Preeclampsia is associated with impaired trophoblast invasion within an ischaemic microenvironment [1]. Although placental development depends on careful coordination of trophoblast proliferation and apoptosis, little is known about the equilibrium that exists between these two key processes that synchronize trophoblast invasion. The aim of this study was to determine whether an imbalance exists between trophoblast apoptosis and proliferation in the placental bed of preeclamptic compared to normal pregnancies. This retrospective study was conducted on true placental bed biopsies obtained from 12 normotensive and 12 hypertensive pregnant women at the Obstetric Unit of King Edward VIII Hospital, Durban, South Africa. Serial sections were immunolabelled with a rabbit anti-human Ki67 antibody, mouse anti-human Cytokeratin 18 and its neo-epitope, a monoclonal M30, Cytodeath antibody. Results demonstrated that the immunoexpression of anti-Ki67 for all trophoblast cell sub populations within the myometrium were non-reactive for both study groups. However, smooth muscle cells of the small caliber microvasculature signify a moderate degree of proliferation in both groups. Additionally, immuno-expression of anti-CK18 indicated intramural trophoblast invasion in the spiral artery of normotensive group was elevated compared to the preeclamptic group (13 vs 0% respectively). Comparative analyses of M30 distribution on corresponding serial sections were 0.06 vs 0% in the normotensive and pre-eclamptic groups respectively. The mean field area of the interstitial trophoblast invasion in the preeclamptic group was 2.87% compared to 10.79% in the normotensive group. However, the serial sections stained with M30 showed elevation of apoptotic invasive interstitial trophoblast in the preeclamptic group compared to the normotensive group (38 vs 17 %). In conclusion, the expression pattern of Ki67 antigen in this study suggests differentiation of invasive trophoblasts at term is coordinated with exit from the cell cycle [2]. In preeclampsia, the invasive trophoblast cells probably exit the cell cycle in G1 phase, directing them towards apoptosis rather than passage in the S phase and mitosis. The balance between trophoblast apoptosis and proliferation, in favour of increased apoptosis may represent an aetiological mechanism in the pathogenesis of preeclampsia.

REFERENCES