This work is made available online in accordance with publisher policies. Please refer to the repository record for this item and our Policy Document available from the repository home page for further information.

To see the final version of this work please visit the publisher’s website. Access to the published online version may require a subscription.

Link to publisher’s version: http://dx.doi.org/10.4172/2161-1165.S1.006

Citation: Anderson D, Najafzadeh M, Ali A et al (2014) DNA damage in lymphocytes from healthy individuals and respiratory disease patients, treated ex vivo/in vitro with aspirin and ibuprofen nanoparticles compared to their bulk forms. Epidemiology. 4(4): 69.

Copyright statement: © The Authors. This is an Open Access article published under the Creative Commons CC-BY license.
DNA damage in lymphocytes from healthy individuals and respiratory disease patients, treated ex vivo/in vitro with aspirin and ibuprofen nanoparticles compared to their bulk forms

Diana Anderson1, Mojgan Najafzadeh1, Aftab Ali1, Badie Jacobs2, Muhammad Isrib1, Rajendran Gopalan1 and Lijun Shang1

1University of Bradford, UK
2Bradford Royal Infirmary, UK

Non-steroidal anti-inflammatory drugs (NSAIDs) inhibit COX enzyme activity, a significant mechanism of action of NSAIDs. Inflammation is associated with increasing cancer incidence. Recent pre-clinical and clinical studies have shown that NSAID treatment could cause an anti-tumour effect in cancers. Such studies are lengthy and expensive. The present study, however, examined DNA damage in the Comet and micronucleus assays in peripheral blood lymphocytes of patients with respiratory diseases and healthy individuals using the nanoparticle (NP) and bulk versions of the NSAIDs, aspirin and ibuprofen. Lymphocytes are suitable surrogate cells for cancers and other disease states. DNA damage decreased in lymphocytes from healthy individuals, asthma, COPD and lung cancer patient groups after treatment with aspirin nano (ASP-N) and ibuprofen nano (IBU-N) compared to their bulk version in both assays. However, when ASP-N was compared to untreated lymphocytes in all groups in the Comet assay, DNA damage significantly decreased in all groups, except the asthma group. When IBU-N was compared to untreated lymphocytes, in healthy individuals and the lung cancer group, DNA damage decreased, but increased in asthma and COPD groups. Similarly, micronuclei (MNi) increased after ASP-N and IBU-N in the healthy individual and lung cancer groups, and decreased in asthma and COPD groups. Furthermore, lymphocytes responses after IBU-N and ibuprofen bulk were investigated by the physiological patch-clamp technique. Patch-clamp recordings demonstrated that IBU-N inhibited ion channel activity by 20%. This molecular epidemiology approach mirrors pre-clinical and clinical findings, and provides further information using nanoparticles.

Biography

Diana Anderson currently holds the Established Chair of the Division of Biomedical Sciences at the University of Bradford, UK. She obtained her first degree in the University of Wales and second degree in the Faculty of Medicine, University of Manchester. After tutoring at the University of Sydney, Australia, she became a research worker in the Department of Cancer Studies at the University of Leeds and at the Paterson Laboratories, Christie Hospital, Manchester. She has organized both National and International meetings and was/is a member of various national (e.g. MRC Advisory Board, Veterinary Products Committee) and was of International committees, including the European Union Scientific Committee for Animal Nutrition (SCAN). She recently won a prize as an Enterprise Fellow. She has hosted and participated in 56 meetings for WHO/IPCS. She is a consultant for many international organizations, such as the WHO, NATO, TWAS, UNIDO and the OECD.

D.Anderson1@bradford.ac.uk