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# Characterization of intestinal inflammation-induced systemic genotoxicity and its potential use as a biomarker of disease activity

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To determine susceptibility of subpopulations of cells in the peripheral blood as well as of peripheral lymphoid organs to several types of DNA damage in genetic mouse models of spontaneous chronic intestinal inflammation and to determine the sufficiency of tumor necrosis factor α (TNF-α) in inducing this genotoxicity. By determining correlations of DNA damage to disease activity, we hope to substantiate systemic genotoxicity as a biomarker of inflammatory activity in intestinal inflammation. Peripheral blood subpopulations were isolated via magnetic bead sorting and cells from peripheral lymphoid organs were isolated incolitic IL-10 mice of various disease activity (3 and 6 months of age), Gαi2 mice (3 months of age) and in wild-type mice with no intestinal inflammation. DNA strand breaks were measured in cells with the alkaline comet assay with hOGG1 incubation to determine oxidative base damage and DNA double strand breaks specifically were quantified by γH2AX foci immune-staining. Recombinant mouse TNF-α or saline was injected (500 ng/mouse) into the tail vein of wild-type mice and peripheral blood was analyzed for DNA damage at several time points post injection. DNA single and double strand breaks were found in subpopulations of cells in the peripheral blood as well as in the peripheral lymphoid organs, which correlated to disease activity of mice with intestinal inflammation. CD4 and CD8 T-cells seemed most sensitive to DNA damage. TNF-α was sufficient to induce DNA damage in wild-type mice. Chronic intestinal inflammation induces systemic DNA damage, in which CD4 and CD8 T-cells are the most sensitive. TNF-α plays a role in inducing this damage though further mechanisms remain investigated. Levels of DNA damage in the peripheral blood correlated strongly to inflammatory activity and severity of disease, making DNA damage to leukocytes a good biomarker to diagnose and monitor disease in inflammatory bowel disease patients.

## Biography

Robert H Schiestl has obtained his PhD from the University of Vienna. He was a Post-doctoral fellow at Edmonton, Alberta, Rochester, NY, and Chapel Hill, NC before working as a Professor at Harvard, where he stayed for 10 years. Since 15 years, he is working as a Professor at UCLA with 190 publications, 6 press releases, 10 patents and 2 startup companies.

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