

# **COLLOQUIA IN PHYSIOLOGY AND VASCULAR BIOLOGY**

Venue: Medical University Vienna, Center for Physiology and Pharmacology,  
Institute of Pharmacology, Waehringerstrasse 13a, 1090 Vienna, "**Leseraum**"  
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Friday 23.5.2014 10:00 s.t. **Susan Wray** (host: Chr. Gruber)  
Professor Susan Wray, FMedSci, FRCOG, MAE  
Department of Molecular and Cellular Physiology  
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## ***"The uterus – from science to translation"***

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**Susan Wray** ([s.wray@liv.ac.uk](mailto:s.wray@liv.ac.uk))

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Many of the problems associated with labour can be attributed to the muscle in the uterus, the myometrium. This excitable tissue has to have its excitability repressed for most of pregnancy. This is done largely by keeping the K conductance high and the connectivity between myocytes and muscle bundles low. If the brakes on excitability are released too early, then uterine contractility is triggered, the cervix starts to be effaced and preterm delivery is threatened. Impaired fetal oxygenation will occur in term labour if the contractions are too strong. Difficult, exhausting labours or even emergency Caesarean section (CS) can occur if uterine contractility is inadequate. It is reasonable therefore to argue that a sound understanding of excitation, contraction and their coupling (EC coupling) in the myometrium, is key to progressing knowledge and developing strategies to better treating the problem labours mentioned above. The importance of EC coupling can be readily appreciated from the experimental and clinical effects of using nifedipine to block L-type Ca channels; uterine contractility rapidly ceases. Much less progress has been made with understanding how to stimulate labours suffering from poor contractility, and hence the excitement around development of new oxytocics – although as I will discuss, a completely different therapeutic strategy may be needed.