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# Immune Exhaustion of Hepatitis C Virus (HCV) Infection and Elucidates Cytokine [IL-1 2, IL-12 and IL-17] and Chemokine-Mediated Pathogenesis

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**Abstract:**
The hepatitis C virus (HCV) infection results in a cascade of immunological responses that influence viral persistence, viral clearance and liver disease formation due to the action of a number of interleukins (ILs). To maintain chronic infection and cause liver damage, the infection of hepatitis C virus alters the immune system drastically generating, suppressing, or utilizing various interleukins (IL). The elevated levels of essential interleukins inflame and fibrosis. This study aimed to shed some light on the role the cytokines and chemokines play in the progression of hepatitis C in patients. A sample of sixty persons who tested HCV-positive and were hospitalized with a diagnostic liver biopsy was analyzed. Eleven people had cirrhosis and 49 had chronic persistent hepatitis. There were forty negative hepatitis test workers working in the laboratory who were healthy. Serum interleukin-17A levels were significantly higher in patients infected with chronic HCV without cirrhosis (318.00±27.04 pg/ml), in patients infected with chronic HCV with cirrhosis (354.81±28.95 pg/ml), and in control subjects (195.00±15.68 pg/ml). The levels of IL-12 were recorded as follows: 59.07±8.06, 85.03±9.75, and 22.05±5.09 pg/ml, respectively. The levels of IL-1β were measured at 16:45±3.71, 28:00±6.09, and 7:00±1.85 pg/ml at the same time. The results of this study demonstrated that compared to the control group, patients with chronic HCV infection and cirrhosis had significantly higher serum chemokine levels in CXCL9 (310.95±26.04 pg/ml, 357.00±29.05 pg/ml, and 226.09±17.13 pg/ml, respectively). Although the CXCL10 levels were measured as 61.00±8.03, 79.05±7.44, and 21.08±5.39 pg/ml, respectively. This has been observed in patients infected with Hepatitis C virus (HCV) where the interleukin (IL-1 β, IL-12 and IL-17) levels are found to be elevated.

**Keywords:** Hepatitis C virus (HCV), Cytokine, Chemokine, Immune Exhaustion.

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# INTRODUCTION

Hepatitis C virus (HCV) infection is an infectious disease that affects the liver, and may lead to cirrhosis and hepatocellular carcinoma. The main ways in which the virus is transmitted include injecting infected fluids, risky dental and surgical procedures, and blood transfusions [1, 2]. About 80% of HCV infections develop into a chronic infection of the hepatitis. HCV infection triggers chronic inflammation, causes fluctuations in serum levels of inflammatory and proinflammatory biomarkers, endothelial invasion, and dysfunction. The innate and adaptive immune systems generate some cytokines as a response to HCV infection. Examples of cellular molecules and soluble cytokines that have been linked to herpes simplex virus infections include immune checkpoint biomarkers such as CTLA-4, PD-1, and PD-L1 that can predict renal problems, and variables related to cardiovascular health such as cardiac troponin-I and intercellular adhesion molecule-1. Th-17 cells produce the cytokine interleukin-17 (IL-17) that encourages inflammation. When it comes to viral infections in particular, this cytokine is crucial in controlling the host immunological response. The dysregulation of IL-17 leads to cirrhosis, fibrosis, and hepatocellular carcinoma, as well as to chronic inflammation and autoimmune diseases. Several investigations suggest that the interleukin-17 (IL-17) pathway can be found in both viral-induced diseases and in antiviral immune response [3]. Not much is known about what IL-17 and IL-35 do in the situation when a person is infected with the chronic hepatitis C virus (HCV). People who had a chronic HCV infection had much higher serum IL-17 levels compared to healthy controls. The elevated blood levels of IL-17 in liver injury in chronic hepatitis and cirrhosis provide support to the notion that IL-17 plays a role in the genesis and/or etiology of liver fibrosis. Inflammation is a protective immune reaction to ensure the damaged tissue is healed and to eliminate damaging stimuli via the host cells. Inflammation induced by microbe infection or tissue injury includes immune cells such as dendritic cells (DCs) and macrophages as well as nonprofessional cells such as endothelial cells, fibroblasts, and epithelial cells. Interleukin-1 (IL-1) and interleukin-18 (IL-18) are important in the body as a natural response to the pathogen invading the body. We wanted to examine the question whether hepatocytes infected with HCV are in a normal state of IL-1 or IL-18 or whether it is induced by the macrophages due to the commonly occurring chronic illness when infected with HCV. Endogenous host and pathogen-associated danger molecules could be present to stimulate the formation of inflammasomes that stimulate IL-1 [4-6]. A group of cytoplasmic PRRs called NOD-like receptors (NLRs) detect viral nucleic acid and/or viral proteins; these receptors make up the inflammasomes. In response to activation the NLRs assemble inflammasomes that activate caspase-1, through a multiprotein complex with apoptosis-associated speck-like protein, which contains a carboxy-terminal CARD. According to some studies, NLRP1, NLRP3, and NLRC4 can recognize viral infections that triggers inflammasomes to release the inflammatory cytokine IL-1. It takes two separate impulses to activate and release IL-1 and IL-18, making their production a highly controlled process. The activation of NF-B and the synthesis of pro-IL-1 mRNA are triggered by the first signal in a manner that is reliant on the Toll-like receptor (TLR) signal [7, 8]. Caspase-1 is activated in the second signal and it cleavages pro-IL-1 into IL-1 and IL-18 that are now biologically active. It has been believed to be an inflammatory property of cancer [9] and inflammatory cells and mediators are often present in tumor environments. Activation of hepatic stellate cells (HSCs) and extracellular matrix (ECM) deposition could be the result of early-stage liver fibrosis following hepatocellular damage, inflammation and activation of innate immune system. The present study discovered that the production of IL-1 5, IL-12 and IL-17 was simultaneously tested on patients infected with HCV.

## Materials and Methods

### Patients and Participants

The subjects of the study were sixty people aged 10-60 years old who were infected with HCV and admitted to have a liver biopsy to determine the diagnosis. Eleven people had cirrhosis and 49 had chronic persistent hepatitis. Control was done on forty healthy laboratory workers who were negative on hepatitis C surface antigen.

### Enzyme-linked immunosorbent assays (ELISA)

HCV serum ELISA antibodies were used to measure the levels of IL-1 2, IL-12 and IL-17 in serum samples of the chronic hepatitis C patients and healthy controls according to the Cusabio Biotech catalog of Life Sciences Advanced Technologies Inc. in USA.

**Statistical Analysis**

Data analysis was done using GraphPad Prism 8.0 developed in the USA. To compare the two groups a t-test was used and the results are presented as means plus SD. One-way ANOVA was used in comparing the four groups. A P-value below 0.05 was considered significant to do statistical uses.

**Results and Discussion**

Figure 1, 2 and Table 1 show the distribution of the numbers and percentages of people with HCV according to age groups. The age group 41-50 (17 or 28.33) appears to have the highest number of patients with chronic HCV with cirrhosis whereas the age group 10-20 (2 or 3.33) had the least number of cases. Our study found that the prevalence of HCV infection changed as people became older. The HCV genotyping was not a component of this study so we cannot assert that the age distribution is due to genotypes. Young people are more likely to receive HCV screening prior to getting married or undergoing surgery, which may be the reason why the age structure of the group will not be similar. Cirrhosis is more likely to develop faster and is more common among elderly patients that become infected later in life. We have concurrently tested (Interleukin-l Beta) production in chronic hepatitis C virus carriers to see if these abnormalities could be due to disordered lymphokine development. The activity of interleukin-1 was significantly increased in the patient supernatants than the normal control supernatants in mononuclear cells cultured with or without lipopolysaccharide ($p<0.01$). The lysates of patient monocytes contained a greater concentration of interleukin-1 in the presence of lipopolysaccharide, silica and both, than controls ($p<0.05$). In contrast, interleukin-2 production was discovered to be decreased in patients with chronic hepatitis B virus infection ($p<0.01$), while these results show that interleukin-lβ production is significantly increased. Because interleukin-1 is capable of stimulating fibroblasts to produce collagen, scientists sought a relationship between fibrosis and interleukin production of liver biopsy samples [10, 11]. Interleukin-l2 was found to be highly correlated ($p<0.001$) with the extent of fibrosis and this implies that the lymphokine may be directly associated with the development of cirrhosis in such individuals.

**Table 1. Prevalence of chronic HCV infections by age, with or without cirrhosis.**

| Age Groups (Years) | Patients Chronic HCV with Cirrhosis | % | Patients Chronic HCV without Cirrhosis | % | Total | % |
|---|---|---|---|---|---|---|
| 10-20 | 2 | 3.33 | 0 | 0 | 2 | 3.33 |
| 21-30 | 4 | 6.66 | 0 | 0 | 4 | 6.67 |
| 31-40 | 10 | 16.67 | 1 | 1.67 | 11 | 18.33 |
| 41-50 | 17 | 28.33 | 2 | 3.33 | 19 | 31.67 |
| 51-60 | 6 | 10 | 2 | 3.33 | 8 | 13.33 |
| ≥60 | 10 | 16.67 | 6 | 10 | 16 | 26.67 |
| Total | 49 | 81.67 | 11 | 18.33 | 60 | 100 |

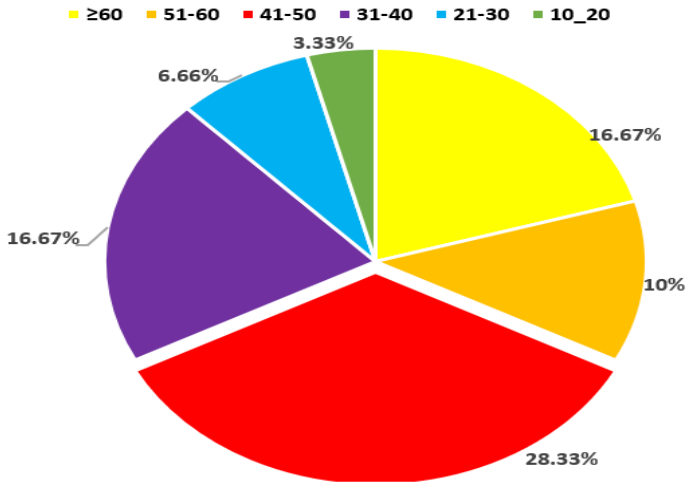


**Figure 1. Distribution of patients infected with chronic HCV with cirrhosis according to age groups.**

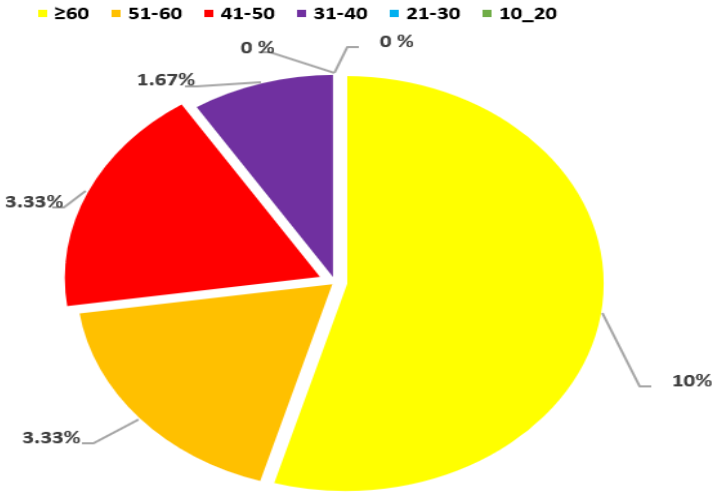


**Figure 2. Distribution of patients infected with chronic HCV without cirrhosis according to age groups.**

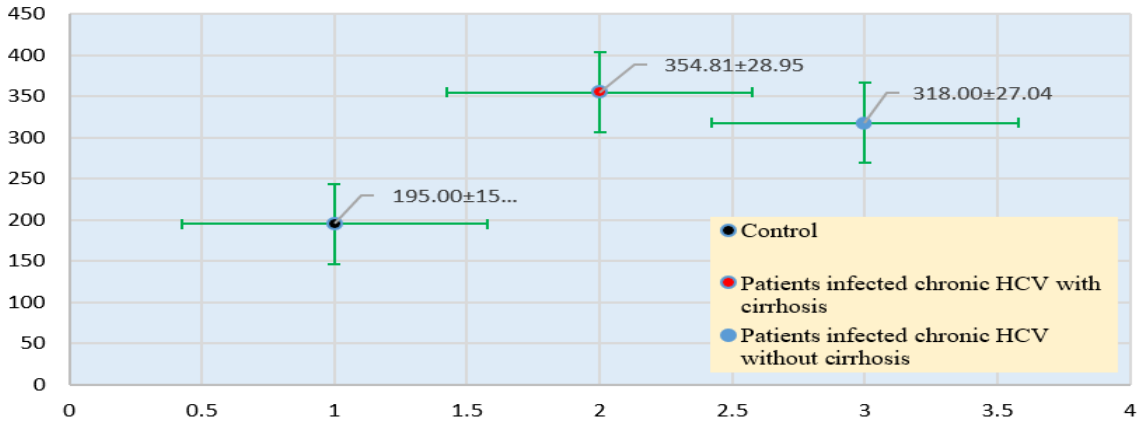


**Figure 3. IL-17A levels in patients infected chronic HCV with cirrhosis and patients infected chronic HCV without cirrhosis comparison with control.**

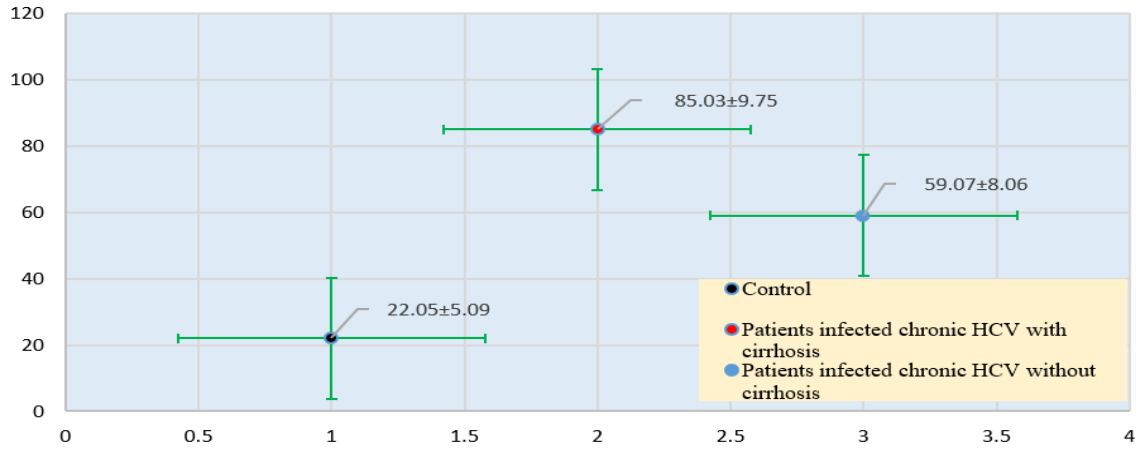


**Figure 4. IL-12 levels in patients infected chronic HCV with cirrhosis and patients infected chronic HCV without cirrhosis comparison with control.**

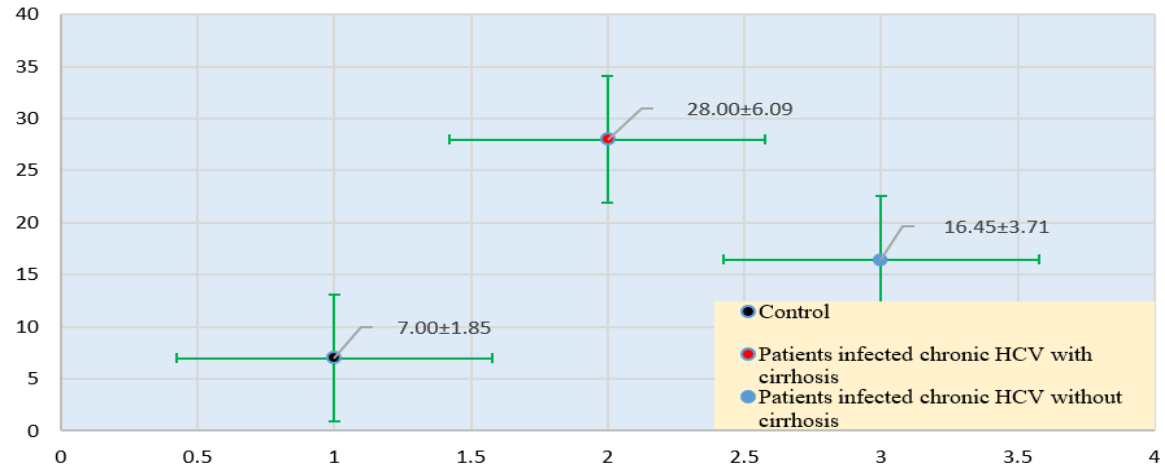


**Figure 5. IL-1β levels in patients infected chronic HCV with cirrhosis and patients infected chronic HCV without cirrhosis comparison with control.**

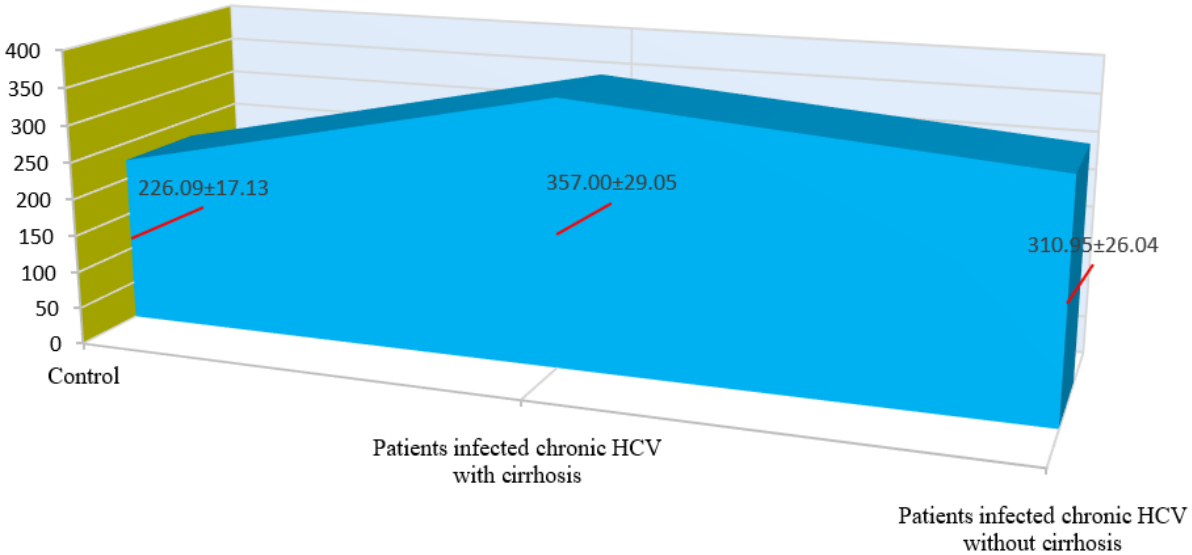


**Figure 6. CXCL9 levels in patients infected chronic HCV with cirrhosis and patients infected chronic HCV without cirrhosis comparison with control.**

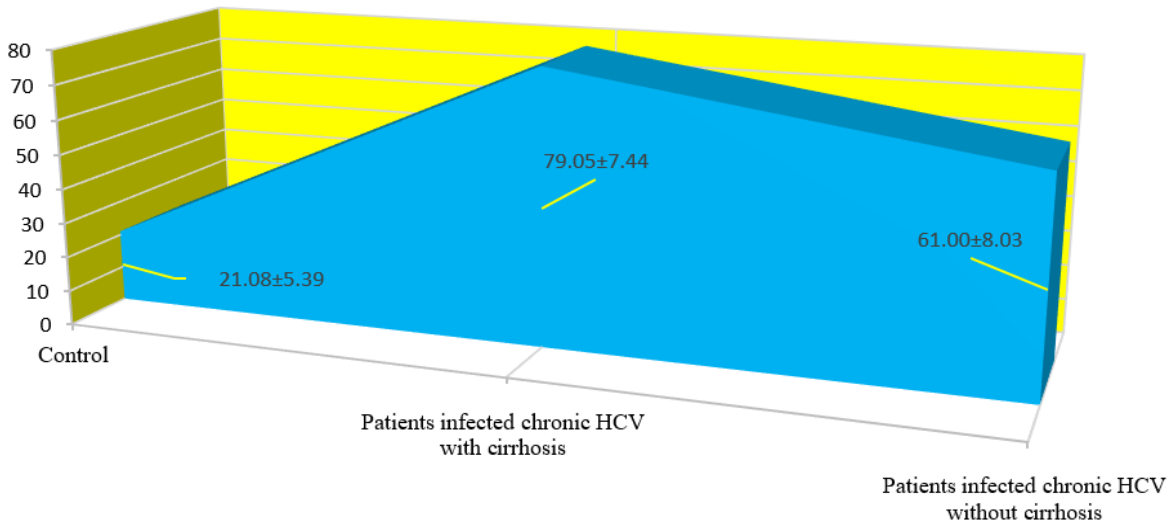


**Figure 7. CXCL10 levels in patients infected chronic HCV with cirrhosis and patients infected chronic HCV without cirrhosis comparison with control.**

There was a significant difference of serum interleukin-17A in patients with chronic HCV who were without cirrhosis (318.00±27.04 pg/ml), patients with chronic HCV with cirrhosis (354.81±28.95 pg/ml) and control subjects (195.00±15.68 pg/ml). The levels of IL-12 were recorded as follows: 59.07±8.06, 85.03±9.75, and 22.05±5.09 pg/ml, respectively. The levels of IL-1β were measured at 16:45±3.71, 28:00±6.09, and 7:00±1.85 pg/ml at the same time. The outcomes of this research showed that patients with chronic HCV infection and cirrhosis with respect to the control group showed significantly elevated serum levels of CXCL9 (310.95±26.04 pg/ml, 357.00±29.05 pg/ml and 226.09±17.13 pg/ml, respectively). Although the CXCL10 levels were measured as 61.00±8.03, 79.05±7.44, and 21.08±5.39 pg/ml, respectively. A chemokine called CXCL9, or monokine produced by interferon-γ (MIG), is vital in regulating the immunological response to HCV infection. Its main functions are the attraction of T cells (Th1 cells in particular) to inflammatory locations. These Th1 cells are needed to develop a robust antiviral response to HCV. Th1 cells express the CXCR3 receptor, which CXCL9 binds to in order to accomplish this recruitment. Upon linking to CXCL9, a signaling cascade is activated, leading these immune cells to the infected liver, where they are able to effectively fight the virus [12-14]. The presence of high CXCL9 in the blood of HCV patients may be the evidence of severity of the disease and an indicator of continuous inflammation. By exploring the role that CXCL9 plays in HCV infection, it would be possible to reveal potential new treatment methods that help to control the immune response and improve treatment outcomes. The main sequence at its N terminus does not contain an ELR sequence not included in the ELR CXC family, and contains four conserved cysteine residues. It is predicted that when the IP-10 gene cleavage site is cleaved by signal peptidase, the main translational product of the gene which is a protein containing two internal disulfide bonds will result in the production of an N-terminal secretory peptide containing four conserved cysteine residues [15, 16]. Th1 cells can release IFN- 7 when infected by a virus and this could cause a rise in the expression of CXCL10 in the endothelial cells that line the liver sinuses. Thus, CXCL10, which is extremely overexpressed in the liver of hepatitis patients can facilitate CTL and other immune cells within the affected region by migrating to the liver via chemotaxis and stimulating monocytes and lymphocytes that secrete its receptor CXCR3. In the meantime, the activated cells release more chemokines that further promote the aggregation of lymphocytes in the liver. Research has increasingly over the years indicated a connection between IP-10 and the effectiveness of CHB treatments [17].

Given that 66 percent of the patients were on anti-HCV medication, the fact that the lowest HCV viral load was 165 copies/ml which is the persistent viremia poses doubts about the effectiveness of these medications. This could be due to the fact that the anti-HCV drugs have been administered not very long ago and the body has not had a sufficient time to develop a good immune response. Although the PCR results showed that HCV patients had high viral load, the new anti-HCV drugs are able to lower the viral load to undetectable levels within two to three weeks after treatment is initiated. The results support earlier studies, which indicated that individuals having chronic HCV infection will have anti-HCV antibodies, which will last forever, as all the patients with HCV infections were found positive of these

antibodies. Interleukin-17 (IL-17) bridges the neutrophil recruitment and activation with T lymphocyte activation by mediating the protective innate immunity to the infections or modulating the pathogenesis of the inflammatory diseases [18-21]. In this study, serum IL-17 was considerably elevated in the HCV group compared to the control group. Reduced interleukin-17 (IL-17) levels in HCV carriers with persistent infections which are similar to that of normal people. They discovered an elevation of IL-17 in hepatocellular carcinoma (HCC) patients with HCV infections but no elevation in IL-17 in HCV patients with liver fibrosis. This result provokes the possibility of an interrelation of IL-17 with HCC progression in patients with HCV. These results point to a potential function of IL-17 in the development of HCV. The role of IL-17 in HCV infection has to be further investigated in future studies. The research has established a weak positive relationship between IL-17 and HCV viral load; this is probably because when the immune response is heightened and secretion of IL-17 as an inflammatory cytokine is increased, the HCV viral load increases. Reduction in the release of IL-17 is however observed in the presence of other regulatory mediators released which control the release of IL-17 and when the infection becomes chronic in HCV [22-25]. Gomaa et al. were able to find a significant correlation between interleukin-17 and hepatitis C virus load. One would assume that an augmentation in viral load would be associated with augmented inflammatory reaction and liberation of inflammatory proteins such as IL-17; thus, the current research observation that serum IL-17 levels rose with augmented mean HCV viral loads with age is in keeping with this forecast. The unavailability of statistically significant relationship between the ages of the patients with HCV and the levels of their interleukins indicates that there could be other factors involved in the case. The production of hydrogen peroxide (ROX) is enhanced in HCV-infected hepatocytes through the inhibited autophagy. ROS does not seem to be an effector mechanism of inflammasome induction, however, because we did not detect an induction of inflammasome mediators in autophagy knockdown HCV-infected hepatocytes. An important acute-phase protein in the liver, human serum amyloid A (SAA) is upregulated in inflammatory responses mainly by the combined effects of proinflammatory cytokines on transcription. The activation of SAA leads to the production of interleukin (IL)-1 and IL-1, and IL-1 secretion by macrophages requires the NLRP3 inflammasome [26, 27]. It is possible that HCV-infected hepatocytes do not activate the inflammasome since, as we have previously noted, SAA stays unchanged in hepatocytes that contain HCV protein. The liver is found at the junction of the blood entering the body via the digestive system and takes part in a very diverse range of metabolic functions. The liver plays a significant role in the digestion of food, production of serum proteins and elimination of both internal and external pollutants among various other functions that this liver performs. Metabolic equilibrium is aided by these functions generally. Inflammation may occur in any tissue whether traumatic, viral, post-ischemic, toxic, or autoimmune, based on a complex web of interactions of cellular and soluble factors. Inflammation usually helps to remove infections and repair damaged tissues, but in most instances, it destroys other tissues and this may make some of the organs to cease functioning. Leukocyte recirculation through the inflamed liver is facilitated by chemokines and their receptors, according to a number of recent articles [28, 29]. Besides their possible contribution in the inflammation-related malignant transformation, chemokines could be involved in liver regeneration and fibrosis. There is emerging evidence that the various stages of liver disease due to chronic infection of the hepatitis C virus can be associated with particular chemokines and chemokine receptors. Extensive phase 3 and 2 clinical trials are underway for a number of small compounds and peptides that target chemokines and their receptors [30, 31]. These drugs will be soon in clinical use and the question arises as to whether they can be used in treating the persistent liver disease related to viral infection.

## CONCLUSION

Cytokines have the potential to promote inflammatory responses in cases of long-term infection. Therefore, it is conceivable that treatment modalities might induce quiescent hepatic stellate cells to become fibrotic due to the stimulation of [IL-1 β, IL-12 and IL-17] by the macrophages and other soluble mediators by the HCV-infected hepatocytes. After an HCV infection, the chemokines bind their receptors, and thus, they can identify specific immune and inflammatory cells in the area of lesion. Such cells then participate in the inflammatory response. The development of the disease is conditional upon a variety of aspects, such as the stage of the HCV infection, the capability of the host immune system to combat the infection, the expression of chemokines and their receptors in the host cells, and the interaction of the factors with each other. Consequently, many critical issues concerning the action

mechanisms of chemokines and their receptors at different stages of HCV infection process are still unresolved. These issues are, but not limited to: how specifically do chemokines play a role in the immune response after an infection of HCV; what is involved in a chronic or severe HCV infection; how different chemokines and their receptors are regulated; and what effect various chemokines have on antiviral treatment. Moreover, other chemokines and their receptors promote the establishment of chronic HCV infection.

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