Production of interleukin-2 in response to synthetic peptides from hepatitis C virus E1 protein in patients with chronic hepatitis C: relationship with the response to interferon treatment


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Abstract of:

The role of cellular immunity in the clearance of hepatitis C virus after interferon therapy has not yet been elucidated. Here, we analyzed the T cell response to peptides from hepatitis C virus E1 protein in untreated and interferon-treated patients with chronic hepatitis C virus infection.

Methods: We used thirty-six 15-mer synthetic peptides from hepatitis C virus E1 protein (genotype 1a) in a sensitive interleukin-2 production assay in two groups of controls (healthy seronegative individuals and patients with liver diseases unrelated to hepatitis C virus), and three groups of patients with chronic hepatitis C: nine patients who cleared the virus after interferon treatment (group 1), nine patients who failed to respond to the therapy (group 2) and nine previously untreated patients (group 3).

Results: None of the controls responded to any of the peptides tested, whereas 8/9 (88%) of patients from group 1 responded positively. In contrast, only 2/9 (22%) of patients from group 2 showed peptide recognition. In group 3, 5/9 patients (55%) displayed positive response against E1 peptides. When E1 peptides from the sequence corresponding to genotype 1b (the commonest in patients who were non-responders to interferon) were tested in nine additional interferon-resistant patients (group 2*) a positive response was detected in only three of them (33%).

Conclusions: T cell recognition of hepatitis C virus E1 peptides in patients with chronic hepatitis C who exhibit sustained response to interferon therapy is increased as compared with interferon-resistant cases, suggesting that T cell immunity to hepatitis C virus structural proteins may play a role in the clearance of this viral infection.

Gene Transfer and Therapy with Adenoviral Vector in Rats with Diethylnitrosamine-Induced Hepatocellular Carcinoma

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Abstract of:

In rats with diethylnitrosamine (DENA)-induced hepatocellular carcinoma (HCC), we studied in vivo gene transfer efficiency using intraportal injections of recombinant adenovirus carrying the lacZ reporter