Biomarkers of Cumulative Exposure


Human serum albumin (HSA) scavenges toxic electrophiles from the blood to produce a constellation of adducts. Since these HSA adducts arise from all reactive chemicals in the serum, they reflect the totality of systemic exposures to electrophiles, including xenobiotics and their metabolites, reactive oxygen and carbonyl species, hormones, etc. Thus, HSA adducts can be used to classify systemic exposures of potential importance to development of chronic diseases – not only from air, water, smoking, and food, but also from stress, obesity, etc.

We are aiming to prove the concept that HSA adducts can be used to quantify exogenous and endogenous exposures of interest using as examples polycyclic aromatic hydrocarbons (PAHs), which produce a host of reactive metabolites, and formaldehyde, which has both exogenous and endogenous sources. Methods have been optimized for characterizing HSA adducts and increasing throughput, and methods have been validated with archived specimens of blood from PAH-exposed workers, formaldehyde-exposed workers, and the general population. One goal is to detect differences in adduct levels between exposed and ‘unexposed’ subjects, and to relate adduct levels with measurements of other relevant endpoints (benzo[a]pyrene diol epoxide [BaPDE]-HSA adducts measured via ELISA and endogenous formyl and acetyl adducts of HSA).

Implications: This work should motivate a new generation of simple, biologically-based methods for assessing human exposures in both prospective and retrospective studies. Given the small amount of HSA required (1 mg or less) the methods should also be ideal for applications involving precious specimens of archived serum, plasma, or whole blood.

Key words: adduct, HSA, cumulative exposure, PAH, formaldehyde

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