Welcome to the Scottish Exhibition and Conference Centre

As President of the British Transplantation Society I am delighted to welcome you to Glasgow for the 15th Annual Congress of the BTS. Glasgow may not have the sand and sea of Bournemouth, but I anticipate the weather to be equally bracing. I hope you find the programme this year as stimulating and educational as last year.

We are very grateful to the members of the Programme Committee for putting together an interesting and imaginative programme, drawing together national and international experts as well as providing a forum for presenting new research from within the UK. We hope that there will be something for everyone throughout the congress. We would like to thank BASL for hosting a joint symposium, and also our colleagues from BSHI whose work we have integrated throughout the congress, a reflection of how integral it has become to the practice of transplantation.

We have endeavoured to build on the success of recent meetings. To that end we have continued the early morning workshops that have been so popular in recent years. We have also copied the format of recent years and combined the welcome reception on Wednesday evening with the moderated poster session. This year's Hoffenberg lecture will be given by Professor Margot Brazier, Professor of Law at Manchester, and former chair of the Retained Organs Commission. On Thursday evening the dinner will be followed by a chance to let your hair down at a traditional Celidh.

We would also like to thank our corporate partners and other industry stakeholders whose support will help make our meeting possible. In particular we would like to thank our two gold corporate partners, Bristol Myers Squibb and Astellas, who will also be hosting sponsored symposia on Wednesday and Thursday lunchtimes. Finally we would like to thank the SECC for hosting the meeting and our secretariat, KSAM, for all their work behind the scenes to make the congress a success.

The Annual Congress is always an excellent opportunity to meet with colleagues within an environment that is both educational and enjoyable – this year should be no different.

We hope you have an excellent congress.

With best wishes

British Transplantation Society Conference, Glasgow, abstract No. 2

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The role of anti-HLA class I antibodies in chemokines mediated leukocytes migration post transplantation

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Background: The development of donor specific HLA class I antibody after organ transplantation has been associated with acute rejection and is a risk factor for chronic rejection. The infiltration of circulating leukocytes into transplanted allograft is associated with antibody mediated rejection. However, the potential of anti-HLA class I antibodies in mediating this process is not fully understood. The aim of this study was to examine the role of anti-HLA class I antibody in modulating endothelium-leukocyte interaction focusing on endothelial transduction pathways, adhesion molecule up-regulation and inflammatory cytokines-chemokines expression.

Methods: Human microvascular endothelial cells (HMEC-1) were typed for the classical HLA class I molecules by using PCR-SSP method. Cells stimulated with anti-HLA class I antibody (W6/32, 12µg/ml) or allospecific antibodies from sensitized patients (n=6) were examined for the upregulation of endothelial adhesion molecules (VCAM-1, ICAM-1) by flow cytometry and induction of cytokines and chemokines by q-PCR. Using *in vitro* flow based adhesion assay (Cellix platform), the potential of HLA class I antibodies in enhancing leukocytes adhesion was assessed.

Results: HMEC-1 cells treated with W6/32 antibodies resulted in the activation of various cell signalling kinases, including transcription factor; CREB in PKA dependent pathway as verified by using PKA inhibitor (H89). Moreover, treatment of cells with W6/32 antibodies significantly induced the expression of cell surface VCAM-1 and ICAM-1 which peaked at 12 and 8 hours respectively, compared to isotype treated group ($p<0.01$ & 0.001). Allospecific antibodies from sensitized patients (A1, A28, B35, B58, CW4, CW6) also induced significant expression of these adhesion molecules after 24 hours treatment. In addition, exposure to W6/32 antibodies upregulated the expression of endothelial cytokines such as IL-6 and various chemokines; CXCL8, CXCL1, CXCL10 and CCL5. The expression of CXCL8 appeared to be dependent on antibody concentration. Allospecific antibodies induced significant expression of CXCL8 (2-11 fold, $p<0.001$). Chemotaxis assay demonstrated that the conditioned media from W6/32 treated endothelial cells stimulated a significant monocyte migration ($p=0.011$) compared to isotype treated group. Under flow based adhesion condition, endothelial cells treated with W6/32 antibodies significantly increased the adhesion of monocytes compared to isotype treated group at 0.5 dyne/cm^2 within 5 minutes ($p<0.0001$).

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ABSTRACTS

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HLA CLASS I ANTIBODIES ACTIVATE ENDOTHELIAL CELLS AND UP-REGULATE ADHESION MOLECULES; A POTENTIAL LINK BETWEEN ANTIBODIES AND CELLULAR REJECTION

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The development of donor specific HLA class I antibody after organ transplantation is associated with acute and chronic rejection. However, the potential of anti-HLA class I antibodies in mediating leukocyte migration is not fully understood. The aim of this study was to examine the role of anti-HLA class I antibody in modulating endothelium-leukocyte interaction, adhesion molecule up-regulation and chemokine expression. Human microvascular endothelial cells (HMEC-1) were genotyped for HLA class I molecules. Cells stimulated with anti-HLA class I antibody (W6/32) or allospecific antibodies from sensitized patients ($n = 6$) were examined for the upregulation of adhesion molecules by flow cytometry and induction of chemokines by q-PCR. Using in vitro flow based adhesion assay the potential of HLA class I antibodies in enhancing leukocytes adhesion was assessed. HMEC-1 cells treated with W6/32 antibodies resulted in the activation of various cell signalling kinases, including transcription factor; CREB in PKA dependent pathway as verified by using PKA inhibitor (H89). Treatment of cells with W6/32 antibodies significantly induced the expression of cell surface VCAM-1 and ICAM-1, compared to isotype treated group ($p < 0.001$ and 0.001). Allospecific antibodies from sensitized