The diagnostic specificity of labial biopsies can be increased by almost 10% when using a cumulative focus score [2]. Multilevel evaluation of a labial gland biopsy may indeed improve the reliability of histopathological grading in SS, which particularly might be of additional value in potential SS cases with a focus score at the cut-off level. Although the amount of tissue available in a parotid biopsy is usually not a problem for SS diagnostics, it occasionally may be in labial biopsies from patients with advanced SS; it might be very interesting to evaluate whether multilevel evaluation will also increase the specificity and/or sensitivity of parotid gland biopsies.

The second point that Morbini and co-workers mentioned in their letter is the low incidence of subjective morbidity after a labial biopsy in their cohort of patients. They recorded adverse events with the aid of a questionnaire [2]. This subjective assessment might underestimate the amount of local hypoesthesia after labial biopsy. That was the reason we performed a thorough neurological examination (two-point discrimination test) in order to evaluate the objective morbidity of a labial or parotid biopsy in addition to a subjective evaluation using a questionnaire [1]. In agreement with the study of Morbini and co-workers [2] the evaluation of the adverse effects was done by independent clinicians. Combining a subjective assessment with an objective assessment is essential as it is not unusual that patients do not complain about local paraesthesia, even when objective assessment indicates some degree of disturbed sensibility. Although this discrepancy between objective and subjective results might not be clinically relevant, such information might be crucial for comparative studies. Moreover, we agree that in skilled hands both a labial and a parotid biopsy will result in minimal adverse effects, but we rather often encounter in our daily practice patients with a permanent disturbed sensibility of the lower lip due to a diagnostic labial biopsy taken by less skilled clinicians.

At the end of their letter Morbini and co-workers mentioned that there is a need for large comparative studies in order to find out the best diagnostic tools for histopathologic evaluation of SS. Morbini and co-workers have a preference for labial biopsies above parotid biopsies because of the specific surgical experience needed for parotid gland biopsies, while labial salivary gland biopsies may be performed directly by clinicians such as rheumatologists. Although taking a parotid gland biopsy as used by Pijpe et al. [1] is a rather simple out-patient technique for e.g. an oral and maxillofacial surgeon, it is indeed not a procedure that is easy to perform in, for example a department of Rheumatology. However, there are some inherent advantages of a parotid biopsy over a labial biopsy in SS patients that are not mentioned by Morbini and co-workers. A parotid biopsy might be preferred for therapy evaluation as repeated biopsies can be taken from the same parotid gland (in combination with saliva samples from the same gland). As such a parotid gland can be used more easily to perform in, for example a department of Rheumatology.

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J. Pijpe, F. K. L. Spikervet, J. E. van der Wal, C. G. M. Kallenber, A. Visser

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Correspondence to: J. Pijpe. E-mail: j.pijpe@kchir.umcg.nl


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Anti-TNF alpha therapy, lipid profile and carotid intimal thickness

Sir, Del Porto et al. [1] report significant reduction in carotid intimal thickness (cIMT) in patients with rheumatoid arthritis (RA) suitable for anti-TNF therapy and who experienced a clinical response to the drug, compared with similar suitable patients who chose to remain on methotrexate and prednisolone. This further underlines the relationship between inflammation and atherosclerosis in general, and systemic inflammation in RA and atherosclerosis in particular.

Several possible mechanisms for this observed effect are discussed, including anti-TNF-mediated down-regulation of T lymphocytes, and effects on lipoprotein metabolism, nitrous oxide production and the coagulation cascade. The effect of alteration of the lipid profile is not directly discussed, and although the lipid profile of the two groups before treatment is tabulated, no results are given for lipids after therapy. Anti-TNF therapy has been shown to favourably alter lipid profiles over time [2], and lipid-lowering drugs, including statins, have been demonstrated to have anti-inflammatory effects [3]. There is currently debate over whether lowering lipids per se might have a positive anti-inflammatory effect, aside from other anti-inflammatory mechanisms of statins; under these circumstances, it would be interesting to see data on the lipid profile of these groups after therapy, and if there was any relationship between the final lipid levels and cIMT changes.

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D. J. ARMSTRONG

Department of Rheumatology, University Hospital of North Durham, UK

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Correspondence to: Dr D. J. Armstrong, MD, MRCP, FRCP, Consultant Rheumatologist, Department of Rheumatology, University Hospital of North Durham, North Road, Durham, DH1 5TW, UK. E-mail: owald17727@hotmail.com


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Effects of tumour necrosis factor alpha blockade on lipid profile in active rheumatoid arthritis

Sir, We would like to thank Dr. Armstrong for his helpful comment regarding lipid profile behaviour during anti-tumour
necrosis factor (TNF-\(\alpha\)) treatment. Such a topic is of increasing interest in the literature, despite lack of long-term definitive supporting evidence [1–4]. Actually, in patients with rheumatoid arthritis (RA) under anti-TNF-\(\alpha\) treatment, early and transient high-density lipoprotein (HDL) level increase [1, 2], but even late atherogenic index (total/HDL cholesterol ratio) worsening has been reported [3].

In our study, significant reduction of common carotid artery intima-media thickness (cIMT) values has been observed in patients with active RA steadily responsive to anti-TNF-\(\alpha\) agents [5]. Despite the fact that data regarding lipid profile have not been shown, total-, HDL-, low-density lipoprotein (LDL)- cholesterol and triglyceride levels were actually measured before, 3, 6 and 12 months after therapy, we did not observe any significant change of lipoprotein pattern both in patients treated with disease modifying anti-rheumatic drugs (DMARDs) plus TNF-\(\alpha\) blockers (group A) and in those treated with only DMARDs (group B) at any time (Table 1). Moreover, no significant differences have been observed in the above-reported parameters between the two groups at any time (Table 1), nor any significant correlation between lipid profile and cIMT (Spearman’s correlation test). In agreement with the literature data [3], we suggest that lipid level modification observed in the above-reported parameters between the two groups at any time (Table 1) is of increasing interest in the literature, despite lack of long-term definitive supporting evidence [1–4]. Actually, in patients with rheumatoid arthritis (RA) under anti-TNF-\(\alpha\) treatment, early and transient high-density lipoprotein (HDL) level increase [1, 2], but even late atherogenic index (total/HDL cholesterol ratio) worsening has been reported [3].

In our study, only five patients (three in Group A and two in Group B) were also treated with statins (simvastatin 20 mg/daily); all of them had started such therapy at least one year before the enrolment. Despite the fact that statin anti-inflammatory effects have been largely demonstrated in atherosclerosis (ATS) [6] and suggested also in RA patients treated with only DMARDs [7], we did not find any significant difference, at any time, in inflammatory marker levels or lipid plasma concentrations between statin-treated and not-treated patients. Our results suggest that in high-grade systemic inflammatory diseases, such as active RA responsive to anti-TNF-\(\alpha\) agents, statin anti-inflammatory effect may hardly be considered playing a crucial role.

In conclusion, although the Armstrong’s remark appears very intriguing, plasma lipid profile do not seem to be significantly influenced by anti-TNF-\(\alpha\) agents; the described statin anti-inflammatory effect also seems to be unapparent in this particular context, as overcome by the potent anti-inflammatory effect of TNF-\(\alpha\) inhibitory treatment.

All the patients gave their written informed consent before being included in the study, which was performed according to the principles reported in the Declaration of Helsinki. This study has been ethically approved by the Faculty of Medicine Committee.

The authors have declared no conflicts of interest.

F. Del Porto, B. Laganà, I. Nofroni, F. Tinti, A. P. Mitterhofer, R. D’Amelio

“Sapienza” Università di Roma, II Facoltà di Medicina e Chirurgia, Azienda Ospedaliera S.Andrea, Dipartimento di Scienze Mediche; I Facoltà di Medicina e Chirurgia, Dipartimento di Medicina Sperimentale e 2Dipartimento di Medicina Clinica

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Correspondence to: Flavia Del Porto, MD, Università ‘La Sapienza’, II Facoltà di Medicina e Chirurgia, Azienda Ospedaliera Sant’Andrea, Dipartimento di Scienze Mediche, Via di Grottarossa 1039, 00189 Roma, Italia. E-mail: flavia.delporto@uniroma1.it

*The authors wish it to be known that, in their opinion, the last two authors contributed equally to this work.


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Time to abandon the rheumatoid factor? Is it not time to rename it?

Sir, We read Professor Symmons editorial [1] with interest. We agree with her conclusions and recommendations and would