peared to attenuate the negative effect of testosterone on the prostate in that subjects who received the dual regimen had no increase in prostate-specific antigen levels and had a significantly lower increase in prostate volume than those treated with testosterone alone (5). While these data are encouraging, they are based on small patient numbers, and the favorable effects on prostate-specific antigen levels may not necessarily translate to a reduction in prostate cancer risk. In addition, while finasteride was shown to reduce the development of prostate cancer in middle-aged men, the incidence of high-grade prostate tumors and sexual side effects was increased (6).

Therefore, I believe that further research is still needed to identify the androgen regimen that confers optimal benefit to older men without compromising prostate health and overall patient safety.

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F.H. has been an advisory board member for Auxilium and has received honoraria from Solvay.

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Hepatitis C Virus Infection: Evidence for an Association With Type 2 Diabetes

Response to Antonelli et al.

Antonelli et al. (1) classified diabetes associated with hepatitis C virus (HCV) infection as type 2. However, these patients show slightly different phenotype than typical type 2 diabetic subjects. Of interest, in our study, HCV diabetic patients presented similar intermediate clinical phenotype with significantly lower BMI (26.5 ± 4.8 vs. 30.9 ± 6.3 kg/m^2), systolic (133.9 ± 14.0 vs. 142.9 ± 25.6 mmHg) and diastolic (84.4 ± 10.2 vs. 88.1 ± 16.0 mmHg) blood pressure, LDL cholesterol (1.9 ± 0.5 vs. 2.7 ± 0.8 mmol/l), and triglycerides (1.4 ± 0.8 vs. 2.6 ± 1.9 mmol/l). Furthermore, these patients showed lower C-reactive protein concentration (1.53 ± 1.23 vs. 3.54 ± 2.53 mg/l).

There is a groundswell of data now to link HCV infection with diabetes. However, serious doubt concerning the true character of diabetes in HCV patients must be emphasized. An autoimmune basis of the HCV-diabetes link is unlikely because no increased prevalence of β-cell autoimmune markers in HCV patients has been found (2). Nonetheless, there is a report of type 1 diabetes 1 year after blood transfusion–related HCV infection (3). Additionally, diabetic HCV patients with mixed cryoglobulinemia are more likely to carry non–organ-specific autoantibodies (4). Interestingly, there is evidence to support the hypothesis that HCV directly damages β-cells or disturbs their function, which ultimately leads to diabetes (5). Finally, there is no question that HCV, by itself, can induce insulin resistance, disturbing the insulin signaling pathway by the function of HCV core protein (6). Moreover, a crucial association between diabetes and the stage of fibrosis in HCV patients, independent of obesity and steatosis, on liver biopsy has also been demonstrated (6).

Diabetes in HCV patients has a unique and complex pathogenesis. Although both insulin resistance and β-cell dysfunction are responsible for the diabetes-HCV association, the specific nature of that link casts doubt on diagnosis of type 2 diabetes in these patients.

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