BASL 2017
British Association for the Study of the Liver
Incl BLTG Transplant Meeting and BASL Nurse Forum Meeting

SPEAKER INFORMATION

20th-22nd September 2017

Warwick Arts Centre, Coventry, UK

Dr Diana Jurk

Dr Diana Jurk is originally from Germany where she graduated with a degree in Natural Sciences from the University of Freiberg in 2004. Following her degree, she conducted biomedical research at Bayer (one of the largest's German multinational pharmaceutical companies), first in Wuppertal and then in Leverkusen (where she was awarded a Master's degree).

Having decided to move to academia, Diana was first a research assistant at the Uniklinik in Freiburg, Germany (2005-2007) and then conducted her PhD studies at Newcastle University and the Max Planck Institute for Stem Cell Ageing in Ulm, Germany (2007-2012), in the area of liver senescence and inflammation. Following her PhD, Diana did a post-doc in the lab of Prof. Thomas von Zglinicki at Newcastle University on the subject of senescence and neurodegeneration.

In 2015, Diana was awarded the prestigious Newcastle University Faculty fellowship, which allows young researchers the opportunity and funds to set up their own lab. She now leads a small team, investigating the mechanisms contributing to neuronal senescence and its impact on age-related cognitive decline.

Diana's work has led to new insights into the mechanisms driving the process of cellular senescence. She demonstrated a novel role for telomeres in stress-induced senescence (*Nature Communications* 2012) and discovered that low grade chronic inflammation can accelerate telomere dysfunction, thereby contributing to organismal ageing (*Nature Communications* 2014).

Since establishing her group in 2015, she has produced her first paper as senior and corresponding author (*Nature Communications* 2017). This work, which involves close collaboration with the Mayo Clinic (Rochester), demonstrates that cellular senescence is a driver of hepatic steatosis.

Selected publications:

- Ogrodnik M, Miwa S, Tchkonia T, Tiniakos D, Wilson C, Lahat A, Cay CP, Burt A, Palmer A, Anstee QM, Grellscheid SN, Hoeijmakers JHJ, Baarnhoom S, Mann DA,

Bird TG,Vermeij WP, Kirkland JL, Passos JF, Von Zglinicki T, and **Jurk D** Cellular senescence drives age-dependent hepatic steatosis. ***Nature Communications*** (2017)

- **Jurk, D**, Fullard, N, Passos, JF, Oakley, F, Greaves, L, Correia-Melo, C, Fox, C, Lawless, C, Anderson, R, Pender, S.L.F, et al. Chronic inflammation induces telomere dysfunction and accelerates ageing in mice. ***Nature Communications*** (2014)
- Hewitt, G [1], **Jurk, D**[1], Marques, FDM[1], Correia-Melo, C, Hardy, T, Gackowska, A, Anderson, R, Taschuk, M, Mann, J, and Passos, JF: Telomeres are favoured targets of a persistent DNA damage response in ageing and stress-induced senescence. ***Nature Communication*** (2012) [1]equal contribution
- **Jurk, D**, Wang, C, Miwa, S, Maddick, M, Korolchuk, V, Tsolou, A, Gonos, ES, Thrasivoulou, C, Jill Saffrey, M, Cameron, K, von Zglinicki, T: Postmitotic neurons develop a p21-dependent senescence-like phenotype driven by a DNA damage response. ***Aging Cell*** (2012)