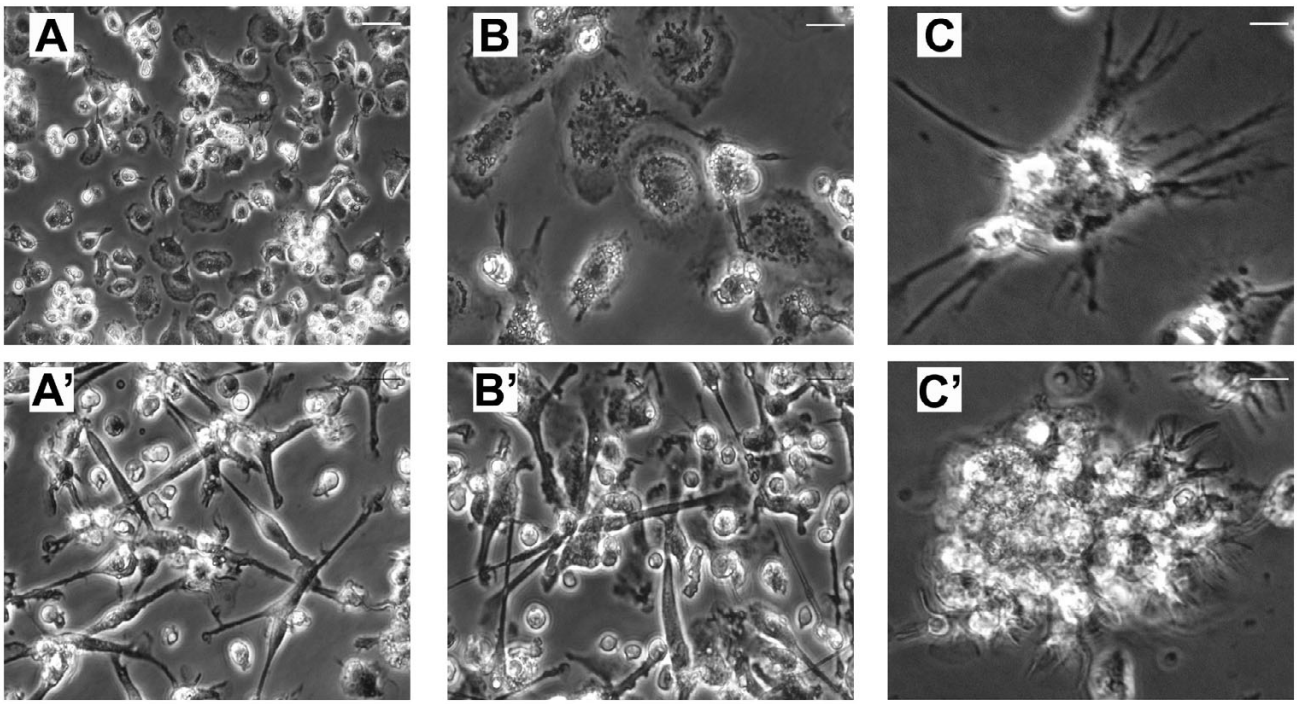

Figure 1
Equine monocyte-derived macrophages (A) and dendritic cells (B) generated *ex vivo*. Isolated peripheral blood monocytes were stimulated (dendritic cells) or not (macrophages) with rEq IL-4 and rHuGM-CSF in DMEM-F12, 5% bovine growth serum. The photomicrogaphs depict the differentiation of adult horse and foal macrophages and dendritic cells in culture. A and B = day 5 adult horse and foal macrophages, respectively; A' and B' = day 5 adult horse and foal dendritic cells, respectively – note their extended shape in contrast to the round macrophages; C = day 6 dendritic cells adhered to the plastic of the cell culture plate; C' = a group of day 6 dendritic cells floating in the supernatant of the cell culture – note the presence of small dendrites. Bars indicate 50 μm.

p65) were comparable ($p < 0.05$) in adult horse and foal macrophages and DCs, independent of treatment.

Discussion

Age-dependent aspects of APCs in the horse

Limitations in the immune system of the foal could be associated with age-dependent development of cell interaction for a primary immune response. The low expression of MHC class II in equine neonate and young foal peripheral blood lymphocytes has been well documented, but the expression of this essential molecule in APCs had not been studied before in the foal [34,35]. Our investigation revealed 2 important observations: a) there was a statistically significant difference in the fluorescence expression of MHC class II in macrophages and DCs of foals with age; and b) median MHC class II fluorescence expression in non-stimulated macrophages and DCs of the foal at birth were 12.5 times and 11.2 times inferior, respectively, to adult horse cells. The median MHC class II fluorescence expression in non-stimulated DCs of 3 month-old-foals was comparable to adult horses, which suggests a greater competence for the priming of T cells at that age. In human fetuses, the percentage of MHC class II-positive monocytes increases significantly over gestation but remains lower than the adult human at term [36]. Limitation in APC number and function in young age has been shown to contribute to poor protective cellular immune responses [37-39]. Human cord blood DCs are less efficient in the activation of T cells *in vitro* and instruction to a Type 1 immune response, likely due to their lower cell surface MHC class I and II, co-stimulatory (CD86), and adhesion molecule expression levels than adult human blood cells [40].

Likewise, the expression of cytokines and co-stimulatory molecules (signal II) in APCs had not been studied before in foals. These important immune mediators are critical for the priming and clone expansion of naïve T cells. There were no statistically significant differences in the expression of CD86 in foal macrophages and DCs. In addition, there were no age-dependent changes in the expression of CD86. Importantly, those values were comparable to the