Diabetologia (2009) 52:373
DOI 10.1007/s00125-008-1222-6

LETTER

Congenital rubella: citation virus or viral cause of type 1 diabetes? Reply to Honeyman MC, Harrison LC [letter] and Burgess MA, Forrest JM [letter]

E. A. M. Gale

Received: 14 October 2008 / Accepted: 14 October 2008 / Published online: 26 November 2008

I thank these correspondents [1, 2] for their interest in my Editorial [3]. Drs Burgess (née Menser) and Forrest [1] chide me gently for missing two references, and I was certainly unaware of the book chapter cited [4]. The other reference [5] relates mainly to an experimental model of teratogenicity in animals, but does indeed contain a table listing eight Australian patients with congenital rubella who developed diabetes between the ages of 18 months and 33 years, all of whom required insulin. Unfortunately, the clinical information provided is minimal, and I was also concerned about the possibility of double-counting individuals reported in previous series. Drs Honeyman and Harrison [2] point out that I did not cite the HLA data reported in a letter to the Lancet in 1974 supporting the case for an HLA association linking congenital rubella to type 1 diabetes [6]. This letter describes HLA serotyping in 87 people with the rubella syndrome, eight of whom had a clinical diagnosis of diabetes, and four of whom were on insulin, plus ten who were said to have 'latent' diabetes on glucose tolerance testing. Nine of the 19, including three of those on insulin, had HLA A8. I acknowledge these findings, but remain unconvinced that they add much to the evidence provided in the New York series.

My Editorial set out to make two main points. The first is the scanty and circumstantial nature of the evidence, and the second is the observation that, while congenital rubella undoubtedly predisposes to diabetes, the clinical picture is very heterogeneous. The spectrum includes both insulin-deficient and insulin-resistant forms of diabetes, presenting across a wide age range. Both sets of correspondents accuse me of accepting the evidence for type 2 diabetes and rejecting it for type 1, but neither statement will be found in what I wrote. Contrariwise, a number of my own conclusions are incorporated in the critique of what I am believed to have written. My argument, in brief, was that diabetes secondary to the congenital rubella syndrome should be considered sui generis, and tells us little or nothing about the pathogenesis of idiopathic type 1 or type 2 diabetes. The International Society for Pediatric and Adolescent Diabetes (ISPAD) was wise to assign congenital rubella-associated diabetes to a category of its own, and there it should remain.

E. A. M. Gale (✉)
Diabetes and Metabolism, Medical School Unit,
Southmead Hospital,
Bristol BS10 5NB, UK
e-mail: Edwin.Gale@bristol.ac.uk

Duality of interest The author declares that there is no duality of interest associated with this manuscript.

References

1. Burgess MA, Forrest JM (2009) Congenital rubella and diabetes mellitus. Diabetologia. doi:10.1007/s00125-008-1227-1
2. Honeyman MC, Harrison LC (2009) Congenital rubella, diabetes and HLA. Diabetologia. doi:10.1007/s00125-008-1213-7
3. Gale EAM (2008) Congenital rubella: citation virus or viral cause of type 1 diabetes? Diabetologia 51:1559–1566
4. Menser MA, Forrest JM, Bransby RD, Hudson JR (1982) Long term observations of diabetes and the congenital rubella syndrome in Australia. In: Mimura G, Baba S, Goto Y, Köbberling J (eds) Clinicopathogenetic genesis of diabetes mellitus. Excerpta Medica, Amsterdam, pp 221–225
5. Menser MA, Forrest JM, Bransby RD (1978) Rubella infection and diabetes mellitus. Lancet 1:57–60
6. Menser MA, Forrest JM, Honeyman MC, Burgess JA (1974) Letter: Diabetes, HL-A antigens and diabetes mellitus. Lancet 2:864–866