we have analyzed patients from one month to more than two years after cardiac transplantation (n = 15). For detection of donor specific tissue components donor and recipient DRB1-alleles were detected by PCR following extraction of DNA from skin biopsies, blood samples, and transplant biopsies. With this approach we could demonstrate chimerism in 9 of the 15 patients. It was shown in 3 of 4 patients studied during the first year after transplantation, in 5 of 7 patients examined during the second year, and in one of 3 patients more than two years after transplantation. Results for skin and blood were always concordant. From the present data it can be concluded that transmission of donor cells into the recipient tissues does occur frequently and that development of allogenic microchimerism after organ transplantation is obviously not restricted to liver transplantation. However, the relevance of development of microchimerism for longterm graft survival is not clear because in 6 patients donor specific tissue components were neither detected in skin nor blood. So far, it is not possible to differentiate whether chimerism is really absent in some patients with a good clinical result or whether it was not detectable because of insufficient sensitivity of our detection system. It is also possible that in some patients transferred donor cells are distributed only focally and escape detection for this reason.

Institute of Immunology, University of Kiel, Kiel, Germany; Dept. of Medical Cell Biology, University of Uppsala, Sweden

M.13 Expression of porcine pancreatic islet antigens varies and determines binding of human natural xenophile antibodies (NXA)

K. Ulrichs, V. Eckstein, O. Korsgren, and W. Müller-Ruchholtz

Natural xenophile antibodies, causing hyperacute and acute graft rejection in immunogenetically discordant species combinations are considered a main cause for the failure of xenografts, including clinical xenografts of porcine fetal pancreatic islet cells (Groth et al. 1993, Transplant. Proc. **25:** 970). An important question is whether fetal islet cells express NXA target epitopes with similar strength as compared to adult tissue. To answer this question, NXA binding to porcine islet cells in various stages of ontogeny was tested in indirect immunofluorescence, using human sera of type-I diabetics (n = 5) with «strong» anti-islet cell NXA reactivity on adult tissue (titer ≥ 1:32) as well as sera from healthy controls (n = 5). Target tissue derived from Swedish and German landrace pigs. **Results:** (1) In contrast to other reports, NXA (IgG) were found to react positively with fetal islet tissue (70 and 115 days gestation) and with newborn tissue. However, binding on these two tissues is «weak» (titer ≤ 1:4) as compared to «strong» (titer ≥ 1:32) binding on adult cells. (2) NXA binding increases from «weak» to «strong» if fetal tissue is cultured *in vitro*. This «strong» binding persists when the cultured tissue is transplanted under the kidney capsule of a nude mouse. (3) «Weak» and «strong» NXA binding correlates with «weak» and «strong» insulin secretion of islet cells, and thus appears to correlate with islet endocrine function. Conclusions: (I) According to the above data, the relevant target for pretransplant NXA screening in xenogeneic islet transplantation appears not to be the native but the functionally activated pancreatic tissue. (II) The probably function-dependent variability of NXA epitope cell surface expression should be taken into consideration for NXA manipulations prior, during and after islet grafting.