Oxidative DNA damage is elevated in renal patients undergoing haemodialysis

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End stage renal disease (ESRD) is associated with an increase in oxidative stress, cardiovascular disease and cancer. The main treatment for ESRD, haemodialysis (HD), itself induces repetitive bouts of oxidative stress through membrane biocompatibility and endotoxin challenge. The resulting higher levels of reactive oxygen species in turn produce increased levels of oxidative DNA damage leading to genomic instability. We measured levels of oxidative DNA damage insss thirty-eight patients receiving HD in the Western Health and Social Services Trust (WHSCT), and 8 age and gender matched control volunteers. Volunteers gave informed consent and non-fasting morning blood samples were taken and assessed for DNA damage using the Modified Comet to identify oxidative specific damage by introducing an enzymatic step with the bacterial enzymes endonuclease III (Endo III, recognise pyrimidine-pyrimidine breaks) and formamidepyrimidine DNA glycosilase (FPG, recognise purine-purine breaks. The study then continued into a 3-month intervention with a novel supplement to determine if levels of oxidative damage could be reduced with this novel supplement. The HD patients had significantly elevated levels of alkaline DNA damage (19.46 ± 8.35 vs 3.86 ± 0.99 % tail DNA, $p<0.05$) and oxidative DNA damage formamidepyrimidine DNA glycosilase (5.81 ± 6.63 vs 1.23 ± 0.39 % tail DNA, $p<0.0$) and endonuclease III (6.04 ± 6.11 vs 1.98 ± 0.85% tail DNA, $p<0.01$) compared to controls, respectively. A positive correlation was observed between the duration on dialysis (months) and levels of Endo III specific damage ($p=0.041$). Following the 3-month intervention we observed a significant reduction in Alkaline, EndoIII and FPG DNA damage in the HD treatment group, while the HD placebo group had DNA damage levels significantly increased from baseline at 3 months. We conclude, the significant increase in oxidative DNA damage and the positive correlation with duration of HD treatment and Endo III damage may contribute to the increased cancer risk observed in this patient group. In addition, treatment with a novel supplement significantly reduced DNA damage in the HD treatment group and could be recommend as a routine treatment in HD patients.

Speaker Biography

Mary Hannon-Fletcher is currently a senior lecturer in Biomedical Sciences at Ulster University and a member of the Biomedical Sciences Research Institute. Having re-joined the School after holding the position of Head of School of Health Sciences for 6 years, where she led a multi- professional team of Allied Health Professions (AHP's) and Healthcare Scientists. Professionally she holds a First Class (Hons) BSc in Biomedical Sciences (1995) and a PhD in Biomedical Sciences, 2000, from Ulster University. She is a Fellow of the Institute of Biomedical Sciences (FIBMS); Chartered Scientist (CSi); Registered Biomedical Scientist with the Health and Care Professions Council (HCPC) and Fellow of the Higher Education Academy (FHEA).

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