

garding age, BMI, and serum levels of TNF-RI and TNF-RII. The authors concluded that the HCV genotype must be included when evaluating insulin resistance in HCV-infected patients and suggest that other factors apart from TNF-α could be involved in the development of insulin resistance during HCV chronic infection. This letter deserves the following comments. First, we would like to specify that all the patients included in our study were genotype 1 (which is largely the most prevalent), and, therefore, a potential bias due to this issue can be absolutely ruled out. Second, and more importantly, it should be stressed that in our study, both groups of patients (anti-HCV negative and anti-HCV positive) were closely matched by the degree of liver fibrosis and several indexes of hepatic insulin extraction. In our view, this is a crucial point that should be considered when comparing patients with liver disease. The HOMA model is based on circulating insulin levels, and, therefore, the higher the degree of hepatic fibrosis the lower the rate of insulin extraction and, in consequence, the higher HOMA. Therefore, the measurement of hepatic insulin extraction should be provided before suggesting any influence of the HCV genotype in insulin resistance measured by HOMA. Finally, it should be noted that apart from TNF-α soluble receptors, we had also determined interleukin-6, and it was significantly higher in anti-HCV-positive than in anti-HCV-negative patients. Therefore, we agree with Petit et al. (1) that other factors apart from TNF-α could be implicated in the insulin resistance associated with HCV infection. In this regard, further studies to evaluate the contribution of other proinflammatory cytokines in the development of insulin resistance associated with HCV infection are warranted.

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DOI: 10.2337/dc06-1098

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The Biogun: A Novel Way of Eradicating Methicillin-Resistant Staphylococcus Aureus Colonization in Diabetic Foot Ulcers

Response to Dang et al.

I applaud the novel approach used by Dang et al. (1) to eradicate methicillin-resistant *Staphylococcus aureus* (MRSA) in patients with a diabetic foot ulcer. These Manchester Royal Infirmary clinicians have been leaders in documenting the problem of MRSA in diabetic foot lesions. As the incidence of colonization and subsequent infection with this virulent pathogen is rising in both the hospital and community, we need approaches to eradication that will not further drive antibiotic resistance (2).

There are several issues, however, that the briefly recorded observation left unclear. First, what was the target of the Biogun? I presume it was directed at a foot ulcer, but the authors only state “. . . patients without clinically infected foot ulcers but with MRSA colonization were treated.” So, was the treatment directed at a clinically uninfected foot ulcer in each case? Was the MRSA colonization of the foot ulcer? Second, MRSA colonization of the skin is most often associated with, and follows, MRSA anterior nares colonization (3). Did the authors check if the enrolled patients had nasal colonization? Did any of the patients receive topical therapy (e.g., mupirocin ointment) to their nares? Finally, antimicrobial therapy can eradicate *S. aureus* colonization (4); did any of the patients receive topical (to the wound) or systemic antibiotic therapy during the time they were treated with the Biogun? If so, did this correlate with MRSA eradication?

The nonantimicrobial method of eradication described by Dang et al. (1), with its promising preliminary results, de-

serves further evaluation. I look forward to the results of the properly designed randomized trial of this new technology that the authors rightfully suggest.

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DOI: 10.2337/dc06-1086

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The Biogun: A Novel Way of Eradicating Methicillin-Resistant Staphylococcus Aureus Colonization in Diabetic Foot Ulcers

Response to Lipsky

We are grateful to Dr. Lipsky (1) for his useful comments and entirely agree that there is a need to develop new ways to eradicate methicillin-

