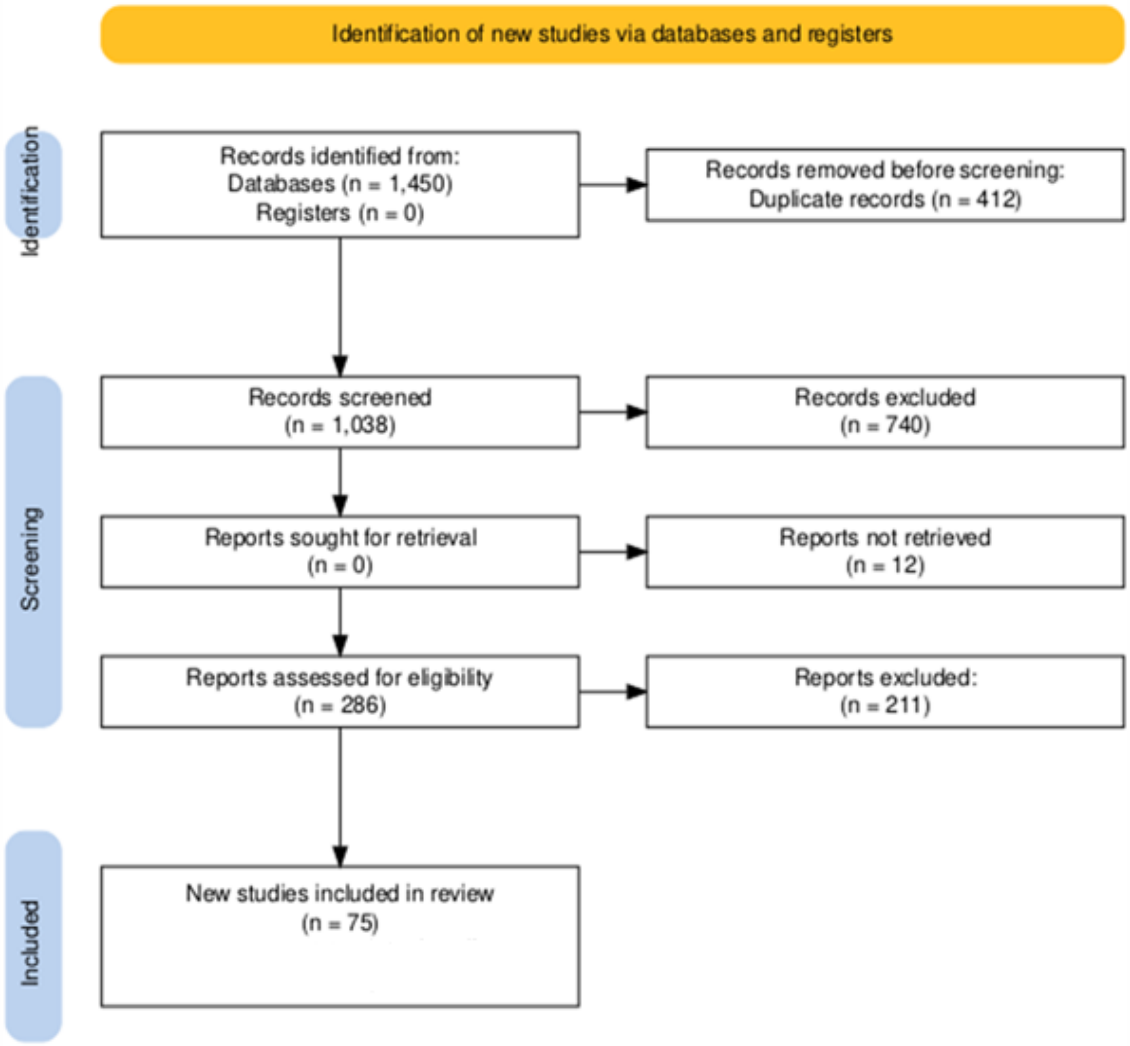

Figure 1. PRISMA flow diagram.

Search Strategy

The search strategy used keywords and combination of terms such as "viral infections," "metabolic reprogramming," "chronic metabolic diseases," "type 1 diabetes," "type 2 diabetes," "insulin resistance," "hepatitis C virus," "HIV," and "SARS-CoV-2." The searches included articles published from January 2015 to October 2025, targeting the most recent and scientifically robust findings to ensure relevance. The studies included addressed the molecular, cellular, and systemic mechanisms by which viruses impact host metabolism, immune-metabolic interactions, and clinical outcomes in metabolic disorders.

The selection criteria were clear: studies investigating the metabolic consequences of viral infections in human subjects or relevant in vivo/in vitro models were to be prioritised. Clinical studies used observational cohort, case-control, and interventional designs to analyse metabolic dysfunction influenced by viral infections. We included preclinical studies that elucidated pathophysiological mechanisms underlying virus-induced metabolic changes. This provides a comprehensive mechanistic context.

Exclusion Criteria:

Studies were excluded if they lacked clear diagnostic or methodological rigor, were not written in English and thus could not be translated, consisted of case reports or series with fewer than five subjects, or did not directly address the relationship between viral infections and metabolic dysfunction. Abstracts without full-text availability were also excluded from the study.

Screening:

After searching the database, about 1,450 records were found. After carefully checking the results, 412 copies that were the same were deleted. Two experienced reviewers then looked at the rest of the 1,038 abstracts on their own to see how relevant they were to chronic metabolic diseases and the effects of viral infections. After looking at lots of research, we chose 286 articles to study more closely. We only chose articles that met our set rules for what to include and what to exclude. After a thorough review, 75 studies were selected for the final summary. These studies were chosen based on how scientific they were, how relevant they were, and the complex connection between viral infections and metabolic diseases (Figure 1).

3. RESULTS

Research from 2018 to 2025 definitively shows that viral infections can influence how our bodies manage energy. However, it is important to understand that these changes can lead to serious long-term metabolic problems (Powell & Gehring, 2023). Viral infections can cause a number of health problems. These include type 1 and type 2 diabetes, obesity, and nonalcoholic fatty liver disease (NAFLD) (Wang et al., 2023; Smith et al., 2020).

The findings definitively show that viruses can influence their host's metabolism. This will undoubtedly have a positive effect on processes such as glycolysis – also known as the Warburg effect – as well as glutaminolysis and lipid synthesis. It is clear that these alterations support the virus's growth and help it to evade the immune system (Palmer, 2022; Darweesh et al., 2025). For example, adenoviruses have been observed to trigger Myc-driven glycolysis, while Hepatitis C virus (HCV) has been seen to interfere with lipid metabolism using SREBP, and Cytomegalovirus (CMV) has been observed to enhance fatty acid production.

Infectious diseases are a big cause of type 1 diabetes (T1D), with enteroviral infections being a main trigger. It is clear that they can directly damage the cells in the pancreas that produce insulin (β-cells). It is clear that this will have a negative effect on the immune system. The body attacks itself. How often this happens depends on the health of the person concerned (Smith et al., 2020). For people with HCV, it can be hard for the body to use insulin well. This causes fat to build up in the liver due to problems with how the body uses insulin and how its energy cells work. This probably leads to type 2 diabetes (Darweesh et al., 2025).

The fact that people can get infected with SARS-CoV-2 shows that there is a link between a virus and how our bodies process food. Infected cells can cause problems with the immune system. This is a key factor in the development of new cases of diabetes and the worsening of existing metabolic diseases (Powell & Gehring, 2023). The virus can cause long-term symptoms that affect the body's metabolism. This can lead to ongoing health problems.

Table 1. Key viral factors influencing the development of chronic metabolic diseases

Factor	Association with Metabolic Disease	Key Evidence Sources
Enteroviruses (e.g., Coxsackieviruses)	Initiation of type 1 diabetes via β-cell destruction and autoimmunity	Perakakis et al., 2023; Strand et al., 2024
Hepatitis C virus (HCV)	Promotes insulin resistance, type 2 diabetes, hepatic steatosis	Perakakis et al., 2023; Powell & Gehring, 2023
Human Immunodeficiency Virus (HIV)	Dysregulated lipid metabolism, adipose inflammation, metabolic syndrome	smith et al., 2020
SARS-CoV-2 (COVID-19)	Predisposes to new-onset diabetes, worsens existing metabolic disorders	Powell & Gehring, 2023; Perakakis et al., 2023
Viral mimicry of host insulin/IGF	Disruption of host metabolic signaling and glucose homeostasis	Perakakis et al., 2023; Girdhar et al., 2021

The results clearly show that metabolic disorders and viral diseases are related. Sometimes, problems with how the body makes energy can make diseases caused by viruses worse. Viruses can also disrupt the body's natural processes, which can lead to long-term

health problems (Darweesh et al., 2025). This complicated connection needs a combination of ways to check for, and treat, the disease. These ways need to focus on how the virus affects the body's energy production. This is the best way to help the people affected.

As summarized in Table 1 how changes to the body's normal processes can play a key role in how viruses cause disease and how the body's immune system responds. This offers new ways to treat the disease (Darweesh et al., 2025). Viral infections use glucose metabolism and other metabolic pathways, such as lipid synthesis and glutaminolysis, to replicate more quickly, evade immune detection, and survive. This makes these pathways good targets for antiviral therapy.

The most important research being done looks at stopping glycolysis using glucose copies like 2-deoxy-D-glucose (2-DG). This stops the virus making copies of itself by taking over the glucose and turning it into energy. Testing on animals has shown that it works against the flu, SARS-CoV-2 and herpes simplex virus. It has been found to reduce the amount of virus in the body and stop the immune system from getting too excited (Darweesh et al., 2025). In the same way, lactate dehydrogenase (LDH) inhibitors stop viruses from getting energy and have been shown to be effective against dengue and cytomegaloviruses.

AMP-activated protein kinase (AMPK) is a clinically relevant strategy to restore metabolic balance by promoting oxidative phosphorylation and reducing inflammation. Metformin, a drug prescribed for diabetes, has been found to help patients with both diabetes and the common cold. These are achieved by strengthening the immune system and increasing energy production in the body (Darweesh et al., 2025). Novel AMPK agonists like AICAR also exhibit antiviral characteristics by changing how cells work and making immune cells stronger.

Other treatments target the substances that cause inflammation, which are involved in the metabolic problems caused by the virus. Monoclonal antibodies against interleukin-6 (tocilizumab) and tumor necrosis factor (infliximab) are being studied to control severe viral infections, such as SARS-CoV-2 and dengue fever. However, these antibodies must be used carefully to make sure that the immune system still works well against viruses.

New treatments include sodium-glucose cotransporter 2 (SGLT2) inhibitors, which reduce glucose and inflammation in the body. These have shown some promise in treating viral infections. Also, we may need to use a combination of different medicines that target several parts of the virus's life cycle, because viruses are able to adapt to them.

Using a patient's genes, metabolism, and immune system to create a personalised medicine plan could help make antiviral therapies much more effective. Advances in metabolic phenotyping (the study of how the body processes energy) and CRISPR-based metabolic engineering (the use of a special enzyme to make very precise changes to genetic material) may further enable precise targeting of pathways that are not working well, enhancing how well treatments work while minimising side effects.

In general, trying to change the way a person's body processes energy is a new and exciting way to develop antiviral medicines. This could help to better manage viral infections and reduce long-term health problems caused by the infection. Researchers should focus on studies that look at how metabolic modulators can be used with conventional antiviral treatments.

4. DISCUSSION

This review definitively shows that viral infections significantly influence the onset of chronic metabolic diseases. It shows how we are learning more and more about how viruses, the body's food metabolism, and immune responses work together. Research has proven that viruses can alter how infected individuals utilise energy. Glucose, lipids and the mitochondria are all included. Mitochondria are the components of cells that generate energy. This energy manipulation helps viruses to reproduce, and it stops the body from regulating itself properly. That can lead to conditions like diabetes (types 1 and 2), metabolic syndrome, which is a group of conditions that make it hard for the body to use energy properly, and non-alcoholic fatty liver disease (NAFLD) (Darweesh et al., 2025).

There is a complicated connection between the body and the disease. Problems with how the body makes energy are part of these long-term diseases. They make it hard for the immune system to fight off viruses. This makes people more likely to get infections and causes more severe disease (Powell & Gehring, 2023; Strand et al., 2024). However, viral infections can make metabolic problems worse in a number of ways. These include direct damage to cells, the virus staying in metabolic tissues for a long time, long-lasting inflammation, and problems with the immune system. All of these things can make it hard for the body to use insulin properly, affect how lipids (fats) are processed, and cause problems with how organs work. Viral persistence can also lead to low-grade inflammation, which can cause tissue damage and metabolic problems.

Research is now being done into how viruses can copy the hormones made by the body, especially insulin. These copies are very similar to the hormones they copy. These viral peptides, found in members of the Iridoviridae family and seen in human samples, can attach to human insulin receptors (IR) and IGF-1 receptors (IGF1R), activating pathways like PI3K/Akt, which control how cells take in

glucose and grow. Some VILPs work as 'producers' to increase the uptake of glucose and the growth of cells. Others work as 'blockers' to stop the effects of IGF1R. This is called molecular mimicry. It helps viruses trick the body into using glucose and lipids in the wrong way. This makes it easier for the virus to grow and spread. But this can disrupt the normal balance of chemicals in the body, which can lead to metabolic diseases. Structural studies reveal that VILPs attach to receptors in a different way compared to normal ligands. This includes the ability to stabilise receptor conformations that prevent normal activation. These viral strategies are an example of how viruses can change to avoid being fought off by the body and change how the body works. This shows that we can learn more about diseases caused by viruses and how we can use drugs to target these viral peptides (Girdhar et al., 2021).

The way in which the body's metabolism and immune system work together is very important. Viruses can change how a cell's genes work, affecting how the cell responds to inflammation and viruses. For example, when the body's metabolism is not working well, it can make it hard for T cells and macrophages to do their job properly. This can lead to a situation where the body can't get rid of viruses and there is ongoing inflammation, which makes metabolic disease worse (Darweesh et al., 2025). This causes a cycle where problems with the immune system and metabolism make each other worse.

The respiratory and hepatotropic viruses show different but similar changes to how their cells use energy. For example, the virus makes the body produce more glycolysis and fat, which helps it spread and causes problems with the immune system. This makes the symptoms of the disease worse (Powell & Gehring, 2023).

The hepatitis C virus (HCV) can also disrupt how the body processes insulin and fat, which can lead to insulin resistance and liver fat accumulation. Basically, HCV messes with the insulin receptor by causing it to become unstable and break down. This affects the way the insulin receptor works, which is part of a process called the PI3K/Akt pathway. The virus also increases the activity of certain enzymes in the liver that help make glucose, which then makes more glucose available. HCV also reduces the amount of glucose transporters (GLUT-2 and GLUT-4) in the body. This makes it harder for the body to take in glucose and leads to a lack of insulin in the blood. HCV makes the body produce more fat by increasing the activity of SREBP-1c and fatty acid synthase, while reducing the body's ability to export fat by decreasing microsomal triglyceride transfer protein activity. These changes lead to fat building up in the liver, which is a key symptom of a long-term HCV infection. Some of these problems with the body's normal functions can be made better (Bose and Ray, 2014; Alaei et al., 2008).

People with ongoing infections, like HIV, have a longer-lasting immune reaction which makes their metabolism worse. This is made worse by antiretroviral therapy (ART). HIV infection makes immune cells produce more of the things that help the virus grow. However, it can also lead to inflammation throughout the body and problems with fat tissue. Antiretroviral therapy (ART) drugs, especially protease inhibitors, can make insulin resistance worse by affecting how glucose (sugar) is transported in the body and causing a condition called lipodystrophy. This can lead to a higher risk of heart disease and a cycle where the immune sistema is weak and metabolic diseases are present. In other words, this shows how viral infections and the body's metabolic health are connected (Darweesh et al., 2025).

While we have more knowledge about how viruses impact the body's metabolism, we are not yet fully aware of why the metabolism changes from short-term problems caused by infection to long-term metabolic diseases. We don't fully understand how these changes happen over time, which makes it hard to predict how things will get worse or better. The symptoms of the disease vary a lot from patient to patient for a number of reasons. A person's genes, environment and the presence of other health problems all play a part. This includes being very fat and having problems with your hormones (Perakakis et al., 2023; Palmer, 2022; Darweesh et al., 2025). People's immune systems can differ greatly due to variations in their genes and the way they process food. They also differ in how much of a virus they can fight off. This means that the virus can spread more quickly, and the body's immune system and response to the infection are weaker. It is clear that these things can lead to different results for patients (Girdhar et al., 2021). Nevertheless, it is still difficult to turn these findings into reliable ways to predict outcomes and develop personalised treatments.

By examining how patients' bodies change over time, doctors can more accurately identify at-risk patients and develop effective treatment plans. New treatments that target the body's metabolism, as we discussed, can stop viruses from multiplying and reduce the metabolic problems they cause. These methods must be thoroughly tested in the clinic to ensure they are both effective and safe. Metabolic pathways are key to how the body fights infection and keeps tissues healthy.

The current literature makes a compelling case that viral infections are both drivers and amplifiers of metabolic dysregulation, setting the stage for chronic disease. A holistic research and clinical approach embracing the immunometabolic interface, personalised medicine and multidimensional treatment strategies is paramount to effectively confronting the intertwined epidemics of viral infections and metabolic diseases.

5. CONCLUSION

This review definitively shows the complex interplay between viral infections and the development of chronic metabolic diseases. Viral -induced metabolic reprogramming can clearly disrupt cellular and systemic homeostasis and lead to conditions such as diabetes, obesity, and non-alcoholic fatty liver disease. It is clear that the relationship is bidirectional: pre-existing metabolic disturbances impair antiviral immune responses, thereby increasing susceptibility to infection and disease severity. On the other hand, viral infections exacerbate metabolic dysfunction through direct tissue damage, inflammatory pathways and immune modulation.

Respiratory viruses such as SARS-CoV-2 and hepatotropic viruses such as hepatitis C clearly demonstrate distinct yet similar mechanisms that disrupt metabolism. Effective management requires integrated approaches targeting both viral replication and host metabolic pathways. Early diagnosis, personalised treatment, and multidisciplinary care are the keys to mitigating the disease burden and improving outcomes. Future research must focus on longitudinal studies and mechanistic insights to fully elucidate these complex interactions and develop targeted therapies.

In conclusion, it is very important to recognise and address the metabolic consequences of viral infections. This will help to manage the growing number of chronic metabolic diseases and improve patient health worldwide. We need to continue to advance our understanding of molecules and develop new treatments to break the cycle of infection and metabolic disorders.

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Michał Grabek- Conceptualization, review and editing, investigation, methodology
Maja Kondratowicz- Methodology, investigation, visualization, supervision
Kamila Kałamarz- Conceptualization, visualization, resources
Kinga Żmuda- Review, data curation, investigation
Maciej Świerczyna- Resources, writing- rough preparation, data curation
Maja Czerniachowska- Visualization, data curation, investigation
Marcin Kaniewski- Review, visualization, formal analis
Martyna Wojnowska- Supervision, writing- rough preparation, data curation
Wiktoria Polkowska- Review and editing, formal analis, supervision
Aleksandra Figzał- Resources, writing- rough preparation, formal analis
Project administration- Michał Grabek

Informed consent

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Ethical approval

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Conflict of interest

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Data and materials availability

All data associated with this study will be available based on reasonable request to the corresponding author.

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