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Direct Effect of Hepatitis B Infection on Insulin Resistance

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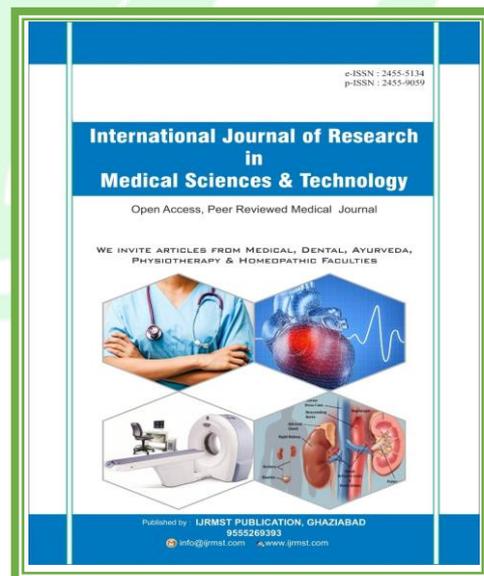
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ABSTRACT

The aim of this research is to find out the targeted effect between insulin resistance and viral hepatitis B (CVHB) Insulin resistance - a blood sugar disorder that precedes diabetes mellitus - is a hallmark of chronic hepatitis C infection, regardless of the severity of the virus or other "metabolic" factors.

Method: 100 samples were collected and a medical examination was performed on the patients. The second step is described as aiming to find out the factors in relation to the metabolism and liver B antigen. As for the determinants that were used in order to know insulin sensitivity, the determinants were HOMA, QUICKI and Mffm.

Conclusion: Our test exhibits that CVHB is related with IR. CVHB may should be observed for event of IR and diabetes mellitus

Keywords: Hepatitis, IR, Aminotransferase, CVHB, HOMA

INTRODUCTION

Hepatitis is an infection that causes hepatitis/liver irritation. The most well-known types of this infection are hepatitis A, B and C. The hepatitis C infection, which is communicated through the blood, is riskier, in light of the fact and exist no fortification for this infection. Hepatitis C keeps the liver from doing its ordinary capacities. An individual has diabetes when the body experiences issues engrossing (glucose). Glucose is a wellspring of energy that additionally influences all organs and muscles in the body. Since glucose is handled through the liver like different supplements, the

connection between hepatitis C and diabetes ought not be astounding.

Hepatitis can cause diabetes in two fundamental manners. Right off the bat, diabetes can happen in light of the fact that somebody has a background marked by persistent hepatitis as the constant hepatitis C infection at last makes it hard for the liver to dispose of abundance glucose. Whenever left untreated, the condition can ultimately cause hyperglycemia. The hepatitis C infection likewise builds insulin obstruction. This is a significant danger factor for type 2 diabetes.

Worldwide, and the absence of a rapid and expanded response, is expected to remain a

number People living with hepatitis B virus:

Its current high levels are the same throughout the 40 years or so the next fifty, as is the cumulative number of deaths that will occur Between 2015 and 2030 there will be 20 million deaths. As for the number People living with the hepatitis C virus are: Really increased, although there is an effective treatment for it. It was no longer possible Postpone strengthening of the global response the five hepatitis viruses (A, B, C, D and E) differ. Great difference between them, and characterized by different transmission patterns, and affect in different populations, which results in a difference Health outcome. An effective response requires a combination of Standardized procedures, with tailored interventions offered for each One of the five viruses at the same time.

Elimination of hepatitis epidemics as one of the major public health threats is possible using:

Currently available tools and approaches are under development and exist Opportunities to improve and expand the response by investing in five Areas for basic interventions, namely

1. Vaccines - effective vaccines are available to prevent infection Each of the viral hepatitis A, B, and E

where they operate A group of countries is already implementing large-scale programs It is inexpensive to vaccinate children against hepatitis B virus

2. Prevention of mother-to-child transmission of hepatitis B virus
Child - timely vaccination at birth with a dose Hepatitis B virus vaccine is an essential intervention for preventing hepatitis B virus Transmission of the virus from mother to child at birth, which is possible Improve it through prenatal screening and use Antiviral drugs.
3. Reduced harm for injecting drug users - can be guaranteed Access to sterile injection tools and addiction treatment services Drug, to prevent epidemics of hepatitis B And combating them among drug users who inject drugs as part of a package A comprehensive range of interventions for the prevention, treatment and care of the injured with human immunodeficiency virus and viral hepatitis and other blood-borne infections of drug user's injection
4. To treat - new oral medications can be tolerated patients well and administered regimens to infected patient's Chronic hepatitis virus

achieving cure rates of over 90% is also available effective treatment for those with chronic infection with hepatitis B virus, provided treatment continues throughout life for most of those infected

Often prevention programs and especially for certain population groups, which are the most affected and exposed to risks, limited in scope and coverage And between 2000 and 2010, there was a decrease of 91% in Hepatitis B virus infection, 83% reduction in infection Hepatitis C virus resulting from unsafe injections However, it is estimated that medical injections and is still responsible for 1.7 million new infections With hepatitis B virus annually and between 157,000 and 315,000 cases New hepatitis C infection annually. The coverage decreases Global harm reduction programs for people who inject drugs, including That's about needle and syringe programs, about 10% by the year In 2014, global HIV vaccination coverage increased Liver B in childhood to more than 82%, but vaccination With a dose of vaccine at birth, hepatitis B virus retarded Far behind its coverage did not exceed 3 And Because of the lack of testing strategies and tools Simple and effective, less than 5% of infected people are known Chronic hepatitis ordered their infection. For this reason, the diagnosis It often comes late,

and appropriate tests are rarely available for evaluation Liver disease guides treatment decisions, including directing them when Starting treatment.

The possibility that liver fat causes insulin resistance in the body, which causes a decrease in its effect on blood sugar, and then type 2 diabetes occurs. Professor Michael Roden of the Austrian Hanus Hospital in the capital, Vienna, explained that liver fat is one of the common consequences of weight gain, noting that medical studies have shown that these fats play a pivotal role in the development of metabolic syndrome, which results from weight gain in the abdominal area in particular and high cholesterol. In addition, blood sugar, and because of high blood pressure, this syndrome may lead to early arteriosclerosis resulting from calcification of blood vessels, heart attacks or strokes.

Insulin resistance is a condition in the body characterized by a decrease in the sensitivity of tissues to the effects of insulin, which leads to an elevated level of insulin in the blood. Increased insulin levels, in turn, have a pathological effect on the vascular wall, which contributes to the formation of atherosclerotic lesions. Moreover, insulin resistance is a significant risk factor for complications, which increases mortality from cardiovascular disease to 65% In addition, prolonged hyperinsulinemia leads to

depletion of the isolated system of the pancreas, leading to type 2 diabetes in addition. In addition, this study is relevant to patients with chronic viral hepatitis - a number of recent studies show an insufficient effect of antiviral therapy if the patient has an increased level of insulin resistance.

MATERIAL AND METHOD

All the necessary tests and requirements for the patients were performed with opinions on them all the medical examinations and all the participants had all the required conditions and met all the accompanying measures: age 18 years, without history of diabetes and hypertension that significant prescription, negative for hepatitis C neutralizer serum aspartate Aminotransferase or alanine aminotransferase (ALT) <80 IU/L, serum gamma glutamyl transferase (GGT) <80 mg/dL, serum creatinine <1.5 mg dL, The success assessment that gave our evaluation data interweaved a certified appraisal, anthropometric examinations, and blood tests. Height and weight were surveyed to the closest 0.1 cm and 0.1 kg utilizing the standard show for subjects wearing a light outfit and without shoes.

Weight list (BMI) was settled as weight (kg) isolated by the square of tallness (m²). Waist circuit was evaluated at the most invulnerable point between the lower line of the rib keep and the iliac top, toward the finishing of a standard sneak past of breathing and to the closest 0.1 cm. Muscle versus fat extent and all out-fat mass were evaluated by bioelectric impedance appraisal. Heartbeat was evaluated utilizing the correct arm of individuals who were in an orchestrated condition, after they had rested for at any rate 10 minutes.

The centralizations of lipid, uric corrosive and GGT were estimated utilizing an autolyser utilizing the enzymatic chromatography strategy, Cystatin C was estimated by turn around immunoassay, and it was estimated by the hepatitis B antigen-connected immunosorbent test technique.

STATISTICAL ANALYSIS

100 samples were collected and the statistical analysis required finding the p-value and find a value was conducted MEAN \pm SD and the statistical analysis was performed using a program SPSS25

RESULTS

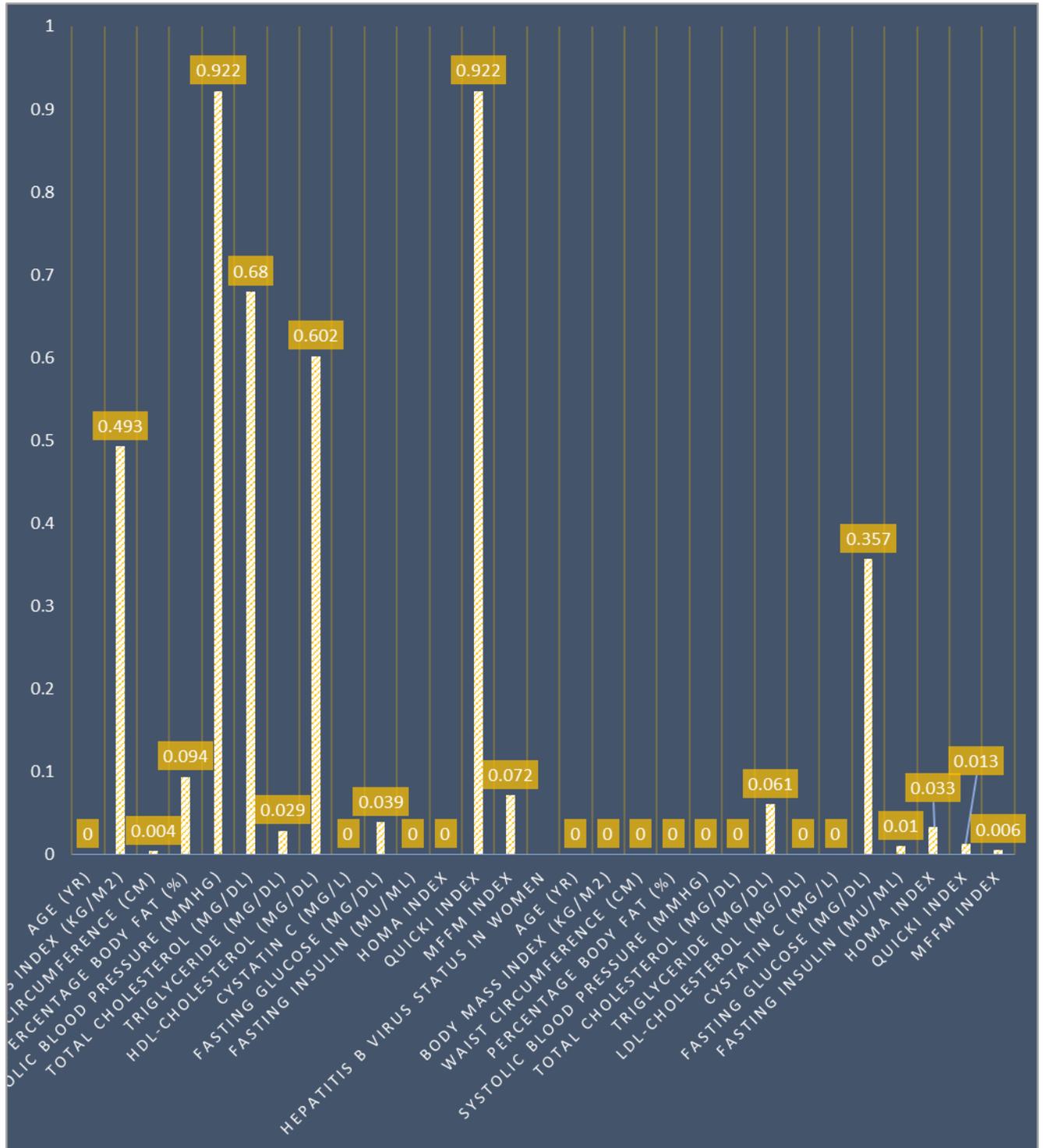
Table 1- Baseline characteristics

Variables	Men	Women
	60	40
AGE (YR)	50.2 ± 11.8	49.7± 11.1
BMI (KG/M ²)	25.6 ± 2.9	24.9 ± 2.7
PERCENTAGE BODY FAT (%)	23.5 ± 5.5	31.7 ± 6.1
SYSTOLIC BP (MMHG)	128.1 ± 16.1	122.3 ± 15.4
AST (IU/L)	24.1 ± 8.2	21.1 ± 11.1
ALT (IU/L)	27.8 ± 17.1	20.0 ± 11.1
GAMMA-GLUTAMYL TRANSFERASE	37.1 ± 20.3	20.0 ± 12.6
FASTING PLASMA GLUCOSE (MG/DL)	92.2 ± 15.5	89.3 ± 15.3
F.INSULIN (MU/ML)	5.50 ± 3.43	5.1 ± 3.22
TC (MG/DL)	198.3 ± 33.1	198.8 ± 35.2
TRI (MG/DL)	140.0 ± 81.1	102.9 ± 65.1
HIGH-DENSITY LIPOPROTEIN (MG/DL)	51.1 ± 12.5	58.8 ± 14.1
LOW-DENSITY LIPOPROTEINS (MG/DL)	125.1± 30.3	123.2 ± 32.2

Table 2- Means \pm SD of metabolic hazard influence relationship with hepatitis B virus

Variables	Negative	Recovery from hepatitis B	Chronic hepatitis B)
AGE	43.2 \pm 10.9	50.9 \pm 10.1	50.8 \pm 10.7
BMI	25.2 \pm 2.8	25.1 \pm 2.9	24.9 \pm 2.6
Waist (cm)	87.0 \pm 7.5	87.3 \pm 7.1	87.7 \pm 7.3
Percentage body fat (%)	23.4 \pm 5.6	23.7 \pm 4.5	23.9 \pm 4.9
Systolic blood pressure (mmHg)	127.3 \pm 16.4	127.3 \pm 15.1	127.1 \pm 16.6
Total cholesterol	197.0 \pm 34.1	197.3 \pm 34.6	196.6 \pm 35.6
Triglyceride	143.1 \pm 85.2	136.2 \pm 80.2	136.1 \pm 79.1
HDL-cholesterol	52.0 \pm 11.9	51.3 \pm 12.5	51.3 \pm 12.5
Fg	90.7 \pm 15.3	92.1 \pm 14.4	91.9 \pm 14.4
FI	5.47 \pm 3.38	5.29 \pm 3.10	5.9 \pm 4.11
HOMA index	1.24 \pm 0.86	1.22 \pm 0.80	1.43 \pm 1.24
QUICKI index	0.31 \pm 0.077	0.34 \pm 0.055	0.362 \pm 0.132
Mf _m index	9.31 \pm 2.43	9.45 \pm 3.44	8.33 \pm 3.11

Figure 1- P Value for risk factors



CORRELATION

Table 3- insulin affect reactivity

	BMI	WC	BFP
FPG	0.144	0.166	0.123
Insulin	0.334	0.431	0.452
HOMA index	0.442	0.452	0.231
QUICKI index	-0.224	-0.331	-0.491
Mf _{fm} index	-0.258	-0.446	-0.399

Table 4 - Logistic regression test

P	IR					
	HOMA			QUICKI		
	β	SE	(95% CI)	β	SE	(95% CI)
Hepatitis B virus status						
Negative			1.000			1.000
Recovery from hepatitis B	-0.032	0.198	0.81 (0.625-1.025)	-0.066	0.143	0.897 (0.644-1.15)
Chronic hepatitis B	0.42	0.120	1.569 (1.108-2.031)	0.347	0.154	1.745 (1.211-2.282)

DISCUSSION

In this test, CVHB apparently was connected with IR in subjects without past diabetes. CVHB self-sufficiently foreseen a clinically enormous development in the odds extent for IR improvement. These outcomes show that patients with CVHB

may require close checking for the rate of infrared radiation and diabetes. Since the ebb and flow, test reports benchmark data on the connection among CVHB and IR in an enormous people, these results maintain past suggestions that CVHB illness is connected with IR It is an in part

abandoned twofold infection that utilizations switch replication in the replication cycle. CVHB contamination is a medical issue that influences around 3.7% of the complete populace with constant hepatitis B. The greater part of them were contaminated straightforwardly from the mother during labor or through contact between the youngsters. CVHB disease may expand the rate of cirrhosis, cirrhosis, and hepatocellular carcinoma. In addition, CVHB contamination is related with sicknesses like PAN, GN, and joint inflammation besides, there is exploratory proof that CVHB disease expands the presence of both IR and related diabetes mellitus. A new creature study demonstrated that HBx weakens the insulin-flagging pathway, The infrared radiation is thought to be brought about by inadequate glucose digestion limit which prompts the discharge of more insulin to accomplish a similar organic reaction and hyperinsulinemia may prompt a huge assortment of anomalies in the veins, kidneys and muscles, which is the principle infection related with metabolic condition. Diabetes and metabolic condition are likewise free danger factors for atherosclerosis, and in this manner, early screening of high-hazard bunches is vital for fruitful wellbeing advancement. The best quality level for deciding insulin

affectability is the insulin hyperglycemia procedure. The HOMA model, QUICKI list, and Mffm utilized in this investigation exhibit a decent relationship with the neural connection strategy and can be effectively utilized in introductory practice.

CONCLUSION

Insulin opposition (IR) is the important sign for improvement of metabolic condition and type 2 diabetes. Constant viral hepatitis B (CVHB) is quite possibly the most widely recognized medical conditions and past clinical examinations additionally propose that hyperinsulinemia happens in CVHB. Notwithstanding, the impact of hepatitis B infection (HBV) contamination on human insulin affectability stays disputable. The creators subsequently examined the theory that HBV disease may connect with IR and metabolic condition, by contrasting occurrence of IR between HBV-contaminated subjects and solid gathering.

REFERENCES

1. Hui JM, Sud A, Farrell GC et al. Insulin resistance is associated with chronic hepatitis C virus infection and, brosis progression. *Gastroenterology* 2003; 125: 1695 – 704

2. Muzzi A, Leandro G, Rubbia-Brandt L et al. Insulin resistance is associated with liver, brosis in non-diabetic chronic hepatitis C patients. *J Hepatol* 2005; 42: 41 – 6
3. Fartoux L, Poujol-Robert A, Guechot J et al. Insulin resistance is a cause of steatosis and, brosis progression in chronic hepatitis C. *Gut* 2005; 54: 1003 – 8
4. Romero-Gómez M, Del Mar Vilorio M, Andrade RJ et al. Insulin resistance impairs sustained response rate to peginterferon plus ribavirin in chronic hepatitis C patients. *Gastroenterology* 2005; 128: 636 – 41
5. Wang CC, Hsu CS, Liu CJ et al. Association of chronic hepatitis B virus infection with insulin resistance and hepatic steatosis. *J Gastroenterol Hepatol* 2008; 23: 779 – 82
6. Jan CF, Chen CJ, Chiu YH et al. A population-based study investigating the association between metabolic syndrome and hepatitis B/C infection (Keelung Community-Based Integrated Screening Study, 10). *Int J Obes (Lond)* 2006; 30: 794 – 9.
7. Misra A, Misra R, Wijesuriya M et al. e metabolic syndrome in south asians: continuing escalation & possible solutions. *Indian J Med Res* 2007; 125: 345 – 54.
8. Alberti KG, Zimmet PZ. De, nition, diagnosis and classi, cation of diabetes mellitus and its complications. Part 1: diagnosis and classi, cation of diabetes mellitus provisional report of a WHO consultation. *Diabet Med* 1998; 15: 539 – 53
9. Matthews DR, Hosker JP, Rudenski AS et al. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; 28: 412 – 9
10. Bonora E, Targher G, Alberiche M et al. Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin sensitivity: studies in subjects with various degrees of glucose tolerance and insulin sensitivity. *Diabetes Care* 2000; 23: 57 – 63.
11. Eckel, R.H., Alberti, K.G., Grundy, S.M. and Zimmet, P.Z., 2010. The metabolic syndrome. *The lancet*, 375(9710), pp.181-183.

12. Lorenzo, C., Okoloise, M., Williams, K., Stern, M.P. and Haffner, S.M., 2003. The metabolic syndrome as predictor of type 2 diabetes: The San Antonio heart study. *Diabetes care*, 26(11), pp.3153-3159.
13. Isomaa, B.O., Almgren, P., Tuomi, T., Forsén, B., Lahti, K., Nissen, M., Taskinen, M.R. and Groop, L., 2001. Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes care*, 24(4), pp.683-689.
14. McMahon, B.J., 2010. Natural history of chronic hepatitis B. *Clinics in liver disease*, 14(3), pp.381-396.
15. Pagnoux, Christian, Raphaële Seror, Corneliu Henegar, Alfred Mahr, Pascal Cohen, Véronique Le Guern, Boris Bienvenu, Luc Mouthon, and Loïc Guillevin. "Clinical features and outcomes in 348 patients with polyarteritis nodosa: a systematic retrospective study of patients diagnosed between 1963 and 2005 and entered into the French Vasculitis Study Group Database." *Arthritis & Rheumatism: Official Journal of the American College of Rheumatology* 62, no. 2 (2010): 616-626.
16. Lai, Kar Neng, Philip KT Li, Siu Fai Lui, Tak Cheong Au, John SL Tam, Kwok Lung Tong, and Fernand Mac-Moune Lai. "Membranous nephropathy related to hepatitis B virus in adults." *New England Journal of Medicine* 324, no. 21 (1991): 1457-1463.
17. Gocke, D.J., 1975. Extrahepatic manifestations of viral hepatitis. *The American journal of the medical sciences*, 270(1), pp.49-52.
18. Reimold, A., & Palmer, B. F. (2010). Viruses and arthritis: new challenges in diagnosis, therapy, and immunization. *The American journal of the medical sciences*, 339(6), 549-556.
19. Moucari, Rami, Tarik Asselah, Dominique Cazals-Hatem, Hélène Voitot, Nathalie Boyer, Marie-Pierre Ripault, Rodolphe Sobesky et al. "Insulin resistance in chronic hepatitis C: association with genotypes 1 and 4, serum HCV RNA level, and liver fibrosis." *Gastroenterology* 134, no. 2 (2008): 416-423.