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Role of the rs1143634 polymorphism in *IL-1β* gene in viral diseases

Papel do polimorfismo rs1143634 no gene *IL-1β* em doenças virais

Papel del polimorfismo rs1143634 en el GEN *IL-1β* en enfermedades virales

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ABSTRACT

Objective: Investigate the relationship between the SNP rs1143634 and viral diseases based on scientific literature, in order to understand the role of this SNP in the context of infection and identify possible risk factors. **Methods:** A scoping review was conducted by analyzing articles from the National Center for Biotechnology Information (NCBI) and databases such as PubMed, Virtual Health Library (VHL), and Web of Science. Search terms included "IL-1β," "Polymorphism," "Single Nucleotide Polymorphism," and "Viral Diseases. **Results:** Initially, 272 studies were found in different databases. However, 266 were excluded for not meeting the inclusion criteria. Thus, six studies were included in the second phase and analyzed in depth, which related the rs1143634 SNP to Hepatitis B (HBV, n=1), Hepatitis C (HCV, n=2), Cytomegalovirus (CMV, n=1), and HIV (n=2). The results showed that the rs1143634 SNP plays an important role in the inflammatory response and the risk of infection for various viral diseases. **Final considerations:** Although limited, studies suggest an association between the rs1143634 SNP and infections by HCMV, HBV, HCV, and HIV. Further research is needed to clarify its role in viral diseases.

Keywords: rs1143634, *IL-1β*, Single nucleotide polymorphism, Viral diseases.

RESUMO

Objetivo: Investigar a relação entre o SNP rs1143634 e doenças virais com base na literatura científica, a fim de compreender o papel deste SNP no contexto da infecção e identificar possíveis fatores de risco. **Métodos:** Foi realizada uma revisão de escopo em bases de dados como PubMed, Biblioteca Virtual em Saúde (BVS) e Web of Science. Foram utilizados termos como "*IL-1β*," "Polimorfismo" e

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"Doenças Virais." **Resultados:** Inicialmente, foram encontrados 272 estudos em diferentes bases de dados. No entanto, 266 foram excluídos por não atenderem aos critérios de inclusão. Dessa forma, seis estudos foram incluídos na segunda fase e analisados em profundidade que relacionavam o SNP rs1143634 com Hepatite B (HBV, n=1), Hepatite C (HCV, n=2), Citomegalovírus (CMV, n=1) e HIV (n=2). Os resultados evidenciaram que o SNP rs1143634 desempenha um papel importante na resposta inflamatória e no risco de infecção por diversas doenças virais. **Considerações finais:** Apesar do número limitado de estudos, há evidências de uma associação entre o SNP rs1143634 e infecções por HCMV, HBV, HCV e HIV. Pesquisas adicionais são necessárias para esclarecer o papel desse polimorfismo nas doenças virais.

Palavras-chave: rs1143634, *IL-1β*, Polimorfismo de nucleotídeo único, Doenças virais.

RESUMEN

Objetivo: Investigar la relación entre el SNP rs1143634 y las enfermedades virales con base en la literatura científica, para comprender su papel en la infección e identificar posibles factores de riesgo. **Método:** Se realizó una revisión de alcance mediante el análisis de artículos del *National Center for Biotechnology Information* (NCBI) y bases de datos como PubMed, la Biblioteca Virtual en Salud (BVS) y *Web of Science*. Se usaron los términos de búsqueda: "IL-1β", "polimorfismo", "polimorfismo de nucleótido único" y "enfermedades virales". **Resultados:** Se encontraron 272 estudios, pero 266 fueron excluidos por no cumplir los criterios de inclusión. Seis estudios fueron analizados en profundidad, asociando el SNP rs1143634 con Hepatitis B (HBV, n=1), Hepatitis C (HCV, n=2), Citomegalovirus (CMV, n=1) y VIH (n=2). Los hallazgos indican que el SNP rs1143634 influye en la respuesta inflamatoria y el riesgo de infección en estas enfermedades. **Consideraciones finales:** Aunque los estudios son limitados, sugieren una asociación entre el SNP rs1143634 y HCMV, HBV, HCV y VIH. Se requiere más investigación para esclarecer su papel en enfermedades virales.

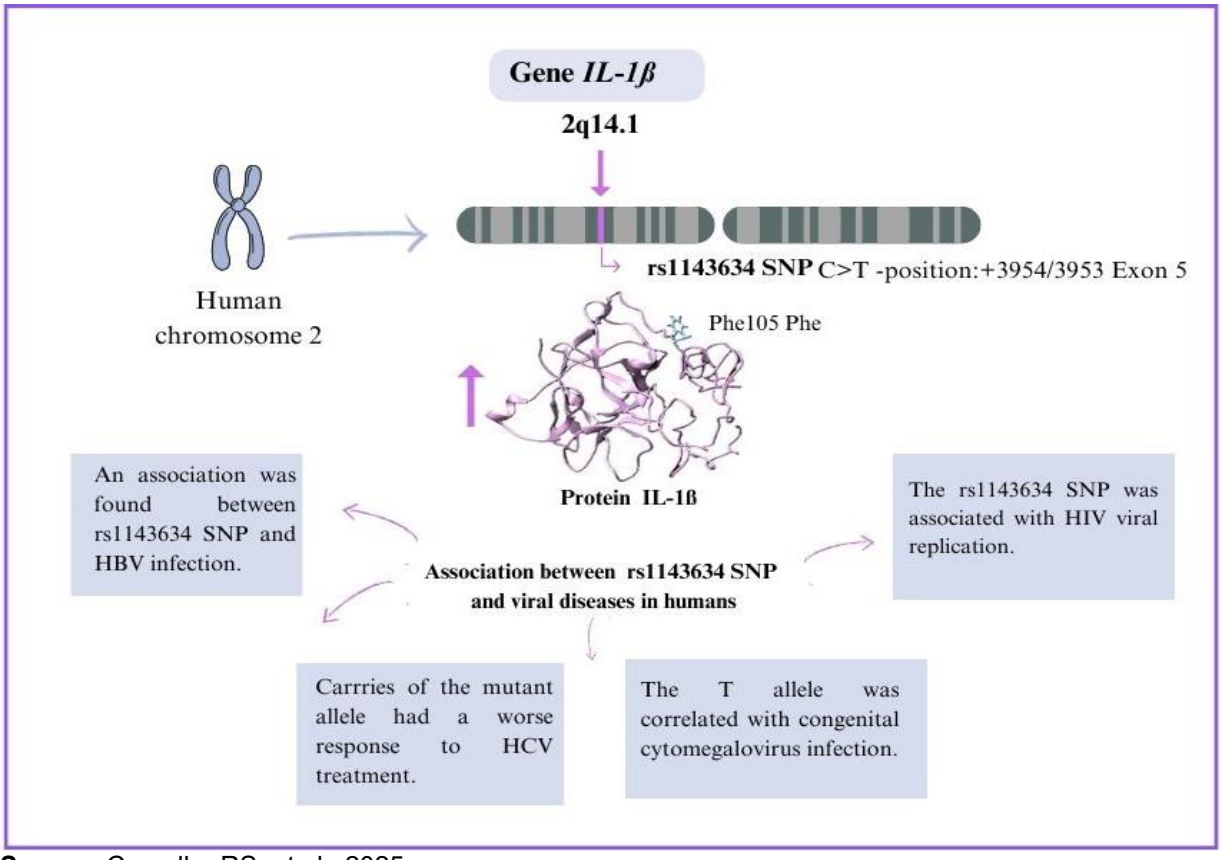
Palabras clave: rs1143634, *IL-1β*, Polimorfismo de nucleótido único, Enfermedades virales.

HIGHLIGHTS

- Single nucleotide polymorphism in the *Interleukin-1β (IL-1β)* gene, rs1143634, is associated with increased risk of some viral diseases;
- The rs1143634 SNP may increase the risk for congenital Human Cytomegalovirus (HCMV) infection and may be a susceptibility factor for Hepatitis B virus (HBV) infection;
- Carries of the mutant allele (T) had a worse response to PEG-IFN plus ribavirin in Hepatitis C virus (HCV) treatment, while those carrying of the wild genotype (C/C) had a better response to hepatitis C treatment;
- The rs1143634 SNP in *IL-1β* increases the risk of HIV acquisition and HIV positive individual's carriers of C/T genotype had a higher risk for tuberculosis.

GRAPHICAL ABSTRACT

Figure 1 - Location of the single nucleotide polymorphism (SNP) rs1143634 in the *IL-1β* gene, located on chromosome 2. In rs1143634 SNP occurs a base substitution between C by T at positions +3954/3953 in exon 5, which the amino acid coded (Phenylalanine - Phe 105 Phe) is not changed, that is a silent mutation.



Source: Carvalho RS, et al., 2025.

INTRODUCTION

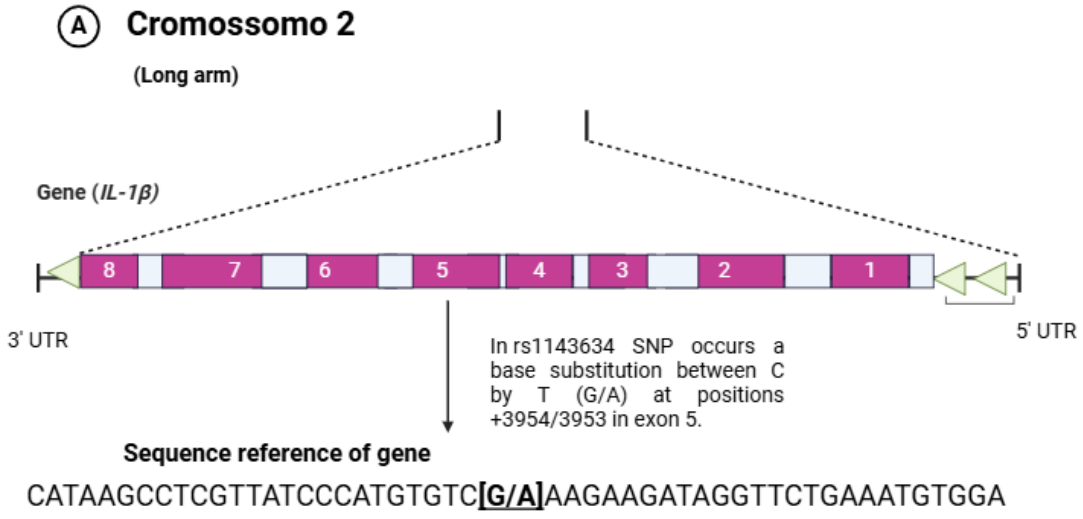
In viral diseases occurs an innate inflammatory response, where Interleukin-1 Beta (IL-1β) is released immediately by monocytes and macrophages, which acts as an immediate line of defense, with inflammatory action enabling the onset of symptoms and activation of new cells in this system, allowing for mechanism of repair and severity of the disease (TSIARA CG, et al., 2018). In this sense, it is known that single nucleotide polymorphisms (SNPs) in cytokine genes might have an effect on the transcription of cytokines and therefore influencing their levels in immune response (WEI Y, et al., 2021). Furthermore, some studies have shown the effect of SNPs in viral diseases (WEI Y, et al., 2021; TSIARA CG, et al., 2018; PACCOUD O, et al., 2019).

Single nucleotide polymorphisms (SNP) are defined as a change in a single nucleotide of the DNA, causing a variation in a specific sequence that affects several people or specific populations. According to the SNP localization different phenotypical results may be seen, where SNPs in encoded regions may produce proteins with altered functions (REIS LM, et al., 2016; CHEN R, VASILAKIS N, 2011).

Thus, SNPs in coding regions are subclassified into two different types: synonyms (or silent) and non-synonyms. The synonyms SNPs generate a nucleotide change that does not alter the sequence of the coded amino acid, however, may impact in the protein conformation, producing a protein with altered phenotype. While the non-synonyms are characterized for a nucleotide substitution that alters the amino acid sequence and may affect the function of the protein or generate a truncated protein (REIS LM, et al., 2016).

The rs1143634 SNP, also known as +3954C>T, is characterized by a synonymous variation in *Interleukin 1-Beta* (*IL-1β)* gene. The rs1143634 is located on exon 5 in chromosome 2, where a base substitution occurs between C and T at positions +3954/3953 (C→T, rs1143634) (REIS LM, et al., 2016) (**Figure 2**). Although the amino acid coded (Phenylalanine - Phe 105 Phe) is not changed, this SNP has been associated with increased expression of the active IL-1β cytokine, which is a proinflammatory cytokine related with increased acute and chronic inflammation and also in cellular response such as proliferation, differentiation and apoptosis (REIS LM, et al., 2016; CHEN R, VASILAKIS N, 2011). Therefore, rs1143634 may be associated with highly susceptible development of worsen pathologies and infection diseases, such as cancers, HIV and Hepatitis (PENA GG, et al., 2017; JAFRIN S, et al., 2021; NCBI, 2023).

Figure 2 - The principle of gene *Interleukin-1βeta (IL-1β)* rs1143634 SNP: On chromosome 2, the Interleukin 1 Beta gene is located in exon 5, where the C>T (G>A) nitrogenous base exchange occurs, promoting the SNP rs1143634.



Created in BioRender.com

Source: Carvalho RS, et al., 2025.

Until now, it is known that SNPs in some cytokine genes, such as *IL-1* gene are polymorphic, having a allelic variation which may produce a distinct translational effect affecting the expression of cytokines, thus influencing the pathogenesis of viral diseases (WEI Y, et al*.,* 2021; AGRAWAL KK, et al., 2023). An study involving *Leishmania guyanensis* showed that the rs1143634 has presented an effect on IL-1Ra levels, where the mutant type genotype (T/T) carriers produced higher levels of IL-1Ra, a protein of the interleukin-1 family which inhibits the action of IL-1β (DA SILVA GA, et al., 2019; WANG B, YUAN F, 2021).

Furthermore, a recent study (AGRAWAL KK, et al., 2023) showed that plasma levels of the IL-1β and IL-6 cytokines were significantly greater in Aseptic Prosthetic Loosening (APL) *T* allele carriers of the rs1143634 SNP than in non-carriers. Moreover, a meta-analysis performed with studies associating several types of cancers identified that the *IL-1β* rs1143634 SNP increased the cancer risk susceptibility (REIS LM, et al., 2016).

This knowledge highlights the importance of understanding the rs1143634 SNP in several pathologies, such as viral diseases. In this sense, this is the first study to review the *IL-1β* SNP rs1143634 in viral diseases, in order to understand the role of this SNP within the context of infection. In addition, it aims to identify possible risk factors for human susceptibility to viruses, contributing to future clinical studies, therapeutic approaches and the diagnosis.

METHODS

This study is characterized as a scoping review performed from a selection of articles published in the National Center for Biotechnology (NCBI) database through the dbSNP section. Furthermore, a supplemental search was conducted in PubMed, Virtual Health Library (BVS) and Web of Science databases with the indexed terms in medical subject headings (MeSH) and health sciences descriptors (DeCS): “IL-1β”, “Polymorphism”, “Single Nucleotide Polymorphism” and “Viral Diseases”. Additionally, the code “rs1143634” was also researched. The boolean operators “AND” and/or “OR” were used to connect the terms.

The searches were performed between October and December of 2023, and all results were described according to the Preferred Reporting Item for Systematic Review and Meta-Analysis (PRISMA) and Joanna Briggs Institute, Reviewers Manual (MOHER D, et al., 2009). To formulate the search strategy, we adopt the PEOS acronym, where: P (population) was individuals (child or adults) with viral diseases; E (exposure): genetic polymorphism rs1143634 in IL-1β cytokine gene in individuals with viral diseases; O (outcome): susceptibility in the development of viral diseases; S (study design): case-controls and meta-analysis articles involving the guiding question.

Among the inclusion criteria were studies associating the presence of the rs1143634 SNP polymorphism in viral diseases, which were full available and written in English, Portuguese or Spanish. Studies approaching the relationship between the SNP and other non-viral and neoplastic diseases, non-humans studies, and studies with unavailable data were excluded.

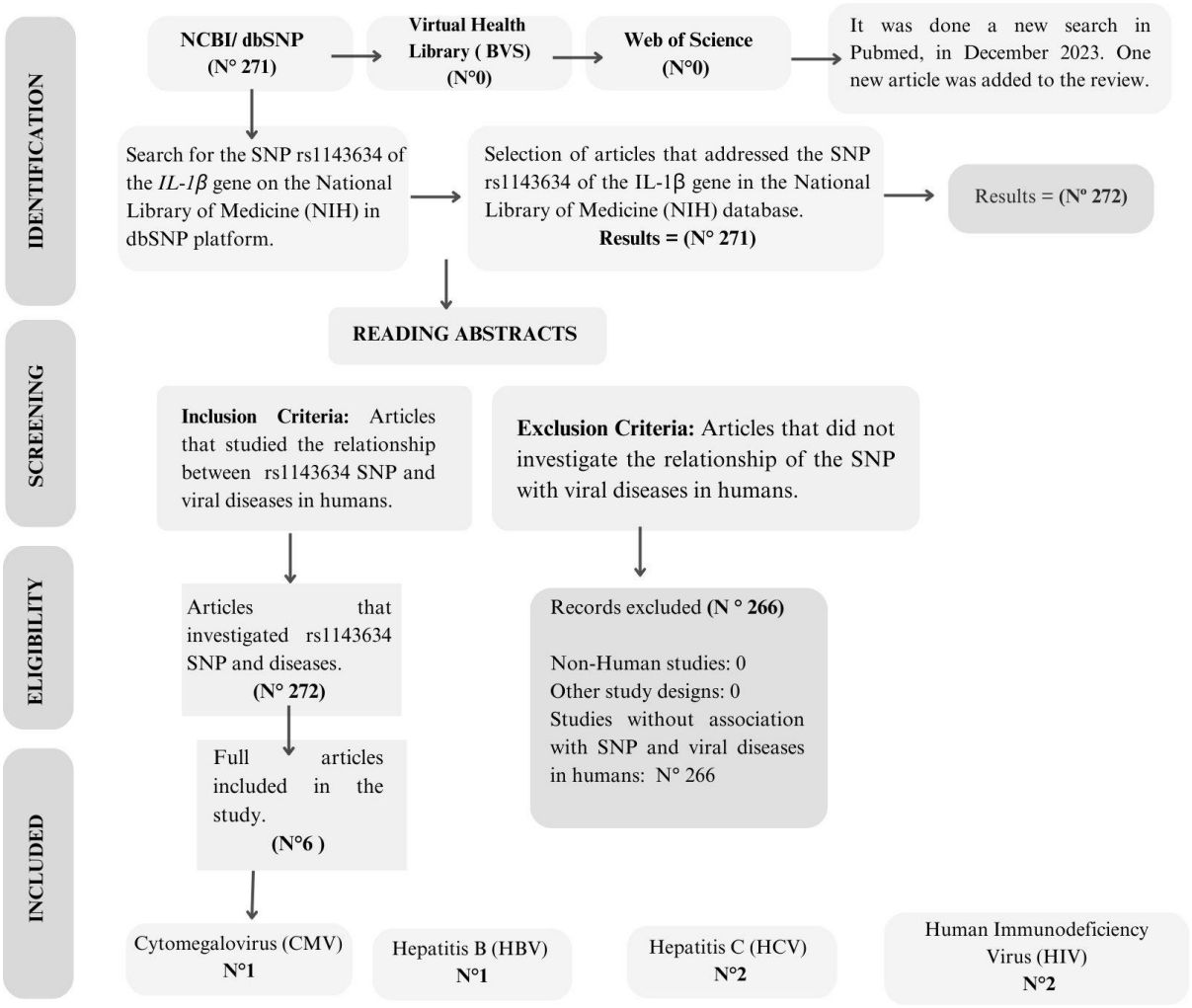
For selection studies (Phase I), the title and abstracts of all articles found in the search were read, including in the next step only those that approached the inclusion criteria. Subsequently, the selected articles were fully read (Phase II). A qualitative analysis was done with the following data extracted from selected studies: (a) author and year of publication, (b) title of article, (c) disease studied (d) country (e) ethnicity (f) study type (g) sample size (g) mean age of patients (h) allele and genotype frequency (i) odds ratio and 95% confidence interval; (j) p-value (k) Hardy-Weinberg equilibrium (l) Genotype

method and (m) key points. Data was collected and entered an Excel software program (Microsoft 365©) table, analyzed qualitatively and then discussed. The data extraction was carried out by two independent reviewers (RSC and JBSB) independently where any disagreement was verified by a third one (FOFC) if necessary. Some information from meta-analysis articles could not be obtained.

RESULTS AND DISCUSSION

From the initial selection, a total of 272 studies were found in all databases, in which, after titles and abstracts reading, a total of 266 articles were excluded for not including criteria, and 6 were included in phase II after full-text reading. Thus, after full-text reading these articles were included in this scope review for presenting *IL-1β* rs1143634 SNP association with viral diseases. Among the viral diseases described in these articles were Hepatitis B (HBV, *n*=1), Hepatitis C (HCV, *n*=2), Cytomegalovirus (CMV, *n*=1) and Human Immunodeficiency Virus (HIV, *n*=2). Below, in **Figure 3**, the entire study selection process is described.

Figure 3 - PRISMA flowchart, which guides the criteria for searching the literature in the databases.



Source: Carvalho RS, et al., 2025.

Table 1 - Characteristics of included studies in this scoping review.

First author and publishing year	Viral disease studied	Study type	Country	Ethnicity	Sample size		Mean age	
					Case	Control	Case	Control
Wujcicka W, et al., 2017.	Human Cytomegalovirus (HCMV)	Case-control	Poland	NA	20 fetuses and neonates	31 uninfected patients	NA	NA
Wei Y, et al., 2021	Hepatitis B Virus (HBV)	Meta-analysis	*	Mixed Caucasian Asian	1.531 patients #	1.854 patients #	NA	NA
Estfanous SZ, et al., 2019	Hepatitis C Virus (HCV)	Case-control	Egypt	NA	201 chronic HCV patients	95 healthy controls	39 years	37 years
Omran MH, et al., 2013	Hepatitis C Virus (HCV)	Case-control	Egypt	NA	50 non-responders to pegylated interferon (PEG-IFN) plus ribavirin	65 Responders to to PEG-IFN plus ribavirin	40.94 years	NA
Tsiara CG, et al., 2018	Human Immunodeficiency Virus (HIV)	Systematic review and meta-analysis	*	Caucasian Other/mixed	782 HIV + patients #	493 HIV – patients#	NA	NA
de Sá NB, et al., 2022	Human Immunodeficiency Virus (HIV)	Case-control	Brazil	Brown - 57 (40.1%) Black - 29 (20,4%) White - 56 (39,4%)	112 patients with HIV and Tuberculosis	30 patients with HIV without Tuberculosis	NA	NA

* Systematic review or meta-analysis involving studies of different countries. # It was considered all population selected for rs1143634 evaluation in the meta-analysis. NA: Not available.
Source: Carvalho RS, et al., 2025.

Table 2 - Main genetic results of studies investigating the *IL-1β* rs1143634 SNP in viral diseases.

First author and publishing year	Allele/genotype or haplotype Comparison		Allelic/ genotypic or Haplotype frequencies (case)	Allelic/ genotypic or Haplotype frequencies (control)	Or - (95% ci) P- value	Hwe (p value)		Genotype method	Key-points
						Case	Control		
Wujcicka W, et al., 2017.	It was available the CT haplotype, composed of: C allele of *IL1A* -889 C>T SNP + T allele of *IL1B* +3954 C>T SNP		2.6%	0.0%	2.5 x 10^8 $p \leq 0.0001$	p=1.000	p=0.650	Nested PCR-RFLP (Restriction enzyme: TaqI)	The CT haplotype increased the risk of congenital cytomegalovirus infection development.
Wei Y, et al., 2021	Allele model: C>T		0.480	NA	1.337 (1.039-1.721) p=0.024*	NA	NA	NA	A significant association was found between the SNP and HBV infection. This is due to the presence of the dominant T allele which showed an association with HBV infection risk.
	Dominant model: C>T				1.942* (1.488-2.535) $p<0.001$				
Estfanous SZ, et al., 2019	Genotype frequencies	CC	97 (48.3%)	47 (49.5%)	0.95 (0.58-1.55) p= 0.85	p=0.055	NA	TaqMan assay	The *IL-1β* rs1143634 SNP showed no risk association with HCV infection.
		CT	85 (42.3%)	33 (34.7%)	1.38 (0.83-2.3) p= 0.22				
		TT	19 (9.5%)	15 (15.8%)	0.56 (0.27-1.15) p=0.11				

First author and publishing year	Allele/genotype or haplotype Comparison		Allelic/ genotypic or Haplotype frequencies (case)	Allelic/ genotypic or Haplotype frequencies (control)	Or - (95% ci) P- value	Hwe (p value)		Genotype method	Key-points
						Case	Control		
	Allele frequencies	C	279 (69.4%)	127 (66.8%)	1.13 (0.78-1.63) p= 0.53				
		T	123 (30.6%)	63 (33.2%)	0.89 (0.61-1.29) p= 0.53				
Omran MH, et al., 2013	CC		6 (11.54%)	46 (88.46%)	17.7544 (6.4884 - 48.5818) p= 0.0001	NA	NA	PCR-RFLP (Restriction enzyme: TaqI)	Carries of the mutant allele (T) had a worse response to PEG-IFN plus ribavirin treatment, while those carrying of the wild genotype (C/C) had a better response to treatment.
	CT		34 (68.00%)	16 (32.00%)					
	TT		10 (76.92%)	3 (23.08%)					
Tsiara CG, et al., 2018	Recessive model: TT versus CT+CC		679	373	4.47 (2.35, 8.52) p=0.00*	NA	NA	NA	The SNP *IL-1β* rs1143634 increases the risk of HIV acquisition in the recessive genetic model analysis.
de Sá NB, et al., 2022	C>T		24 (27,27%) #	2 (10%) #	5,5 (1,04-29.02) p=0.044 +	0.560**	NA	TaqMan assays	HIV+ individuals' carriers of C/T genotype had a higher risk for tuberculosis onset.

Abbreviations: OR= Odds Ratio; 95% CI= 95% Confiance Interval; HWE= Hardy-Weinberg Equilibrium; NA: Not available or not applicable.
* OR calculated from the 4 studies included in the meta-analysis.
# Case group was composed by HIV+ patients that also have tuberculosis, while the control group was only composed by HIV+ individuals.
+ Odds ratios were adjusted by skin color, education, site of tuberculosis, HIV transmission route, and CD8 count.
** In this comparison it was considered HIV + patients without TB versus with TB. **Source:** Carvalho RS, et al., 2025.

Regarding the type of study, two meta-analyses and four case-control studies were analyzed. The studies were published between 2013 and 2022, and the case-control studies were performed in three different countries: Poland, Egypt and Brazil (DOS SANTOS NC, et al., 2020; PAWLOTSKY JM, et al., 2020; PACCOUD O, et al., 2019). In general, the articles do not present the age of participants, however two case-control studies had the mean age of 39 and 40 years of the case group. Only the Brazilian study (PACCOUD O, et al., 2019) described the groups divided by ethnicities, which was brown population (*n*=57; 40,1%), white (*n*=56; 39,4%) and black (*n*=29; 20,4%). Three studies did not describe any detail about the ethnicity of the population. The main characteristics of these studies are presented in **Table 1**.

About genetic characteristics, **Table 2** describes the main important details of the rs1143634 SNP. Through the analysis, it was seen that CT haplotype, composed of C allele of *IL1A* -889 C>T SNP and T allele of *IL1*β +3954 C>T SNP increased the risk of HCMV infection development (OR=2.5x10^{8}; $p \leq 0.0001$). Therefore, Wujcicka W, et al. (2017), concludes that there is a relationship between the T allele of the *IL1β* +3954 C>T polymorphism and the occurrence of congenital CMV infection in the neonatal population.

CMV is an infection that can be transmitted through the blood-brain barrier and therefore may affect the fetus during pregnancy. A virus is part of the *Herpesviridae* family and has symptoms such as pharyngitis or fever; and may stay into latency for many years until the development of infectious mononucleosis. Wujcicka W, et al. (2017), reported that newborns who had the T allele (mutant) of the rs1143634 SNP showed greater severity of congenital cytomegalovirus infection.

Grove J, et al. (2014), showed that children with the presence of a rs7902091 SNP in the *CTNNA3* gene hPPPad an increased risk of schizophrenia in 5-fold if their mothers had CMV infection, reporting the important role of SNPs in CMV infection. The same was observed in the study by Jedlińska-Pijanowska D, et al. (2020), which quantified the viral load in patients with congenital cytomegalovirus (cCMV) and correlated it with the presence of the SNP rs16944 of the *IL12β* gene. According to their results it was found a significant relationship between the rs16944 SNP and the viral load in newborns with CMV.

Regarding HBV, we found only one study that evaluates the SNP rs1143634. Wei Y, et al. (2021), investigated the association between the rs1143634 SNP and HBV infection, where it was evaluated the allele (OR=1.337, CI 1.039-1.721, p=0.024) and dominant genotype model (OR= 1.942, CI 1.488-2.535, p<0.001), being compared C by T. Their findings showed a significant association between the rs1143634 SNP and HBV infection risk, due to the presence of the dominant T allele.

The hepatitis virus (HV) are classified into HBV or HVC, causes liver damage that may progress to cirrhosis in severe forms (SILVA JM, et al., 2020). Considering the immunological context of the disease, it is known that hepatocytes cell death is induced due to recurrent inflammation where the virus promotes a disorganizing tissue regeneration, which can lead, in some cases, to hepatocellular carcinoma. The presence of hepatitis B antigen in the liver causes high expression of pro-inflammatory cytokines such as IL-1β, Interleukin-4 (IL-4) and Interferon Gamma (IFN-***γ***), where they are more notable in high viral load (PENA GG, et al., 2017; GROVE J, et al., 2014).

In HBV infection, rs1143634 polymorphism may promote an exacerbated stimulus of the IL-1β, that is involved in regulating the innate immune response in defense against the virus (PENA GG, et al., 2017). In the study by Wei et al., 2021 it was found a significant association between IL-1β rs1143634

SNP and HBV infection in allele and dominant models. Evidence has shown that T allele might become greater the IL-1β production, but there are still discrepancies (WEI Y, et al., 2021; WANG B, YUAN F, 2021; LÓPEZ-ANGLADA E, et al., 2022).

In patients with HBV infection there is a higher expression of IL-1β, which becomes active; while individuals with the presence of the rs1143634 SNP, may have a higher risk of severe infection (PENA GG, et al., 2017). HBV has the ability to inhibit the expression of IL-1β where its occurs after the suppression of the *IL-1β* gene through the deregulation of the maturation of pro-IL1β. The ability to potentiate this infection is related to the T allele, present in the TTC codon of the rs 1143634 SNP (PENA GG, et al., 2017; PAWLOTSKY JM, 2020).

In relation to HCV, Estfanous SZ, et al. (2019), evaluated the presence of the SNP rs1143634 in individuals with hepatitis C compared to other polymorphisms, rs1539019 and rs35829419 in gene *NLRP3*, rs2043211 in gene *CARD8*, and rs1946518 in gene *IL-18*, they detected that there is no relation between the SNP rs1143634 and severe HCV. This differs from other studies, which reported an association between the presence of some SNPs and liver damage (WUJCICKA W, et al., 2017; SILVA JM, et al., 2020). Was found an association between the genotype groups CT/AC/GG (*IL-28β*/ *IL-6R*/ *IL-6P*) and HCV viremia (OR: 5.4) when compared with the other genotypes (ALKHARSAH KR, et al., 2020).

On the other hand, Omran MH, et al. (2017) investigated the CC-versus CT-TT genotype of the rs1143634 SNP in HCV patients during treatment with Pegylated Interferon (PEG-IFN) plus Ribavirin (RBV) and found a frequency of 6.06% (OR: 17.7544; CI: 6.4884 - 48.5818; *p*=0.0001). Thus, their results showed that carriers of the mutant allele (T) had a worse response to treatment, while those carrying the wild genotype (C/C) had a better response to treatment.

Investigating the presence of the HIV in a Brazilian population, it was possible to identify that the T allele of the *IL-1β* rs1143634 SNP increases the risk of infection with HIV and was associated with a high production of IL-1β, which occurs due to the increased expression of the *IL-1β* pro-cytokine that becomes active, and setting in motion monocytes that have the virus incubated in their nucleus. Thus, this cytokine leaves to enhance viral replication, and therefore in HIV-positive patients it is found at high levels, as well as the viral replication (ALKHARSAH KR, et al., 2020).

Furthermore, other studies have shown a relationship between the presence of the rs1143634 SNP and non-viral diseases (NATANA HM, et al., 2021; DE CASTRO ALVES CE, et al., 2022). In endometriosis, the evaluation of rs1143634 and the severity of the symptoms it was not identified as a risk association. However, in Mexican mestizo women with severe forms of endometriosis, it was found a higher frequency of rs1143634 allele T mutant, which may explain the severity of the inflammatory signs of the disease in these population (JEDLIŃSKA-PIJANOWSKA A, et al., 2020).

On the other hand, there are some studies that demonstrate a protective effect of rs1143634 against diseases (REIS LM, et al., 2016; JEDLIŃSKA-PIJANOWSKA A, et al., 2020; FREITAS L, et al., 2021). In Malaria, an infection with the parasite *Plasmodium falciparum* investigated the association between the genetic variation rs1143634 SNP and susceptibility to infection in children in the first year of life. It was found to have an association of rs1143634 and a protective effect against *Plasmodium* infection (NATAMA HM, et al., 2021). After allogeneic transplantation of hematopoietic stem cells was observed a reduction in the expression of Il- 1β and an increase in interleukin 10, which is one of the cytokines that inhibit inflammation (STINCO M, et al., 2021). Thus, new studies investigating the relationship between genetic variations of the gene *in IL1β* rs1143634 and diseases and their immunological mechanisms are highlighted.

FINAL CONSIDERATIONS

Even though studies on the IL1β rs1143634 SNP have been conducted on numerous diseases, such as periodontitis, carcinomas, pericarditis, and parasitic diseases where it acts as a risk and severity factor its correlation with viral diseases remains underexplored. This highlights the importance of further research, as it may help identify new factors that potentially contribute to disease severity in different populations. The genetic diversity resulting from population miscegenation allows for variations that can influence susceptibility to diseases. Therefore, studies focusing on SNPs are crucial for identifying protective or risk factors in viral infections. Up to this review, only six studies have investigated this SNP in the context of recurrent viral diseases and public health, reinforcing the need for further research in this area. In conclusion, our findings suggest that the IL1β rs1143634 SNP may be a potential risk factor for viral diseases, including Hepatitis, Cytomegalovirus, and Human Immunodeficiency Virus. Understanding the genetic mechanisms underlying disease risk and severity can aid in the development of preventive measures to ensure a more favorable course of viral infections.

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