Accepted Manuscript

Using untargeted metabolomics for detecting exposome compounds

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PII: S2468-2020(17)30138-9

DOI: 10.1016/j.cotox.2018.03.002

Reference: COTOX 129

To appear in: Current Opinion in Toxicology

Received Date: 27 October 2017

Revised Date: 18 December 2017

Accepted Date: 2 March 2018

Please cite this article as: C.S. Bloszies, O. Fiehn, Using untargeted metabolomics for detecting exposome compounds, *Current Opinion in Toxicology* (2018), doi: 10.1016/j.cotox.2018.03.002.

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2 compounds

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16 Highlights

- Chronic diseases are influenced by gene-environment interactions.
- While the genome has already been characterized, a better understanding of the
- 19 exposome is necessary to fully understand disease phenotypes.
- Untargeted metabolomics is perfectly suited to handle the complexity and breadth of
 the chemical exposome, and should be used to complement existing targeted methods.
- Through various mass spectrometric techniques, untargeted metabolomics allows for
 the detection of both exposure compounds and the phenotypic variation caused by exposure
 compounds.

2526 Abstract

The exposome is the summary of all chemical and non-chemical exposures over an 27 individual's lifetime that collectively describe all non-genetic factors that may influence 28 29 phenotype. While advances in genomics have significantly improved the understanding of 30 chronic disease, they have also highlighted the need for better characterization of exposure. 31 Untargeted metabolomics should complement targeted methods for quantitative and reliable analysis of exposome compounds in biological matrices. Using an existing workflow consisting 32 33 of untargeted instrumental acquisition, analyte annotation using library matching, unknown 34 identification, and data visualization, environmental effects on endogenous metabolites can be assessed by accurate and comprehensive exposure analysis. 35

36

37 Genome wide association studies do not explain complex disease phenotypes

38 In recent years, it has become clear that the environment has more impact on disease

39 phenotype than originally thought [1]. While the Human Genome Project and the subsequent

- 40 Genome-wide Association Studies (GWAS) have been successful in elucidating associations
- 41 between genotype and phenotype [2], much of phenotypic variation remains unexplained for
- 42 chronic diseases [3]. GWAS studies are designed to get a better understanding of heritability, a
- 43 measure of the proportion of total phenotypic variability explained by genomic variability.

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