

PostScript

CORRESPONDENCE

Potential viral pathogenic mechanism for new variant inflammatory bowel disease

I write in relation to our paper that was published in the April 2002 issue of *Molecular Pathology*.¹ When generating data for this manuscript, we tested samples and controls provided by our collaborators at the Royal Free Hospital, London.

Before the paper was published (January 2002), we became concerned that four samples included in the "control category" might not have been correctly categorised. We immediately sought to clarify the situation. While our investigation was ongoing, we decided to test four additional controls, which matched the diagnostic classification of the control samples under investigation. They tested negative, so the overall data remained unchanged.

We later established that we were correct in our suspicion that the original four controls had been inaccurately grouped. For the avoidance of doubt, those four cases tested negative for measles virus. They were as follows:

- Three "normal" biopsies that should have been classified as "autistic enterocolitis".
- One "Crohn's" biopsy that should have been classified as "normal".

These samples did not form part of the testing cohorts described in the paper, having been substituted by controls whose diagnosis matched that described in the text.

To avoid confusion, if these samples had been included in our paper, the results of table 2 would have read as follows.

For comparison, the original table 2 is attached below. We apologise for any confusion caused but are anxious to document precisely the data that were presented to us and the results that we obtained.

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Reference

- 1 Uhlmann V, Martin CM, Sheils O, et al. Potential viral pathogenic mechanism for new variant inflammatory bowel disease. *Mol Pathol* 2002;55:84-90

Does leptin resistance contribute to infections in patients with diabetes?

Leptin is a protein hormone structurally similar to interleukin 2, which regulates food intake and metabolic and endocrine function.¹ It also has an important regulatory role in the immune response, mainly acting as a T cell growth factor.² Moreover, it also effects cytokine production, monocyte/macrophage activation, wound healing, angiogenesis, and haemopoiesis.¹ Leptin concentrations rapidly increase during infection and inflammation. The low concentrations of leptin that can occur during starvation or malnutrition are associated with impaired cellular immunity, impaired delayed-type hypersensitivity responses, and thymic atrophy, and thereby contribute to increased susceptibility to infection.²

High leptin concentrations are seen in obese and diabetic individuals, with the concentrations of leptin rising with increasing insulin resistance.³ However, despite high leptin concentrations, these individuals have leptin resistance or defective leptin.⁴ Therefore, leptin resistance, along with many other factors, may also contribute to the increased incidence of infection seen among patients with diabetes, who are more susceptible to infections such as tuberculosis and candidiasis, which require a good cellular immune response to overcome infection. Because leptin is important in the regulation of cellular immune responses, leptin resistance may be a contributing factor for the high incidence of such infections. Therefore, the role of leptin in infections in patients with diabetes should be further examined.

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- 2 Matarese G. Leptin and the immune system: how nutritional status influences the immune response. *Eur Cytokine Netw* 2000;11:7-14.
- 3 Fischer S, Hanefeld M, Haffner SM, et al. Insulin-resistant patients with type 2 diabetes mellitus have higher serum leptin levels independently of body fat mass. *Acta Diabetol* 2002;39:105-10.
- 4 Lee JH, Reed DR, Price RA. Leptin resistance is associated with extreme obesity and aggregates in families. *Int J Obes Relat Metab Disord* 2001;25:1471-3.

Table 1 Summary of TaqMan RT-PCR and RT in situ PCR results (revised)

	TaqMon RT-PCR positive	In situ PCR positive
Affected patients	70 (94)	42 (57)
Controls		
Normal controls	0 (20)	1 (5)
Mild non-specific change	0 (13)	NT
Lymphonodular hyperplasia	0 (3)	NT
Appendicectomies	4 (26)	NT
Ulcerative colitis	0 (1)	NT
Crohn's disease	0 (8)	NT

Total number of patients tested in parentheses.
NT, not tested; RT-PCR, reverse transcription polymerase chain reaction.

Table 2 Summary of TaqMan RT-PCR and RT in situ PCR results (original)

	TaqMon RT-PCR positive	In situ PCR positive
Affected patients	70 (91)	42 (57)
Controls		
Normal controls	0 (19)	1 (5)
Mild non-specific change	0 (13)	NT
Lymphonodular hyperplasia	0 (3)	NT
Appendicectomies	4 (26)	NT
Ulcerative colitis	0 (1)	NT
Crohn's disease	0 (8)	NT

Total number of patients tested in parentheses.
NT, not tested; RT-PCR, reverse transcription polymerase chain reaction.

CORRECTIONS

van den Brink GR, Hardwick JCH, Nielsen C, et al. Sonic hedgehog expression correlates with fundic gland differentiation in the adult gastrointestinal tract. *J Clin Pathol: Mol Pathol* 2003;56:150-5. This article was originally published in *Gut* 2002;51:628-33. Any citations of this article should refer to the original article in *Gut*.

Ryan BM, Lefort F, McManus R, et al. A prospective study of circulating mutant KRAS2 in the serum of patients with colorectal neoplasia: strong prognostic indicator in postoperative follow up. *J Clin Pathol: Mol Pathol* 2003;56:172-9. This article was originally published in *Gut* 2003;52:101-8. Any citations of this article should refer to the original article in *Gut*.



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