SUPPLEMENTARY BIBLIOGRAPHY

Lymphomas/non-Hodgkin lymphomas

1. Akdoğan M, Mert A, Tabak F et al (1998) Hepatitis C infection in non-Hodgkin's lymphoma. Turk J Gastroenterol 1: 73-75
2. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
3. Amin J, Gidding H, Gilbert G et al (2004) Hepatitis C prevalence--a nationwide serosurvey. Commun Dis Intell Q Rep 28: 517-21
4. Anderson LA, Pfeiffer R, Warren JL et al (2008) Hematopoietic malignancies associated with viral and alcoholic hepatitis. Cancer Epidemiol Biomarkers Prev 17: 3069-75
5. Arcaini L, Paulli M, Boveri E et al (2004) Splenic and nodal marginal zone lymphomas are indolent disorders at high hepatitis C virus seroprevalence with distinct presenting features but similar morphologic and phenotypic profiles. Cancer 100: 107-15
6. Arcaini L, Varettoni M, Boveri E et al (2011) Distinctive clinical and histological features of Waldenstrom's macroglobulinemia and splenic marginal zone lymphoma. Clin Lymphoma Myeloma Leuk 11: 103-5
7. Arican A, Sengezer T, Bozdayi M et al (2000) Prevalence of hepatitis-G virus and hepatitis-C virus infection in patients with non-Hodgkin's lymphoma. Med Oncol 17: 123-6
8. Aviles A, Valdez L, Halabe J et al (2003) No association between lymphoma and hepatitis C virus. Med Oncol 20: 165-8

9. Bauduer F, Katsahian S, Blanchard Y et al (1999) Descriptive epidemiology of non-Hodgkin lymphomas in a southwestern French hematology center: absence of significant relationship with hepatitis C virus infection. Hematol Cell Ther 41: 191-3
10. Besson C, Canioni D, Lepage E et al (2006) Characteristics and outcome of diffuse large B-cell lymphoma in hepatitis C virus-positive patients in LNH 93 and LNH 98 Groupe d'Etude des Lymphomes de l'Adulte programs. J Clin Oncol 24: 953-60
11. Bianco E, Marcucci F, Mele A et al (2004) Prevalence of hepatitis C virus infection in lymphoproliferative diseases other than B-cell non-Hodgkin's lymphoma, and in myeloproliferative diseases: an Italian Multi-Center case-control study. Haematologica 89: 70-6
12. Boffetta P, Armstrong B, Linet M et al (2007) Consortia in cancer epidemiology: lessons from InterLymph. Cancer Epidemiol Biomarkers Prev 16: 197-9
13. Brind AM, Watson JP, Burt A et al (1996) Non-Hodgkin's lymphoma and hepatitis C virus infection. Leuk Lymphoma 21: 127-30
14. Bronowicki JP, Bineau C, Feugier P et al (2003) Primary lymphoma of the liver: clinical-pathological features and relationship with HCV infection in French patients. Hepatology 37: 781-7
15. Bruzzi P, Green SB, Byar DP et al (1985) Estimating the population attributable risk for multiple risk factors using case-control data. Am J Epidemiol 122: 904-14
16. Catassi C, Fabiani E, Coppa GV et al (1998) [High prevalence of hepatitis C virus infection in patients with non-Hodgkin's lymphoma at the onset. Preliminary results of an Italian multicenter study]. Recenti Prog Med 89: 63-7
17. Cavanna L, Sbolli G, Tanzi E et al (1995) High prevalence of antibodies to hepatitis C virus in patients with lymphoproliferative disorders. Haematologica 80: 486-7
18. Chindamo MC, Spector N, Segadas JA et al (2002) Prevalence of hepatitis C infection in patients with non-Hodgkin's lymphomas. Oncol Rep 9: 657-9
19. Chuang SS, Liao YL, Chang ST et al (2010) Hepatitis C virus infection is significantly associated with malignant lymphoma in Taiwan, particularly with nodal and splenic marginal zone lymphomas. J Clin Pathol 63: 595-8
20. Cocco P, Piras G, Monne M et al (2008) Risk of malignant lymphoma following viral hepatitis infection. Int J Hematol 87: 474-83
21. Collier JD, Zanke B, Moore M et al (1999) No association between hepatitis C and B-cell lymphoma. Hepatology 29: 1259-61
22. Cowgill KD, Loffredo CA, Eissa SA et al (2004) Case-control study of non-Hodgkin's lymphoma and hepatitis C virus infection in Egypt. Int J Epidemiol 33: 1034-9
23. Cucuianu A, Patiu M, Duma M et al (1999) Hepatitis B and C virus infection in Romanian non-Hodgkin's lymphoma patients. Br J Haematol 107: 353-6
24. Dal Maso L and Franceschi S (2006) Hepatitis C virus and risk of lymphoma and other lymphoid neoplasms: a meta-analysis of epidemiologic studies. Cancer Epidemiol Biomarkers Prev 15: 2078-85
25. Dal Maso L, Talamini R, Montella M et al (2004) Hepatitis B and C viruses and Hodgkin lymphoma: a case-control study from Northern and Southern Italy. Haematologica 89: ELT17
26. De Re V, De Vita S, Marzotto A et al (2000) Pre-malignant and malignant lymphoproliferations in an HCV-infected type II mixed cryoglobulinemic patient are sequential phases of an antigen-driven pathological process. Int J Cancer 87: 211-6
27. De Renzo A, Persico E, de Marino F et al (2002) High prevalence of hepatitis G virus infection in Hodgkin's disease and B-cell lymphoproliferative disorders: absence of correlation with hepatitis C virus infection. Haematologica 87: 714-8; discussion 718
28. De Rosa G, Gobbo ML, De Renzo A et al (1997) High prevalence of hepatitis C virus infection in patients with B-cell lymphoproliferative disorders in Italy. Am J Hematol 55: 77-82
29. de Sanjose S, Benavente Y, Vajdic CM et al (2008) Hepatitis C and non-Hodgkin lymphoma among 4784 cases and 6269 controls from the International Lymphoma Epidemiology Consortium. Clin Gastroenterol Hepatol 6: 451-8
30. de Sanjose S, Nieters A, Goedert JJ et al (2004) Role of hepatitis C virus infection in malignant lymphoma in Spain. Int J Cancer 111: 81-5
31. De Vita S, Zagonel V, Russo A et al (1998) Hepatitis C virus, non-Hodgkin's lymphomas and hepatocellular carcinoma. Br J Cancer 77: 2032-5

32. Domingo JM, Romero MS, Palomera L et al (2001) [Hepatitis C virus infection in patients with non Hodgkin's lymphoma]. Med Clin (Barc) 117: 638
33. Duberg AS, Nordstrom M, Torner A et al (2005) Non-Hodgkin's lymphoma and other nonhepatic malignancies in Swedish patients with hepatitis C virus infection. Hepatology 41: 652-9
34. El-Serag HB, Hampel H, Yeh C et al (2002) Extrahepatic manifestations of hepatitis C among United States male veterans. Hepatology 36: 1439-45
35. Ellenrieder V, Weidenbach H, Frickhofen N et al (1998) HCV and HGV in B-cell non-Hodgkin's lymphoma. J Hepatol 28: 34-9
36. Engels EA, Chatterjee N, Cerhan JR et al (2004) Hepatitis C virus infection and non-Hodgkin lymphoma: results of the NCI-SEER multi-center case-control study. Int J Cancer 111: 76-80
37. Ennishi D, Maeda Y, Niitsu N et al (2010) Hepatic toxicity and prognosis in hepatitis C virus-infected patients with diffuse large B-cell lymphoma treated with rituximab-containing chemotherapy regimens: a Japanese multicenter analysis. Blood 116: 5119-25
38. Ferri C, Caracciolo F, Zignego AL et al (1994) Hepatitis C virus infection in patients with non-Hodgkin's lymphoma. Br J Haematol 88: 392-4
39. Ferri C, La Civita L, Caracciolo F et al (1994) Non-Hodgkin's lymphoma: possible role of hepatitis C virus. JAMA 272: 355-6
40. Ferri C, La Civita L, Monti M et al (1996) Chronic hepatitis C and B-cell non-Hodgkin's lymphoma. QJM 89: 117-22
41. Franceschi S, Lise M, Trepo C et al (2011) Infection with hepatitis B and C viruses and risk of lymphoid malignancies in the European Prospective Investigation into Cancer and Nutrition (EPIC). Cancer Epidemiol Biomarkers Prev 20: 208-14
42. Fwu CW, Chien YC, You SL et al (2011) Hepatitis B virus infection and risk of intrahepatic cholangiocarcinoma and non-Hodgkin lymphoma: a cohort study of parous women in Taiwan. Hepatology 53: 1217-25
43. Gasparotto D, De Re V and Boiocchi M (2002) Hepatitis C virus, B-cell proliferation and lymphomas. Leuk Lymphoma 43: 747-51
44. Gasztonyi B, Par A, Szomor A et al (2000) Hepatitis C virus infection associated with B-cell non-Hodgkin's lymphoma in Hungarian patients. Br J Haematol 110: 497-8
45. Gentile G, Mele A, Monarco B et al (1996) Hepatitis B and C viruses, human T-cell lymphotropic virus types I and II, and leukemias: a case-control study. The Italian Leukemia Study Group. Cancer Epidemiol Biomarkers Prev 5: 227-30
46. Genvresse I, Spath-Schwalbe E, Meisel H et al (2000) Primary hepatic or splenic diffuse large B-cell lymphoma and hepatitis C virus infection: a non-fortuitous association? Ann Hematol 79: 530-2
47. Germanidis G, Haioun C, Pourquier J et al (1999) Hepatitis C virus infection in patients with overt B-cell non-Hodgkin's lymphoma in a French center. Blood 93: 1778-9
48. Giordano TP, Henderson L, Landgren O et al (2007) Risk of non-Hodgkin lymphoma and lymphoproliferative precursor diseases in US veterans with hepatitis C virus. JAMA 297: 2010-7
49. Gisbert JP, Garcia-Buey L, Pajares JM et al (2003) Prevalence of hepatitis C virus infection in B-cell non-Hodgkin's lymphoma: systematic review and meta-analysis. Gastroenterology 125: 1723-32
50. Goldman L, Ezzat S, Mokhtar N et al (2009) Viral and non-viral risk factors for non-Hodgkin's lymphoma in Egypt: heterogeneity by histological and immunological subtypes. Cancer Causes Control 20: 981-7
51. Grudeva-Popova J, Nenova I, Mateva N et al (2013) Non-Hodgkin lymphomas and carrier state of viral hepatitis B and C. J BUON 18: 239-44
52. Guida M, D'Elia G, Benvestito S et al (2002) Hepatitis C virus infection in patients with B-cell lymphoproliferative disorders. Leukemia 16: 2162-3
53. Hanley J, Jarvis L, Simmonds P et al (1996) HCV and non-Hodgkin lymphoma. Lancet 347: 1339
54. Harakati MS, Abualkhair OA and Al-Knawy BA (2000) Hepatitis C Virus infection in Saudi Arab patients with B-cell non-Hodgkin's lymphoma. Saudi Med J 21: 755-8
55. Harris NL, Jaffe ES, Stein H et al (1994) A revised European-American classification of lymphoid neoplasms: a proposal from the International Lymphoma Study Group. Blood 84: 1361-92
56. Hausfater P, Cacoub P, Sterkers Y et al (2001) Hepatitis C virus infection and lymphoproliferative diseases: prospective study on 1,576 patients in France. Am J Hematol 67: 168-71

57. Herrinton LJ (1998) Epidemiology of the Revised European-American Lymphoma Classification subtypes. Epidemiol Rev 20: 187-203
58. Hwang JP, Suarez-Almazor ME, Torres HA et al (2014) Hepatitis C virus screening in patients with cancer receiving chemotherapy. J Oncol Pract 10: e167-74
59. Imai Y, Ohsawa M, Tanaka H et al (2002) High prevalence of HCV infection in patients with B-cell non-Hodgkin's lymphoma: comparison with birth cohort- and sex-matched blood donors in a Japanese population. Hepatology 35: 974-6
60. Isikdogan A, Ayyildiz O, Dursun M et al (2003) Hepatitis C virus in patients with non-Hodgkin's lymphoma in southeastern Anatolia region of Turkey: a prospective case-control study of 119 patients. Leuk Lymphoma 44: 1745-7
61. Iwata H, Matsuo K, Takeuchi K et al (2004) High incidences of malignant lymphoma in patients infected with hepatitis B or hepatitis C virus. Haematologica 89: 368-70
62. Izumi T, Sasaki R, Miura Y et al (1996) Primary hepatosplenic lymphoma: association with hepatitis C virus infection. Blood 87: 5380-1
63. Izumi T, Sasaki R, Tsunoda S et al (1997) B cell malignancy and hepatitis C virus infection. Leukemia 11 Suppl 3: 516-8
64. Kalaitzakis E, Gunnarsdottir SA, Josefsson A et al (2011) Increased risk for malignant neoplasms among patients with cirrhosis. Clin Gastroenterol Hepatol 9: 168-74
65. Karavattathayyil SJ, Kalkeri G, Liu HJ et al (2000) Detection of hepatitis C virus RNA sequences in B-cell non-Hodgkin lymphoma. Am J Clin Pathol 113: 391-8
66. Kaya H, Polat MF, Erdem F et al (2002) Prevalence of hepatitis C virus and hepatitis G virus in patients with non-Hodgkin's lymphoma. Clin Lab Haematol 24: 107-10
67. King PD, Wilkes JD and Diaz-Arias AA (1998) Hepatitis C virus infection in non-Hodgkin's lymphoma. Clin Lab Haematol 20: 107-10
68. Kocabas E, Aksaray N, Alhan E et al (1997) Hepatitis B and C virus infections in Turkish children with cancer. Eur J Epidemiol 13: 869-73
69. Kuniyoshi M, Nakamuta M, Sakai H et al (2001) Prevalence of hepatitis B or C virus infections in patients with non-Hodgkin's lymphoma. J Gastroenterol Hepatol 16: 215-9
70. Luppi M, Longo G, Ferrari MG et al (1998) Clinico-pathological characterization of hepatitis C virus-related B-cell non-Hodgkin's lymphomas without symptomatic cryoglobulinemia. Ann Oncol 9: 495-8
71. Matsuo K, Kusano A, Sugumar A et al (2004) Effect of hepatitis C virus infection on the risk of non-Hodgkin's lymphoma: a meta-analysis of epidemiological studies. Cancer Sci 95: 745-52
72. Mazzaro C, Zagonel V, Monfardini S et al (1996) Hepatitis C virus and non-Hodgkin's lymphomas. Br J Haematol 94: 544-50
73. McColl MD, Singer IO, Tait RC et al (1997) The role of hepatitis C virus in the aetiology of non-Hodgkins lymphoma--a regional association? Leuk Lymphoma 26: 127-30
74. Mele A, Pulsoni A, Bianco E et al (2003) Hepatitis C virus and B-cell non-Hodgkin lymphomas: an Italian multicenter case-control study. Blood 102: 996-9
75. Merli M, Visco C, Spina M et al (2014) Outcome prediction of diffuse large B-cell lymphomas associated with hepatitis C virus infection: a study on behalf of the Fondazione Italiana Linfomi. Haematologica 99: 489-96
76. Mizorogi F, Hiramoto J, Nozato A et al (2000) Hepatitis C virus infection in patients with B-cell non-Hodgkin's lymphoma. Intern Med 39: 112-7
77. Montella M, Crispo A, de Bellis G et al (2001) HCV and cancer: a case-control study in a high-endemic area. Liver 21: 335-41
78. Montella M, Crispo A, Frigeri F et al (2001) HCV and tumors correlated with immune system: a case-control study in an area of hyperendemicity. Leuk Res 25: 775-81
79. Morton LM, Engels EA, Holford TR et al (2004) Hepatitis C virus and risk of non-Hodgkin lymphoma: a population-based case-control study among Connecticut women. Cancer Epidemiol Biomarkers Prev 13: 425-30
80. Musto P, Dell'Olio M, Carotenuto M et al (1996) Hepatitis C virus infection: a new bridge between hematologists and gastroenterologists? Blood 88: 752-4
81. Negri E, Little D, Boiocchi M et al (2004) B-cell non-Hodgkin's lymphoma and hepatitis C virus infection: a systematic review. Int J Cancer 111: 1-8

82. Nicolosi Guidicelli S, Lopez-Guillermo A, Falcone U et al (2012) Hepatitis C virus and GBV-C virus prevalence among patients with B-cell lymphoma in different European regions: a case-control study of the International Extranodal Lymphoma Study Group. Hematol Oncol 30: 137-42
83. Nieters A, Kallinowski B, Brennan P et al (2006) Hepatitis C and risk of lymphoma: results of the European multicenter case-control study EPILYMPH. Gastroenterology 131: 1879-86
84. Ogino H, Satomura Y, Unoura M et al (1999) Hepatitis B, C and G virus infection in patients with lymphoproliferative disorders. Hepatol Res 14: 187-194
85. Ohsawa M, Shingu N, Miwa H et al (1999) Risk of non-Hodgkin's lymphoma in patients with hepatitis C virus infection. Int J Cancer 80: 237-9
86. Omland LH, Jepsen P, Krarup H et al (2012) Liver cancer and non-Hodgkin lymphoma in hepatitis C virus-infected patients: results from the DANVIR cohort study. Int J Cancer 130: 2310-7
87. Panovska I, Georgievski B, Stojanovic A et al (2000) Low prevalence of chronic hepatitis C virus infection in B-cell non-Hodgkin's lymphoma patients from a population with a high prevalence of healthy hepatitis c virus carriers. Br J Haematol 109: 249-50
88. Park SC, Jeong SH, Kim J et al (2008) High prevalence of hepatitis B virus infection in patients with B-cell non-Hodgkin's lymphoma in Korea. J Med Virol 80: 960-6
89. Paydas S, Kilic B, Sahin B et al (1999) Prevalence of hepatitis C virus infection in patients with lymphoproliferative disorders in Southern Turkey. Br J Cancer 80: 1303-5
90. Pellicelli AM, Marignani M, Zoli V et al (2011) Hepatitis C virus-related B cell subtypes in non Hodgkin's lymphoma. World J Hepatol 3: 278-84
91. Pioltelli P, Gargantini L, Cassi E et al (2000) Hepatitis C virus in non-Hodgkin's lymphoma. A reappraisal after a prospective case-control study of 300 patients. Lombart Study Group of HCV-Lymphoma. Am J Hematol 64: 95-100
92. Pioltelli P, Zehender G, Monti G et al (1996) HCV and non-Hodgkin lymphoma. Lancet 347: 624-5
93. Prati D, Zanella A, De Mattei C et al (1999) Chronic hepatitis c virus infection and primary cutaneous B-cell lymphoma. Br J Haematol 105: 841
94. Rabkin CS, Tess BH, Christianson RE et al (2002) Prospective study of hepatitis C viral infection as a risk factor for subsequent B-cell neoplasia. Blood 99: 4240-2
95. Ramos-Casals M, Trejo O, Garcia-Carrasco M et al (2004) Triple association between hepatitis C virus infection, systemic autoimmune diseases, and B cell lymphoma. J Rheumatol 31: 495-9
96. Rosenberg SA, Berard CW and Brown BW (1982) National Cancer Institute sponsored study of classifications of non-Hodgkin's lymphomas: summary and description of a working formulation for clinical usage. The Non-Hodgkin's Lymphoma Pathologic Classification Project. Cancer 49: 2112-35
97. Salem Z, Nuwaiyri-Salti N, Ramlawi F et al (2003) Hepatitis C virus infection in Lebanese patients with B-cell non-Hodgkin's lymphoma. Eur J Epidemiol 18: 251-3
98. Sansonno D, De Vita S, Cornacchiulo V et al (1996) Detection and distribution of hepatitis C virus-related proteins in lymph nodes of patients with type II mixed cryoglobulinemia and neoplastic or non-neoplastic lymphoproliferation. Blood 88: 4638-45
99. Schollkopf C, Smedby KE, Hjalgrim H et al (2008) Hepatitis C infection and risk of malignant lymphoma. Int J Cancer 122: 1885-90
100. Seve P, Renaudier P, Sasco AJ et al (2004) Hepatitis C virus infection and B-cell non-Hodgkin's lymphoma: a cross-sectional study in Lyon, France. Eur J Gastroenterol Hepatol 16: 1361-5
101. Shariff S, Yoshida EM, Gascoyne RD et al (1999) Hepatitis C infection and B-cell non-Hodgkin's lymphoma in British Columbia: a cross-sectional analysis. Ann Oncol 10: 961-4
102. Shirin H, Davidovitz Y, Avni Y et al (2002) Prevalence of hepatitis C virus infection in patients with lymphoproliferative disorders. Isr Med Assoc J 4: 24-7
103. Silvestri F, Barillari G, Fanin R et al (1997) Hepatitis C virus infection among cryoglobulinemic and non-cryoglobulinemic B-cell non-Hodgkin's lymphomas. Haematologica 82: 314-7
104. Silvestri F, Pipan C, Barillari G et al (1996) Prevalence of hepatitis C virus infection in patients with lymphoproliferative disorders. Blood 87: 4296-301
105. Singer IO, Cumming RL and Hogg RB (1997) Is hepatitis C associated with non-Hodgkin's lymphoma? Leuk Lymphoma 26: 633-4

106. Sonmez M, Bektas O, Yilmaz M et al (2007) The relation of lymphoma and hepatitis B virus/hepatitis C virus infections in the region of East Black Sea, Turkey. Tumori 93: 536-9
107. Spinelli JJ, Lai AS, Krajden M et al (2008) Hepatitis C virus and risk of non-Hodgkin lymphoma in British Columbia, Canada. Int J Cancer 122: 630-3
108. Sung VM, Shimodaira S, Doughty AL et al (2003) Establishment of B-cell lymphoma cell lines persistently infected with hepatitis C virus in vivo and in vitro: the apoptotic effects of virus infection. J Virol 77: 2134-46
109. Takai S, Tsurumi H, Ando K et al (2005) Prevalence of hepatitis B and C virus infection in haematological malignancies and liver injury following chemotherapy. Eur J Haematol 74: 158-65
110. Takeshita M, Sakai H, Okamura S et al (2006) Prevalence of hepatitis C virus infection in cases of B-cell lymphoma in Japan. Histopathology 48: 189-98
111. Talamini R, Montella M, Crovatto M et al (2004) Non-Hodgkin's lymphoma and hepatitis C virus: a case-control study from northern and southern Italy. Int J Cancer 110: 380-5
112. Teng CJ, Liu HT, Liu CY et al (2011) Chronic hepatitis virus infection in patients with multiple myeloma: clinical characteristics and outcomes. Clinics (Sao Paulo) 66: 2055-61
113. Thalen DJ, Raemaekers J, Galama J et al (1997) Absence of hepatitis C virus infection in non-Hodgkin's lymphoma. Br J Haematol 96: 880-1
114. Timuraglu A, Colak D, Ogunc D et al (1999) Hepatitis C virus association with non-Hodgkin's lymphoma. Haematologia (Budap) 29: 301-4
115. Tkoub EM, Haioun C, Pawlotsky JM et al (1998) Chronic hepatitis C virus and gastric MALT lymphoma. Blood 91: 360
116. Tursi A, Brandimante G, Chiarelli F et al (2002) Detection of HCV RNA in gastric mucosa-associated lymphoid tissue by in situ hybridization: evidence of a new extrahepatic localization of HCV with increased risk of gastric malt lymphoma. Am J Gastroenterol 97: 1802-6
117. Udomsakdi-Auewarakul C, Auewarakul P, Sukpanichnant S et al (2000) Hepatitis C virus infection in patients with non-Hodgkin lymphoma in Thailand. Blood 95: 3640-1
118. Vajdic CM, Grulich AE, Kaldor JM et al (2006) Specific infections, infection-related behavior, and risk of non-Hodgkin lymphoma in adults. Cancer Epidemiol Biomarkers Prev 15: 1102-8
119. Vallisa D, Bernuzzi P, Arcaini L et al (2005) Role of anti-hepatitis C virus (HCV) treatment in HCV-related, low-grade, B-cell, non-Hodgkin's lymphoma: a multicenter Italian experience. J Clin Oncol 23: 468-73
120. Vallisa D, Berte R, Rocca A et al (1999) Association between hepatitis C virus and non-Hodgkin's lymphoma, and effects of viral infection on histologic subtype and clinical course. Am J Med 106: 556-60
121. Varma S, Menon MC, Garg A et al (2011) Hepatitis C virus infection among patients with non-Hodgkin's lymphoma in northern India. Hepatol Int 5: 688-92
122. Veneri D, Franchini M, Zanotti R et al (2007) Prevalence of hepatitis C virus infection among patients with lymphoproliferative disorders: a single center survey. Am J Hematol 82: 1031
123. Viswanatha DS and Dogan A (2007) Hepatitis C virus and lymphoma. J Clin Pathol 60: 1378-83
124. Yamac K, Aydemir S, Ozturk G et al (2000) Hepatitis C infection in lymphoma patients in a Turkish center. Eur J Epidemiol 16: 685
125. Yenice N, Gulluk F, Arican N et al (2003) HCV prevalence in Hodgkin and non-Hodgkin lymphoma cases. Turk J Gastroenterol 14: 173-6
126. Yoshida EM, Shariff S and Shenkier T (2000) Hepatitis C and B-cell non-Hodgkin's lymphoma: a geographically variable association? Am J Med 108: 350-1
127. Yoshikawa M, Imazu H, Ueda S et al (1997) Prevalence of hepatitis C virus infection in patients with non-Hodgkin's lymphoma and multiple myeloma. A report from Japan. J Clin Gastroenterol 25: 713-4
128. Yu SC and Lin CW (2013) Early-stage splenic diffuse large B-cell lymphoma is highly associated with hepatitis C virus infection. Kaohsiung J Med Sci 29: 150-6
129. Zucca E, Roggero E, Maggi-Solca N et al (2000) Prevalence of Helicobacter pylori and hepatitis C virus infections among non-Hodgkin's lymphoma patients in Southern Switzerland. Haematologica 85: 147-53
130. Zuckerman E, Zuckerman T, Levine AM et al (1997) Hepatitis C virus infection in patients with B-cell non-Hodgkin lymphoma. Ann Intern Med 127: 423-8

Biliary ducts/cholangiocarcinoma

1. Abdel Wahab M, Mostafa M, Salah T et al (2007) Epidemiology of hilar cholangiocarcinoma in Egypt: single center study. Hepatogastroenterology 54: 1626-31
2. Barusrux S, Nanok C, Puthisawas W et al (2012) Viral hepatitis B, C infection and genotype distribution among cholangiocarcinoma patients in northeast Thailand. Asian Pac J Cancer Prev 13 Suppl: 83-7
3. Choi D, Lim JH, Lee KT et al (2006) Cholangiocarcinoma and Clonorchis sinensis infection: a case-control study in Korea. J Hepatol 44: 1066-73
4. Donato F, Gelatti U, Tagger A et al (2001) Intrahepatic cholangiocarcinoma and hepatitis C and B virus infection, alcohol intake, and hepatolithiasis: a case-control study in Italy. Cancer Causes Control 12: 959-64
5. El-Serag HB, Engels EA, Landgren O et al (2009) Risk of hepatobiliary and pancreatic cancers after hepatitis C virus infection: A population-based study of U.S. veterans. Hepatology 49: 116-23
6. Fwu CW, Chien YC, You SL et al (2011) Hepatitis B virus infection and risk of intrahepatic cholangiocarcinoma and non-Hodgkin lymphoma: a cohort study of parous women in Taiwan. Hepatology 53: 1217-25
7. Hai S, Kubo S, Yamamoto S et al (2005) Clinicopathologic characteristics of hepatitis C virus-associated intrahepatic cholangiocarcinoma. Dig Surg 22: 432-9
8. Jarnagin WR, Weber S, Tickoo SK et al (2002) Combined hepatocellular and cholangiocarcinoma: demographic, clinical, and prognostic factors. Cancer 94: 2040-6
9. Kalaitzakis E, Gunnarsdottir SA, Josefsson A et al (2011) Increased risk for malignant neoplasms among patients with cirrhosis. Clin Gastroenterol Hepatol 9: 168-74
10. Koshiol J, Pawlish K, Goodman MT et al (2014) Risk of hepatobiliary cancer after solid organ transplant in the United States. Clin Gastroenterol Hepatol 12: 1541-9 e3
11. Kuper H, Lagiou P, Mucci LA et al (2001) Risk factors for cholangiocarcinoma in a low risk Caucasian population. Soz Praventivmed 46: 182-5
12. Lee CH, Chang CJ, Lin YJ et al (2009) Viral hepatitis-associated intrahepatic cholangiocarcinoma shares common disease processes with hepatocellular carcinoma. Br J Cancer 100: 1765-70
13. Lee MH, Yang HI, Lu SN et al (2012) Chronic hepatitis C virus infection increases mortality from hepatic and extrahepatic diseases: a community-based long-term prospective study. J Infect Dis 206: 469-77
14. Lee TY, Lee SS, Jung SW et al (2008) Hepatitis B virus infection and intrahepatic cholangiocarcinoma in Korea: a case-control study. Am J Gastroenterol 103: 1716-20
15. Li T, Li D, Cheng L et al (2010) Epithelial-mesenchymal transition induced by hepatitis C virus core protein in cholangiocarcinoma. Ann Surg Oncol 17: 1937-44
16. Liu X, Zou S and Qiu F (2002) [Pathogenesis of hilar cholangiocarcinoma and infection of hepatitis virus]. Zhonghua Wai Ke Za Zhi 40: 420-2
17. Lu H, Ye MQ, Thung SN et al (2000) Detection of hepatitis C virus RNA sequences in cholangiocarcinomas in Chinese and American patients. Chin Med J (Engl) 113: 1138-41
18. Mohammad-Alizadeh AH, Ghobakhlou M, Shalmani HM et al (2012) Cholangiocarcinoma: an-eight-year experience in a tertiary-center in Iran. Asian Pac J Cancer Prev 13: 5381-4
19. Nuzzo G, Giuliante F, Ardito F et al (2010) Intrahepatic cholangiocarcinoma: prognostic factors after liver resection. Updates Surg 62: 11-9
20. Palmer WC and Patel T (2012) Are common factors involved in the pathogenesis of primary liver cancers? A meta-analysis of risk factors for intrahepatic cholangiocarcinoma. J Hepatol 57: 69-76
21. Parkin DM, Srivatanakul P, Khlat M et al (1991) Liver cancer in Thailand. I. A case-control study of cholangiocarcinoma. Int J Cancer 48: 323-8
22. Peng NF, Li LQ, Qin X et al (2011) Evaluation of risk factors and clinicopathologic features for intrahepatic cholangiocarcinoma in Southern China: a possible role of hepatitis B virus. Ann Surg Oncol 18: 1258-66
23. Portolani N, Baiocchi GL, Coniglio A et al (2008) Intrahepatic cholangiocarcinoma and combined hepatocellular-cholangiocarcinoma: a Western experience. Ann Surg Oncol 15: 1880-90

24. Qu Z, Cui N, Qin M et al (2012) Epidemiological survey of biomarkers of hepatitis virus in patients with extrahepatic cholangiocarcinomas. Asia Pac J Clin Oncol 8: 83-7
25. Sempoux C, Jibara G, Ward SC et al (2011) Intrahepatic cholangiocarcinoma: new insights in pathology. Semin Liver Dis 31: 49-60
26. Shaib YH, El-Serag HB, Davila JA et al (2005) Risk factors of intrahepatic cholangiocarcinoma in the United States: a case-control study. Gastroenterology 128: 620-6
27. Shaib YH, El-Serag HB, Nooka AK et al (2007) Risk factors for intrahepatic and extrahepatic cholangiocarcinoma: a hospital-based case-control study. Am J Gastroenterol 102: 1016-21
28. Shin HR, Lee CU, Park HJ et al (1996) Hepatitis B and C virus, Clonorchis sinensis for the risk of liver cancer: a case-control study in Pusan, Korea. Int J Epidemiol 25: 933-40
29. Shin HR, Oh JK, Masuyer E et al (2010) Comparison of incidence of intrahepatic and extrahepatic cholangiocarcinoma--focus on East and South-Eastern Asia. Asian Pac J Cancer Prev 11: 1159-66
30. Shin HR, Oh JK, Masuyer E et al (2010) Epidemiology of cholangiocarcinoma: an update focusing on risk factors. Cancer Sci 101: 579-85
31. Shirakawa H (1996) [Analysis of hepatitis C virus (HCV) genotypes in hepatocellular carcinoma]. Hokkaido Igaku Zasshi 71: 677-88
32. Songsivilai S, Dharakul T and Kanistanon D (1996) Hepatitis C virus genotypes in patients with hepatocellular carcinoma and cholangiocarcinoma in Thailand. Trans R Soc Trop Med Hyg 90: 505-7
33. Srivatanakul P, Honjo S, Kittiwatanachot P et al (2010) Hepatitis viruses and risk of cholangiocarcinoma in northeast Thailand. Asian Pac J Cancer Prev 11: 985-8
34. Taguchi J, Nakashima O, Tanaka M et al (1996) A clinicopathological study on combined hepatocellular and cholangiocarcinoma. J Gastroenterol Hepatol 11: 758-64
35. Tanaka M, Tanaka H, Tsukuma H et al (2010) Risk factors for intrahepatic cholangiocarcinoma: a possible role of hepatitis B virus. J Viral Hepat 17: 742-8
36. Tao LY, He XD, Qu Q et al (2010) Risk factors for intrahepatic and extrahepatic cholangiocarcinoma: a case-control study in China. Liver Int 30: 215-21
37. Tomimatsu M, Ishiguro N, Taniai M et al (1993) Hepatitis C virus antibody in patients with primary liver cancer (hepatocellular carcinoma, cholangiocarcinoma, and combined hepatocellular-cholangiocarcinoma) in Japan. Cancer 72: 683-8
38. Torbenson M, Yeh MM and Abraham SC (2007) Bile duct dysplasia in the setting of chronic hepatitis C and alcohol cirrhosis. Am J Surg Pathol 31: 1410-3
39. Uenishi T, Nagano H, Marubashi S et al (2014) The long-term outcomes after curative resection for mass-forming intrahepatic cholangiocarcinoma associated with hepatitis C viral infection: a multicenter analysis by Osaka Hepatic Surgery Study Group. J Surg Oncol 110: 176-81
40. Welzel TM, Graubard BI, El-Serag HB et al (2007) Risk factors for intrahepatic and extrahepatic cholangiocarcinoma in the United States: a population-based case-control study. Clin Gastroenterol Hepatol 5: 1221-8
41. Wu TT, Levy M, Correa AM et al (2009) Biliary intraepithelial neoplasia in patients without chronic biliary disease: analysis of liver explants with alcoholic cirrhosis, hepatitis C infection, and noncirrhotic liver diseases. Cancer 115: 4564-75
42. Yamamoto M, Takasaki K, Nakano M et al (1998) Minute nodular intrahepatic cholangiocarcinoma. Cancer 82: 2145-9
43. Yamamoto S, Kubo S, Hai S et al (2004) Hepatitis C virus infection as a likely etiology of intrahepatic cholangiocarcinoma. Cancer Sci 95: 592-5
44. Yano Y, Yamamoto J, Kosuge T et al (2003) Combined hepatocellular and cholangiocarcinoma: a clinicopathologic study of 26 resected cases. Jpn J Clin Oncol 33: 283-7
45. Yin F and Chen B (1998) Detection of hepatitis C virus RNA sequences in hepatic portal cholangiocarcinoma tissue by reverse transcription polymerase chain reaction. Chin Med J (Engl) 111: 1068-70
46. Zhang H, Tang Y and Lu X (1996) [Detection of hepatitis B virus DNA and hepatitis C virus RNA in human hepatocellular carcinoma by polymerase chain reaction]. Zhonghua Bing Li Xue Za Zhi 25: 70-2
47. Zhou Y, Zhao Y, Li B et al (2012) Hepatitis viruses infection and risk of intrahepatic cholangiocarcinoma: evidence from a meta-analysis. BMC Cancer 12: 289

48. Zhou YM, Yin ZF, Yang JM et al (2008) Risk factors for intrahepatic cholangiocarcinoma: a case-control study in China. World J Gastroenterol 14: 632-5
49. Zou SQ, Liu XF, Guo RX et al (2003) [The retrospective analysis of HBV and HCV infection in cholangiocarcinoma]. Zhonghua Wai Ke Za Zhi 41: 417-9
50. Zuo HQ, Yan LN, Zeng Y et al (2007) Clinicopathological characteristics of 15 patients with combined hepatocellular carcinoma and cholangiocarcinoma. Hepatobiliary Pancreat Dis Int 6: 161-5

Kidney

1. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
2. Budakoglu B, Aksoy S, Arslan C et al (2012) Frequency of HCV infection in renal cell carcinoma patients. Med Oncol 29: 1892-5
3. Gonzalez HC, Lamerato L, Rogers CG et al (2015) Chronic Hepatitis C Infection as a Risk Factor for Renal Cell Carcinoma. Dig Dis Sci
4. Gordon SC, Moonka D, Brown KA et al (2010) Risk for renal cell carcinoma in chronic hepatitis C infection. Cancer Epidemiol Biomarkers Prev 19: 1066-73
5. Hofmann JN, Torner A, Chow WH et al (2011) Risk of kidney cancer and chronic kidney disease in relation to hepatitis C virus infection: a nationwide register-based cohort study in Sweden. Eur J Cancer Prev 20: 326-30
6. Malaguarnera M, Gargante MP, Risino C et al (2006) Hepatitis C virus in elderly cancer patients. Eur J Intern Med 17: 325-9
7. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
8. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2:

Pancreas

1. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
2. Ben Q, Li Z, Liu C et al (2012) Hepatitis B virus status and risk of pancreatic ductal adenocarcinoma: a case-control study from China. Pancreas 41: 435-40
3. Chang MC, Chen CH, Liang JD et al (2014) Hepatitis B and C viruses are not risks for pancreatic adenocarcinoma. World J Gastroenterol 20: 5060-5
4. El-Serag HB, Engels EA, Landgren O et al (2009) Risk of hepatobiliary and pancreatic cancers after hepatitis C virus infection: A population-based study of U.S. veterans. Hepatology 49: 116-23
5. Fiorino S, Chili E, Bacchi-Reggiani L et al (2013) Association between hepatitis B or hepatitis C virus infection and risk of pancreatic adenocarcinoma development: a systematic review and meta-analysis. Pancreatology 13: 147-60
6. Hassan MM, Li D, El-Deeb AS et al (2008) Association between hepatitis B virus and pancreatic cancer. J Clin Oncol 26: 4557-62
7. Hong SG, Kim JH, Lee YS et al (2010) [The relationship between hepatitis B virus infection and the incidence of pancreatic cancer: a retrospective case-control study]. Korean J Hepatol 16: 49-56
8. Huang J, Magnusson M, Torner A et al (2013) Risk of pancreatic cancer among individuals with hepatitis C or hepatitis B virus infection: a nationwide study in Sweden. Br J Cancer 109: 2917-23
9. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
10. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755

11. Woo SM, Joo J, Lee WJ et al (2013) Risk of pancreatic cancer in relation to ABO blood group and hepatitis C virus infection in Korea: a case-control study. J Korean Med Sci 28: 247-51
12. Xing S, Li ZW, Tian YF et al (2013) Chronic hepatitis virus infection increases the risk of pancreatic cancer: a meta-analysis. Hepatobiliary Pancreat Dis Int 12: 575-83
13. Xu JH, Fu JJ, Wang XL et al (2013) Hepatitis B or C viral infection and risk of pancreatic cancer: a meta-analysis of observational studies. World J Gastroenterol 19: 4234-41
14. Xu P, Huang Q, Liu C et al (2011) Risk factors for pancreatic cancer: a case-control study. Cancer (Chinese J) 31: 653-657
15. Zhu F, Li HR, Du GN et al (2011) Chronic hepatitis B virus infection and pancreatic cancer: a case-control study in southern China. Asian Pac J Cancer Prev 12: 1405-8

Thyroid

1. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
2. Antonelli A, Ferri C, Fallahi P (1999) Thyroid cancer in patients with hepatitis C infection. JAMA 281:1588
3. Antonelli A, Ferri C, Fallahi P et al (2002) Thyroid cancer in HCV-related mixed cryoglobulinemia patients. Clin Exp Rheumatol 20: 693-6
4. Antonelli A, Ferri C, Fallahi P et al (2007) Thyroid cancer in HCV-related chronic hepatitis patients: a case-control study. Thyroid 17: 447-51
5. Giordano TP, Henderson L, Landgren O et al (2007) Risk of non-Hodgkin lymphoma and lymphoproliferative precursor diseases in US veterans with hepatitis C virus. JAMA 297: 2010-7
6. Malaguarnera M, Gargante MP, Risino C et al (2006) Hepatitis C virus in elderly cancer patients. Eur J Intern Med 17: 325-9
7. Montella M, Crispo A, Pezzullo L, et al (2000) Is hepatitis C virus infection associated with thyroid cancer? A case-control study. Int J Cancer 87:611-612
8. Montella M, Crispo A, de Bellis G et al (2001) HCV and cancer: a case-control study in a high-endemic area. Liver 21: 335-41
9. Montella M, Pezzullo L, Crispo A et al (2003) Risk of thyroid cancer and high prevalence of hepatitis C virus. Oncol Rep 10: 133-6
10. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
11. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755

Breast

1. Bruno G, Andreozzi P, Graf U et al (1999) Hepatitis C virus: a high risk factor for a second primary malignancy besides hepatocellular carcinoma. Fact or fiction? Clin Ter 150: 413-8
2. Gower E, Estes C, Blach S et al (2014) Global epidemiology and genotype distribution of the hepatitis C virus infection. J Hepatol 61: S45-57
3. Larrey D, Bozonnat MC, Kain I et al (2010) Is chronic hepatitis C virus infection a risk factor for breast cancer? World J Gastroenterol 16: 3687-91
4. Malaguarnera M, Gargante MP, Risino C et al (2006) Hepatitis C virus in elderly cancer patients. Eur J Intern Med 17: 325-9
5. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
6. Su FH, Chang SN, Chen PC et al (2011) Association between chronic viral hepatitis infection and breast cancer risk: a nationwide population-based case-control study. BMC Cancer 11: 495
7. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2:

Lung

1. Malaguarnera M, Gargante MP, Risino C et al (2006) Hepatitis C virus in elderly cancer patients. Eur J Intern Med 17: 325-9
2. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
3. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755

Stomach

1. Amin J, Gidding H, Gilbert G et al (2004) Hepatitis C prevalence--a nationwide serosurvey. Commun Dis Intell Q Rep 28: 517-21
2. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755

Colon

1. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
2. Bruno G, Andreozzi P, Graf U et al (1999) Hepatitis C virus: a high risk factor for a second primary malignancy besides hepatocellular carcinoma. Fact or fiction? Clin Ter 150: 413-8
3. Malaguarnera M, Gargante MP, Risino C et al (2006) Hepatitis C virus in elderly cancer patients. Eur J Intern Med 17: 325-9
4. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
5. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755

Skin/oral

1. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
2. Eftekharian A, Khajavi M, Shokoofi S et al (2012) Hepatitis C virus in patients with squamous cell carcinoma of the head and neck in Iran: is there any relation? Eur Arch Otorhinolaryngol 269: 2571-3
3. Gandolfo S, Richiardi L, Carrozzo M et al (2004) Risk of oral squamous cell carcinoma in 402 patients with oral lichen planus: a follow-up study in an Italian population. Oral Oncol 40: 77-83
4. Nagao Y, Sata M, Noguchi S et al (2000) Detection of hepatitis C virus RNA in oral lichen planus and oral cancer tissues. J Oral Pathol Med 29: 259-66
5. Nagao Y, Sata M, Tanikawa K et al (1995) High prevalence of hepatitis C virus antibody and RNA in patients with oral cancer. J Oral Pathol Med 24: 354-60
6. Nobles J, Wold C, Fazekas-May M et al (2004) Prevalence and epidemiology of hepatitis C virus in patients with squamous cell carcinoma of the head and neck. Laryngoscope 114: 2119-22
7. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
8. Su FH, Chang SN, Chen PC et al (2012) Positive association between hepatitis C infection and oral cavity cancer: a nationwide population-based cohort study in Taiwan. PLoS One 7: e48109
9. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755
10. Takata Y, Takahashi T and Fukuda J (2002) Prevalence of hepatitis virus infection in association with oral diseases requiring surgery. Oral Dis 8: 95-9

Bladder

1. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
2. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
3. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755

Prostate

1. Amin J, Dore GJ, O'Connell DL et al (2006) Cancer incidence in people with hepatitis B or C infection: a large community-based linkage study. J Hepatol 45: 197-203
2. Bruno G, Andreozzi P, Graf U et al (1999) Hepatitis C virus: a high risk factor for a second primary malignancy besides hepatocellular carcinoma. Fact or fiction? Clin Ter 150: 413-8
3. Malaguarnera M, Gargante MP, Risino C et al (2006) Hepatitis C virus in elderly cancer patients. Eur J Intern Med 17: 325-9
4. Omland LH, Farkas DK, Jepsen P et al (2010) Hepatitis C virus infection and risk of cancer: a population-based cohort study. Clin Epidemiol 2: 179-86
5. Swart A, Burns L, Mao L et al (2012) The importance of blood-borne viruses in elevated cancer risk among opioid-dependent people: a population-based cohort study. BMJ Open 2(5): e001755

SUPPLEMENTARY MATERIAL AND METHODS

The used MESH terms and keywords were: 'chronic hepatitis C', *'pancreatic neoplasm', 'kidney neoplasm', 'renal neoplasm', 'biliary duct neoplasm', 'mouth neoplasm', 'oral neoplasm', 'skin neoplasm', 'haematological neoplasm', 'breast neoplasm', 'lung neoplasm', 'thyroid neoplasm', 'prostate neoplasm'. In addition, the following key words were searched: 'pancreatic malignancy', 'pancreatic cancer', 'pancreatic carcinoma', 'pancreatic adenocarcinoma', 'pancreatic tumor' 'kidney cancer', 'kidney carcinoma', 'kidney malignancy, 'kidney tumor', 'renal cancer', 'renal carcinoma', 'renal malignancy', 'renal tumor', 'cholangiocarcinoma', 'intrahepatic cholangiocarcinoma', 'extra-hepatic cholangiocarcinoma' 'thyroid cancer', 'thyroid carcinoma', 'thyroid tumor', 'thyroid malignancy', 'prostate carcinoma', 'prostate cancer', 'prostate carcinoma', 'prostate tumor', 'prostate malignancy', 'oral cancer', 'oral carcinoma', 'oral tumor', 'oral malignancy', 'skin cancer', 'skin carcinoma', 'skin tumor', 'skin malignancy', 'lymphomas', 'non-*

Hodgkin's lymphomas', 'Hodgkin's lymphomas', 'hematologic malignancies', 'hematologic neoplasm', 'hematologic tumor'.

The PubMed 'related articles' features and the reference lists of retrieved articles were also searched to find additional pertinent studies. If a study was considered potentially eligible by either of the two reviewers, the full-text of this study was further evaluated. Full-text assessment was performed according to eligibility criteria developed to systematically include studies into this review. The aim of our paper was to assess the possible association between the HCV infection alone and different types of human cancers. Therefore, we excluded all trials designed with the primary aim to evaluate the risk of malignancies in patients with HCV/HIV co-infection or we did not consider the subgroup of individuals with HIV co-infection in studies carried out to assess the association between HCV and different types of human cancers. In particular, this possibility occurred in cohort studies. However, in most of trials, HIV co-infected patients were excluded in advance. Furthermore, most of identified studies assessed the association of both HCV and HBV infection and risk of the different types of human malignancies. In this case also we did not considered the cohort of HBV positive individuals or we reported the number of HBV/HCV co-infected patients. We also searched and considered in our paper systematic reviews and meta-analyses, concerning each of the above mentioned tumours, when they were available.

SUPPLEMENTARY TABLES

Supplementary Table 1. Characteristics of available studies, reported in English, designed to assess the association between HCV infection and Haematopoietic Malignancies (HM)

Author/ Journal/ Publication Year	Country	Study Design/	Diagnosis	Type of HCV test	HCV positive HM/ total HM (%)	Control Source	HCV positive controls/ total controls (%)	Matching factors	Percentage of HCV-positive cases with 95 % CI	Main conclusions
Akdogan M Turk J Gastroenterol 1998	Turkey	Case series study with control group Period: NR	All lymphomas : NHLs: 30 HL: 18NHLs NHL classification :	IIG ELISA HCV-RNA	a) NHL : 4/30 (13.3%) b) Patients with Hodgkin Lymphoma	b) Healthy blood donors	a) 17/9488 (0,8%)	NR	13.3 (3.8-30.7)	Increased prevalence of HCV persistent infection in patients with NHL, but not in patients with HL, in

			Working Formulation							comparison with general population
Amin J, J Hepatol 2006	Australia	Community-based cohort-study Period: 1990-2002	NHLs Cohort of HCV positive patients:75,834, Cohort of HBV/HCV positive patients:2604 Incidence of LNHs observed in the study cohort was compared to expected incidence derived from New South Wales population cancer rates by calculating standardised incidence ratios	Identification of LNHs cases by means of ICD-10-diagnosis codes (C82–C85 and C96)	Individuals with HCV infection: 75834 LNH cases detected :33	Incidence observed in the study cohort was compared to expected incidence derived from NSW population cancer rates by calculating standardised incidence ratios (SIR)	SIR : 0.9 (0.6-1.2)	NR	0.04 (0.03-0.05)	In HCV infection group no increased overall risk of NHL-cell lymphoma, but, a number of B-cell NHLs (diffuse NHL, immunoproliferative malignancies and chronic lymphocytic leukaemias) had SIRs greater than one.
Anderson LA Epidemiol Biomarkers Prev. 2008	USA	Population-based nested case-control study of hematopoietic malignancies Period: 1993 and 2002	Subjects with hematopoietic malignancies identified, using SEER-Medicare data. SEER program: a cancer surveillance program supported by the National Cancer Institute and covering about 25% of USA population NHL classification : World Health Organization classification Myeloproli	Hemato-poietic malignancies identified using the third edition of the International Classification of Diseases for Oncology (ICD-O3) morphology codes 9590–9989	195/ 61,464 (0.3%) cases with Hemato-poietic malignancies identified **NHLs: 103/33,940 (0.3%)** DLBCL: 34/10,144 (0.3%) BL: 2 /197 (1.5%) MZL: 12/1,908 (0.6%) FL: 19 / 4,491 (0.4%) CLL: 23/10,170 (0.2%) LL: 2/1,148 HL: 3/1,155 (0.3%) PCM: 31/9,995 (0.3%) **Myeloid neoplasm: 47/11,945 (0.4%)** AML:	Controls were identified by means of Medicare, a federally funded program administered by the Centres for Medicare and Medicaid Services, For each included case, two controls were selected at random from the 5% random sample of Medicare beneficiari	264/122,531 (0.2%) population-based controls identified	Gender, age and year (1993–2002)	0.3 (0.2-0.4)	Association between HCV and elevated risk of NHLs and acute myeloid leukemia. HCV may induce lymphoproliferative malignancies through chronic immune stimulation.

			ferative malignancies classification: acute- and chronic myeloid leukaemia, myelodysplastic syndrome, chronic myeloproliferative disease		23/6068 (0.4%) CML: 1/1528 (0.1%) MS: 18/3084 (0.6%) CMD: 1/1346 (0.1%)	es				
Arcaini L Clinical Lymphoma, Myeloma & Leukemia, 2011	Italy	Case series study with control group Period: NR	Splenic MZLs NHL classification : World Health Organization (WHO) classification	NR	25/92 Splenic MZL patients (27.2%)	Patients (122) with WMc 66/122 subjects with HCV markers	6 /66 WMc patients (9%)	NR	27.2 (18.1-36.2)	Despite similar outcomes among SMZL and WM, SMZL appears as a disease with distinct clinical and histologic characteristics, and a peculiar association with HCV infection
Arican A Med Oncol 2000	Turkey	Case series Period: February-October 1997	NHLs Low-grade: 12 (27%) Intermediate grade: 24 (55%) High-grade: 8 (18%) NHL classification : Working Formulation	III G MEIA HCV-RNA	2/44 (4.5%)	NR	NR	NR	4.5 (0-10.7)	No association between HCV chronic infection and NHL development in this study. The prevalence of HCV infection reported to be 0.3-1.5% in healthy Turkish-blood donors in previous studies b1.5% in healthy Turkish-blood donors in previous

Aviles A Med Oncol 2003	Mexico	Case-control study Period: January 1997-December 1999	B-cell NHLs: 416 Diffuse large cell: 236 Follicular: 97 Marginal B-cell zone: 83 NHL classification : World Health Organization (WHO) classification	III G ELISA III G RIBA HCV-RNA	B-cell NHLs 2/416 (0.5%)	Group 1: 682 first-degree relatives (spouses, children, fathers, and brothers of the patient) living in the neighboring area of the patient. Group 2: 832 healthy blood donors, donating during the same period of time at the central blood bank. Group 3: Neoplastic disease group, with 408 patients with solid tumors, breast cancer:127 colon cancer :94 gastric cancer;79 lung cancer :98 Group 4: 353	Prevalence of HCV equal to: a) 0 among first-degree relatives of patients b) 0.12 (0.02–0.88) among healthy blood donors c) 0.56, (0.28–0.75). among patients with solid tumors d) No patients with HCV chronic liver disease developed malignant lymphoma in a median follow-up of 7.9 yr.	Group 2: sex and age (+ 5 yr)	0.5 (0-1.1)	Association between HCV infection and development of malignant lymphoma represents an hazardous observation, the close association reported in areas with a higher prevalence of HCV infection has to be considered with caution, because other epidemiological factors have not been considered, such as a high prevalence of HCV infection compared to other areas.

						patients with HCV-positive related chronic liver disease				
Bauduer F Hematol Cell Ther 1999	Southwestern France	Case series Period: January 1995-June 1998	NHLs: 136 subjects Bcell-NHLs: 110 patients NHL classification : Revised European American Lymphoma (REAL) histological scheme	III G ELISA III G RIBA HCV-RNA	2/136 (1.5%)	NR	NR	NR	1.5 (0.-3.4)	No evidence of relationship between HCV and NHLs
Besson C J Clin Oncol. 2006	France	Case control Period: March 1993 - June 2002	B-NHL (DLBCL) NHL classification : Working Formulation	HCV serology results need for inclusion in the LNH98 program but not mandatory in the LNH 93 program ELISA II G HCV-RNA	26/5586 (0.5%)	a) HCV negative patients with DLCL enrolled in the present study b) individuals with DLCL randomly chosen among HCV-negative patients included in the GELA program	a) 5586 b) 35	Age, sex, arm protocol, stage, and performance status	0.5 (0.29-0.64)	HCV-positive patients with DLBCL differ from other patients both at presentation and during chemotherapy. Specific protocols evaluating antiviral therapy should be designed for these patients.
Bianco E Haematologica 2004	Italy (10 Hospital Centres)	Italian multi-center case-control study Period: January 1998 - February 2001	All lymphomas: 637 HD: 157 CLL: 100 ALL: 54 MM: 107 AML:140 CML: 49 T-NHL: 30 NHL classificati	III G ELISA III G RIBA HCV-RNA	44/637 (6.9%) HD: 5/157 (3.2%) CLL:, 9/100 (9%) ALL,: 4/54 (7.6%) MM: 5/107 (4.7%) AML,:11/140 (7.9%) CML: 6/49 (12.2%)	Patients from other departments of the same hospitals: the departments of dentistry, dermatology, general	22/396 (5.6%)	Gender, age (ble, in ten-year groups), level of education, and place of birth.	6.9 (4.9-8.8)	Possible association of HCV infection not only with B-NHL but also with some other lymphoid and myeloid malignancies, however no

			on for T-NHLs:: REAL/WHO classification		T-NHL 4/30 (13.8%)	surgery, gynecology, internal medicine, ophthalmology, orthopedics, otorhinolaryngology, and traumatology				definitive significant results, due to the absence of large groups of patients to confirm this assumption
Bronowicki J-P Hepatology 2003	France	Case records Data obtained from the hepatology gastroenterology, hematology, internal medicine and pathology departments of 64 French hospitals Period: 1992 -1999	All PLL: 31 cases, 27/ 31 patients with a B-cell lymphoma: -DLBCL: 22, -BL: 1 , -EMZBL of mucosa-associated lymphoid tissue type:3, unclassified, small B-cell lymphoma: 1, T-cell lymphomas: 4 NHL classification : World Health Organization (WHO) classification	NR	HCV-test available for 28 subjects, HCV test available in 23 patients with B-cell PLL 1 HCV positive patient with peripheral T-cell lymphoma 5/23 (21.7%)	NR	NR	NR	21.7 (7.5-43.7)	This study confirms the rarity of PLL and demonstrates an increased prevalence of HCV infection
Cavanna L Haematologica 1995	Italy	Case-control study Period: 1985-1990	All LPDs: 300 patients Anti-HCV positive patients 57/300 (19.7%) NHLs:	II G ELISA II G RIBA	NHL: 38/150 (25.3%) HL: 2/20 (10%) CLL: 2/40 (5%) Plasma cell discrasias:	Blood donors	53/3,108 (1.7%)	Age and sex	25.3 (18.3-32.3)	High prevalence of anti-HCV antibodies among patients with lymphoprol

			150; HL: 20 CLL: 40 Plasma cell discrasias: 90		15/90(16%)					ipherative disorders as compared with the control group of healthy blood donors.
Caviglia GP J Gastroenterol Hepatol. 2014	Italy (Turin)	Cohort study Period: January 2006 - December 2013	1313 patients with chronic HCV hepatitis 121 patients with extra-hepatic manifestations: B-NHL: 41/1323 (3.1%) MCS: 25/1323 (1.9%) MGUS: 55/1323 (4.2%) NHL classification : World Health Organization (WHO) classification	NR	B-cell NHL: 41 MZL: 15 (36.6%) , had DLBCL: 10 (24.4%) , FL: 4 (9.8%) LPL;1 (2.4%), MM: 1 (2.4%), CLL: 1 (2.4%) and B-NHL not otherwise specified: 9 (22%)	Controls selected on the basis of the absence of extra-hepatic manifestation of HCV infection	130 HCV positive subjects without extrahepatic manifestation	Age, gender and HCV genotype	3.1 (2.2-4)	Cirrhosis is an additional risk factor for the development of lymphoproliferative disorders in patients with chronic HCV infection
Chindamo MC Oncol Rep 2002	Brazil	Case series with control group Period: May 1995 - September 1998	All lymphomas : 207 -HL: 67 -B-NHL: 87 -T-NHL: 22 -CLL: 31 NHL classification : Revised European American Lymphoma (REAL) histological scheme	II and III G ELISA HCV-RNA	B-cell NHL: 8/87 (9.2%)	a) Blood donors b) Other haematological malignancies (Hodgkin's disease and chronic lymphocytic leukaemia)	a) 472/39371 (1.2%) b) 2/98 (2%)	NR	9.2 (3.1-15.2)	Association between HCV infection and NHLs
Chuang SS J Clin Pathol. 2010	Taiwan	Case-control study Period: January 2004 - December 2008	All malignancies: 346 -HL: 25 (3HCV+) -B-NHL: 321 (DLBCL, FC CLL, MZL,BL,ot	II and III G EIA HCV-RNA	All NHL: 35/321 (11%) B-cell NHL: 34/266 (12.8%) (3 /38 with HBV coinfection).	Healthy Taiwanese subjects	15/824 (1.8%)	Age and sex	12.8 (8.7-16.8)	The incidence of HCV infection among lymphoma patients in Taiwan was

			hers) -T- or NK/T- cell NHL: 55 NHL classification : World Health Organization (WHO) classification							significantly higher than that for healthy controls. Non-MALT (nodal and splenic) MZL was the only group significantly associated with HCV
Cocco P Int J Hematol 2008	Italy, (Cagliari and Nuoro)	Case-control study Period: -February 1999-October 2002 -January 2002 –July 2003	All malignancies (277): -HL: 13 -NHL: 264 (DLBCL, FC CLL, MZL, MM, T-cell NHL, others) NHL classification : World Health Organization (WHO) classification	III G MEIA HCV-RNA	a) All B cell- NHL: 20/237 (8.4%) b) NHLs (excluding CLL and MM): 15/177 (8.5%)	Randomly selected controls from population registrars	9/217 (4.1%)	Age, gender, andprovince of residence	a) 8.4 (4.9-11.9) b) 8.5 (4.3-12.5)	Acute or chronic hepatitis C is associated with a consistent risk increase in all lymphoma subtypes, but follicular lymphoma
Collier JD Hepatology 1999	Canada (Toronto)	Case series with control group Period: February 1997 and May 1997	B-cell NHLs: 100 NHL classification : Working Formulation	III G ELISA III G RIBA HCV-RNA	1/100 (1%)	In-Hospital patients with nonhematologic malignancies, treated at the Princess Margaret Hospital	1/100 (1%)	NR	1 (0-3)	No Association Between Hepatitis C and B-Cell Lymphoma
Cowgill KD Int J Epidemiol 2004	Egypt, (Cairo)	Case-control study Period: October 1999- and January 2003	B-cell NHL: 220 NHL classification: NR	II G EIA HCV-RNA)	Total: 106/220 (48.1%) a) anti-HCV+/RNA- 12 /220 (5.4%) b) anti-HCV+/RNA+ 94 /220 (42.7%)	In-Hospital patients with fractures, treated at the Kasr El-Aini Orthopaedic Hospital,	Total : 80/222 (36%) a) anti-HCV+/RNA-28/222 (12.6%) b) anti-HCV+/RNA+ 52/222 (23.4%)	rural versus urban birthplace, gender, and 5-year age category	48.2 (41.5-54.7)	Strong association between chronic HCV infection and risk of developing NHL, persisting after

										adjustment in multivariate models and after several sensitivity analyses.
Cucuianu A Br J Haematol 1999	Romania	Case series with control group Period: December 1997 and March 1999	All B-cell NHL: 68 NHL classification : Working Formulation	II G ELISA	20/68 (29.5%)	Non-hospitalized Romanian individuals	46/ 943 (4.9%)	NR	9.1 (5.3-12.9)	Detection of high prevalence (29.5%) of anti-HCV in patients with NHL, especially in low-grade types
De Renzo A Haematologica 2002	Italy (Napoli)	Case-control Period: NR	All LPDs: 227 -B-cell LPds : 127 -HL 100 NHL classification : Revised European American Lymphoma (REAL) histological scheme	II G RIBA HCV-RNA	B-cell LPds : 22/127 (17.3%) B-NHL 12 /61(19.7%) MM 4/48 (8.3%) WM 4/9 44.4%) CLL 2/9 (22.2%)	A group of occasional blood donors from the same geographical area, studied as healthy controls	-HL 2/100 (2%) -Controls : 2/ 110 (1.8%)	NR	19.7 (9.7-29.6)	Detection, in Southern Italy, of a higher prevalence of HCV infection in patients suffering from B-LPD in comparison with healthy subjects, particularly in patients with Bcell-NHL, CLL and WMc
De Renzo A European Journal of Haematology 2008	Italy (Napoli)	Case series Period: 1990 - 2005	All NHLs patients observed : 550 Primary hepatic lymphomas (PHL): 6 Primary splenic Lymphomas (PSL): 19 NHL classification : World Health Organization (WHO) classification	II G RIBA HCV-RNA	PHL: 4/6 PSL: 13/19	NR	NR	NR	PHL 66.7 (22.3-95.7) PSL 68.4 (43.5-87.4)	High prevalence of HCV infection among patients with rare haematologic malignancies (PHL and PSL), favourable outcome of these subjects

De Rosa G Am J Hematol. 1997	Italy (Napoli)	Case series with control group Period: November 1994 - November 1995	All Lympho-prolipherative Disorders (315): 1) No-B LPD: 52 HD: 43 (1 HCV+) T-NHL: 9 2) B LPD : 272, including; NHL- B-cell lymphoma , CLL, HCL, MGUS, WMc, MM, (59 HCV+) NHL classification : Working Formulation	III G ELISA III G RIBA HCV-RNA	B-cell NHL : 21/91 (23.1%)	a) Patients with Hodgkin Lymphoma b) Healthy blood donors	a) 1/43 (2.3%) 0/9 b) 30/1568 (1.9%)	NR	23.1 (14.4-33.7)	Detection of a higher prevalence of anti-HCV antibodies patients with B-Lymphoproliferative disorders, as compared to the normal population and to patients with a non-B-lymphoproliferative disorders.
De Vita S Br J Cancer 1998	Italy	Case-control study Period: January 1994 - June 1997	All malignancies 84 NHLs NHL classification : Working Formulation	II G ELISA II G RIBA HCV-RNA	20/84 (23.8%)	Controls recruited at Aviano, with cancers in: ovary: 13 uterus:14, colon-rectum:13, pancreas:10, lung: 8, stomach: 6, oesophagus : 4 other sites: 5 HCC: 27	Controls :3 /73 (4.1%) HCC : 11/27 (40.7%)	Gender and age at diagnosis (± 5 years)	23.8 (15.2-34.3)	Detection of a higher than expected prevalence of HCV infection in B-cell NHL patients.
Duberg AS Hepatology 2005	Sweden	Nationwide cohort of HCV-infected persons Cancer Registry used to identify all incident	All malignancies : Patients with B-Cell NHLs, after exclusion of patients with HIV coinfection : 16 CLL : 4	ICD-7 codes used:200.1 , 200.2 200.3, 202.1 202.2), CLL (204.1), ALL(204.0), HL (201),	B-Cell NHLs : 16 in 27,150 HCV positive patients included in the cohort, HCV infection diagnosis	NR	NR	NR	0.06 (0.04-0.1)	A significantly increased risk of NHL and MM observed in this study, although an

		cancers diagnosed in the cohort malignant NHL Period: 1990-2000	MM :7 ALL : 1 HL : 1 NHL classification : NR	MM (203.0), and TC (194.0)	made to the Swedish Institute for Infectious Disease Control (SMI)					underestimation of the risk may have been caused by the delayed diagnosis of HCV
Ellenrieder V J Hepatol 1998	German, Ulm	Case series Period: 1991- 1995	B-cell NHLs : Low-grade B-cell NHL: 55 High- low-grade B-cell:14 NHL classification : Kiel Classification	HCV-RNA	3/69 (4.3%) CLL : 1/14 CC : 0/4 CB: 1/14 CCBC: 1/19 IC= 0/18	NR	NR	NR	4.3 (0.9-12.2)	No aetiological role of HCV in the development of NHL in German.
El-Serag HB Hepatology 2002	USA	Cohort study Period: 1992- 1999	Identification of LNHs cases by means of ICD-9-CM diagnosis codes NHL classification : NR	HCV-infected subjects identified by means of ICD-9-CM diagnosis codes (070.41, 070.44, 070.51, 070.54 and V02.62)	421/ 34,204 (1.23%)	34,204 HCV positive patients and 136,816 randomly selected patients without HCV (controls)	1669/136,816 (1.22%)	Year of admission, (to ensure similarity between cases and controls in coding practice, indications for hospital admission, guidelines of testing and management)	1.23 (1.1-1.3)	Significant high association between HCV infection and NHL, after adjustment for age
Engels EA Int J Cancer 2004	USA	Case-control study Period: July 1998 - June 2000	All NHL subtypes: a) 32/ 813 (3.9) b)Low-grade B-cell NHL 18/411 (4.4%) c)Intermediate-and high-grade	III G ELISA III G RIBA HCV-RNA	26/686 (3.8%)	Eligible cases and controls sampled from individuals 20–74 years old, prospectiv	14 /684 (2.1%)	Residence, age, sex, and race	3.8 (2.3-5.2)	Detection of an association between HCV infection and NHL in the United States.

			B-cell NHL 8/275 (2.9%) d) T-cell NHL 2/50 (4.0%) e)other/unknown 4/77 (5.2%) NHL classification : Revised European American Lymphoma (REAL) histological scheme			ely identified by using Surveillance, Epidemiology and End Results (SEER) program of the National Cancer Institute (NCI)				HCV infection may be a cause of NHL.
Ferri C Br J Haematol 1994	Italy	Case series with control group Period: NR	B-cell NHL : 50 NHL classification : Working Formulation	II G ELISA II G RIBA HCV-RNA	B-cell NHL : 17/50 (34%)	a) Patients with Hodgkin Lymphoma b) Healthy subjects c) anti-HCV negative patients with type B or delta chronic active hepatitis	a) 1/30 (3%) b) 30 c) 15 HCV prevalence in in the healthy Italian population : 1,3%	a) no matching b) age c) no matching	34 (20.8-47.1)	Presence of HCV infection in a substantial number of unselected NHL patients, particularly in comparison with HCV prevalence in control groups and in healthy Italian population.
Franceschi S Cancer Epidemiol Biomarkers Prev 2011	Eight countries participating in the EPIC prospective study Italy	Nested case–control study Period: standardized lifestyle and personal history questionnaires collected between 1991 and 2000. Vital status followed up to 2004 and 2006	All lymphomas: 1,023 cases NHL: 739 MM: 238 HL: 46 HCV positive: 12/1,023 (1.17%) NHL classification : World Health Organization (WHO) classification	III G ELISA	B-cell NHLs: 628/1,023 (61.4%) Number of HCV positive patients in B-NHLs not reported 9/730 HCV positive patients in all NHL.s 14/1,454 HCV positive in controls HL:2/46 (4.3%) MM:1/238 (0.4%)	Lymphoid tissue Malignancies classified according to the second revision of the International Classification of Diseases for Oncology (ICD-O-2) and to the WHO Classification of Tumours of Haematop	18/2,028 controls (0.9%)	center, sex, age (±12months at blood collection), date (±3months), time of day at blood collection, and fasting status	61.4 (58.4-64.3)	The present study neither weakened nor strengthened the evidence of an association between HCV and NHL or other lymphoid tissue malignancies

						oietic and Lymphoid Tissues, Third Edition				
Gentile G. Cancer Epidemiol Biomarkers Prev 1996	Italy	Hospital-based case-control study of risk factors for acute leukemias Period:. 1 November 1986- 3 1 March 1990	All acute leukemias: 430 Diagnosis performed by means of: French-American-British classification of bone marrow aspirates for acute leukemias and RAEB, whereas diagnosis for CML was based on typical dinical and cytogenetic laboratory features.	II G ELISA III G RIBA	All acute leukemias: 27/430 (6.3%): AML: 15/172 (8.7%) ALL: 5/67 (7.5%) CML: 2/125 (1.6%) RAEB: 5/66 (7.6%)	Controls recruited in the region of the three hospitals (Rome, Bologna, Pavia) during the study period among outpatients without hematobogical malignancies who were seen in the same hospitals at which cases had been identified.	44/857 (5.1%)	Control groups selected by taking the first five outpatients in Rome and the first three in Bologna and Pavia, seen on a random day each week.	6.3 (3.9-8.5)	Association between acute leukemias, RAEB, and CML Possible association between hepatitis B virus, AML, RAEB, and CML, but further confirmation required.
Genvresse I Ann Hematol 2000	German	Case series Period: 1995-2000	All lymphomas : 119 a) B-NHLs : 105 b) T-NHLs : 14 NHL classification : Revised European American Lymphoma (REAL) histological scheme	III G ELISA	a) 2/105 (1.9%) b) 0/14	NR	NR	NR	1.9 (0-4.5)	Possible HCV involvement in NHLs development via a continuous antigenic stimulation, leading to a B-cell clonal expansion.
Germanidis G. Blood 1999	France	Case series with control gorup	B-NHL: 201 HD: 94	II G ELISA II G RIBA HCV-RNA	B-NHL: 4/201 (2%)	Hematologic malignancies different	1/94 (1.1%)	NR	2 (0-3.9)	No existence

		Period: January 1994 -July 1997	NHL classification : Revised European American Lymphoma (REAL) histological scheme			from B-cell NHL (HD)				of a significant relationship between HCV infection and B-NHL in France
Giordano TP. JAMA 2007	USA	Cohort study Period: 1997-2004	Identification of LNH cases by means of ICD-9-CM diagnosis codes NHL classification : NHL (200, 202.0-202.2, 202.8), WMc (273.0, 273.3), HL (201), MM (203.0-203.1, 238.6), ALL (204.0),CLL (204.1) AnLLs 205.0, 206.0), CML (205.1), other leukemia (204.2, 204.8-204.9, 205.2, 205.8-205.9, 206.1-206.2, 206.8-206.9, 207.8, 208.0-208.2, 208.88.9), MGUS (273.1, 273.2)	HCV infected subjects, were identified by means of *ICD-9-CM* diagnosis codes of HCV infection (070.41, 70.44, 070.51, 070.54, V02.62)	HCV-positive cohort: 146,394 patients During follow-up, 813 patients in HCV-infected cohort (0.5%) had a HIV diagnosis NHL: 319 HL: 65 MM: 95 CCL:69 ALL: 27 WMc: 67 CML: 30	Inpatients records from more than 150 USA Veterans Affairs (VA) hospitals in the Patients' treatment file and outpatients records from any VA facility in the Output Clinic File	HCV-negative cohort: 572,293 patients. During follow-up, 35 696 uninfected HCV patients (6.2%) had a recorded HCV diagnosis and 1539 patients (0.3%) a HIV diagnosis NHL: 1040 HL: 295 MM: 431 CCL:343 ALL; 184 WMc: 98 CML: 163	HCV-infected patients, matched according to sex and age on the baseline date	0.2 (0.19-0.25)	An increased risk of : a) non-Hodgkin lymphoma overall (20% - 30%), b) Waldenström macroglobulinemia, a low-grade lymphoma (3-fold higher risk), c) cryoglobulinemia, in subjects with HCV infection. An etiological role for HCV, in causing lymphoproliferation and non-Hodgkin lymphoma, supported by these results
Goldman L Cancer Causes Control. 2009	Egypt (Cairo)	Case-control study Period:	All lymphomas : 139/296 (47%)	III G ELISA HCV-RNA	B-NHL: 131/272 (48.2%)	Cancer-free subjects,	283/786 (37.4%)	Rural versus urban birthplace, gender, and	48.2 (42.2-54.1)	HCV is a risk factor for diffuse

		October 1999 - March 2004	-T-NHL: 8/24 (34.8%) - DLBCL:79 /146 (54.9%) -MZL: 14/24 (58.3%) -CLL: 24/58 (41.4%) -FC: 9/23 (40.9%) -MCL: 5/16 (31.3%) NHL classificati on : World Health Organizati on (WHO) classificati on			sampled from the Kasr El Aini Faculty of Medicine Orthopaedi c Hospital in Cairo		five-year age category		large B cell, marginal zone, and follicular lymphoma s in Egypt.
Guida M Leukemia 2002	Italy (Bari)	Case-control study Period: September 1999 - October 2001	All Lymphoma s: 12/60 (20%) MM: 5/60 B-NHL: 55/60 NHL classificati on : Working Formulatio n	III G MEIA. HCV-RNA	B-NHL: 12/55 (21.8%)	Control patients with non-hematologi cal malignanci es recruited from the Surgery Departmen t the Oncology Institute of Bari (Italy).	9/63 (14.2%)	Similar age, sex, ethnicity	21.8 (10.9-32.7)	Moderate increase of prevalence of HCV infection among patients with B cell lymphopro liferative disorders in a very homogene ous population of southern Italy
Hanley J Lancet 1996	United Kingdom (Edinburgh)	Case series Period: NR	All LPDs: 72. B-cell NHLs: 38 MM: 24 MGUS: 10 NHL classificati on : Working Formulatio n	II G EIA HCV-RNA	0/72	NR	NR	NR	0 (0-4.9)	No association between chronic HCV infection and risk of NHLs developme nt.
Harakati MS. Saudi Med J 2000	Saudi Arabia	Case series with control group	B-cell NHLs: 56 patients NHL	II G ELISA II G RIBA	B-NHL: 12/56 (21.4%)	1) Blood donors and general medical patients)	1) 3/104 (3%), 2) 2/41	NR	21.4 (10,6-32.7)	Higher prevalence of

		Period:	classification :			2) Other hematologic malignancies other than B-cell NHL.	(5%)			Hepatitis C virus infection in Saudi Arab patients with B-cell non-Hodgkin's lymphoma than in the control groups.
Hausfater P Am J Hematol 2001	France	Prospective controlled study Period: June to September 1998	All LPD: 394 B-NHL: 164 HD: 34 CLL: 107 MM: 54 WMc:12 NHL classification : NR	III G ELISA	B-NHL: 3/164 (1.8%)	1) In-Hospital patients without cancers 2) Nonmalignant hematological diseases 3) Hematological malignancies other than B-cell NHL	1) 3/694 (0.43%) 2) 8/224 (3.6%) 3) 9/425 (2.1%)	NR	1.8 (0-3.8)	No increased prevalence of HCV infection in patients admitted to the Hematology department for B-NHL. No major pathophysiologic role of HCV in lymphoproliferative disorders in Paris
Hwang JP J Oncol Pract. 2014	USA	Cohort-study Period: January 2004 - April 2011	Patients'data, obtained from four institutional sources: *Tumor registry:* to assess patients' demographic characteristics *Pharmacy informatics :* to evaluate chemotherapy drugs and dates administer	NR	141,877 patients with cancer, who were newly registered at MD Anderson Cancer during the study period. Patients considered in the study: 16, 773. HCV screened subjects: 1628/16, 773 (9.7%) with NHLs, 1400 patients with anti-HCV test 42 NHLs antiHCV-	NR	**NR**	NR	3 (2.1-3.9)	HCV screening rates were low, even among patients with risk factors, and the groups with the highest rates of screening did not match the groups with the highest rates of a positive

			ed. *Patient accounts:* to identifystudy patients' International Classification of Diseases (ninth edition; ICD-9) codes *Laboratory informatics :* to determine HCV antibody (anti-HCV) and ALT test dates and results		positive (3%)					test result.
Imai Y Hepatology 2002	Japan	Cohort study Period: February 1992 -July 1992	NHLs: 187 B-cell NHLs: 156 T-cell NHL:31 NHL classification : World Health Organization (WHO) classification	II G ELISA III G ELISA HCV-RNA	21/156 (13.5%)	Use of screening data of 197,600 first-time voluntary blood donors to the Osaka Red Cross Blood Center	Expected numbers of anti- HCV–positive patients with NHL categorized by gender and phenotype in general population : 4.64	Birth cohort–and sex	13.5 (8.1-18.8)	A significantly higher frequency of HCV infection in B cell NHL in comparison with that in birth cohort- and sex-matched blood donors; chronic HCV infection may be associated with B-cell NHL in Japan.
Isikdogan	Turkey	Case series	NHLs: 119	II G	0/119		117	NR	0 (0-3)	No

A Leuk Lymphoma. 2003		with control group Period: December 1997 – September 2001	High-grade NHLs: 10 Intermediate-grade: 64 Low-grade : 45 NHL classification: Working formulation	ELISA HCV-RNA		Subjects admitted as outpatients at Internal Medicine of Dicle Univeristy, Diyarbakir, without history of haematological disordres, during the same period				relationship between HCV and NHLs in the Southeastern Anatolia of Turkey
Iwata H Haematologica 2004	Japan	Hospital-based case control Study Period: 1995 - 2001	All NHLs: 145, 140 with anti-HCV test NHL classification : World Health Organization (WHO) classification	NR	16/140 (11.4%)	Randomly selected controls from patients admitted to the a) orthopedics (290 patients, 286 with anti-HCV markers) or b) ear, nose and throat (284 patients, 282 with anti-HCV markers) departments of the hospital	a) 9//286 (3.1%) b) 20/282 (7%)	Age, sex and year of visit with the controls in a 1:2 ratio	11.4 (6.1-16.7)	Significant association between HCV infection, and malignant lymphoma by multivariate analysis
Izumi T Blood 1996	Japan	Case series Period: 1992-1997	All lymphomas : 83 patients, B-cell NHLs: 54 Non-Bcell NHLs: 20 HLs: 9 NHL classification : NR	II G ELISA	B-cell NHLs: 12/54 (22.2%) Non-Bcell NHLs: 0/20 HLs: 0/9	NR	NR	NR	22.2 (11.2-33.3)	Direct causal relationship between the occurrence of PHSL and chronic HCV infection

Karavattathayyil SJ Am J Clin Pathol 2000	USA (New Orleans)	Case series Period: January 1993 - December 1996	Patients with B-cell NHLs: 31 NHL classification : World Health Organization (WHO) classification	Amplification of HCV RNA from paraffin-embedded tissues	Positive HCV-RNA strands: 8/31 (25.8%) Negative HCV-RNA strands: 6/31 (19.4%)	a)T-cell NHLs: 2 cases b) HL: 2 cases c) Patients with lymph nodes removed for reasons other than lymphoma: 28	0/32	similar sex and age in B-NHL patients and in control group	Positive HCV-RNA strands: 25.8 (10.4-41.2) Negative HCV-RNA strands 19.4 (5.4-33.2)	Presence of HCV infection in a significant percentage of paraffin-embedded tissue from B-cell NHLs patients, compared with control subjects; detection of negative-strand RNA suggests HCV replication in these tissues, excluding the possibility of contamination with viral RNA or blood
Kashyap A Ann Intern Med 1998	USA (Los Angeles)	Case series with control group Period: February 1992-December 1995	All NHLs: 312 36 HCV positive patients NHL classification : NR	NR	NHLs: 36/312 (11.5%)	a) Healthy USA blood donors b) Black and Hispanic patient population at City of Hope National Medical Center	a) (0.4%) b) approximately 25%	NR	11.5 (8-15.1)	Prevalence of HCV positivity is still much higher than expected, even after adjustement for differences in patient demographic charcaterists.
Kaya H Clin Lab Haematol 2002	Turkey	Case-control study Period: NR	All NHLs : 70 patients Low-grade NHLs: 22, Intermediate- grade NHLs: 17 high-grade NHLs: 31 NHL	III G ELISA	1/70 (1.4%)	Healthy-subjects admitted at Departments of Haematology, Ataturk University, Erzurum	1/ 70 (1.4%)	Age and sex	1.4 (0-4.2)	No aetiologic role of HCV in NHL developmnt

			classification : Working Formulation							
Kim JH Jpn J Cancer Res 2002	South Korea	Case-control study Period: January 1997 - December 1998	NHLs: 233 patients 214 patients with anti-HCV positivity NHL classification : Working Formulation	II G ELISA	7/214 (3.3%)	Control groups comprised patients with a) non-hematological malignancy (control group 1) and subjects with b) non-malignant conditions (control group 2) diagnosed at Seoul National University Hospital during the same period. For each case, four controls selected.	a) 7/426 (1.6%) b) 12/439 (2.7%)	age, sex, and date of admission.	3.3 (0.8-5.6)	No association between NHL and HCV infection
King PD Clin Lab Haematol 1998	USA	Case series series with control group Period: June 1995-May 1997	All lymphomas : 93 patients. NHLs : 73 patients HL: 20 patients 438 HCV positive patients NHL classification: Working Formulation	II G ELISA HCV-RNA	1/73 (1.4%)	Patients with HL admitted at Department of Gastroenterology, University of Missouri Hospital	0/20 1/438 (0.22%) patients developed NHL	NR	1.4 (0-4)	No association between NHL and HCV infection
Kocabaş E Eur J Epidemiol. 1997	Turkey	Case series with control group Period: October 1993-March1994	137 Children with malignancies: Acute leukemia: 48 Lymphoma	II G ELISA II G RIBA HCV-RNA	8/137 children were anti HCV positive, 129 patients were anti-HCV	Children admitted,at Balcah Hospital, Adana, during the same period with diseases	1/45	NR	5.8 (1.9-9.7)	HCV infection is common among Turkish children with different types of

			: 51 Solid tumours: 38 NHL classificati on:		negative, but 7/129 were HCV-RNA positive	other than malignanci es				cancer
Kuniyoshi M J Gastroenter ol Hepatol 2001	Japan	Case-control Study Period:Jan uary 1990-March 1998	NHLs 348 patients 20/348 (8.1%) HCV positive patients with NHLs NHL classificati on : Working Formulatio n	III G ELISA HCV-RNA	B-cell NHLs: 15/348 (4.3%)	1,658,234 blood donors, representin g general population in the area (Fukuoka, Japan)	11,922/1,6 58,234 (0.72%)	Age- and sex-	4.3 (2.1-6.4)	Involveme nt of HCV infection in the developme nt of a subgroup of NHL, in males
Luppi M Ann Oncol 1998	Italy	Case series Period: January 1989-August 1993	B-cell NHLs: 157 patients NHL classificatio n : Revised European American Lymphoma (REAL) histological scheme	II G ELISA II RIBA HCV-RNA	35/157 (22.3%) HCV positive B-cell NHLs: LDBCL 8/35 (23%) FC: 14/35 (40%) LPL: 2/35 (6%) 122/157 (67.7%) HCV negative B-cell NHLs	NR	NR	NR	22.3 (15.8-28.8)	Associatio n of HCV infection with the malignant proliferatio n of defined B-cell subsets other than the immunoglo bulin Mk B-cell subset involved in the pathogenes is of mixed cryoglobuli nemia type II and associated lymphoplas macytoid lymphoma type
Markovic Hepato-Gastroenter ology 1999	Slovenia	Case-series Period: January 1991-April 1996	All lymphomas : 305 patients NHLs: 300 patients HL: 5 patients 181 patients with anti-HCV test NHL classificati on : NR	II G ELISA II RIBA HCV-RNA	3/181 (1.6%)	NR	NR	NR	1.7 (0–3.5)	No association between HCV infection and non-Hodgkin's lymphoma s, because of low HCV prevalence in Slovenia
Mazzaro C Br J Haematol 1996	Italy	Case-series with control group	All lymphomas : 199 patients Low-grade	II G ELISA II RIBA HCV-RNA	57/199 (28.6%) Low-grade NHLs:	a)Patients with other haematolog ical malignanci	a) 5 /153 (3.1%) b)	NR	28.6 (22.4-34.9)	Important role of HCV in the

		Period: NR	NHLs: 105 (52.7%) Intermediate grade NHLs: 48 (24.1%) High-grade: 39 (19.6%) MALT : 5 (2.5%) T-cell NHLs: 2 (1%) NHL classification : Working Formulation		40/110 (36.47%) Intermediate grade NHLs: 6/48 (12.5%) High-grade: 9/39 (23.1%)	es, including HL (21 patients), CLL (41), myelodysplastic syndrome (72), plasma cell myeloma (19) b) general population of two towns in the same geographical area (Cormons and Campogalliano) in the cohort study called Dyonisos project	199/6917 (2.9%)			development of low-grade non-Hodgkin's lymphomas
McColl MD Leuk Lymphoma 1997	Scotland	Case series Period: NR	B-Cell NHL: 72 patients Low-grade: 41 Intermediate-grade: 23 High grade: 8 NHL classification : Working Formulation	III G ELISA	0/72	Patients with CLL, recruited at two Hospital in the West of Scotland	0/38	NR	0 (0-9.2)	Possible role of HCV infection in the aetiology of certain subgroups of NHLs, although this effect may be regional
Mele A Blood 2003	Different cities in Italy (Bari, Bergamo, Monte-Fiascone Napoli, Palermo, Reggio Calabria, Roma [2 hospitals], San Giovanni Rotondo, Sassari)	Multicenter case- study with control group Period: 1998 – 2001	B-Cell NHL: 400 patients NHL classification : Revised European American Lymphoma (REAL)/ World Health Organization (WHO) classifications	III G ELISA III G RIBA HCV-RNA	70/400 (17.5%) Aggressive B-NHL: 43/230 (18.7%) Indolent NHL:27/170 (15.9%)	Patients recruited n other departments of the same Hospitals: the departments of dentistry, dermatology, general surgery, gynecology, internal medicine, ophthalmology, orthopedics, otorhinolar	22/396 (5.6%)	NR	17.5 (13.8-21.2)	Detection of an association between HCV and B-NHL

						yngology and traumatology				
Mizorogi F Intern Med 2000	Japan	Case series with control group Period: January 1993-December 1998	Patients with LPDs: 161, subdivided into 2 groups: a)patients with B-cell LPDs, including Bcell-NHLs: 100 MM : 17 CLL: 4 b) patients with non B-cell LPDs: 38 NHL classification : Working Formulation	II G ELISA HCV-RNA	B-cell NHLs: 17/100 (17%)	Subjects with miscellaneous diseases other than liver diseases or LPDs, used as controls	nonB-cell LPDs: 0/25 34/516 (6.6%)	NR	17 (9.6-24.3)	Higher prevalence of HCV infection in patients with B-cell NHLthan in those with non-B-cell NHL and the control group, frequent primary liver involvement and liver-related causes of death in HCV-positive patients with B-cell NHL
Montella M Leuk Res 2001	Italy Naples	Case-control study Period: January 1997 and December 1999	-Bcell-NHLs: 101 -HL: 63 -T-cell NHLs: 10 -MM: 41 NHL classification : Working Formulation / Revised European American Lymphoma (REAL)	III G ELISA HCV-RNA	25/101 (24.8%)	Controls: patients with no history of malignant tumor, admitted to the National Cancer Institute and Cardarelli Hospital of Naples, in the same period	-Controls: 17/226 (8%) -HL: 6/63 (10%) -T-cell NHLs: 3/10 (30%) -MM: 13/41 (32%)	NR	24.8 (16.3-33.1)	Detection of a significant association between HCV infection and B-cell NHLs in the extranodal localization, and also indicate an association for the nodal seat
Morton	USA		All	III G	B cell 7	A	5/534 (1%)		1.9 (0.5-	

LM Cancer Epidemiol Biomarkers Prev 2004		Population -based case-control study of women in Connecticut The Yale Comprehensive Cancer Center's Rapid Case Ascertainment Shared Resource (RCA), a part of the Connecticut Tumor Registry (CTR), a population -based tumor registry Period: 1995 - 2001.	lymphomas : B cell 362 T cell 34 Others: 60 NHL classification : World Health Organization (WHO) classification Incident cases of NHL identified by means of (ICD)-O: M-9590–9595, 9670–9687, 9690–9698, 9700–9723.	ELISA III G RIBA HCV-RNA	/362 (1.9%) T cell 0/4 Others 1 /60 (1.6%) Total: 8/464 (2%)	population -based control group of female residents of Connecticut, aged 21–84, assembled using two methods: - Random digit dialing used to contact women less than 65 years of age, - random selection from the files of the Centers for Medicare and Medicaid Services for women aged 65 years and older		age within 5-year groups	3.3)	Indirect HCV involvement in the development of B-NHL, this risk varying by B-NHL subtype among women.
Musolino C Haematologica 1996	Italy	Case series Period: NR	All-NHLs:24 HCV positive: 2 patients HCV-RNA positive: 5 patients NHL classification: Working Formulation	II G RIBA HCV-RNA	5/24 HCV-RNA positive/NHLs	NR	NR	NR	20.8 (7.1-42.2)	Possible HCV involvement in NHL development.
Musto P Blood 1996	Italy	Case series with control group Period: NR	B-LPDs B-NHL: 150 HCL: 9 CLL: 41 MM: 90 WMc :13 MGUS: 47 NHL classificati	III G Assays HCV-RNA	B-NHLs: 40/150 (26.7%) HCL:1/9 (11.1%) CLL: 8/41 (19.5%) MM: 10/90 (11.1%) WMc: 3/13 (23%)	Patients hospitalized for acute trauma	25/466 (5.4%)	NR	26.7 (19.6-33.7)	A significantly higher prevalence of anti-HCV in patients with B-NHLs than in controls

			on : NR		MGUS:6/47 (12.8%)					and independent of age
Nicolosi Guidicelli S Hematol Oncol. 2012	Italy	Case-control study Period: July 2001 to March 2002	All lymphomas : 137 NHL classification : World Health Organization's (WHO) classification	III G ELISA HCV-RNA	6/137 (4.4%)	Patients observed in Hospital Clinic,, Barcelona and San Giovanni Hospital, Bellinzona, (ideally in traumatology and orthopaedic divisions	7/125 (5.6%)	age, gender, country of origin	4.4 (0.9-7.8)	Existence of marked geographic differences in the prevalence of HCV in NHL but no significant evidence for an association between HCV and B-cell NHLs
Nieters A Gastroenterology 2006	7 countries (Germany, Italy, Spain, Ireland, France, Finland, and Czech Republic)	European Multicenter Case-Control Study Period: 1998 -2004	Total Lymphomas: 1807 NHL classification : World Health Organization's (WHO) classification	III G ELISA HCV-RNA	53/1807 (2.9%)	Controls drawn randomly from population registers of the study regions in Germany and Italy. In the remaining countries, controls recruited from the same hospital as cases	41/1788 (2.3%)	age (5-years), sex, and study center.	2.9 (2.1-3.7)	Positive association between HCV infection and B-cell lymphoma and a role of viral replication in lymphoma genesis.
Ogino H. Hepatol Res 1999	Japan	Case-control sudy Period: 1991-1997	All LPDs: 43 patients NHLs: 33 ALL:10 NHL classification : Working Formulation	II G EIA HCV-RNA	4/33 (12.1%)	a) 45 patients, undergoing colonscopy from July 1995 to June 1996 b) 10599 healthy subjects, receiving a general medical check-up	2/45 (4.4%)	a)Age and sex b) no matching factors	12.1 (3.4-28.2)	High prevalence of HCV infection in patients with NHL in Toyama prefecture in Japan

						in Toyama prefecture from April 1996 to March 1997				
Ohsawa M Int J Cancer 1999	Japan	Cohort-study Period: 1957-1997	Patients with HCV chronic infection, included in the present study: 2162 NHL classification : World Health Organization's (WHO) classification	III G ELISA HCV-RNA	Patients developing B-cell NHLs: 4/2162 During follow-up	Expected number of cases of NHLs in the sex-, age- and calendar year-matched general population: 1.90	NR	Sex-, age- and calendar year-matched general population	0.2 (0-0.3)	Chronic HCV infection moderately associated with increased risk of NHL
Okan V Int J Hematol 2008	Turkey	Case series with control group Period: NR	All Lymphomas: 334 NHL classification : World Health Organization's (WHO) classification	III G ELISA HCV-RNA	9/334 (2.7%) MM: 1/67 (3.1%) CLL: 2/78 (2.5%) DLBCL: 4/67(6%) Follicular 0/9 Mantle: 1/11 (9%) Other: 0/26 T-cell lymphoid tumors: 1/16 (6.2%) HL: 0/60	Controls recruited, using records from the University blood center in Gaziantep	9 /802 (1.1%)	NR	2.7 (0.9-4.4) 6 (0.3-11.6)	Higher HCV-seropositivity rate in patients with DLBCL in comparison with controls. No significant differences in the prevalence of HCV seroposity between patients with lymphoproliferative disorders and controls
Omland LH Int J Cancer. 2012	Denmark	Cohort-study Period:1991-2006 Patients and subjects with HCV infection	10 digit civil registration number assigned to all individuals in Denmark	ICD-7 ICD-10 Codes:	-11,975 anti- HCV-positive patients LNH cases detected: 12 12/11,975: 0.1%	Comparison cohort, which consisted of 6 age- and gender-matched individuals (without a HCV	-71,850 anti- HCV-positive patients LNH cases detected: 24	age- and gender	0.1 (0.04-0.15)	Possible increased risk of NHLs in patients with chronic HCV infection

		identified by means of : - Danish HCV cohort (DANVIR). - Civil registratio n system (CRS) - Danish cancer registry (DCR). - Danish national patient registry (DNPR).	Analysis of the association between HCV and risk of NHL (ICD-10 codes: C82.0–85.9 and C96_) NHL classificatio n : Cancers classified according to the 'Internatio nal Classificati on of Diseases'' 7th revision (ICD-7) for the period 1943–1977 and the 10th revision (ICD-10) for the period 1978–2006			diagnosis) from the general population randomly selected from the CRS, on the day HCV-infection was diagnosed in the correspond ing DANVIR cohort member				
Panovska I Br J Haematol. 2000	Macedonia	Case-series with control group Period: NR	Bcell-NHLs: 112 NHL classificati on : Revised European American Lymphoma (REAL) histological scheme	III G ELISA HCV-RNA	1/112 (0.9%)	Patients with other B-cell malignanci es HL: 38 CLL: 43, ALL: 9 MM: 26 WMc : 1 Prevalence of HCV carriers in Republic of Macedonia within the general population is equal to	1/137 (0.72%)	NR	0.9 (0-2.6)	Low prevalence of HCV infection in patients with B-cell NHL from Macedonia and a lack of association between the two disorders.

						2´0%..				
Park SC J Med Virol 2008	South Korea	Case-control study Period: January 1998-December 2001	235 patients with NHLs, B-cell subtypes: 168 T-cell subtypes: 57 not identified subtypes: 10 NHL classificatio n : NR	II G ELISA	5/235 (2.1%) No informatio n about number of patients with HCV infection and B-NHL cases	Patients with advanced gastric cancer diagnosed at the Korea Cancer Center Hospital	7/235 (3%)	age- and sex-	2.1 (0.3-3.9)	No association between HCV infection and non-Hodgkin's lymphoma.
Paydas S Br J Cancer 1999	Turkey	Case series Period: NR	LPDs: 228 patients NHL: 98 CLL: 38 MM: 47 HD: 36 ALL: 9 NHL classificati on : NR	III G ELISA	NHL: 9/98 (9.2%) CLL: 4/38 (10.5%) MM: 5/47 (10.6%) HD: 7/36 (19.4%) ALL: 1/9 (11.1%)	NR	NR	NR	9.2 (3.4-14.9)	HCV infection as a causative and/or contributin g factor in lymphoprol iferation in this study
Pellicelli WJG 2011	Italy	Case-series Period: January 2008 - January 2009	125 patients with B-cell NHLs NHL classificati on : World Health Organizati on's (WHO) classificati on	III G ELISA HCV-RNA	24/125 (19.2%)	NR	NR	NR	19.2 (12.3-26.1)	HCV genotypes and duration of HCV infection differed between B-NHL subtypes. Indolent lymphoma s can be managed with antiviral treatment, while DLBCL is not affected by the HCV infection.

Pioltelli P Lancet 1996	Italy	Case-series with control groups Period: January-June 1995	All Lymphomas: 204 NHLs: 126 HL: 78 28HCV positive lymphomas NHL classification : Working Formulation	III G ELISA II G RIBA HCV-RNA	26/126 (20.6%)	a)HL b) candidated blood donors c) elderly people	a) 2/78 b) 9/832 c) 9/ 94	NR	20.6 (13.5-27.7)	High prevalence of HCV infection in NHLs, in the absence of an increased risk for HCV infection and of a clinical history of MC
Pioltelli P Am J Hematol 2000	Italy	Case-control study Period: 01/01/96 - 30/06/97	Patients with B-cell NHLs: 300 NHL classification : Working Formulation (WF) and Revised European American Lymphoma (REAL) histological scheme	III G ELISA III G RIBA HCV-RNA	48/300 (16%)	Individuals consecutively recruited during routine visits at medicine, surgery, or traumatology departments during the recruitment period of the study population a) Patients with internal and surgical diseases b) Patients with solid neoplasm c) Patients with autoimmune disorders	a) 51/ 600 b) 15/247 c) 6/122	age- and sex	16 (11.8-20.1)	The prevalence of HCV infection is higher in patients with NHLs than in non-neoplastic people and in patients with non-lymphoproliferative malignancies or receiving immunosuppressive treatment, but the small difference among these groups, the identical genotype pattern between NHL and controls do not support the hypothesis that HCV plays a role in lymphoma genesis.
Pivetti S Br J Haematol 1996	Italy	Case-series with control group	Patients with LPDs: 167 patients	II G ELISA II G RIBA HCV-RNA	7/47 (14.9%)	a) Patients with connective tissue	a) 26/100 (26%)	NR	14.9 (4.7-25)	HCV may link

		Period: NR	(30 HCV positive) HL: 30 NHLs: 47 CLL: 29 MM: 18 MGUS: 31 WMc: 12 NHL classification : NR			diseases b) Patients with idiopathic thrombocytopenic purpura	b) 12/33 (36.4%)			lymphoid malignancies and autoimmune diseases by skewing the activity of the immune system toward the production of autoAbs.
Pozzato G Blood 1994	Italy	Case series Period: NR	31 patients with MC. 12 patients/31 with low-grade NHLs. 26/31 HCV positive NHL classification : Working Formulation	II G EIA II G RIBA	10/12 patients with low-grade NHLs were anti-HCV positive	NR	NR	NR	83.3 (51.6-97.9)	HCV associated with a high prevalence of low-grade non-Hodgkin's lymphomas
Prati D Br J Haematol 1999	Italy, Milan	Case series Period: January 1989 - August 1998.	Primary cutaneous B-cell NHL. NHL classification : European Organisation for Research and Therapy of Cancer (EORTC)	III G ELISA III G RIBA HCV-RNA	1/34 (2.9%)	NR	NR	NR	2.9 (0-8.6)	Primary cutaneous B-cell NHL might represent a distinctive group among B-cell NHLs
Rabkin CS Blood 2002	USA	Cohort study Period: June 1959 and September 1966	All LPDs: 95 B-cell NHL: 57 MM: 24 HL: 14. NHL	III G ELISA III G RIBA HCV-RNA	4/95 (4.2%) 0/95 at RIBA 0/95 at HCV-RNA	Study subjects (48 420 individuals) recruited from the Child Health and Development Study (CHDS) cohort established in 1959 at	1/48.420 at ELISA 0/48420 at RIBA	Age, sex, smoking and race	4.2 (0.1-8.2)	Not substantial role of chronic HCV infection in the etiology of B-cell neoplasia.

			classification : Tumors classified according to the International Classification of Diseases for Oncology, second edition, as NHL (histologic classifications 9590 through 9642 and 9670 through 9698), multiple myeloma (9730 through 9732), or Hodgkin disease (9650 through 9667)			the Kaiser Foundation Health Plan, Oakland, CA				
Ramos-Casals M, J Rheumatol 2004	Spain	Case series Period: 1994-2000	NHL classification : World Health Organization's (WHO) classification	III G ELISA HCV-RNA	6/98	NR	NR	NR	6.1 (1.3-10.8)	Description concerning a triple association of HCV infection, autoimmune diseases and NHLs
Salem AK Gulf J Oncol 2009	Yemen	Case series with control-group Period: January 2005-January 2007	All NHLs : 192 patients NHL classification : NR	III G EIA	29/192 (15.1%)	Patients checked for HCV infection with several acute medical conditions and coming from different parts of the country	814/20,329 (4%)	NR	15.1 (10-20.1)	Higher prevalence of HCV infection among Yemeni patients with NHL than among persons in the control group
Salem Z Eur J Epidemiol	Lebanon	Case-series with control	B-cell NHL: 35 patients.	III G ELISA	0/35	a) Patients with different	a) 0/63 b) 0/220	NR	0 (0 -10)	No association between

2003		group Period: NR	NHL classification : NR			malignancies (malignant myeloproliferative disorders: 12, malignant lymphoproliferative disorders: 28, non haematological cancers: 23 patients) b) Healthy blood donors and patients without malignant conditions, attending General Medicine of American university, Beirut				HCV infection and B-cell NHLs development in Lebanese patients
Sansonno D, Blood 1996	Italy	Case series Period: January 1991 to December 1995	12 HCV-positive patients with MC and 2 HCV-positive patients with reactive lympho-adenopaties NHL classification: Working Formulation	II G ELISA II G RIBA	3/12 (25%)	NR	NR	NR	25 (0.5-49.5)	These data emphasize that lymphoid organs may be a site of HCV infection. The demonstration of HCV-related proteins in a nonmalignant condition, namely HRL, indicates that HCV infection precedes the neoplastic transformation and possibly plays a major role in lymphomagenesis in MC.
Schöllkopf C Int J Cancer 2008	Denmark	Nation-wide Danish-Swedish case-control study (Scandinavi	All lymphomas: 2819	III G ELISA III G RIBA HCV-RNA	HCV positive NHLs:57(2.4%) HL: 6 (1%) at III G ELISA test,only	Controls randomly sampled from the entire Danish and Swedish	21/1856(1%)	Sex and age in 10-year intervals	2.4 (1.8-3)	Positive association between HCV and risk of NHL, in particular

		an Lymphoma Etiology study, SCALE) Period: The SCALE study population includes the entire Danish population between June 1, 2000-August 30, 2002, and the Swedish population between October 1, 1999-April 15, 2002	NHLs: 2353 HL:466 NHL classification : World Health Organization's (WHO) classification		NHLs:7/ 2353 (0.7%) HL: 0 positive at ELISA test and positive or intermediate at RIBA test for anti-HCV antibodies	population s using continuously updated, computerized population registers.				of B-cell origin
Seve P Eur J Gastroenterol Hepatol 2004	France	Cross-sectional study Period: January 1997-December 1998	B-NHL:212 patients BL 6 DLBCL 109 FC 31 LL 7 LPL 5 MALT 17 MCL 21 MZL16 NHL classification : Revised European American Lymphoma (REAL) classification	II G ELISA II G RIBA	a) 6/212 (2.8%) b) MALT 3/17	Transfusion patients from surgical emergency, internal medicine pneumology, endocrinology, gastroeterology, nephrology, oncology, general surgery, orthopaedics, rheumatology, obstetrics and gynaecology, and intensive care wards.	20/974 (2.05%)	NR	. a) 2.8 (0.6–5) b) 17.6 (3.8–43.4)	Possible association between HCV and MALT lymphoma.
Shariff S Ann Oncol	Canada	Case series with	patients	III G EIA III G RIBA	2/88 (2.3)	a) patients	0/37 11/1085	NR	2.3 (0-5.3)	Chronic

1999		control group Period: 1996 and part of 1997	with B-cell NHL NHL classification : Working Formulation/ Revised European American Lymphoma (REAL) classification	HCV-RNA		with aT-cell NHL b) second control group, including health-care workers, recruited between 1995 and 1997.	(1%)			HCV infection as a risk factor for B-cell NHL in certain populations or with certain genotypes of the virus, no significant association in British Columbia.
Shirin H Isr Med Assoc J 2002	Israel (Tel Aviv)	Case control group Period: May 1997 - September 1999	B-NHL (DLCL FC CLL) NHL classification : Revised European American Lymphoma (REAL) classification	IIIG ELISA	Total: 212 patients Lymphoproliferative disorders: 10/128 (7.8%)	a) Patients with Myeloproliferative and myelodisplastic disorders: b) Israeli blood donors:	a) 1/84 (1.1%) b) HCV prevalence equal to 0.64%	Age,gender and percentage of patients born in the former Soviet Union	7.8 (3.1-12.4)	Significant association between HCV infection and diffuse large B cell lymphoma .
Silvestri F Bood 1996	Italy, Udine	Case series with control group Period: NR	537 unsekected patients with LPDs B-cell NHLs: 311 T-cell NHLs: 57 MM: 78 HL:88 ALL: 23 NHL classification : Kiel classification/ Revised European American Lymphoma (REAL) classification	II G ELISA II G RIBA HCV-RNA	29/311 (9%)	NR	T-cell NHLs: 2/57 (4%) MM: 3/78 (4%) HL:0/88 ALL: 1/23 (4%)	NR	9 (6-12.5)	High prevalence of HCV infection in patients with B-cell NHL
Silvestri F Haematologica 1997	Italy	Case series Period: NR	B-cell NHLs NHL classification : Revised European American Lymphoma (REAL) classificati	II G ELISA II G RIBA HCV-RNA	42/470 (8.9%) 21/22 (95.4%) B cell-NHLs patients with cryo-globulinemia	NR	NR	NR	8.9 (6.3-11.5)	Close association between HCV infection and B-cell NHLs

			on		21/448 (4.6%) B cell-NHLs patients without cryo-globulinemia					
Singer IO Leuk Lymphoma 1997	United Kingdom	Case-series with control group Period: NR	All Lymphomas: 50 unselected patients B-cell NHLs: 31 T-cell NHLs: 6 HL: 13 NHL classification : Working Formulation	III G ELISA HCV-RNA	0/31	No information about control groups	0/19	NR	0 (0-11.2)	No evidence supporting an association between HCV infection and LNH development
Sonmez M. Tumori 2007	Turkey	Case-control study Period: 2002-2005	B-cell NHLs: 109 DLBCL: 71 Small-cell LL: 38 NHL classification : World Health Organization's (WHO) classification	III G ELISA HCV-RNA	3/109 (2.8%) Low grade: 1/38 (2.6%) High grade: 2/71 (2.9%)	Patients selected from orthopedics, general surgery, urology, ophthalmology, otorhino-laryngology clinics with irrelevant diseases.	28/551 (5.1%)	NR	2.8 (0-5)	No difference in the incidence of HCV infection between NHL- and control-group
Spinelli JJ Int J Cancer 2008.	Canada (Vancouver)	Population-based case-control study Period: March 2000 and February 2004	All-NHL cases:795, from the Greater Vancouver Regional District (GVRD) and the Capital Regional District (CRD), including the city of Victoria, enrolled from the BC Cancer Registry	II G ELISA IIIG Line-immunoassay	NHLs:19/795 (2.4%) B-cell NHLs:18/717 (2.5%) T-cell NHLs: 1/78	Controls selected from the Client Registry of the BC Ministry of Health	5/697 (0.7%)	Sex, age (within 5-year age group)	2.4 (1.3-3.4)	HCV infection contributes to increase NHL risk.

			NHL classification : World Health Organization's (WHO) classification							
Swart A BMJ Open 2012	Australia	Cohort-study Period: 1 January 1993 - 31 December 2007	Individuals registered on the Pharmaceutical Drugs of Addiction System, a record of all NSW Health Department authorities that administer methadone or buprenorphine to opioid-dependent people as opioid substitution therapy. Solid cancers classified according to the International Classification of Diseases (ICD), 10th revision, haematopoietic neoplasms and Kaposi sarcomas classified according to the ICD for Oncology, 3rd edition	NR	Patients considered in the study: 29 613 Subjects with HCV infection alone: 14,892 Observed number of LNH in HCV-positive cohort: 75	Calculation of expected number of incident LNHs	Expected number of LNH: 49.6	NR	0.5 (0.4-0.6)	Association between HCV infection and LNHs
Takai S Eur J Haematol. 2005	Japan	Case series Period: January 1996 to September	All haematological malignancies: 601	CLISA HCV-RNA	37/601 patients were anti-HCV positive **NHL: 22/218**	NR	NR	NR	NHLs: 10.1 (6.1-14.1) DLBCL: 11.8 (5.8-17.8) FCL: 8 (2.7-	High prevalence of HCV infection in NHL Possible role of HCV

		200	**NHL: 218** DLBCL: 110 FCL: 100 MCL: 3 PTCL: 5 Acute Leukemia: 246 AML: 193 ALL: 53, Adult T-cell Leukaemia: 13 MM:124		**(10.1%)** DLBCL: 13/110 (11.8%) FCL: 8/100 (8%) MCL: 1/3 (33%) PTCL:1/ 5 (20%) AML: 5/193 (2.6%) ALL: 2/53 (1.8%) adult T-cell Leukaemia: 0/13 MM: 8/124				13.3) MCL: 33 (0-86..2) PTCL:20 (0-63) AML: 2.6 (0.4-4.8) ALL: 1.8(0-8.9) MM: 6.5 (2.2-10.8)	in the pathogenesis of NHs.
Takeshita M Histopathology 2006	Japan	Case series with control group Period: NR	All-Lymphomas: 537 a) HL: 18 -B-NHL: 400 (DLBCL, FC CLL, MALT, PCM, MCL, MZL,BL,others) -T- cell NHL: 96 -NK/T-cell NHL: 23 NHL classification : World Health Organization's (WHO) classification	II G ELISA HCV-RNA	B-cell NHL 45/400 (11.3%) Primary Effusion Lymph: 3/6 (50%) BL: 1/7 (14.3%) DLCL:28 /161 (17.4%) FL: 3/47 (6.4%) MALTOMA: 5/52 (9.6%) MM: 4/81 (4.9%) CLL, SMZL, Mantle cell Lymp: 0	a) Other haematological malignancies b) Blood donors	a) HL: 1/18 (5.6%) T-cell NHL:5/96 (5.2%) NK-Tcell Lymphomas: 2/23 (8.7%) b) 396/15,567 (2.5%)	NR	11.3 (8.1-14.3)	HCV infection may play a role in lymphoma genesis of splenic and gastric DLBCL.
Talamini R Int J	Italy	Case-control	Total NHL:	II G MEIA	44 /225 HCV	Patients	45/504 (8.9%)		17.8 (12.8-22.8)	HCV infection

Cancer 2004		study Period: January 1999 -July 2002	225 cases 44 /225 HCV positive patients NHL classification : *International Classification of Diseases for Oncology*, updated to include categories in the Revised European–American Lymphoma (REAL)/World Heath Organization (WHO) classification	HCV-RNA	positive patients 40/225 (17.8%) patients with B-cell NHLs a) Low-grade B-cell: 24 b) Intermediate- and high-grade B-cell: 16 c) T-cell: 2 d) Unknown: 2	with a wide spectrum of acute conditions admitted at National Cancer Institute, Aviano; the "Santa Maria degli Angeli" General Hospital, Pordenone; the "Pascale" National Cancer Institute, Naples and 4 general hospitals, Naples		Gender, age (in 5-year bands) Center (Aviano/ Pordenone and Naples)		associated with an increased NHL risk,
Teng CJ Clinics 2011	Taiwan	Case series Period: 2003-2008	MM: 155 patients 30 patients with chronic hepatitis MM diagnosis: : International Myeloma Working Group	III G MEIA	14/155 (9%) 1/155 with HBV/HCV co-infection	NR	NR	NR	9 (4.5-13.5)	High prevalence of cytogenetic abnormalities in patients with HCV chronic hepatitis
Thalen DJ Br J Haematol 1997	The Netherlands	Case series Period: NR	NHLs: 115 patients B-cell NHLs: 99/115 (86%) T-cell NHLs: 15 (13%) Unclassified: 1 (1%) NHL classification : Working Formulation	II G EIA II G RIBA	B-cell-NHLs: 0/99 T cell NHLs: 0/15	NR	NR	NR	0 (0-3.7)	No association between HCV infection and B-cell NHLs in the study
Timuragaoğlu A. Haematologia 1999	Turkey	Case series with control group Period: NR	NHLs: 48 patients NHL classification : Working Formulation	II G EIA HCVRNA	Anti HCV positive: 0/48 HCV-RNA positive: 3/35 (8.6%)	Patients with various haematological disorders (MM, HL, acute myeloblastic leukaemia,	0/28	NR	8.6 (1.8-23.1)	Association between HCV infection and B-cell NHLs in the study

						acute lymphoblastic leukaemia, chronic myelogenous leucemia, idiopathic thrombocytopenic purpura, myelodysplastic syndrome)				
Tkoub EM Blood 1998	France	Case series with control group Period: NR	46 patients with gastric MALT: High grade: 21 Low-grade 25 37/46 patients with Helicobacter Pylori NHL classification : NR	III G ELISA III G RIBA	1/46 (2.2%)	Patients with gastroduodenal disease: 84 with duodenal ulcer 43 with gastric ulcer 38 with dispepsia	4/165 (2.4%)	Both groups comparable in terms of the sex ratio, age, prevalence of H. Pylori. Risk factors for HCV infection and geographical origin	2.2 (0-6.3)	No link between HCV infection and gastric MALT in France
Tursi A. Am J Gastroenterol 2002	Italy	Case series Period: NR	25 HCV positive patients with gastric MALT. -20/25 (80%) with grade 2 - 5/25 (20%) with grade 3. 18/25 patients with Helicobacter Pylori NHL classification : World Health Organization's (WHO) classification	III G ELISA III G RIBA HCV-RNA	NR	NR	NR	NR	MALT grade 2: 80 (59.3-93.2) MALT grade 3: 20 (6.8-40.7)	HCV may colonize gastric MALT, allowing the development of a grade of acquired MALT, this represents the first step toward a MALT lymphoma
Udomsakdi-Auewarakul, C Blood 2000	Thailand	Case series Period: NR	All malignancies: 130 Intermediate- to high-grade NHL: 98	II ELISA HCV-RNA	2/98 (2%) 1/32 (3.1%)	NR	NR	NR	2.(0-4.8)	No association between HCV infection and NHLs in this study from

			Low-grade NHL 32 patients NHL classification : Working Formulation							Thailand, where HCV infection is highly prevalent
Vajdic CM Cancer Epidemiol Biomarkers Prev 2006	New South Wales, and the Australian Capital Territory	Population -based case-control study Period: January 2000 and August 2001	Total Lymphomas:694 -B-cell NHLs: 659 (95%) -T-cell NHLs: 28 (4%) - Undetermined: 7 (1%) NHL classification : World Health Organization's (WHO) classification	III G EIA **CLISA** HCV-RNA	NHLs: 3/694 (0.4%)	Potential participants (both cases and controls) received a letter t inviting their participation in research about the development of NHL	2/694 (0.3%)	Age, sex, and residential area	0.4 (0-0.9)	No strong evidence for an association between any infection and non-Hodgkin lymphoma risk in immunocompetent people, but increased risk between HCV infection and non-Hodgkin lymphoma in subjects with injecting drug use
Vallisa D Am J Med 1999	Italy	Case-control study Period: 1990-1996	B-cell-NHLs: 175 patients NHL classification : Working Formulation/ Revised European American Lymphoma clasificatio n	II G ELISA II G RIBA HCV-RNA	65/175 (37.1%)	Subjects without lymphoma selected from: 1) inpatients (175) 2) outpatients (175) cared at Civil Hospital, Piacenza, subdivided into 2 groups	a) 17 /175 (10%) b)15/175 (9%)	Age (within 2 years) and sex	37 .1 (30-44.3)	Possible HCV role as an etiologic agent in non-Hodgkin's B-cell lymphoma.

Varma S Hepatol Int 2011	Northern India	Case-control study Period: NR	B-NHLs: 57 patients High-grade disease (DLBCL): 44 (77.2%) Intermediate-disease (FC): 6 (10.5%) Low grade disease: (small lymphocytic): 7 (12.3%) NHL classification : World Health Organization's (WHO) classification	III G ELISA HCV-RNA	1/57 (1.7%)	Patients with non-hematological conditions admitted to Departments of Ophthalmology, Otorhinolaryngology, Dermatology, and Internal Medicine in the Hematology Clinic, Institute of Medical Education and Research,, Chandigarh	2/171 (1.2%)	Age and sex-matched controls	1.7 (0-5.1)	No significant association between HCV infection and NHL in Northern India
Veneri D Am J Hematol 2007	Italy	Case series Period: January 1995 – December 2006	947 patients with lymphoproliferative disorders: DLBCL: 361 MM: 139 B-cell MZL: 62 HL: 103 B-CLL: 186 FL. 96 NHL classification : World Health Organization's (WHO) classification	NR	55/947 patients were HCV positive DLBCL: 27/361 (7.5%) MM: 1/139 (0.7%) B-cell MZL: 15/62 (24.2%) HL: 4/ 103 (3.9%) B-CLL: 4/186 (2.1%) FL: 4/96 (4.2%)	NR	NR	NR	DLBCL: 7.5 (4.7-10.2) B-cell MZL: 24.2 (13.5-34.8)	Confirmed association between a subset of B-cell lymphomas and HCV infection
Yamac K Eur J Epidemiol 2000	Turkey	Case series Period: August 1996 - June 1998,	All Lymphomas:92 NHLs: 73 HL 19 NHL classification : Revised	II G ELISA HCV-RNA	1/92 (1.1%)	NR	NR	NR	1.1 (0-3.2)	No significant association between HCV and NHL in the study

			European American Lymphoma clasificatio n							
Yenice N Turk J Gastroenter ol 2003	Turkey	Case series with control group	All Lymphoma s:134 B cell NHLs: 84 HLs: 50	III G ELISA	B-cell NHLs: 6/84 (7.1%) HLs: 1/50 (2%)	Healthy blood donors	1/100 (1%)	NR	7.1 (1.6-12.6)	HCV may play a role in the developme nt of B-cell non-Hodgkin lymphoma, but not in Hodgkin lymphoma
Yoshikawa M J Clin Gastroenter ol 1997	Japan	Case series with control group Periood: NR	All Lymphoma s: 100 B-NHLs: 55 T-NHLs: 10 HL: 5 MM: 25 B-CLL: 2 MGUS: 3 NHL classificati on : Working Formulatio n	II G ELISA III G ELISA HCV-RNA	B-NHLs: 9/55 (16.4%) MM:5/25 (20%) MGUS: 1/3 (33.3%)	Patients with any cancer in digestive organs except liver enrolled at Nara Medical University	1/25 (4%)	NR	16.4 (6.5-26.1)	High rates of HCV infection detected in B-NHL and MM
Yu SC Kaohsiung J Med Sci. 2013	Taiwan	Case series Period: 1988 - 2011	All lymphomas : 74 patients: -B-cell lymphomas : 69 -T-cell lymphomas : 3 - Lymphobla stic Lymphoma : 1 - Unspecifie d high-grade lymphoma: 1 41/74 patients with serology for HCV infection NHL	NR HCV-RNA	Patients with B-cell-NHL and with serology for HCV infection: 39 Patients with B-cell-NHL and HCV positive 10/39 (25.6%)	NR	NR	NR	25.6 (11.9-39.3)	High HCV sero-prevalence in patients with early-stage DLBCL suggests a role of HCV in the pathogene sis of primary DLBCL.

			classification : World Health Organization's (WHO) classification							
Zucca E Haematologica 2000	Switzerland	Case series Period: 1990 and 1995	B-cell NHLs: 180 Anti-Helicobacter antibodies detected in 81/180 (45%) patients. NHL classification : Revised European American Lymphoma (REAL) histological scheme	EIA	17/180 (9.4%) Gastric lymphoma: 2 Non gastric lymphoma: 15	A survey of 5,424 subjects new blood donors from the same area tested between 1992 and 1997 (Swiss Red Cross Transfusional Medicine Service for Canton Ticino)	49/5,424 (0.9%)	NR	9.4 (5.1-13.7)	High prevalence of HCV infection detected in NHL lymphoma patients and associated with a shorter time to lymphoma progression. HCV infection not correlated with primary gastric presentation or with MALT-type histology.
Zuckerman E *Ann Intern Med* 1997	USA (Los Angeles)	Controlled, cross-sectional study. Period: October 1994 and May 1996	B-cell NHLs: 120 patients NHL classification : Working Formulation	II G ELISA HCV-RNA	B-cell NHLs 26/120 (22%)	a) Patients with hematologic malignancies other than B-cell NHLs b) Patients without hematologic malignancies, attending the general medicine clinic at LAC-USC	268 patients a)7/ 154 (4.5%) b) 6/114 (5%)	NR	21.7 (14.3-29)	Increased prevalence of HCV infection among patients from the United States with B-cell lymphoma, but uncertain generalizability to other populations, because of high number of patients,

						and with: systemic hypertension or ischemic heart disease: 69 diabetes mellitus:35 primary hypothyroidism:10				belonging to Hispanic ethnicity .

ALL: Acute Lymphoblastic Leukemia, AML: acute myeloid leukemia, AnLLs= Acute non-Lymphocitic Leukemia, B-LPD=B-cell lymphoproliferative disorders, BL= Burkitt Lymphoma, CC= centrocitic, CB= centroblastic, CBCC= centroblastic/centrocitic, CLL= Chronic lymphocytic leukemia, CML=chronic myeloid leukemia, CMD= Chronic myeloprolipherative disease, DLBCL =Diffuse Large B-cell Lymphoma, CLISA= chemiluminescence immunoAssay, ELISA= enzyme-linked immunosorbent assay, EIA= enzyme – immunoassay, EMZBL= extranodal marginal zone B-cell lymphoma, FC=Follicular Lymphoma, FCL=Follicle Center Lymphoma, HCL, hairy cell leukemia, HL= Hodgkin Lymphoma, IC: immunocytoma, LAC-USC=Los Angeles County-University of Southern California, LL=Lymphocitic Lymphoma, LPds= Lymphoproliferative diseases, LPD= Lymphoproliferative disorders, LPL= Lymphoplasmocitoid Lymphoma, MCL= Mantle Cell Lymphoma, MGUS=Monoclonal Gammopathy of uncertain significance, MEIA= Microparticle enzyme immunoassy; MCL=Mantle Cell Lymphoma, MCS= mixed cryoglobulinemia syndrome, MGUS= monoclonal gammopathy of undetermined significance, MM =Multiple Myeloma, MS= myelodisplastic syndrome, MZL = Marginal Zone Lymphoma, PCM=Plasma Cell Myeloma, PHL= Primary Hepatic non-Hodgkin's Lymphoma, PLL= Primary Liver Lymphoma, PSL= Primary Splenic non-Hodgkin's Lymphoma, PTCL= peripheral T-cell lymphoma, RAEB= refractory anemia with excess of blasts; SIR,=standardised incidence ratio, WMc= Waldenström's microglobulinemia, Y=determined, N=not determined, NR= not reported

Supplementary Table 2. Characteristics of available studies, reported in English, assessing the association between HCV infection and cholangiocarcinomas (A) or bile duct dysplasia

Author/ Journal/ Publication Year	Country	Study Design Study period	CCA Diagnosis	HCV positive colangiocarcinoma (n)/ total colangiocarcinoma Cases (N)	Total patients enrolled and Control Source	HCV positive controls (n)/ Controls (N)	Matching factors	
(A)								
Abdel Wahab M 2007	Egypt	Case series Period: Januiary 1995-October 2004	Histologic confirmation/ CT/MRI/ERCP/PTD	440 patients with hilar cholangiocarcinoma 238 anti-HCV positive patients 238/440 (54%)	NR	NR	NR	5 5
Barusrux S Asian Pacific J Cancer Prev 2012	Northeast Thailand	Case series with control group Period: NR	Histologic confirmation	8/295 (2.7%)	Total patients: 6120 Controls randomly selected from people in 4 provinces in Thailand, representing 4 geographically distinct areas and thus, populations in the North, North-east, South and Center of the country, respectively	125/5825 (2.15%) HCV-Ab prevalence in Thailand ranging from 1.5% to 2.15%. Sunanchaikarn S, Theamboonlers A, Chongsrisawat V, et al (2007). Seroepidemiology and genotypes of hepatitis C virus in Thailand. *Asian Pac J Allergy*, 25, 175-82.	NR	2 4
Chantajitr S J Hepatobiliary Pancreat Surg 2006	Thailand	Case series with control group Period: 2000-2004	Histologic confirmation	HCC-CCA=25 15 patients with test for anti- HCV 2/15 (13.3%)	Total patients: 75. 50 individuals, diagnosed with HCC at Ramathibodi Hospital	HCC= 50 32 patients with test for anti- HCV 1/32(3.1%)	NR	1 4
Donato F Cancer Causes and control 2001	Italy	Hospital-based case-control study Period: January 1, 1995-July 31, 2000	Histologic confirmation	6/24 (25%)	Total individuals : 848. Subjects unaffected by liver diseases or malignant neoplasms, admitted to the Department of Ophtalmology, Dermatology, Urology, Surgery, Cardiology, Internal Medicine in the two main Hospitals in Brescia, enrolled as controls	50/824 (6%)	Age (±5 years), sex, date and hospital admission	2 4
El-Serag H Hepatology	USA	Cohort study	Identification of PAC cases by means	HCV-infected cohort: 146,394	718,687 patients (146,394 HCV-	HCV-uninfected cohort: 572,293	HCV-uninfected and	I (

2009		Period: October 1, 1988, and September 30, 2004	of ICD-9-CM diagnosis codes (157.0, 157.1, 157.2, 157.3, 157.8, 157.9) Identification of HCV infected subjects by means of ICD-9-CM diagnosis codes (070.41, 070.44, 070.51, 070.54 and V02.62)	patients ICC= 14 ECC= 15	infected cohort, 572,293 HCV-uninfected cohort) , ICC: 37 and ECC: 75 (14 ICC and 15 ECC in HCV infected patients, 23 ICC and 60 ECC in HCV uninfected subjects)	patients ICC= 23 ECC:=60	HCV-infected subjects matched by age (± 1 year)and sex	E (
Hai S Dig Surg 2005;	Japan	Case series with control group Period: January 1997 - December 2002	Histologic confirmation	19/50 (38%)	Total patients: 50 Subjects admitted to the Osaka City University Hospital or the Osaka City General Hospital,	31/50 (62%)	NR	3 5
Hsing AW Int. J. Cancer: 2008	China	Population- based case-control study Period: June 1997 - May 2001,	Histologic confirmation or by means of ERCP	3/234 (2%) with galdbladder cancers 2/134 (1.5%) with extrahepatic bile duct cancers 1/49 (2%) with Ampulla of Vater carcinomas	Total patients :1696 Controls represented by biliary stone case patients and by healthy subjects without a history of cancer, randomly selected from all permanent residents listed in the Shanghai Resident Registry	2/301 (0.7%) patients with gallbladder stones, 5/216 (2.3%) with bile duct stones and 15/762 (2%) healthy individuals	age (5-year intervals) and gender	1
Kobayashi M Cancer 2000	Japan	Case series with control group Period: 1980-1997	Cirrhosis confirmation by means of liver biopsy, peritoneoscopy, or both	14/600 (2.3%) developed CCA 11/14 patients with CCA 3/14 patients with CCA-HCC	600 HCV positive patients in follow-up between 1980 to 1997	206/600 (34.3%) patients developed HCC in the same period	NR	2 3
Kuper H Soz Praventivmed. 2001	Greece	Case-control study Period: January 1995-December 1998	Histologic confirmation	0/6 with CCA	Total subjects : 699 Controls represented by patients with injuries or eye, ear, nose and throat conditions admitted to three teaching Hospitals in Athens	52/333 (16%) with HCC 1/360 (0.3%) controls	Age and gender	0
Lee CH Br J Cancer 2009	Taiwan	Case-control study Period: 1991-2005	Histologic confirmation	21/160 (13.1%)	Individuals generally surveyed for any disease Chang Gung Memorial Hospital at the Lin-Ko Medical Center.	10/160 (6.3%)	Age and sex	1 1

Lee TY Am J Gastroenterol 2008	Korea	Hospital-based case-control study Period: 2000- 2004	Histologic confirmation	12/622 (1.9%)	Total subjects:3110 2,488 healthy controls selected from 192,655 individuals undergoing routine health examinations at the health promotion center at Asan Medical Center, Seoul	47/2488 (1.9%)	Age, sex, and date of admission or visit	1
Lee WS Surg Today 2006	Korea	Case series with control group Period: November1994- December 2003	Histologic confirmation	ICC= 3/79 (3.8%) HCC-CCA= 4/33 (12.1%)	Total patients: 952, subjects, undergoing surgical resection at Samsung Medical Center, because of: HCC-CCA= 33 ICC= 79 HCC= 832	HCC= 61/832 (6.5%)	NR	3
Matsumoto K Intern Med 2014	Japan	Case series with control group Period: NR	Histologic confirmation	145 patients undergoing surgical resection because of ICC: 50 ECC: 95 a)ECC: 7/95 (7.4%) b)ICC: 10/50 (20%)	General Japanese population (individuals ≥20 years of age)	HCV-Ab prevalence equal to 1,2% in the Japanese individuals ≥20 years of age	NR	a 1 b 3
Mohammad-Alizadeh AH Asian Pac J Cancer Prev. 2012	Iran	Case series with control group Period: 2004-2011	Histologic confirmation ERCP MRCP	CCA : 43/283 (15.2%) No distinction between HCV and number of ICC and ECC cases	Total subjects: 566 Patients with the primary or final diagnosis of CAA, admitted to gastroenterology ward of a tertiary academic center in Tehran-Iran	Gallstones 72/283 (25.4%), diabetes 70/283 (24.6%), HBV infection 52/283 (18.3%),%), primary sclerosing cholangitis 16/283 (5.6%) smoking 120/283 (42.3%).	NR	1 1
Nuzzo G Updates Surg (2010)	Italy	Case series with control group Period: 1997- 2008	Histologic confirmation	8/55 (14.5%) (2 patients with HBV coinfection), undergoing surgical resection at Policlinico Gemelli, Rome	Total subjects: 55	47/55 (76.5%)	NR	1 2
Perumal V Human Pathology 2006	USA	Case series with control group Period: NR	Histologic confirmation	2/11 (18.2%)	10 liver specimens from anti-HCV negative individuals and	Total subjects : 21	NR	1 5

					13 liver specimens from individuals who were negative for HBV surface antigen by serologic testing, used as negative controls HCV RNA–positive liver tissues from HCV positive cases used as positive controls for HCV RNA detection , at Johns Hopkins Hospital, Baltimore			
Portolani N Annals of Surgical Oncology 2008	Italy	Case series with control group Period: 1990-2006	Histologic confirmation or typical findings on ultrasound., CT-, MRI- examination	ICC= 33 patients undergoing resection and 16 not resected 6/33 (18.1%)	Total subjects : 51 Patients diagnosed with ICC-HCC at the Surgical Clinic of Brescia University, Italy	ICC-HCC=18 patients undergoing resection 11/18 (61.1%)	NR	1 3
Qu Z Asia–Pacific Journal of Clinical Oncology 2012	China	Case series with control group Period: January 1990 - June 2001	Histologic confirmation of ECC	ECC: 305, 139 with test for anti- HCV ECC:6/139 (4.3%)	Total subjects: 353 Patients with BBD with cholelithiasis or acute cholangitis, undergoing surgical intervention selected as controls at Tianjin Nankai Hospital, Tianjin Third Central Hospital, Tianjin Medical University General Hospital and The Second Hospital of Tianjin Medical University hospitals in the corresponding time period.	BBD:480, 214 with test for anti-HCV BBD:12/214 (5.6%)	NR	4 7
Shaib YH Gastroenterology 2005	USA	Hospital-Based Case–Control Study Period: 1993-1999	Histologic confirmation HCV defined by using ICD-9 codes for HCV (ICD-9 codes 070.41, 070.44, 070.51, 070.54, and V02.62) or for unspecified	Data obtained from the National Cancer Institute (NCI)'s Surveillance, Epidemiology and End Results program SEER–Medicare database, linking SEER registry	Controls included in the study derived from the 5% random sample of Medicare-enrolled beneficiaries with no cancer of any type residing in the geographic regions of SEER registries	90,834 controls c) HCV-specific codes: 161 (0.2%) a) HCV (including unspecified hepatitis): 940	Years of search for risk factors to minimize the possibility of differing testing and diagnosis trends	0 1

			hepatitis (ICD-9 codes 070.9, 571.4, 571.8, and 571.9).	information with Medicare claims data, it is a program of the NCI to collect population-based cancer incidence and survival data, including population-based cancer registries in 5 states and 6 metropolitan areas (about 14% of the USA population). ICC cases: 625 c) HCV-specific codes: 5/625 (0.8%) a) HCV (including unspecified hepatitis):35/625 (5.6%)		(1%)		
Shaib YH Am J Gastroenterol 2007	USA	Hospital-Based Case–Control Study Period: 1992-2002	Histologic confirmation	246 patients undergoing surgical resection because of ICC: 5/83 (6%) ECC: 6/163 (3.7%)	Total patients : 482 Controls randomly selected from an existing database of healthy individuals at M.D. Anderson	2/236 (0.8%)	Gender, ethnicity, and age (±5 yr)	
Shin RH Int J Epidemiol 1996	Korea	Case-control study Period: August 1990-August 1993	Histologic confirmation or typical findings on ultrasound., CT-, MRI- examination	41 patients with CCAs 203 patients with HCC a)29/41 patients with tests for antiHCV/HBV status. 4/29 (13.8%) HCV positive b) 128/203 patients with test for antiHCV/HBV status 17/128 (13.3%) HCV positive	a)Inpatients without liver disease, systemic disease, and malignant disorders from the Departments of Ophthalmology or Otorhinolaryngology b) healthy people who had visited the Non-Communicable Disease Control Center All subjects were visited at the Tnje University Pusan Paik Hospital	c) 203 d) 203 394/406 subjects with tests for anti-HCV status. 23/394 (6.6%) HCV positive	Sex and age (± 4 years)	
Songsivilai S Trans R Soc	Thailand	Case series with control group	Histologic confirmation	0/30	Total subjects : 110 Patients with HCC,	9/80 (11.2%)	NR	

Trop Med Hyg. 1996		Period: July 1993 – June 1995			undergoing surgical resection at Siriraj Hospital, Mahidol University, Bangkok			
Srivatanakul P Asian Pacific J Cancer Prev, 2010	Northeast Thailand	Case-control study Period: September 1999 -2001	Histology, or typical findings on ultrasound examination with an elevated titre (≥40units/ml) of CA 19-9 and normal level of alpha-fetoprotein (AFP< 20ng/ml)	7/103 (6.8%)	Total subjects : 206 Community hospitals in Nakhon Phanom Province and Nakhon Phanom Provincial Hospital	0/103	Sex, age (± years) and place of residence.	
Taguchi J J Gatroenterol Hepatol 1996	Japan	Case series with control group Period: January 1988- July 1995	Histologic confirmation	14/20 (70%)	Total subjects : 367 HCC-CCA : 23/367, 20 patients with anti-HCV markers	6/20 (30%)	NR	
Tanaka M J Viral Hepat. 2010	Japan	Cohort study Period: 1991-1993	ICC cases identified by the ICD-10 code (C22.1). diagnosis of ICC was based on histological examination and/or combined clinical, radiological (echography, CT and endoscopic retrograde cholangio-pancreatography) and laboratory findings	ICC: 11 cases 1/11 (9.1%)	154 814 study subjects voluntary blood donors	1927/154 814 (1.2%)	NR	
Tomimatsu M Cancer 1993	Japan	Case series with control group Period: January 1985 - December 1990	Histologic confirmation	a) CCA : Anti-HCV + : 4/13 (30.8%) HBsAg+ : 3/13 (23.1%) Anti-HCV- /HBsAg- : 6/13 (46.1%) b) CCA-HCC : Anti-HCV + : 5/7 (71.4%) HBsAg+ : 1/7 (14.3%) Anti-HCV- /HBsAg- : 1/7 (14.3%)	Total subjects : 141 Patients with HCC, undergoing surgical resection at the Institute of Gastroenterology of Tokyo Women's Medical College	Anti-HCV + : 85/121 (70.3%), Anti-HCV+ /HBsAg+ : 5/121 (4.1%) HBsAg + : 16/121 (13.2%) HBsAg-/anti-HCV - : 15/121 (12.4%)	NR	
Uenishi T Journal of Surgical Oncology 2014	Japan	Case series with control group Period: January 2000 - December 2011	Histological confirmation	33/90 (36.7%)	Total subjects : 90 Patients enrolled at Hirakata and Osaka University Hospital	57/90 (63.4%)	NR	

Yamamoto M Cancer 1998	Japan	Case-series Period: February 1990- March 1996	Histologic confirmation	50 patients with ICC Anti-HCV positive : 16/50 (32%) HBsAg+/Anti-HCV positive : 1 (2%)	NR	NR	NR	3 4
Yamamoto S Cancer Sci 2004	Japan	Hospital case-control based study Period: January 1991 - December 2002	Histologic confirmation	18/50 (36%)	Total subjects : 255 Control patients enrolled at the two major medical centers of Osaka City	7/205 (3%)	Gender, 5-year age group, and operation date (within 1 year)	3 4
Yano Y Jpn J Clin Oncol 2003	Japan	Case-control study Period: January 1978-December 1998	Histologic confirmation	HCV alone : a) HCC-CCA= 10/26 (38.5%) b) CCA= 5/53 (9.4%) HCV+HBV: 1/53 (2%)	Total subjects : 1172 Patients with HCC, undergoing surgical resection at the Department of Surgery, National Cancer Center Hospital, Tokyo	HCV alone: HCC=526/1093 (48%) HCV+HBV: 16/1093 (1%)	Patients compared for: age, sex, serum HBsAg, anti-HCV status and serum AFP and CEA levels	a (b 1
Wahab A M Hepatogastro-enterology 2007	Egypt	Case series Period: January 1995-October 2004	Histologic confirmation or typical findings on CT, ERCP, MRI and PTD	Total patients : 440 238/440 (54.1%)	NR	NR	NR	5 5
Welzel TM Clin Gastroenterol Hepatol. 2007	USA	Population-based case-control study Period: 1993-1999	Identification of CAA cases from the Surveillance, Epidemiology and End Results-Medicare databases by means of ICD-9-CM diagnosis codes: (C22.0, C22.1, C24.0, 8010, 8020, 8041, 8070, 8140, 8144, 8160, 8161,	a) ICC=5/ 535 (0.9%) b) ECC= 5/ 549 (0.9%)	102,782 cancer-free controls identified using the Surveillance, Epidemiology and End Results-Medicare databases.	142/102,782	Cases/controls matched on the year of search for risk factors. Risk factors for ICC or ECC categorized into five diagnostic	I 0 1 E 0 1

			8260, 8310, 8480, 8490, 8560) . Identification of HCV infection by means of ICD-9-CM diagnosis codes 070.41, 070.44, 070.51, 070.54 and 070.7				groups based on ICD-9 or CPT- codes	
Zhou HQ Hepatobiliary Pancreat Dis Int 2007	China	Case-series Period: January 1996 - November 2005	Histologic confirmation	a)HCC : 132 patients Anti-HCV positive : 26/132 (19.7%) b)CCA : 44 patients Anti-HCV positive : 4/44 (9.1%) c)cHCC-CCA : 15 anti-HCV positive : 3/15 (20%)	NR	NR	NR	a (b 1 c 4
Zhou YM World J Gastroenterol 2008	China	Hospital-based-case control Study Period: February 2004 - May 2006	Histologic confirmation	9/312 (2.9%)	Total patients : 750 Controls were selected from patients who were unaffected by liver diseases in the Changhai Hospital of the Second Military Medical University	6/438 (1.4%)	age (± 5 years), sex, and date of hospital admission	2 4
(B)								
Torbenson M Am J Surg Pathol 2007	USA	Review of liver explants with control group from 3 transplant centers Period: 1995 - 2005	Histologic confirmation in explanted livers	a) HCV alone= 10/511 (2%) b) HCV + alcohol = 4/85 (5%)	1058 total liver explants Control groups included: (1) alcohol cirrhosis, (2) chronic hepatitis B infection, (3) nonviral causes of cirrhosis such as cryptogenic cirrhosis, (4) noncirrhotic livers that were transplanted for fulminant liver failure	1) Alcohol cirrhosis= 5/ 112 (4%) 2) HBVchronic hepatitis = 0/67 (0%) 3) Cirrhosis from nonviral and non alcohol causes= 0/149 (0%) 4) Noncirrhotic =/134 (0%)	NR	a 3 b 9
Wu TT Cancer 2009	USA	Review of liver explants with control group at Mayo Clinic Rochester, Minnesota Period: 1995 -	Histologic confirmation in explanted livers	a) Alcohol-related and HCV-related cirrhosis : 24 26 (92%) b) HCV-related cirrhosis: 27/44 (61%)	244 total liver explants Causes: 94 alcohol-related cirrhosis, 44 HCV-related	Noncirrhotic 27/80 (34%) alcohol-related cirrhosis 86/ 94 (91%)	NR	a (b (

		2007			cirrhosis, 26 alcohol- and HCV-related cirrhosis, 28 massive hepatic necrosis, 24 correction of metabolic conditions, 16 primary or metastatic tumors, 8 nodular regenerative hyperplasia, 2 subacute Budd- Chiari syndrome, 2 liver failure during the first week after transplantation			

BIN=Biliary Intraepithelial Neoplasia, BBD=benign biliary disease, CT= Computed Tomography, ERCP=endoscopic retrograde cholangiopancreatography, HBsAg= hepatitis surface antigen, cHCC-CC patients= combined HCC and CCA, ICC= intrahepatic colangiocarcinoma, ECC= extrahepatic colangiocarcinoma colangiocarcinoma, CCA= colangiocarcinoma, HCC=hepatocellular carcinoma, MRI=Magnetic Resonance Imaging, PTD= Percutaneous Transhepatic Cholangiography, NR= not reported, NA=not available

Supplementary Table 3. Characteristics of available studies, reported in English, designed to assess the association between HCV infection and PAC risk.

First author/ Journal/ Publication Year	Country	Study design/ Study period	PAC diagnosis	HCV positive PAC (n)/ total PAC Cases (N)	Control source	HCV positive controls (n)/ Controls (N)	Matching criteria	Percentage of HCV-positive cases with 95 % CI	Main conclu
Amin J, J Hepatol 2006	Australia	Community-based cohort-study Period: 1990-2002	Identification of pancreatic cancer cases by means of ICD-10-diagnosis codes	-Individuals with HCV infection: 75834 PAC detected: 17/75834 (0.02%)	Incidence observed in the study cohort was compared to expected incidence derived from NSW population cancer rates by calculating standardised incidence ratios	SIRs: 1.4 (0.8-2.2):	NR	**0.02 (0.01-0.03)**	No evidence supporting a association between HCV infection and development
Chang MC World J Gastroenterology 2014 (49)	China	Case-control study Period: 2000-2013	Histological or citological	22/585 (3.8%)	Controls were individuals recruited from a free screening program in a community located in Northern Taiwan	45/1,716 (2.6%)	age (±5 years) and sex	3.8 (2.2-5.3)	HCV infectio associated wi higher risk of PAC development, after adjusten for age, sex, diabetes and smoking (independent factors for PA
El Serag Hepatology 2009 (47)	USA	Cohort study Cohort: 718,687 patients PAC detected: 617 Period: 1988-2004	Identification of PAC cases by means of ICD-9-CM diagnosis codes (157.0, 157.1, 157.2, 157.3, 157.8, 157.9) Identification of HCV infected subjects by means of ICD-9-CM diagnosis codes (070.41, 070.44, 070.51, 070.54 and V02.62)	146,394 patients in HCV-infected cohort PAC detected:140/146,394 (0.09%)	Sources included inpatients records from more than 150 of USA Veterans Affairs (VA) hospitals in the Patients treatment file and outpatients records from any VA facility in the Output Clinic File	572,293 patients in HCV-uninfected cohort PAC detected: 477	HCV-uninfected and HCV-infected subjects matched by age (± 1 year) and sex	**0.09 (0.08-0.11)**	Higher risk o PAC in patie HCV-infecte cohort , but th association w attenuated af adjustement f alcohol use, pancreatitis, choledocholit cholelithiasis primary scler cholangitis
Hassan MM J Clin Oncol. 2008 (43)	USA	Hospital-based case-control study Period: 2000-2007	Histological confirmation	6/474 (1.5%)	Community-based (healthy genetically unrelated family members of patients with cancer other	9/872 (1%)	age (± 5 years), sex and race	0.8 (0.02-1.6)	HCV infectio associated wi higher risk of PAC develop

					than pancreatic, GI, lung or head cancers)				
Huang J Br J Cancer 2013	Sweden	Retrospective Nationwide cohort study 197,208 participants: Period: 1990-2006	Identification of PAC cases from the Swedish Cancer Register (International Classification of Disease ICD-7: 157) and from the Cause of Death Register (ICD-9: 157; ICD-10: C25)	Individuals in HCV reference cohort : 39,442 PAC detected: 34 /39,442 (0.09%)	Control population obtained from the national surveillance database at the Swedish Institute for Infectious Disease Control. The expected numbers of calculated PAC from the observed person-time in each 5-year age group by sex and the corresponding Swedish population incidence rates.	Expected number of PAC: 16. 5	age (± 5 years) and sex	**0.09 (0.05-0.11)**	Statistically significant increased risk PAC develop
Omland LH Clinical Epidemiology 2010:	Denmark	Cohort-study Period: 1994 -2003	Patients and subjects with HCV infection identified by means of : -The Danish National Hospital Registry (DNHR) -The Danish Cancer Registry People listed in DNHR with at least one diagnosis of acute or chronic HCV infection (ICD-10 B17.1 and 18.2) were included Cancer diagnoses based on the Danish version of the international classification	4,349 patients with HCV infection in the DNHR 4/4,349 PAC detected (0.1%)	The expected number of cases of cancer after a diagnosis of HCV infection using Danish incidence rates of first cancer diagnoses according to sex, age, and year of diagnosis in 1-year intervals was calculated.	Expected number of PAC: 1.01	NR	**0.1 (0-0.18)**	Association between HCV infection and higher risk of PAC develop

			of diseases, 8th revision (ICD-8) until Dec 31, 1993, and 10th version (ICD-10) thereafter						
Qiwen Ben Pancreas 2012 (46)	China	Double-centre ongoing hospital-based case-control study. Period: January 1,2004-August 31, 2008 January 1,2003-Octobet 31, 2009	Histological or citological confirmation	14/943 (1.5%)	Patients admitted to the same Hospitals (Ruijin Hospital and Changai Hospital, Shangai for any acute conditions)	12/1128 (1,1%)	age (±3 years) and sex	1.5 (0.7-2.2)	No higher HC prevalence in patients with in comparison with controls
Swart A BMJ Open 2012	Australia	Cohort-study Patients considered in the study: 29 613 1 January 1993 - 31 December 2007	Individuals registered on the Pharmaceutical Drugs of Addiction System, a record of all NSW Health Department authorities that administer methadone or buprenorphine to opioid-dependent people as opioid substitution therapy. Solid cancers classified according to the International Classification of Diseases (ICD), 10th revision, haematopoietic neoplasms and Kaposi sarcomas classified according to the ICD for Oncology, 3rd edition	Subjects with HCV infection alone: 14,892 Observed number of PAC in HCV-positive cohort: 20/14,892 (0.1%)	Calculation of expected number of incident PAC:	Expected number of PAC: 7.12	NR	**0.13 (0.08-0.21)**	Increased risk PAC in patien with HCV infection
Woo SM J Korean Med Sci 2013 (49)	Korea	Case-control study Period: 2001-2011	Histological or radiological/ clinical confirmation	753 patients with PAC 724/753 with available anti-HCV test 21/724 (2.8%)	Individuals subjected to routine health examination in the Cancer Screening Cohort	36/3,012 (1.2%)	age (±5 years) and sex	2.9 (1.7-4.1)	Seropositivity anti-HCV, infection, ma increase the r of developing in Korea

NR= not reported, SIR=standardised incidence ratio

Supplementary Table 4. Characteristics of available studies, reported in English, designed to assess the association between HCV infection and breast cancer risk.

Author/ Journal/ Publication Year	Country	Study Design/ StudyPeriod	Diagnosis	Sample size (HCV positive breast cancer cases)	Control source	HCV positive controls/ controls	Matching factors	Percentage of HCV-positive cases with 95 % CI	Main conclusions
Amin J, J Hepatol 2006	Australia	Community-based cohort-study Period: 1990-2002	Patients'data obtained from: -New South Wales (NSW) Australia Health Department's Notifiable Diseases Database (NDD) for notification of newly diagnosed HCV infection -NSW Central Cancer Registry (CCR) for notification of incident cancer cases -National Death Index (NDI) database, containing records of all deaths in Australia since 1980 Identification of breast cancer cases by means of ICD-10- diagnosis codes	Individuals with HCV infection : 75,834 Breast cancers detected: 50 50/75,834	Incidence observed in the study cohort was compared to expected incidence derived from NSW population cancer rates by calculating standardised incidence ratios	SIR: 0.3 (0.4-0.5)	NR	**0.07 (0.05-0.09)**	No evidence supporting an association between HCV infection and breast cancer development
Hwang JP J Oncol Pract. 2014	USA	Cohort-study Period: January 2004 -April 2011	Patients'data, obtained from four institutional	141,877 patients with cancer, who were newly registered at MD	NR	NR	NR	a)25 (5.5-57.2) b)9.9 (8.1-	HCV screening rates were low, even among patients

			sources: *Tumor registry:* to assess patients' demographic characteristics *Pharmacy informatics:* to evaluate chemotherapy drugs and dates administered. *Patient accounts:* to identify study patients' International Classification of Diseases (ninth edition; ICD-9) codes *Laboratory informatics:* to determine HCV antibody (anti-HCV) and ALT test dates and results	Anderson Cancer during the study period. Patients considered in the study: 16, 773. HCV screened subjects: 2,330/16, 773 (13.9%) HCV screened females: 1038 HCV-positive patients with cancers: 35/2330 (1.5%) HCV-positive females with cancers: 12 a)HCV-positive females with breast cancers: 3/12 b)HCV-negative females with breast cancer: 102/1026				11.8)	with risk factors, and the groups with the highest rates of screening did not match the groups with the highest rates of a positive test result.
Larrey D World J Gastroenterol 2010(46)	France	Case serie with control gorup Period: NR	Females with history of HCV-related chronic infection, observed in Liver Unit of Montpellier School of Medicine, France, for chronic liver diseases in several occasions for a period longer than 1 year. Chronic hepatitis proved by liver biopsy and/or	17/294 (5.8%)	Females sequentially and prospectively seen during the same period with chronic liver disease over 1 year, with well defined clinical, radiological and histological characteristics [chronic- HBV, alcoholic-liver disease, auto-immune hepatitis, hemochromatosis, non alcoholic fatty liver disease (NAFLD), cholangitis]	5/107 (4.7%)	NR	5.8 (3.1-8.4)	Chronic HCV infection is not a strong promoter of breast carcinoma in adult females of any age

			biological markers of inflammation and fibrosis.						
Omland LH Clinical Epidemiology 2010:	Denmark	Cohort-study Period: 1994 -2003	Patients and subjects with HCV infection identified by means of : -The Danish National Hospital Registry (DNHR) -The Danish Cancer Registry People listed in DNHR with at least one diagnosis of acute or chronic HCV infection (ICD-10 B17.1 and 18.2) were included Cancer diagnoses based on the Danish version of the international classification of diseases, 8th revision (ICD-8) until Dec 31, 1993, and 10th version (ICD-10) thereafter	4,349 patients with HCV infection in the DNHR 2 breast cancer detected 2/4,349 (0.05%)	The expected number of cases of cancer after a diagnosis of HCV infection using Danish incidence rates of first cancer diagnoses according to sex, age, and year of diagnosis in 1-year intervals was calculated.	Expected number of breast cancers 8.05	NR	**0.05 (0-0.1)**	No association between HCV infection and higher risk of breast cancer development
Su FH, BMC Cancer 2011 (43)	Taiwan	Population-based study Period: 2000-2008	Data retrieved from National Health Insurance Research Database (NHIRD), which is maintained by the National Health	56/1958 (2.9%)	Randomly selected and matched individuals without a history of breast cancer (control to patient ratio was 4:1).	178/7832 (2.3%)	age- and sex	2.9 (2.1-3.5)	HCV infection associated with early onset risk of breast cancer in areas endemic for HCV

			Research Institute (NHRI), Taiwan. Newly diagnosed breast cancer identified from the registry for Catastrophic Illness Patients Database (ICD-9-CM code 174 and 175). Identification of HCV infected subjects by means of ICD-9-CM diagnosis codes (ICD-9- CM 070.41, 070.44, 070.51, 070.54, and V02.62)						
Swart A BMJ Open 2012	Australia	Cohort-study 1 January 1993 - 31 December 2007	Individuals registered on the Pharmaceutical Drugs of Addiction System, a record of all NSW Health Department authorities that administer methadone or buprenorphine to opioid-dependent people as opioid substitution therapy. Solid cancers classified according to the International Classification of Diseases (ICD), 10th revision, haematopoietic neoplasms and Kaposi sarcomas classified according to the ICD for Oncology, 3rd	Patients considered in the study: 29, 613 Subjects with HCV infection alone: 14,892 Observed number of breast cancer in HCV-positive cohort: 48 48/14,892 (0.03%)	Calculation of expected number of incident breast cancer	Expected number of breast cancers: 101	NR	**0.03 (0.02-0.04)**	No evidence supporting an association between HCV infection and breast cancer development

			edition						

NR= not reported; SIR, standardised incidence ratio

Supplementary Table 5. Characteristics of available studies, reported in English, assessing the association between HCV infection and renal cancer.

Author/ Journal/ Publication Year	Country	Study Design/ StudyPeriod	Diagnosis	Sample size (HCV positive RCC cases)	Control source	HCV positive controls/ controls	Matching Factors	Percentage of HCV-positive cases with 95 % CI	Main conclusions
Amin J, J Hepatol 2006	Australia	Community-based cohort-study Period: 1990-2002	Identification of renal cancer cases by means of ICD-10-diagnosis codes	Individuals with HCV infection: 75834 RCC detected: 19 19/75,834	Incidence observed in the study cohort was compared to expected incidence derived from NSW population cancer rates by calculating standardised incidence ratios	SIR: 0.9 (0.6-1.4)	NR	0.02 (0.01-0.03)	No evidence supporting an association between HCV infection and kidney cancer development
Budakoğlu B Med Oncol 2012	Turkey	Case series with control group 2005 - 2010	Histological confirmation	15/903 (1.7%)	Data collected in previous prevalence studies in healthy subjects in three different geographical areas of the Turkey, used as control group	81/5267 (1.5%)	NR	1.7 (0.8-2.4)	No higher frequency of HCV positivity in RCC patients in comparison with healthy people
Gonzalez HC Dig Dis and Sci 2015	USA	Case series with control group January 2011 - August 2013	Histological confirmation	a)Anti-HCV positive : 11/140 (7.9%) b)HCV-RNA positive : 9/140 (6.4%)	Consecutive individuals newly diagnosed with colon cancer. The control group recruited simultaneously and from the same health care system (Henry Ford Health System in Detroit, Michigan)	Anti-HCV positive : 1/100 (1%) HCV-RNA positive : 0/100	NR	a) 7.9 (3.4-12.3) b) 2.3 (10.5)	Increased risk of RCC in subjects with HCV chronic infection

Gordon SC Cancer Epidemiol Biomarkers Prev 2010	USA	Cohort study Period: 1997-2006	Use of administrative data from Henry Ford Hospital, an integrated healthcare delivery system serving southeastern Michigan. Cancer diagnosis codes in administrative databases [International Classification of Diseases, 9th ed., Clinical Modification (ICD-9-CM) codes in the range of 140 through 208.9]	72,487 patients tested for anti-HCV 3057/72,487 anti-HCV positive patients 17/3057 (0.6%) with RCC	Control cohort of patients who tested negative for anti-HCV	64006/72,487 anti-HCV negative patients 177/64006 (0.3%) with RCC	NR	0.6 (0.3-0.8)	Chronic infection with HCV confers an increased and independent risk for developing RCC
Hofmann JN Eur J Cancer Prev. 2011	Sweden	Nationwide register-based cohort-study Period: 1990 - 2008	HCV diagnosis extracted from the national surveillance database at the Swedish Institute for Infectious Disease Control (SMI). Cancer diagnoses were coded using the seventh revision of the International Classification of Diseases (ICD-7) (ICD-7 codes 180.0 and 180.9).	43,000 Lag period after HCV notification a) None: 38, Expected: 27.1 b) Three months 33 Expected: 26.5 c) One year :29 Expected: 24.9	A non-HCV-infected cohort selected from the general population	215,000	Year of birth, sex, and county of residence in Sweden, five subjects never diagnosed with HCV infection were matched to each HCV-infected subject	a) 0.06 (0.09-0.21) b) 0.05 (0.08-0.21) c) 0.05 (0.07-0.21)	In the cohort of HCV-infected subjects, no increased risk of developing kidney cancer but an enhanced risk of non-cancer chronic kidney disease, particularly among women.
Malaguarnera M Eur J Int Medicine (2006)	Italy	Case-control study Period: NR	All cancer patients: 236 HCV diagnosis performed with II G ELISA test. Cancers diagnosed at Garibaldi	15 patients with RCC 8/15 (53%) HCV positive patients	Elderly volunteers evaluated at Garibaldi Hospital, Catania	30/300 (10%)	Age, sex and previous blood transfusions	53.3 (26.5-78.7)	High prevalence of anti-HCV antibodies in patients with renal cancer.

			Hospital						
Omland LH Clinical Epidemiology 2010:	Denmark	Cohort-study Period: 1994 -2003	Patients and subjects with HCV infection identified by means of : -The Danish National Hospital Registry (DNHR) -The Danish Cancer Registry People listed in DNHR with at least one diagnosis of acute or chronic HCV infection (ICD-10 B17.1 and 18.2) were included Cancer diagnoses based on the Danish version of the international classification of diseases, 8th revision (ICD-8) until Dec 31, 1993, and 10th version (ICD-10) thereafter	4,349 patients with HCV infection in the DNHR 4 renal cancer detected 4/4,349	The expected number of cases of cancer after a diagnosis of HCV infection using Danish incidence rates of first cancer diagnoses according to sex, age, and year of diagnosis in 1-year intervals was calculated.	Expected number of kidney cancers: 1.11	NR	0.1 (0-0.2)	Association between HCV infection and higher risk of renal cancer development
Swart A BMJ Open 2012	Australia	Cohort-study 1 January 1993 - 31 December 2007	Individuals registered on the Pharmaceutical Drugs of Addiction System, a record of all NSW Health Department authorities that administer methadone or	Patients considered in the study: 29, 613 Subjects with HCV infection alone: 14,892 Observed number of RCCs in HCV-positive cohort: 20	Calculation of expected number of incident RCCs	Expected number of RCCs: 18.1	NR	0.1 (0.08-0.20)	No evidence supporting a strong association between HCV infection and RCC development

			buprenorphine to opioid-dependent people as opioid substitution therapy. Solid cancers classified according to the International Classification of Diseases (ICD), 10th revision, haematopoietic neoplasms and Kaposi sarcomas classified according to the ICD for Oncology, 3rd edition	20/14,892					

RCC=renal cell carcinoma, SIR,=standardised incidence ratio

Supplementary Table 6. Characteristics of available studies, reported in English, assessing the association between HCV infection and oral or skin cancer.

Author/ Journal/ Publication Year	Country/ Ethnicity	Study Design/ Study Period	Diagnosis	Sample size (Cases/ controls)	Control source	HCV positive controls/ controls	Matching factors	Percentage of HCV-positive cases with 95 % CI	Ma con
Amin J, J Hepatol 2006	Australia	Community-based cohort-study Period: 1990-2002	Identification of skin/oral cancer cases by means of ICD-10-diagnosis codes	Individuals with HCV infection: 75,834 Skin/oral cancer: 19 * * including , mouth (7 cases), tongue (6 cases), tonsil (6 cases) no skin cancers described	Incidence observed in the study cohort was compared to expected incidence derived from NSW population cancer rates by calculating standardised incidence ratios	SIR: Mouth: 1.5 (0.7-3.2) Tongue: 1.1 (0.5-2.4) Tonsil: 2.1 (1-4.8)	NR	0.02 (0.01-0.03)	No sup ass bet infe ski can dev low risk can
Eftekharian A Eur Arch Otorhinolaryngol 2012	Iran	Case-series 107 patients with SCCHN Period: October 2008-June2010	Histological confirmation: SCCHN	1/107 (0.9%)	NR	NR	NR	0.9 (0-2.7)	HC in I risk SC
Gandolfo S Oral Oncol 2004	Italy	Case-series 402 patients with OLP Patients with available HCV test: 357 HCV positive patients: 69/357 (19.3%) Period: January 1988 - July 1999	During the follow-up period: 9 patients developed an oral squamous cell carcinoma. Histological confirmation: OSCC	HCV positive patients with OSCC: 4/9 (44.5%)	NR	NR	NR	44.5 (11.9-76.9)	Pos inc for HC infe pati lich (OI
Nagao Y J Oral Pathol Med 1995	Japan	Case-series 100 patients with oral cancer enrolled Period: January 1989-October 1993	Histological confirmation: Different histotypes	24/100 (24%)	Patients with non-malignant disease receiving dental treatment at the Department of Oral Surgery of the Kurume University Patients with gastric cancer	a) 11/104 (10.6%) b)12/113 (10.6%)	a) Age and sex	24 (15.6-32.3)	HCV pat cha the cav HCV in can
Nagao Y J Oral Pathol Med 2000	Japan	Biopsies of 36 patients, including: a) OLP: 19 b) Oral cancer: 17 Period: NR	Histological confirmation: Well-differentiated SCCHN	a) 14/19 (73.7%) b) 7/17 (41. 2%)	Biopsies of 10 patients, including: c) Non-malignant disease with HCV d) Non-malignant disease without HCV	c): 6 d): 4	NR	a)73.7 (53.8-93.4) b) 41. 2 (17.8-64.5)	HCV pat cha the cav HCV in

									can
Nobles J Laringoscope 2004	USA	Case-series 100 patients with SCCHN enrolled. Period: June 1991 - December 2002	Histological confirmation: SCCHN	21/100 (21%)	NR	NR	NR	21 (13-28.9)	A la nur pat (21 SC incl this coi wit Thi pre sig inc wh con wit gen pop (1.4 pop VA (9.9
Omland LH Clinical Epidemiology 2010	Denmark	Cohort-study Period: 1994 - 2003	Patients and subjects with HCV infection identified by means of : -The Danish National Hospital Registry (DNHR) -The Danish Cancer Registry People listed in DNHR with at least one diagnosis of acute or chronic HCV infection (ICD-10 B17.1 and 18.2) were included Cancer diagnoses based on the Danish version of the international classification of diseases, 8th	4,349 patients with HCV infection in the DNHR 4 oropharyngeal cancers detected	The expected number of cases of cancer after a diagnosis of HCV infection using Danish incidence rates of first cancer diagnoses according to sex, age, and year of diagnosis in 1-year intervals was calculated	Expected number of oropharyngeal cancers: 1.73	NR	0.1 (0-0.2)	No ssso bet infe hig oro can dev

			revision (ICD-8) until Dec 31, 1993, and 10th version (ICD-10) thereafter						
Su FH PlosOne 2012	Taiwan	Nationwide Population-Based Cohort Study HCV positive patients:5,311 HCV and HBV positive patients: 3,519 Period: 1996-2008	Data obtained from the Taiwan National Health Insurance Research Database (NHIRD). HCV cases identified by means of ICD-9-CM diagnosis codes (ICD-9-CM: 070.41, 070.44, 070.51, 070.54, V02.62)	a)21/5,311 b)9/3,519	Controls identified by means of a systematic random sampling method to select 4 insured people without viral hepatitis for every insured person with viral hepatitis during the same period	147/84,796	age-, sex, index-date and year	a)0.4 (0.2-0.5) b)0.3 (0.09-0.4)	HC is a for can add sub HC ten ear risk cav ma
Swart A BMJ Open 2012	Australia	Cohort-study 1 January 1993 - 31 December 2007	Individuals registered on the Pharmaceutical Drugs of Addiction System, a record of all NSW Health Department authorities that administer methadone or buprenorphine to opioid-dependent people as opioid substitution therapy. Solid cancers classified according to the International Classification of Diseases (ICD), 10th revision, haematopoietic neoplasms and Kaposi sarcomas classified according to the ICD for Oncology, 3rd edition	Patients considered in the study: 29 613 Subjects with HCV infection alone: 14,892 Observed number of following cancer in HCV-positive cohort: a)Tonsil : 10 b)Mouth ;8 c) Salivary gland :4 d) Tongue : 9 Total : 31	Calculation of expected number of incident tonsil/mouth//salivary gland/tongue cancers	Expected number of oral cancers: Tonsil : 2.96 Mouth ;3.54 Salivary gland : 2.75 Tongue :5.35	NR	0.2 (0.1-0.3)	Pos ass bet and mo can No ass bet infe ton sali can

Takata Y Oral Diseases 2002	Japan	Case series Patients with anti-HCV antibodies: 2613 HCV positive patients:151/2613 (5.8%) Period: January 1989 - December 1998	Histological confirmation Histotype not reported	25/245 (10.2%)	NR	NR	NR	10.2 (6.4-13.9)	Hig ant pre in p wit can Pos imp ass bet can HC infe wit incr pre dep hig ant pos pat

OLP= Oral Lichen Planus; OSCC=oral squamous cell carcinoma, SCCHN= squamous cell carcinoma of the head and neck, SIR,=standardised incidence ratio VA= veterans administration

Supplementary Table 7. Characteristics of available studies, reported in English, assessing the association between HCV infection and thyroid cancer.

Author/ Journal/ Publication Year	Country	Study Design/ StudyPeriod	Diagnosis	Sample size	Control source	Controls	Matching Factors	Percentage of HCV-positive cases with 95 % CI	Main conclusions
Amin J, J Hepatol 2006	Australia	Community-based cohort-study Period: 1990-2002	Identification of thyroid cancer cases by means of ICD-10-diagnosis codes	Individuals with HCV infection: 75,834 Thyroid cancers detected: 9	Incidence observed in the study cohort was compared to expected incidence derived from NSW population cancer rates by calculating standardised incidence ratios	SIR: 0.3 (0.2-0.7)	NR	0.01 (0-0.02)	No evidence supporting an association between HCV infection and thyroid cancer development
Antonelli A. Clin Exp Rheumat 2002	Italy	Case-control study Period: 1999-2001	FNA PTC	94 patients with HCV-associated MC Patients with PTC and HCV-associated MC / patients with HCV-associated MC : 2/94 (2.1%)	Control group obtained from a sample (2401 individuals) of the general population, 5 controls were randomly associated with each MC patient	0/470	Sex	2.1 (0-5)	Possible association between HCV-related MC and thyroid cancer, careful monitoring of the thyroid opportune, during the clinical follow-up of H C V-associated MC patients
Antonelli A. Thyroid 2007	Italy	Case-control study Period: January 1995 - December 2001	FNA PTC	308 HCV positive patients PTC and HCV positive cases/ all HCV positive cases : 6/308 (1.9%)	a) subjects from an iodine deficient area b) subjects from an iodine-sufficient	PTC cases/all HCV negative controls: a) 0/616 b) 1/616	Gender and age (±5 years)	1.9 (0.4-3.4)	High prevalence of thyroid papillary cancer in HCV+ patients, overall in presence of thyroid autoimmunity;

					area				careful thyroid monitoring is indicated during the follow-up of these patients
Giordano TP. JAMA 2007	USA	Cohort study Period: 1997-2004	Identification of HCV infected subjects by means of *ICD-9-CM ,* diagnosis codes of HCV infection (070.41, 70.44, 070.51, 070.54, V02.62) Identification of thyroid cancer by menas of ICD-9-CM diagnosis codes: 193	HCV-positive cohort: 146,394 patients During follow-up, 813 patients in HCV-infected cohort (0.5%) had a HIV diagnosis. 46 patients developed thyroid cancer	Inpatients records from more than 150 USA Veterans Affairs (VA) hospitals in the Patients' treatment file and outpatients records from any VA facility in the Output Clinic File	HCV-negative cohort: 572,293 patients. During follow-up, 35 696 uninfected HCV patients (6.2%) had a recorded HCV diagnosis and 1,539 patients (0.3%) a HIV diagnosis 274 patients developed thyroid cancer	HCV-infected patients, matched according to sex and age on the baseline date	0.03 (0.02-0.04)	No increased, risk for thyroid cancer in HCV-positive cohort
Montella M Oncol Rep 2003	Italy	Case-control study Period 1997 - 1999	Histological confirmation PTC	HCV positive PTC cases/ all PTC cases : 16/130 (12.3%)	Control group including subjects, operated for benign diseases. Cases and controls selected from the hospital tumor registry	242 controls and 311 surgical procedures. HCV positive controls/total controls 18/311	NR	12.3 (6.6-17.9)	Association between HCV and thyroid cancer. This malignancy more readily detectable in countries with a high prevalence of HCV.
Omland LH Clinical Epidemiology 2010:	Denmark	Cohort-study Period: 1994 -2003	Patients and subjects with HCV infection identified by means of : -The Danish	4,349 patients with HCV infection in the DNHR	The expected number of cases of cancer after a diagnosis	Expected number of thyroid cancers: 0.46	NR	0.02 (0-0.06)	No association between HCV infection and higher risk of thyroid cancer

			National Hospital Registry (DNHR) -The Danish Cancer Registry People listed in DNHR with at least one diagnosis of acute or chronic HCV infection (ICD-10 B17.1 and 18.2) were included Cancer diagnoses based on the Danish version of the international classification of diseases, 8th revision (ICD-8) until Dec 31, 1993, and 10th version (ICD-10) thereafter	1 thyroid cancer detected	of HCV infection using Danish incidence rates of first cancer diagnoses according to sex, age, and year of diagnosis in 1-year intervals was calculated				development
Swart A BMJ Open 2012	Australia	Cohort-study 1 January 1993 - 31 December 2007	Individuals registered on the Pharmaceutical Drugs of Addiction System, a record of all NSW Health Department authorities that administer methadone or buprenorphine to opioid-dependent people as opioid substitution therapy. Solid cancers classified according to the International Classification of Diseases (ICD), 10th revision, haematopoietic neoplasms and Kaposi	Patients considered in the study: 29 613 Subjects with HCV infection alone: 14,892 Observed number of thyroid cancer in HCV-positive cohort: 48	Calculation of expected number of incident thyroid cancer	Expected number of thyroid cancers: 34.4	NR	0.3 (0.2-0.4)	No evidence supporting an association between HCV infection and thyroid cancer development

			sarcomas classified according to the ICD for Oncology, 3rd edition						

FNA= Fine neeedle-aspiration, MC=mixed cryoglobulinemia,, PTC= papillary thyroid cancer, SIR,=standardised incidence ratio