The Exposome
Why does this matter for Public Health?

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APHL Annual Meeting

Indianapolis, May, 2015

What is “Exposure”?

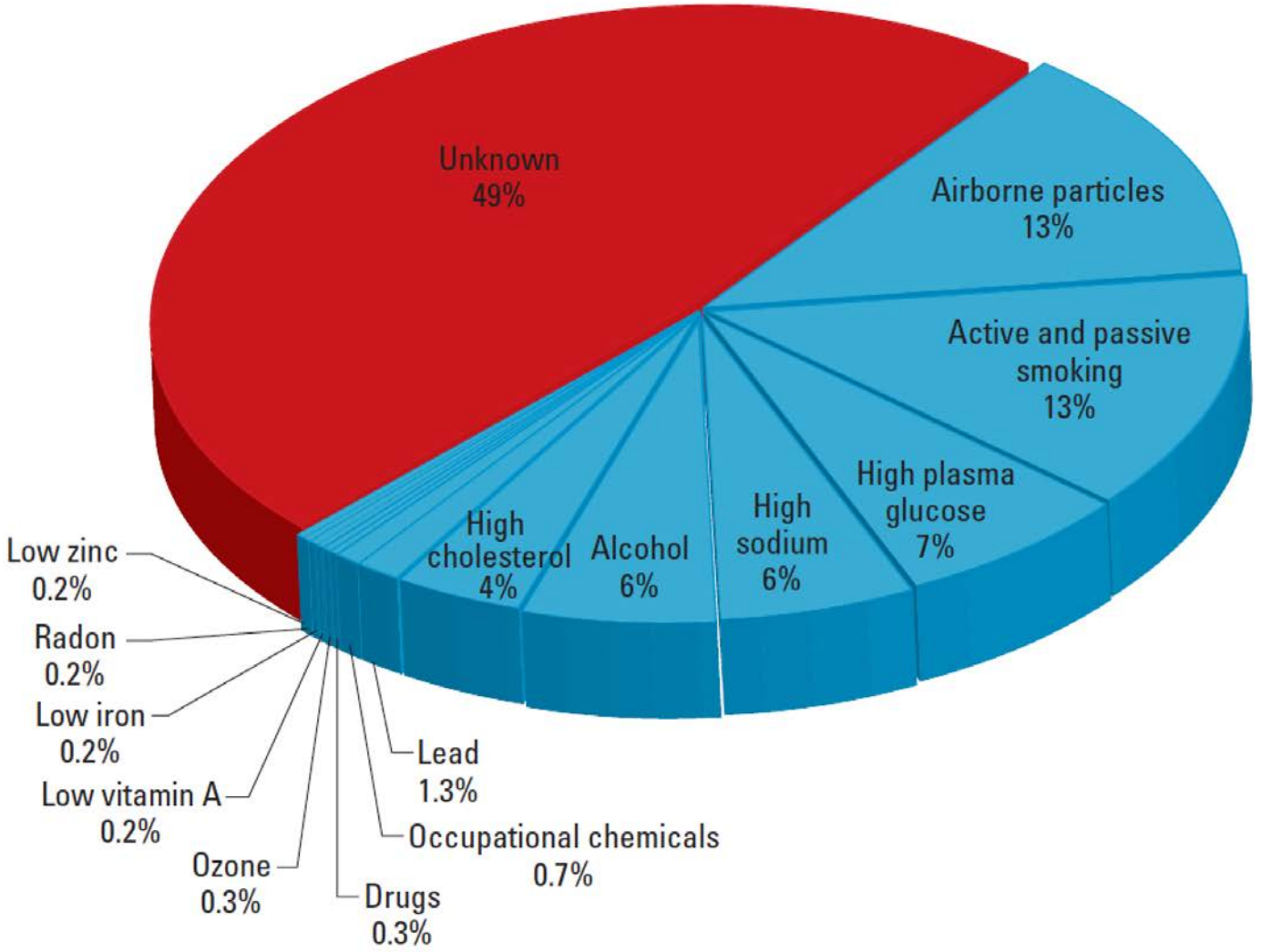
- The **epidemiologist**’s view : something a person can tell you (location, diet, behavior, lifestyle, etc.)
  - Indirect , categorical surrogate for a predictor of disease risk
- The **molecular epidemiologist**’s view: something (biomarker) measured inside a person
  - Relates directly or indirectly to internal dose
- **Exposure scientist**’s view : something measured or predicted outside a person
  - External level(s) across media (air, water, dermal contact, etc.)

Exposure is Dynamic

- Levels vary
  - Within and between persons and across populations
  - Internal and external doses
  - 10-fold to 10,000-fold, depending upon the context
- Variability makes it impossible to accurately predict exposure levels without empiric data
  - Need to measure something – repeatedly!

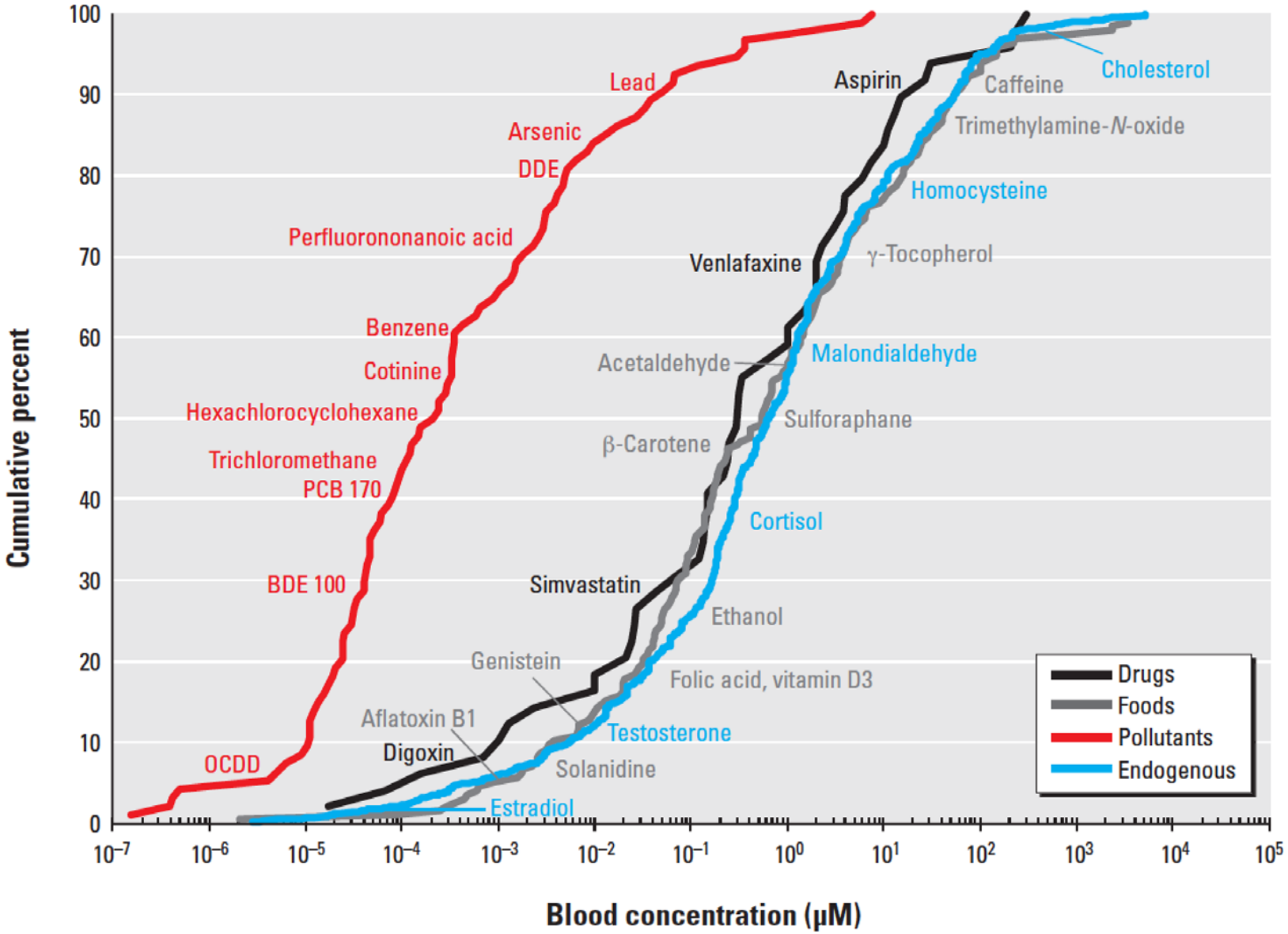
Risk factors for exposures that contribute to chronic-disease mortality

Rappaport et al., EHP, 2014



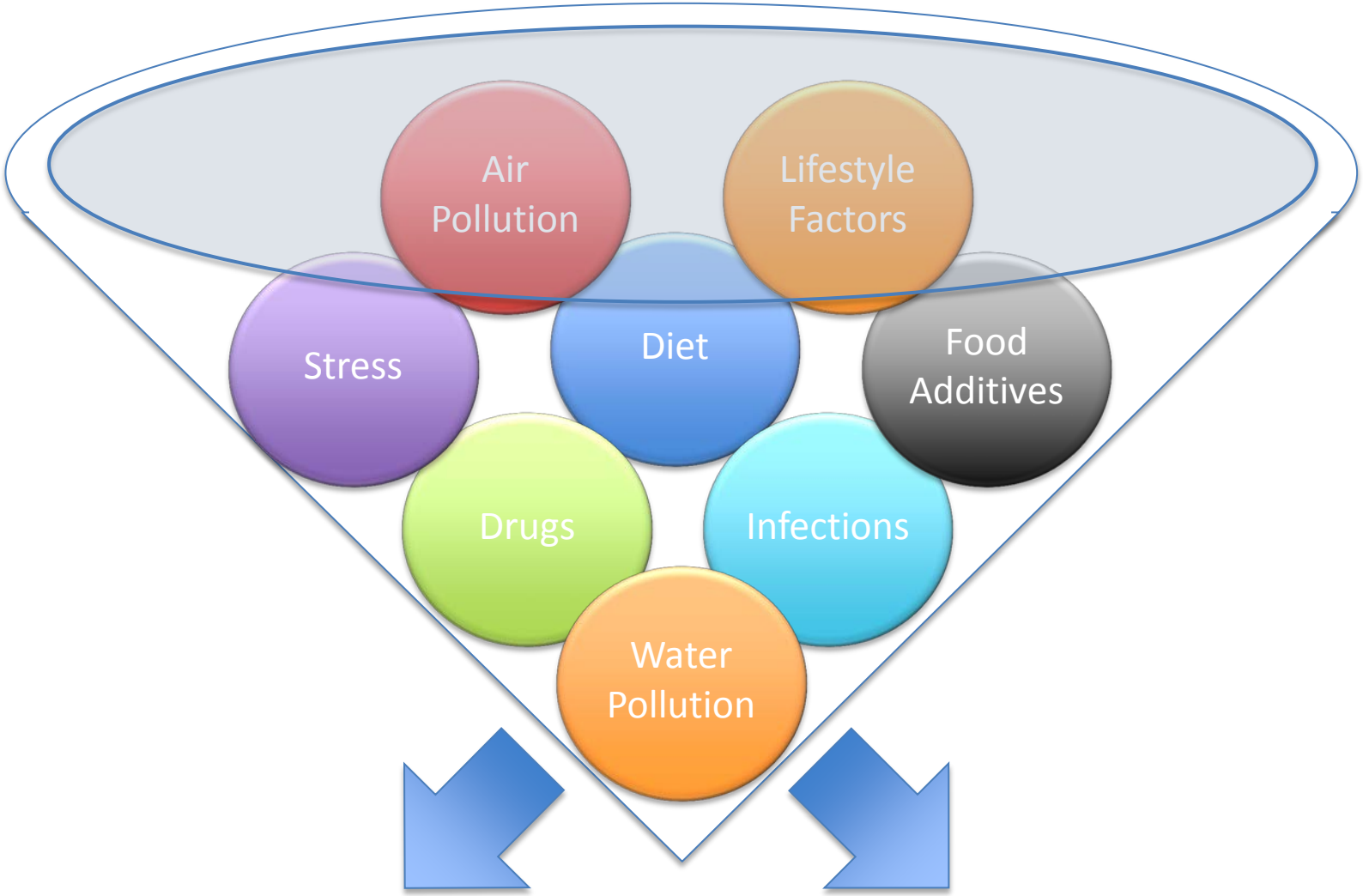
Small molecules and metals in human blood

Rappaport et al., EHP, 2014



What is the “Exposome”?

- “At its most complete, the exposome encompasses life-course environmental exposures (including lifestyle factors) from the prenatal period onwards” *Chris Wild, Cancer Epidemiology Biomarkers 2005*
- A comprehensive measurement of all exposure events (exogenous and endogenous) from conception to death



Direct impacts on health

Indirect impacts on health

Challenges in Characterizing the Exposome

- Scale and complexity
  - Lifecourse environmental exposures
  - Lifestyle, nutrition, occupation etc.
  - Endogenous events at different target sites within the body
- Dynamic
  - The “exposome” changes over time (unlike genome)
  - Critical lifestage windows

Partial characterization is beneficial !

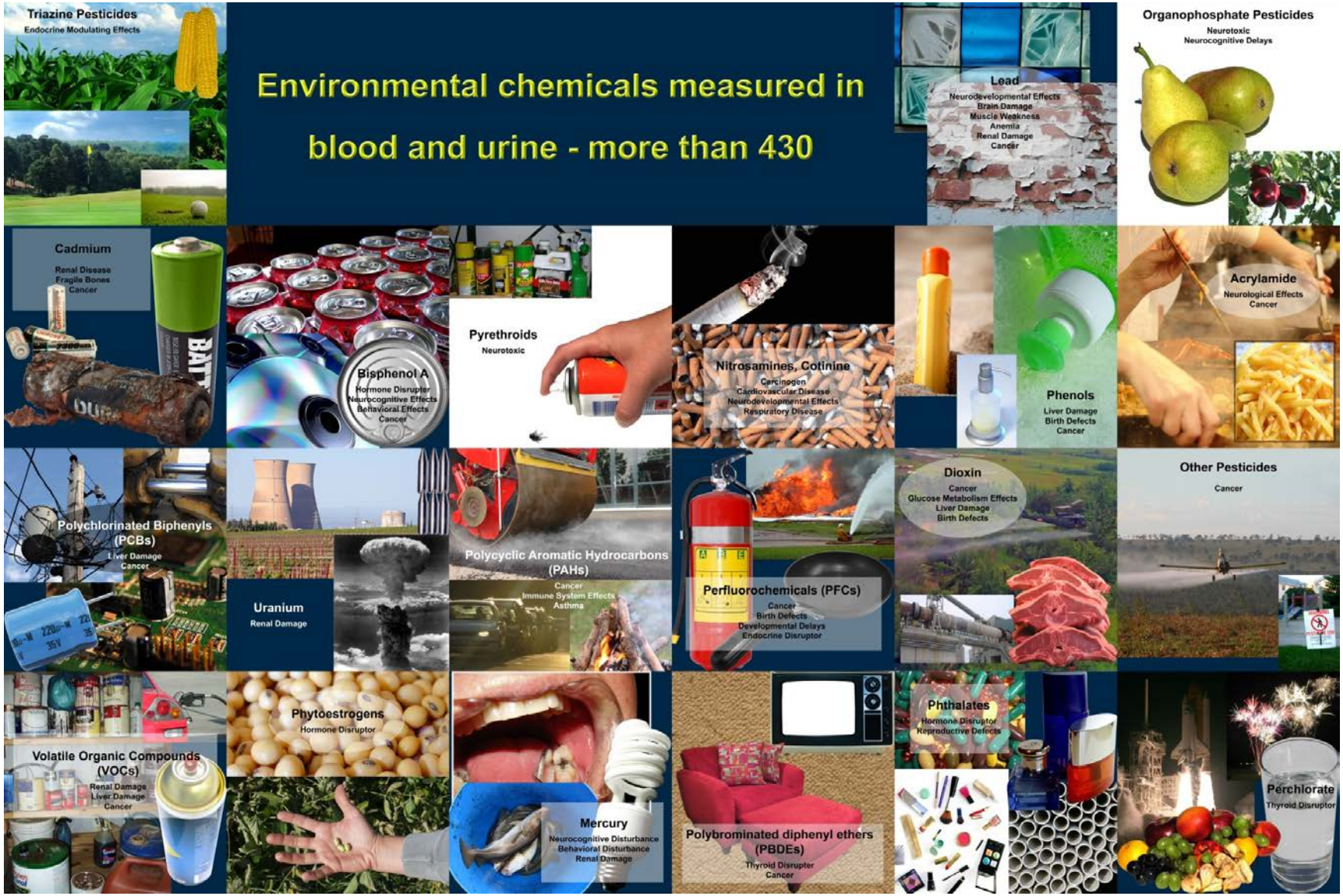
Advances in Exposure Assessment

- Biomonitoring
- Biomarkers
  - "omics" revolution
- Personal and environmental monitoring
  - Cheap sensors
  - Crowd-sourcing
- Increasingly sophisticated questionnaires
  - Social media

	Partially «unbiased» approaches (some selection inevitably necessary)		Selective approach
approach	 patient cohort / healthy control cohort questionnaire-based EWAS, evaluation of socio-demographic factors (e.g. breastfeeding)	 "-omics" EWAS, evaluation of blood/tissue/ exhalation air levels of potential chemicals and factors in patients and control cohorts	 Single factor studies, animal models and selective experiments
advantages	• "unbiased" approach • Retrospective analysis possible • Factors before onset of disease "accessible" • Relatively cheap • Large sample possible	• "unbiased" approach • quantitative measurement • definite factors • Some causation possible, especially for pollution factors	• Hypothesis driven • Causative relationship can be established • Animal models possible • Interventions and therapies easier
disadvantages	• Recall bias • No causative relationships	• Selection bias • Expensive • Only possible after onset of disease • Limited time points	• Selection bias • Low number of factors accessible

Rogler et al, Inflamm Bowel Dis, 2015

National Biomonitoring Program



National Biomonitoring Program targets both the general population and special groups

General population

- *National Exposure Report* (NHANES measurements)
- National Children's Study

Higher exposed or vulnerable groups

Higher or potentially higher exposed groups

Newborns

Women of childbearing age

Elderly

- 50-75 studies each year

PAHs and Obesity

Table 2. Multivariate linear regression β coefficient (95% CI)[a] association between BMI *z*-score, waist circumference, and quartile[b] of ∑molPAHs, or ∑NAPHT.

	BMI *z*-score		Waist circumference	
Exposure	β coefficient (95% CI)	*p*-Value	β coefficient (95% CI)	*p*-Value
ALL (6–19 years)	*n* = 3,189		*n* = 3,189	
∑molPAHs Q1	Referent		Referent	
∑molPAHs Q2	0.18 (0.04, 0.32)	0.01	1.37 (–0.11, 2.85)	0.07
∑molPAHs Q3	0.18 (0.01, 0.35)	0.04	1.34 (–0.28, 2.96)	0.10
∑molPAHs Q4	0.25 (0.08, 0.43)	0.01	2.24 (0.25, 4.23)	0.03
∑NAPHT Q1	Referent		Referent	
∑NAPHT Q2	0.22 (0.06, 0.39)	0.01	1.79 (0.15, 3.43)	0.03
∑NAPHT Q3	0.24 (0.08, 0.40)	< 0.01	1.78 (0.24, 3.32)	0.02
∑NAPHT Q4	0.31 (0.15,0.50)	< 0.01	2.68 (0.88, 4.49)	< 0.01

Scincariello and Buser, EHP, 2014

Pthalates and Obesity

Phthalate	nmol/min: β (SE), p-value	nmol/mL: β (SE), p-value	nmol/mL + crt: β (SE), p-value	nmol/g crt: β (SE), p-value	nmol/kg-day: β (SE), p-value
	Outcome is ln(Body Mass Index)				
DBP	**0.022 (0.005)****	**0.023 (0.004)*****	**0.014 (0.006)***	0.007 (0.006)	**0.040 (0.006)******
BBzP	**0.019 (0.005)****	**0.021 (0.004)*****	**0.011 (0.005)***	0.006 (0.006)	**0.033 (0.006)*****
DEHP[a]	**0.019 (0.005)****	**0.025 (0.004)*****	**0.017 (0.005)***	0.008 (0.006)	**0.033 (0.005)*****
DiNP	**0.020 (0.004)*****	**0.023 (0.004)******	**0.017 (0.004)****	**0.013 (0.004)***	**0.028 (0.004)******
DiBP	**0.022 (0.005)****	**0.025 (0.005)*****	**0.014 (0.006)***	0.003 (0.007)	**0.045 (0.007)******
DEP	**0.013 (0.004)****	**0.016 (0.003)****	**0.010 (0.004)***	0.005 (0.004)	**0.018 (0.004)****
	Outcome is ln(Waist Circumference)				
DBP	**0.011 (0.004)***	**0.014 (0.003)****	0.007 (0.004)	0.001 (0.005)	**0.024 (0.005)*****
BBzP	**0.012 (0.004)****	**0.014 (0.003)****	**0.008 (0.004)***	0.004 (0.004)	**0.023 (0.004)*****
DEHP[a]	**0.012 (0.003)****	**0.017 (0.003)*****	**0.013 (0.004)****	0.006 (0.004)	**0.024 (0.004)*****
DiNP	**0.011 (0.003)****	**0.014 (0.003)*****	**0.011 (0.003)****	**0.008 (0.003)***	**0.018 (0.003)*****
DiBP	**0.012 (0.004)***	**0.016 (0.004)****	0.009 (0.005)	0.0006 (0.005)	**0.029 (0.005)*****
DEP	**0.007 (0.003)***	**0.010 (0.003)****	**0.007 (0.003)***	0.003 (0.003)	**0.011 (0.003)****

[a] Represents the molar sum of 4 DEHP metabolites (MEHP, MEHHP, MEOHP, MECPP).

* $p < 0.05$.

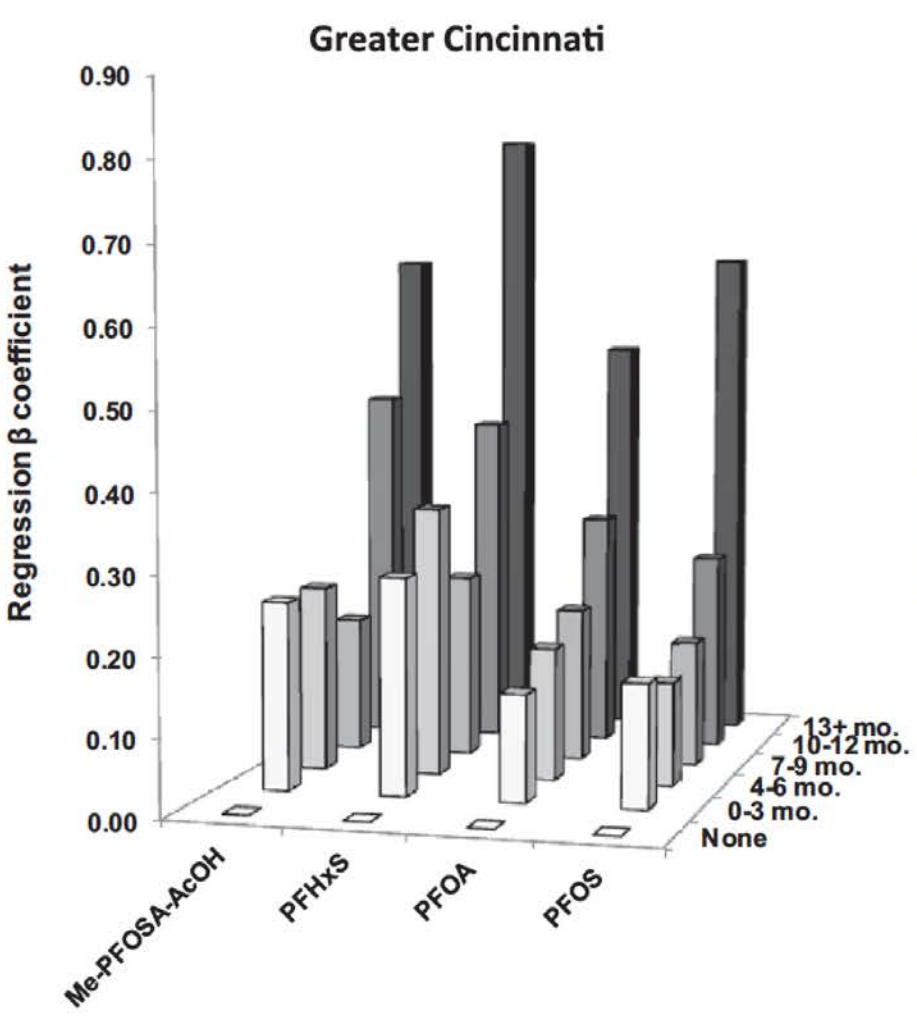
** $p < 0.001$ (1×10^{-3}).

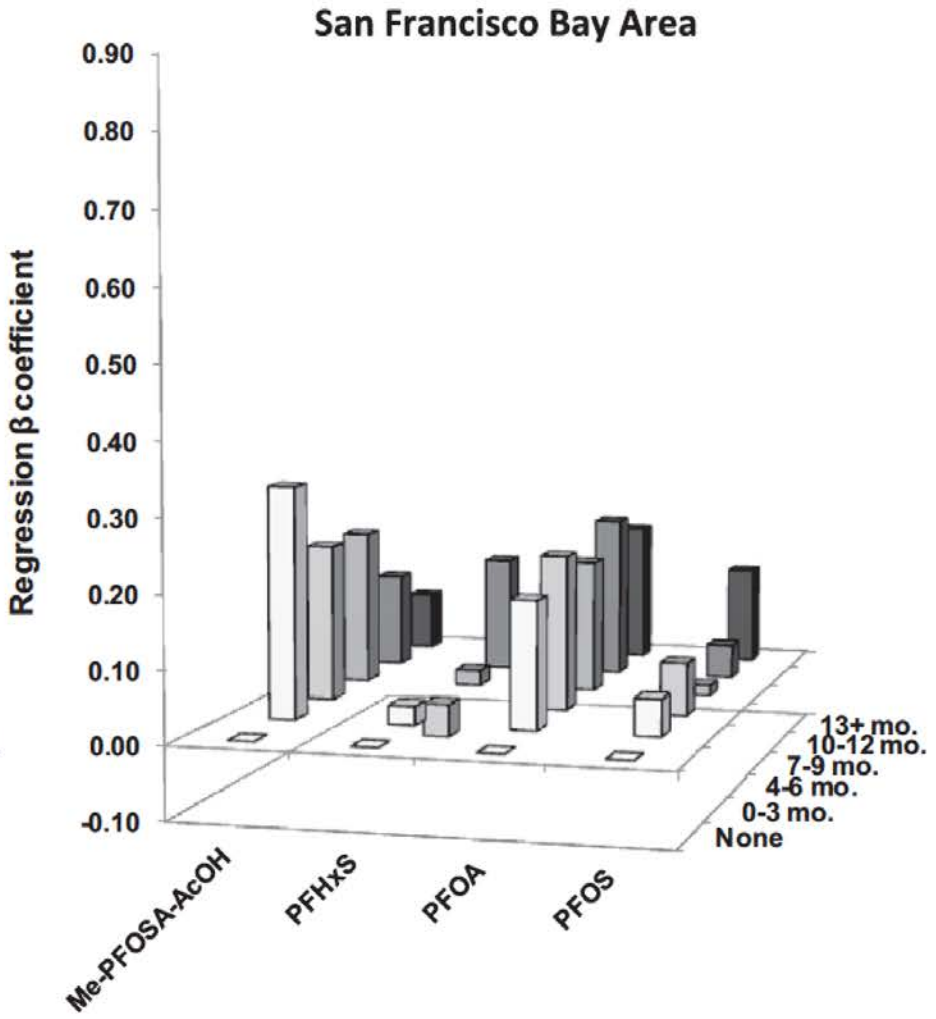
*** $p < 0.000001$ (1×10^{-6}).

**** $p < 0.000000001$ (1×10^{-9}).

Christenson et al., Env Int, 2014

PFCs and Duration of Breast Feeding

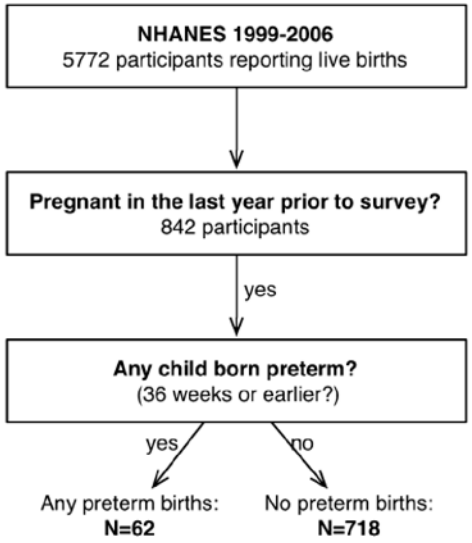




Pinney, et al., Env Pollut, 2014

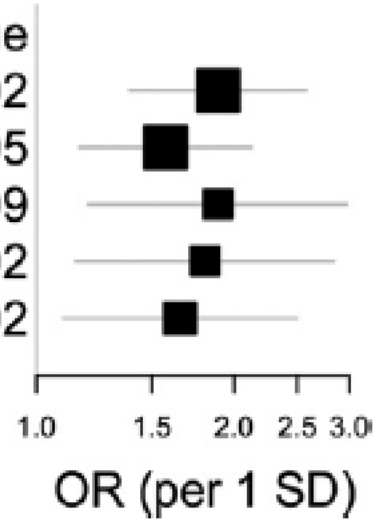
NHANES Exposures and Low Birth Weight

Patel et al., Repro Tox 2014



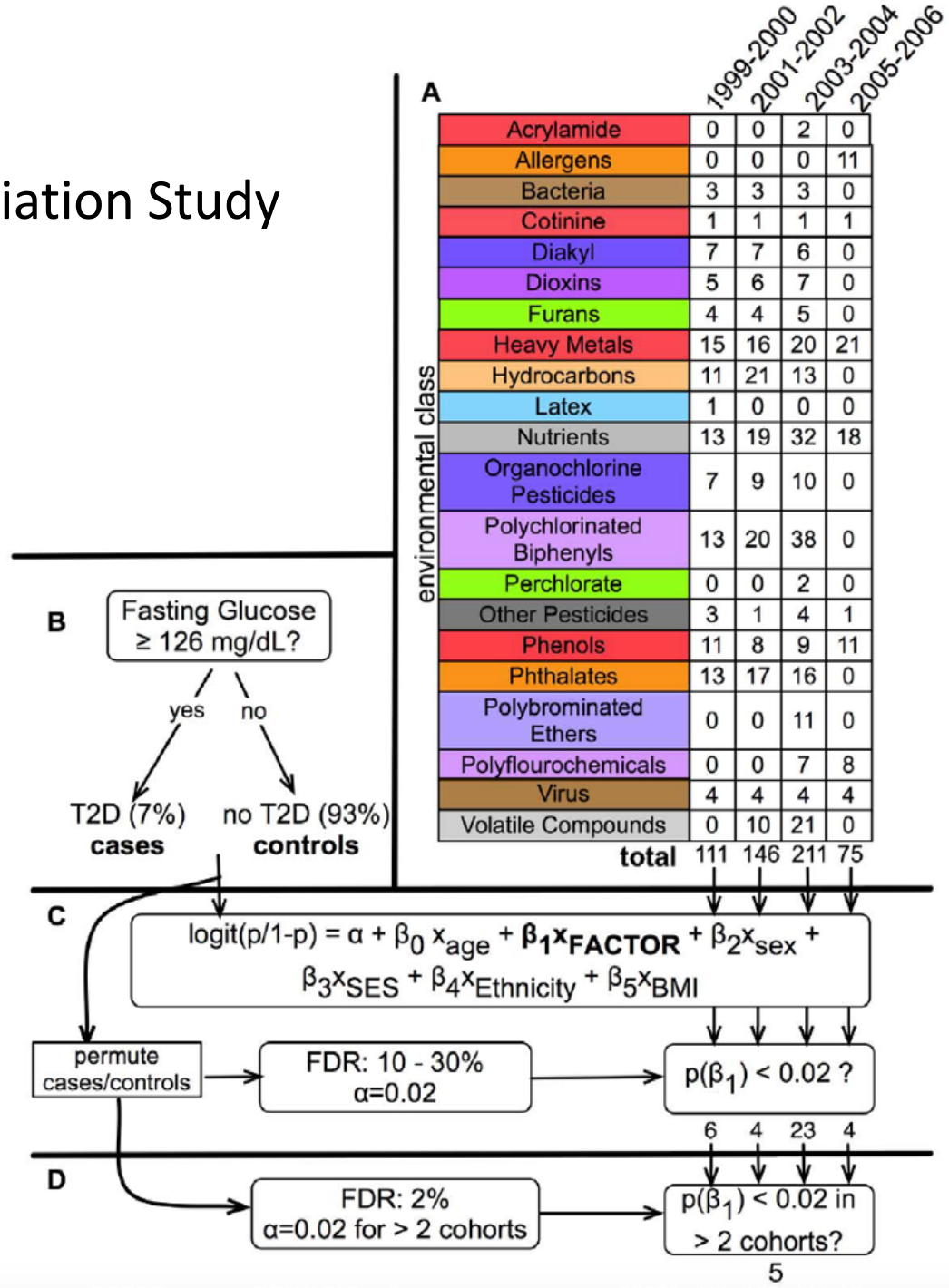
exposure category	number	sample sizes
bacteria	13	129-691
cotinine	1	754
diakyl	7	140-195
dioxins	6	106-163
furans	4	158-162
heavy metals	21	126-762
hydrocarbons	9	171-179
nutrients	32	164-762
polychlorinated biphenyls	23	126-193
perchlorate	3	198-254
pesticides	22	109-250
phenols	3	109
phthalates	11	114-242
phytoestrogens	6	233-245
polyflourochemicals	9	175
virus	11	151-161
volatile compounds	20	135-241

Factor	N(cases)	OR [95% CI]	pvalue
Urinary Bisphenol A	109(10)	1.9[1.4,2.6]	0.002
Serum Iron	761(62)	1.6[1.2,2.1]	0.005
Urinary Cesium	245(20)	1.9[1.2,3]	0.009
Urinary 1-hydroxypyrene	179(11)	1.8[1.1,2.8]	0.02
Serum Beta-cryptoxanthin	586(51)	1.7[1.1,2.5]	0.02

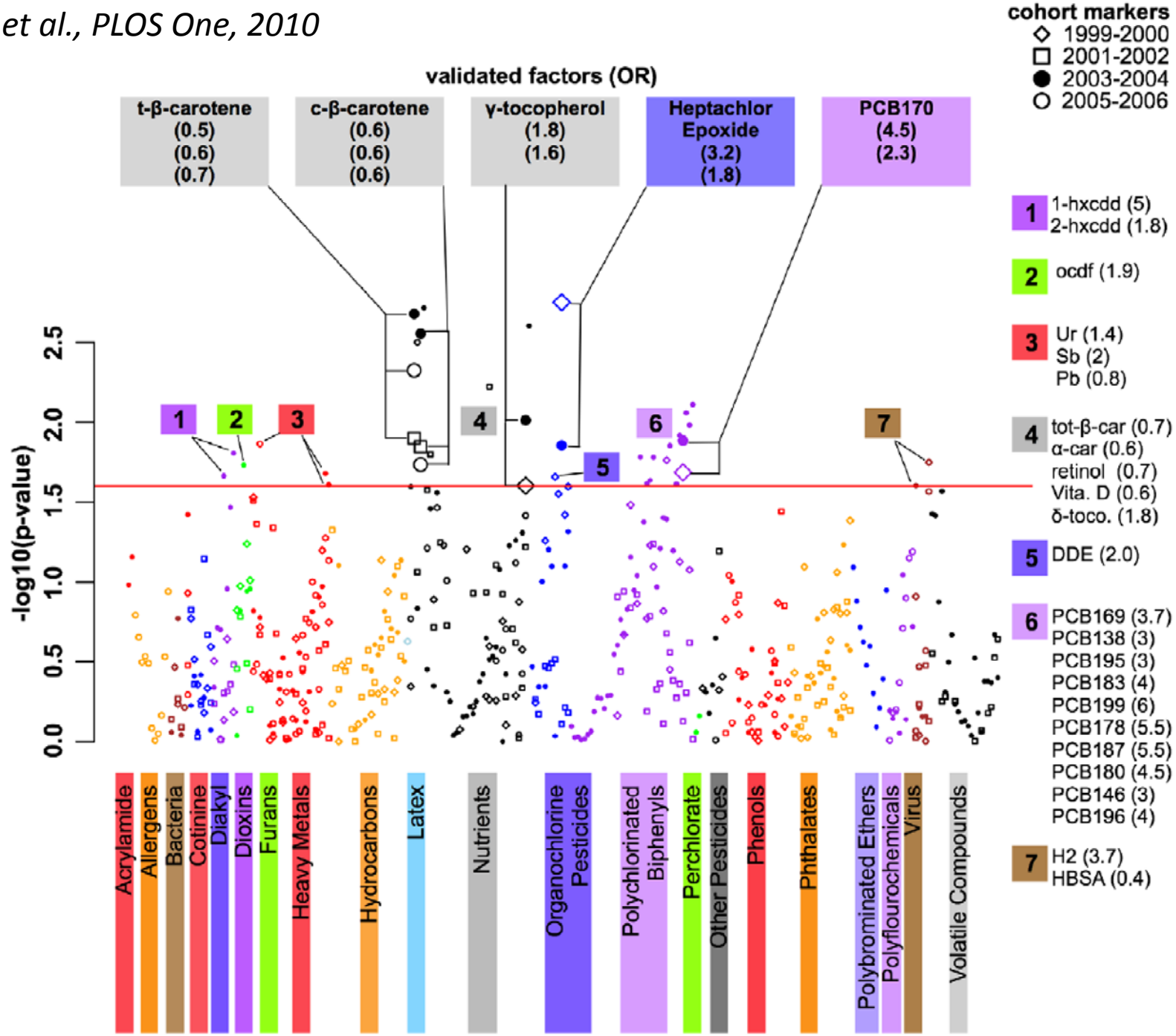


Environment-Wide Association Study for Type 2 Diabetes

Patel et al., PLOS One, 2010

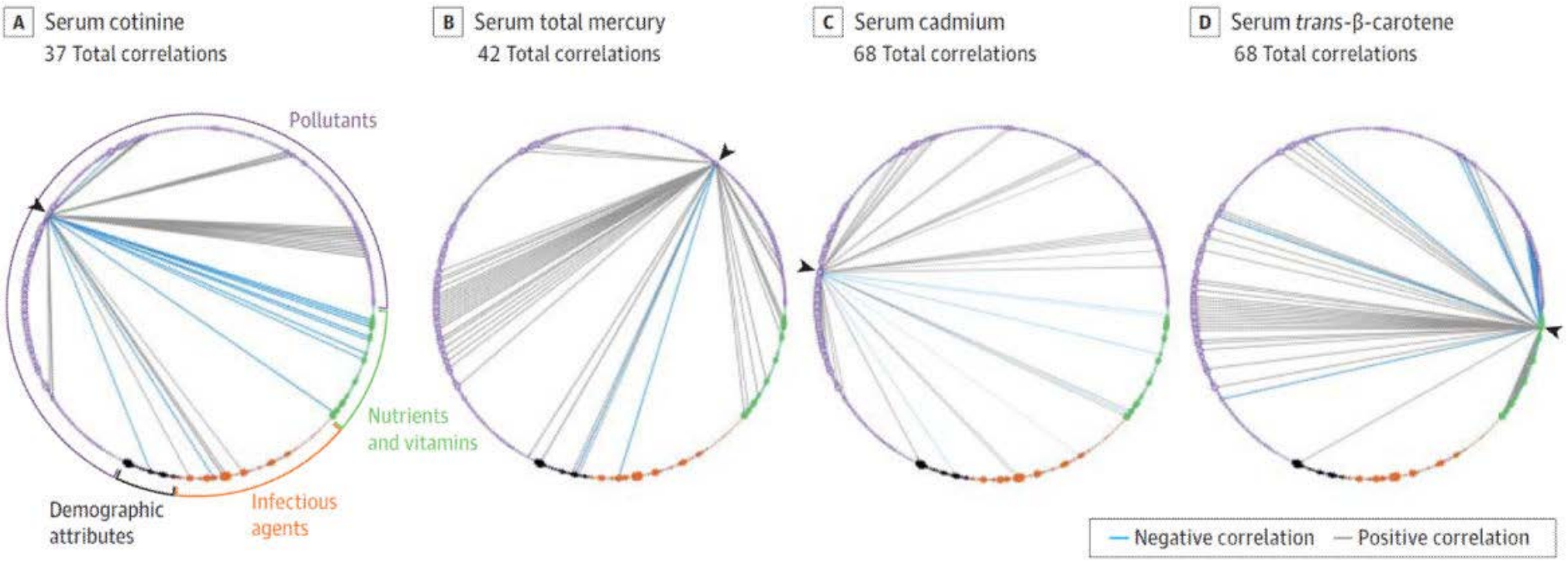


Patel et al., PLOS One, 2010



Correlation Interdependency Globes for 4 Environmental Exposures (Cotinine, Mercury, Cadmium, Trans -β -Carotene) in National Health and Nutrition Examination Survey (NHANES) Participants, 2003–2004

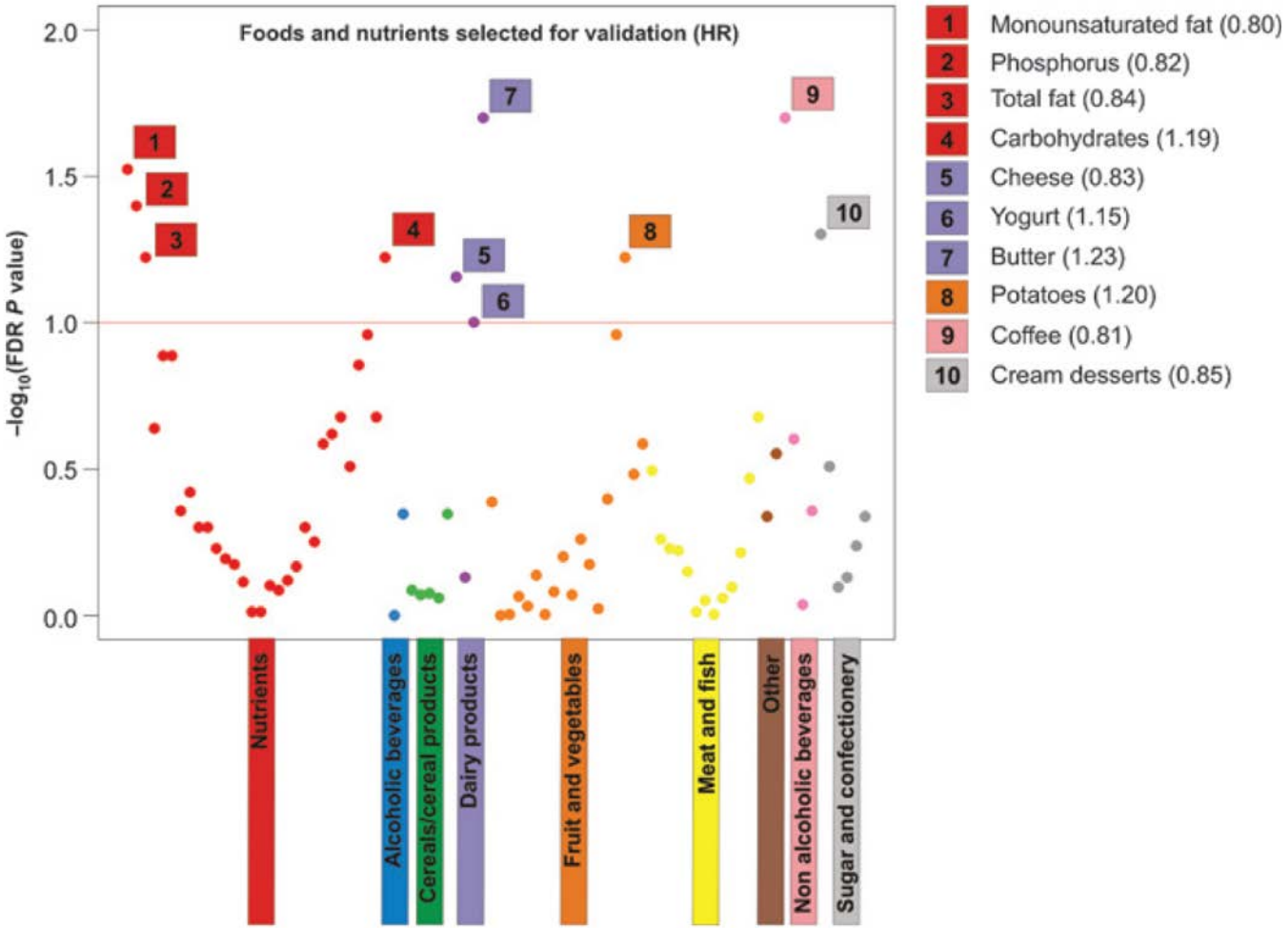
Patel et al, JAMA 2014



Each correlation interdependency globe includes 317 environmental exposures represented by the nodes around the periphery of the globe. Pairwise correlations are depicted by edges (lines) between the node of interest (arrowhead) and other nodes. Correlations with absolute values exceeding 0.2 are shown (strongest 10%). The size of each node is proportional to the number of edges for a node, and the thickness of each edge indicates the magnitude of the correlation.

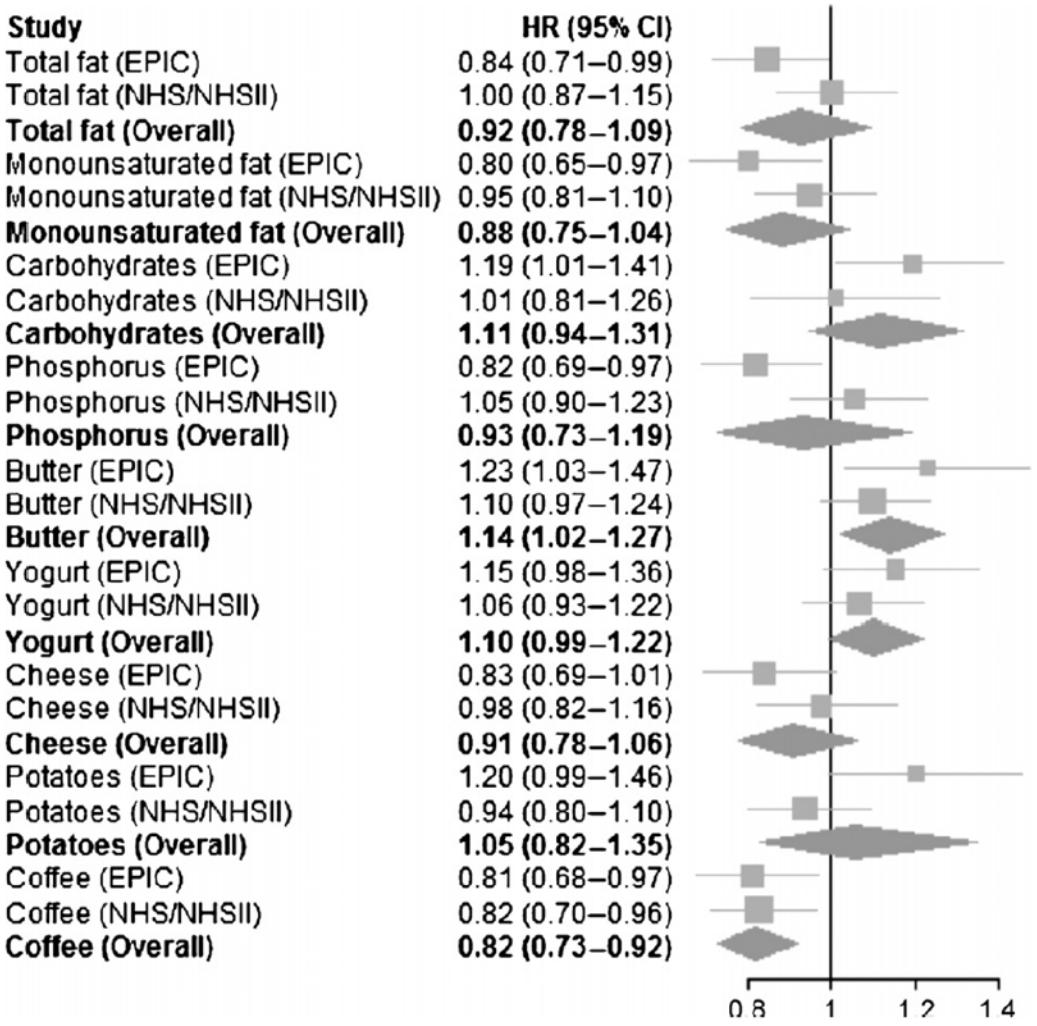
Endometrial Cancer and Food Nutrients in the European Prospective Investigation into Cancer and Nutrition (EPIC) study

Merritt et al. Cancer Epi, Biomarkers and Prevention, 2015



Comparison of EPIC and NHSII for Endometrial Cancer and Food Nutrients

Merritt et al. Cancer Epi, Biomarkers and Prevention, 2015



Biomonitoring

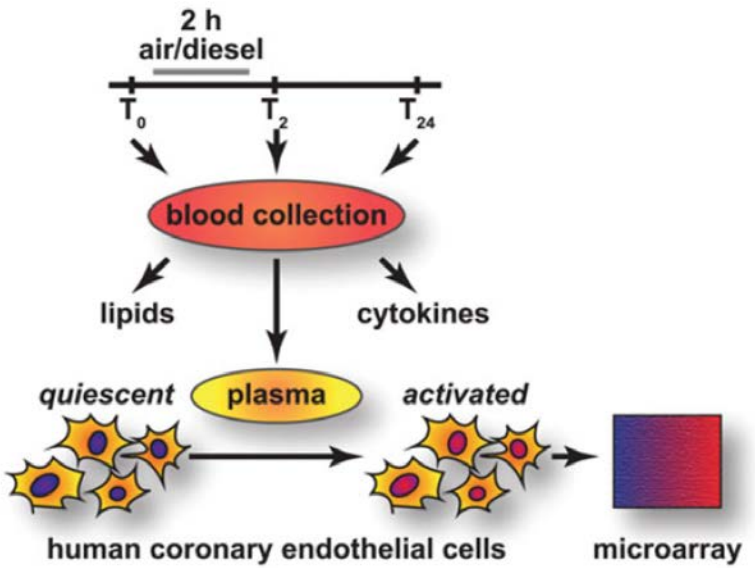
- Advantages
  - Direct measure of exposure
  - Can assess all sources of exposure (chem, drugs, nutrient, etc.)
  - Unbiased
- Disadvantages
  - Short- versus long-term exposures
  - Expensive
  - Identifying unique biomarkers

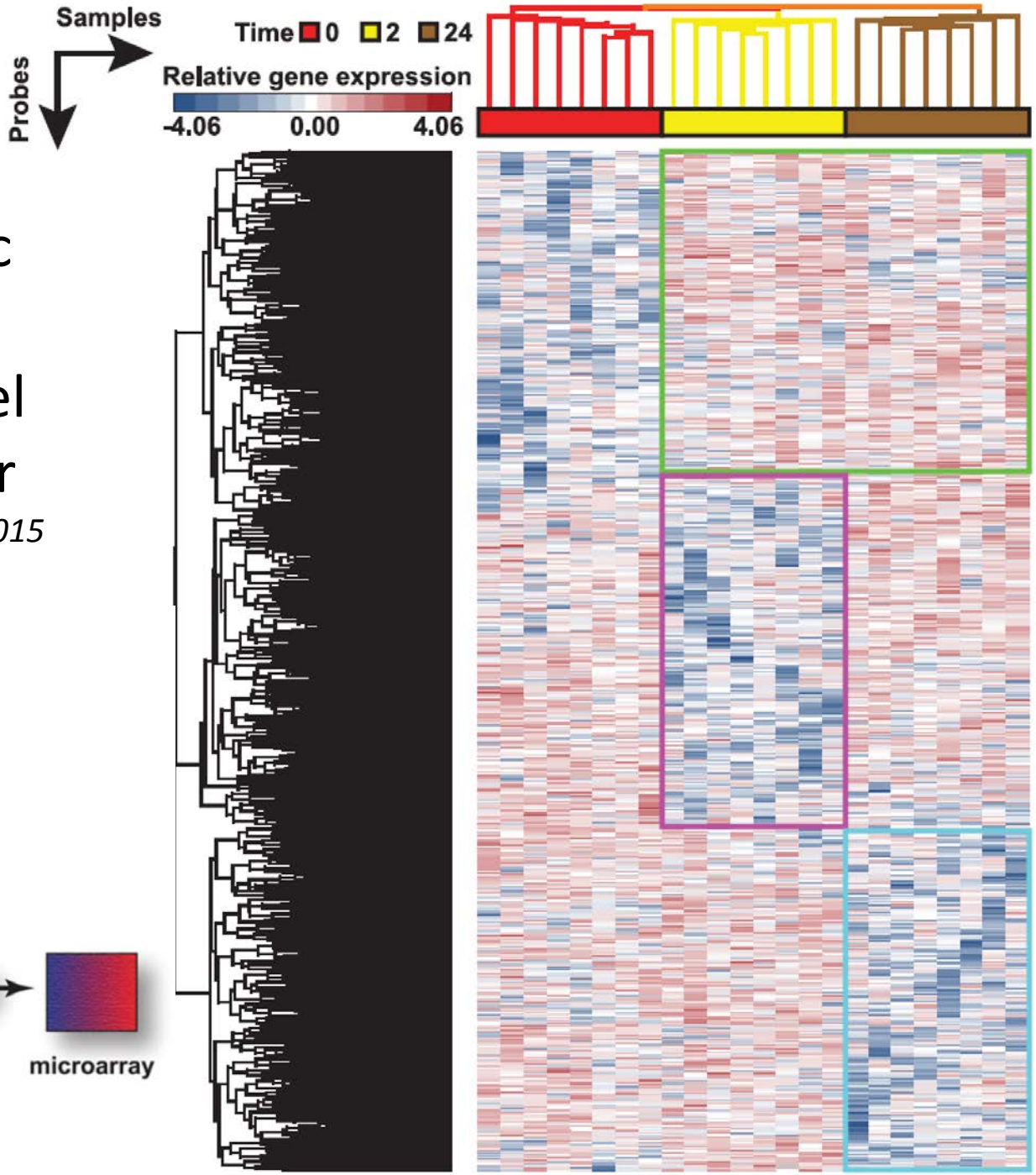
How does “omics” improve exposure assessment?

- Specific exposures, or categories of exposure can alter the expression of specific groups of genes, proteins or metabolites (“exposure fingerprint”)?
  - How do such alterations relate to dose?
  - How stable are the alterations over time?
  - How do potential confounding factors affect the association between exposure and “omics” biomarkers
    - Can confounders confound “omics” biomarkers?

Ex-Vivo Transcriptomic Fingerprint following Diesel Exposure in Air

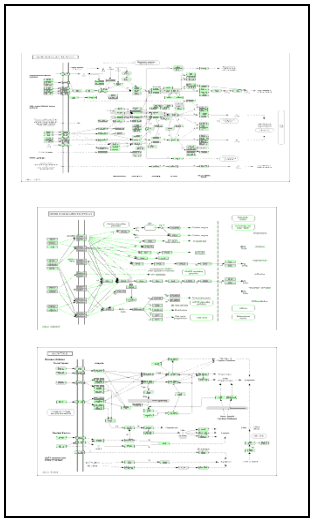
Schissler et al, Inhal. Tox. 2015





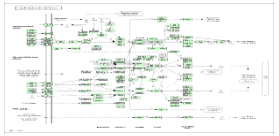
Identifying Important Disease Pathways

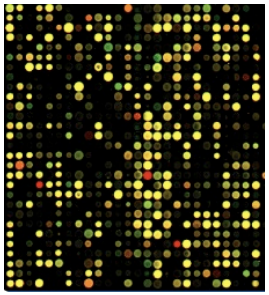
Human Genetics
+
Human Disease



Sets of Pathways

Identify Pathways Related to Human Disease

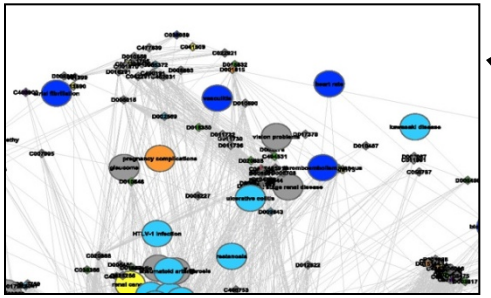




High Throughput
High Content
Chemical-Specific
Data

New
‘omics &
HTS Data

Predict
Chemical-Gene-
Disease Interactome



Predict
Risks

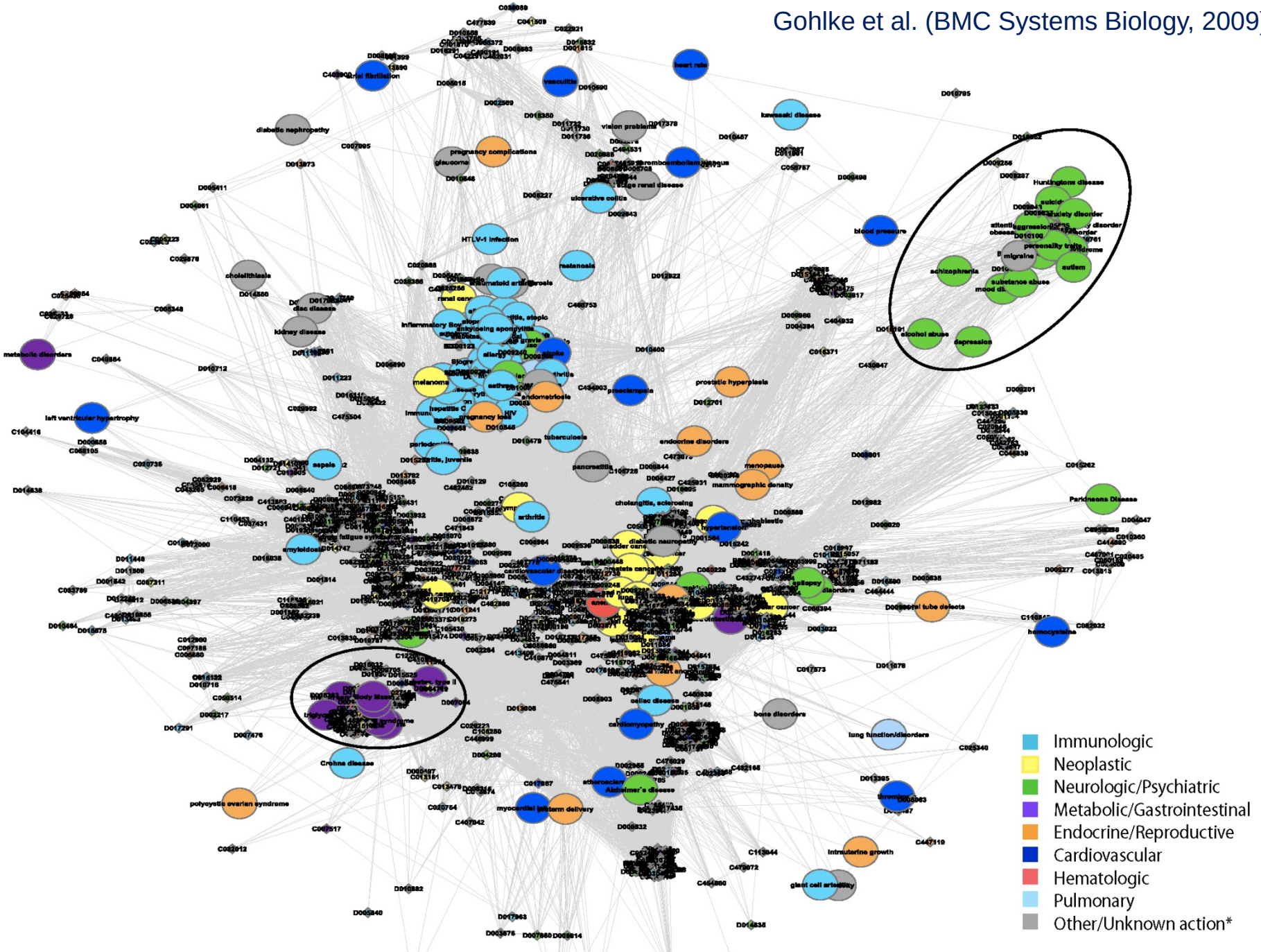
Develop
Screens

Set
Priorities

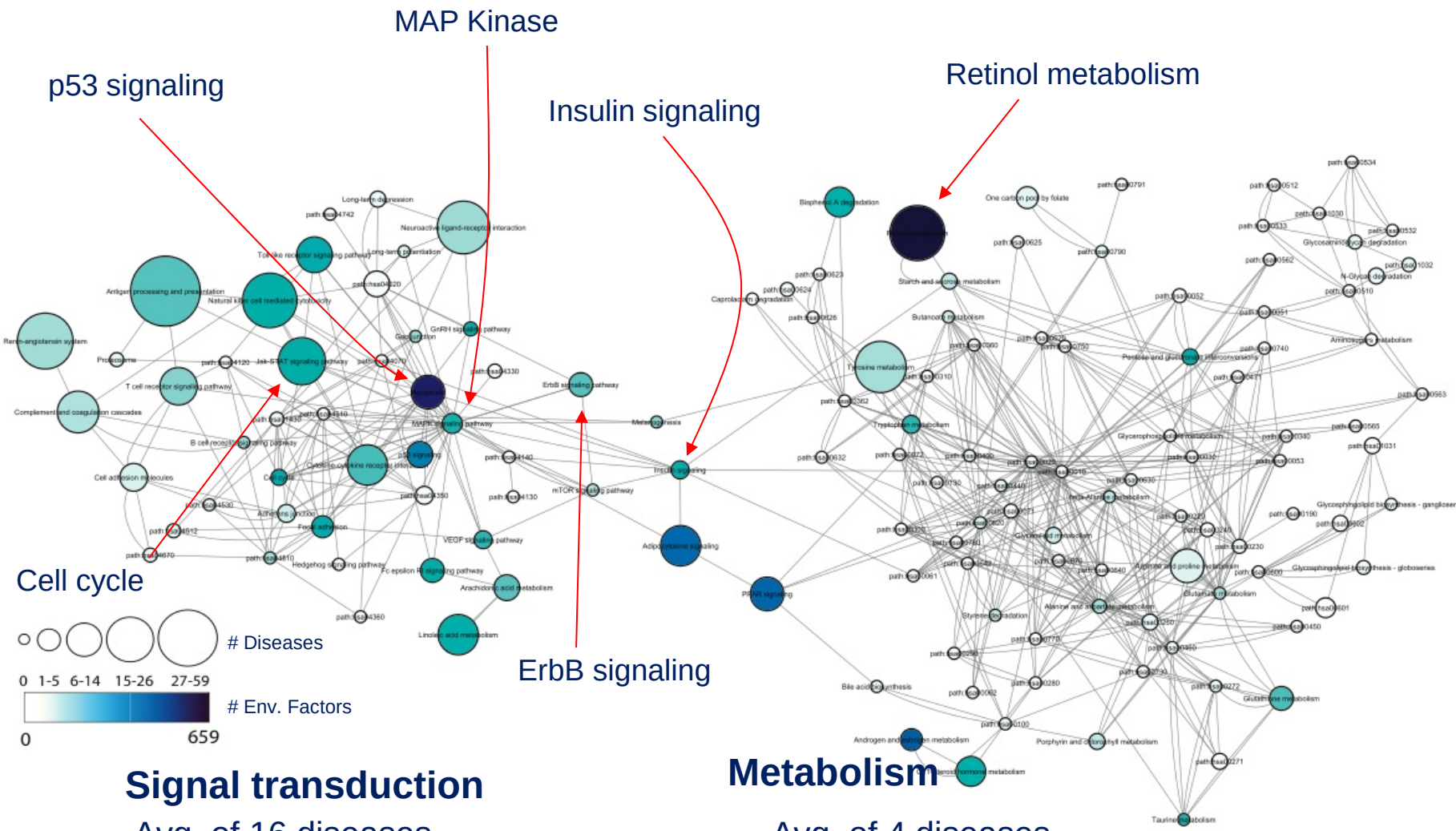
Fingerprint
Toxicants

Develop
Hypotheses

Gohlke et al. (BMC Systems Biology, 2009)



Relating Across Pathways



Signal transduction

Avg. of 16 diseases associated with each signaling pathway

Metabolism

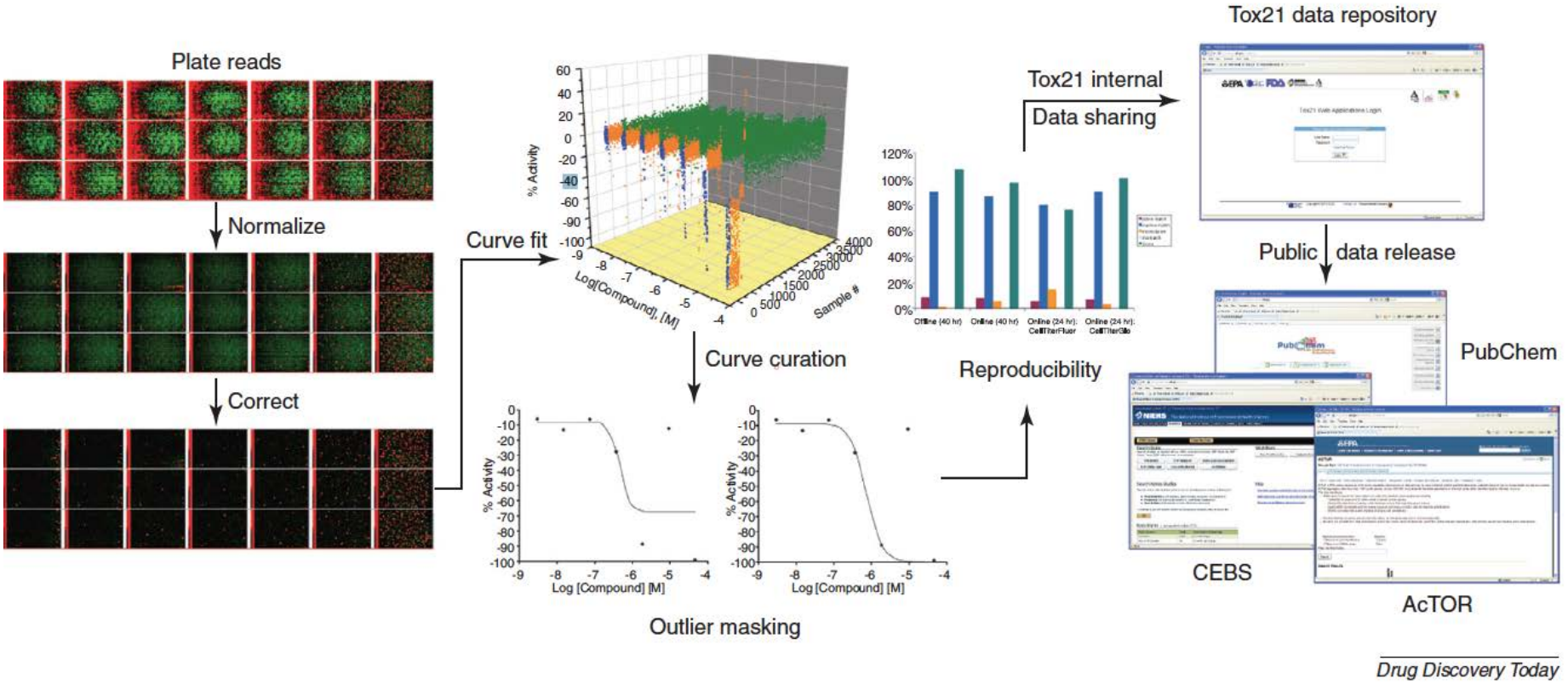
Avg. of 4 diseases associated with each metabolic pathway

Metabolomics Profiling

Table 6
Metabolite pattern for liver enzyme induction in female rats. Red colour indicates statistical significance $p < 0.05$.

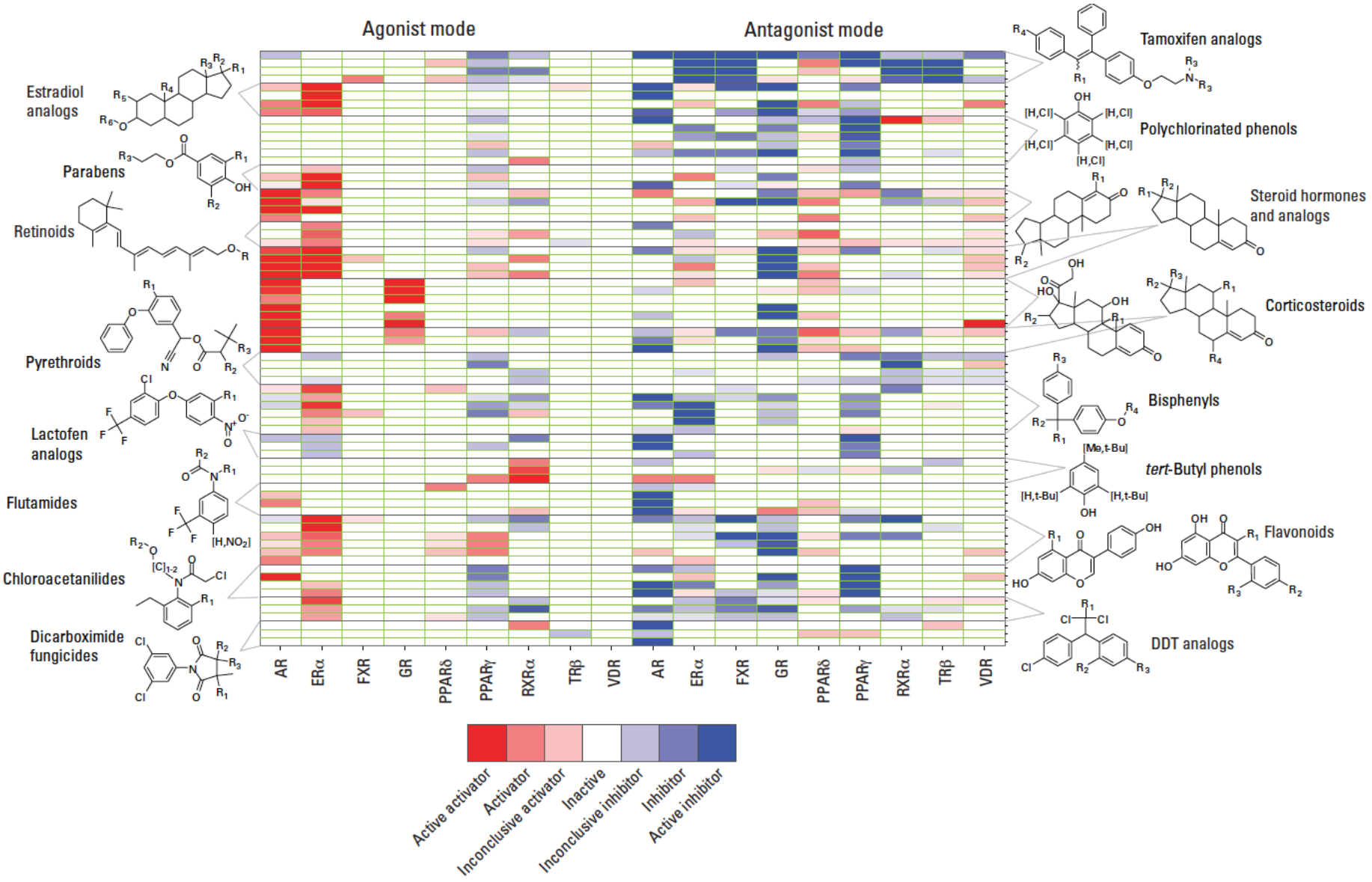
		2-Acetylamino-fluorene			4-Acetylamino-fluorene			Aroclor 1254			Ethylbenzene			Pentachloro-benzene			Phenobarbital sodium			Vinclozolin		
Metabolite	**Direction**	**f7**	**f14**	**f28**	**f7**	**f14**	**f28**	**f7**	**f14**	**f28**	**f7**	**f14**	**f28**	**f7**	**f14**	**f28**	**f7**	**f14**	**f28**	**f7**	**f14**	**f28**
Glycerol, lipid fraction	up	1,62	1,97	1,47	1,20	1,26	1,17	1,01	1,24	1,16	2,92	2,31	2,04	2,38	7,37	3,30	1,38	1,25	1,00	1,62	1,94	1,55
Palmitic acid	up	1,29	1,37	1,42	1,21	1,16	1,25	1,16	1,27	1,19	2,39	2,00	1,70	1,59	3,46	1,86	1,50	1,34	1,14	1,54	1,51	1,65
Linoleic acid	up	1,37	1,45	1,38	1,16	1,24	1,27	1,34	1,34	1,54	3,04	2,46	2,00	2,11	5,23	2,69	1,73	1,41	1,35	1,46	1,62	1,69
Stearic acid	up	1,16	1,19	1,15	1,24	1,23	1,25	1,34	1,54	1,91	1,73	1,92	1,51	1,30	1,87	1,65	1,48	1,41	1,23	1,75	1,99	2,07
Arachidonic acid	up	1,18	1,20	1,14	1,22	1,22	1,28	1,25	1,48	1,53	1,99	2,26	1,52	1,27	1,89	1,50	1,46	1,25	1,15	1,80	1,98	2,08
Cholesterol	up	1,19	1,32	1,01	1,31	1,25	1,38	1,43	1,45	1,53	2,13	2,39	1,82	1,23	1,64	1,62	1,40	1,26	1,12	1,70	1,92	2,08
Lignoceric acid	up	1,07	1,22	1,24	1,12	1,19	1,14	1,07	1,40	1,18	1,83	2,13	1,47	1,39	1,60	1,75	1,36	1,38	1,03	1,76	1,74	1,91
Eicosanoic acid	up	1,03	1,17	1,19	0,96	1,45	1,27	1,87	2,25	1,40	2,05	1,73	2,22	1,13	2,72	1,98	1,26	1,43	1,17	1,38	1,61	1,69
Behenic acid	up	0,97	1,20	1,39	1,15	1,11	1,18	1,57	1,27	1,24	1,75	2,11	2,00	1,30	1,66	1,47	1,75	1,47	1,19	1,49	1,73	1,84

Van Ravenzwaay et al., Mutat Res 2012



Drug Discovery Today

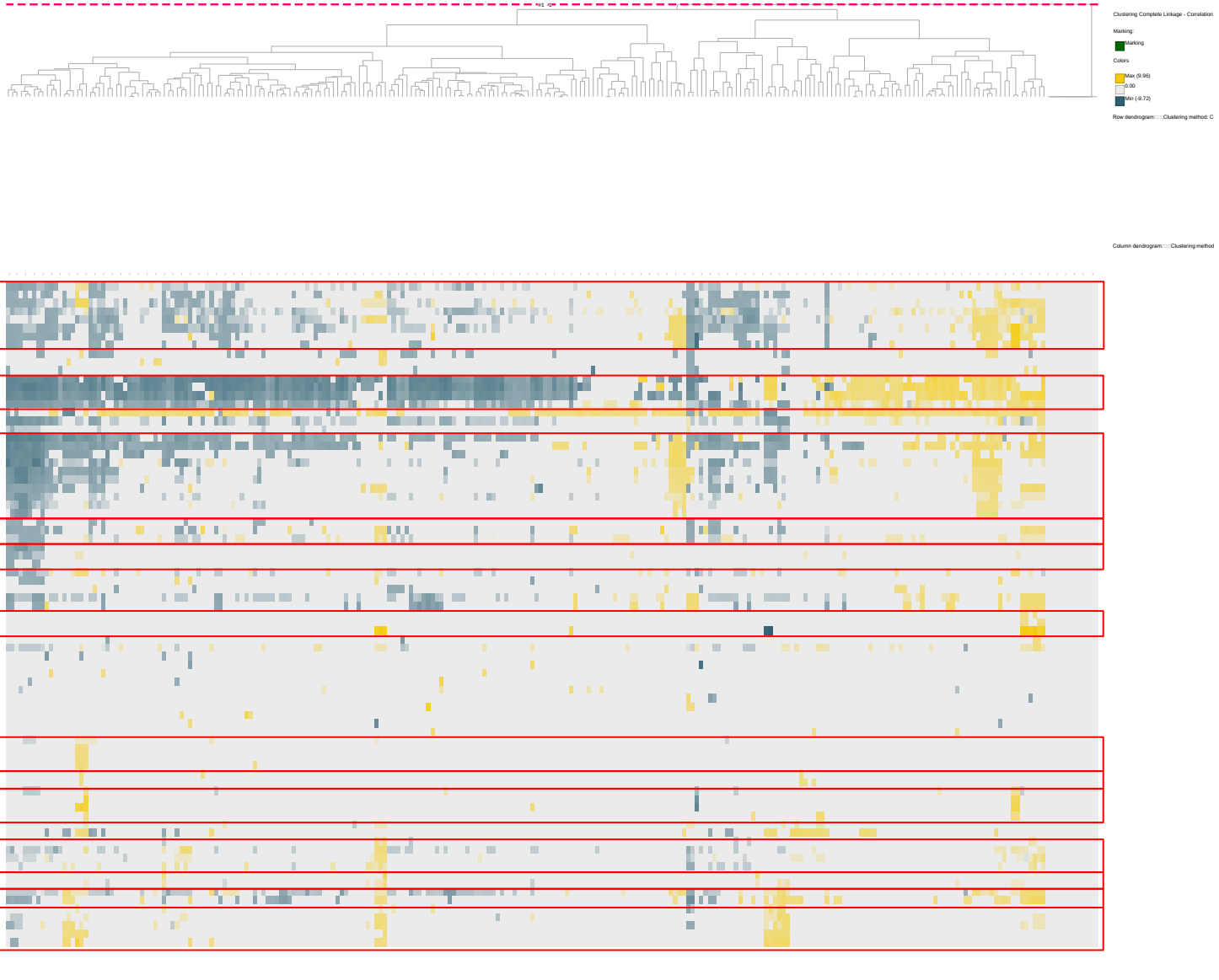
Attene-Ramos et.al, Drug Disc. Today 18, 2013

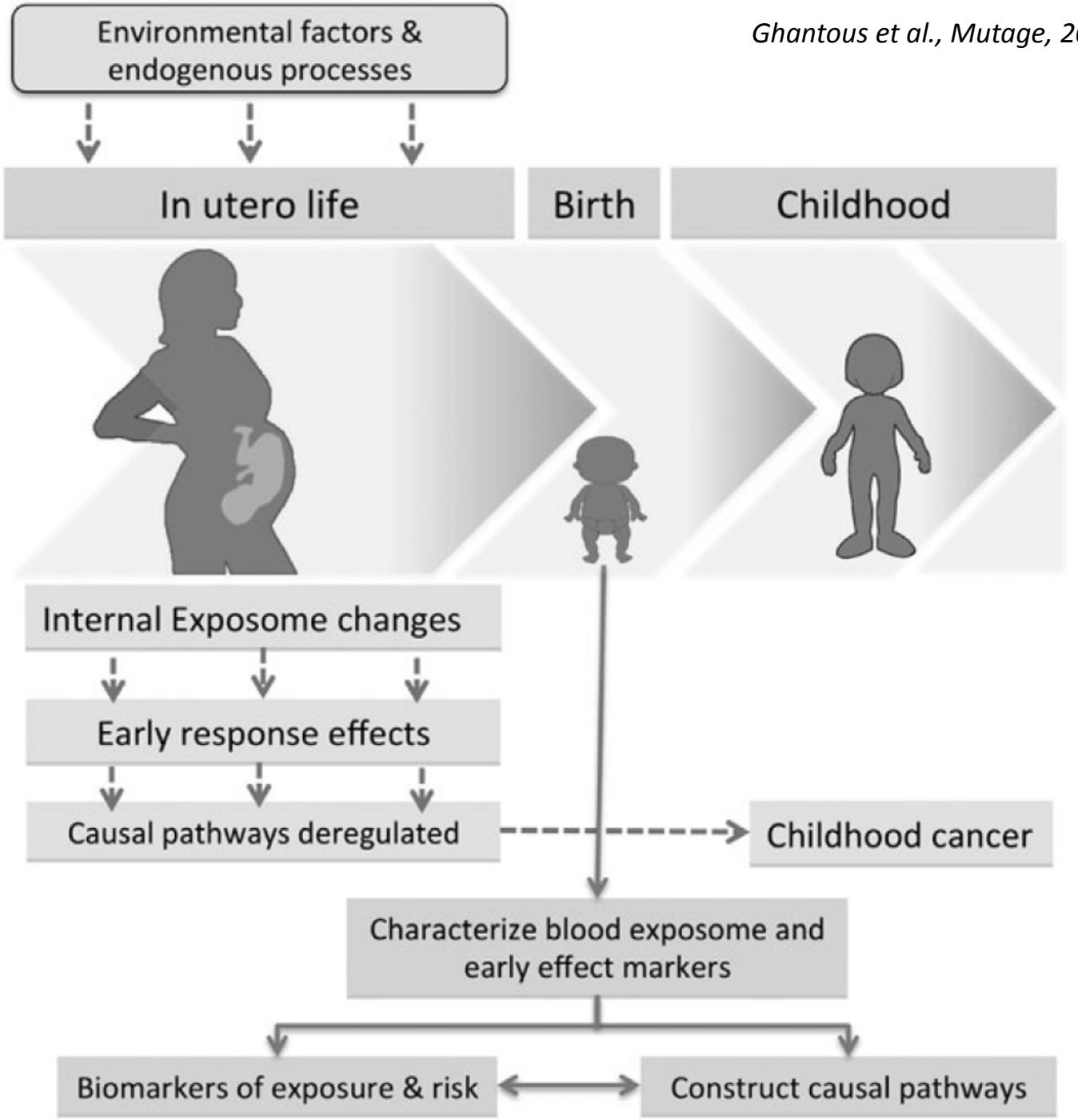


Huang et al., EHP 2011

AHR – Ah receptor
AR – androgen receptor
ARE – antioxidant response element
Aromatase – aromatase inhibitors
DT40 – cytotoxicity
ER – estrogen receptor alpha
FXR – farnasoid X receptor
GH3 – thyroid receptor
GR – glucocorticoid receptor
HSE – heat shock response
MITOTOX – mitochondrial membrane
P53 – P53 signaling
PPARD – PPAR delta
PPARG – PPAR gamma
SPEC – test for autofluoresence
VDR – vitamin D receptor

Heat Map of Group 1 and Advisory Group Chemicals that are in Tox21-v2 Database

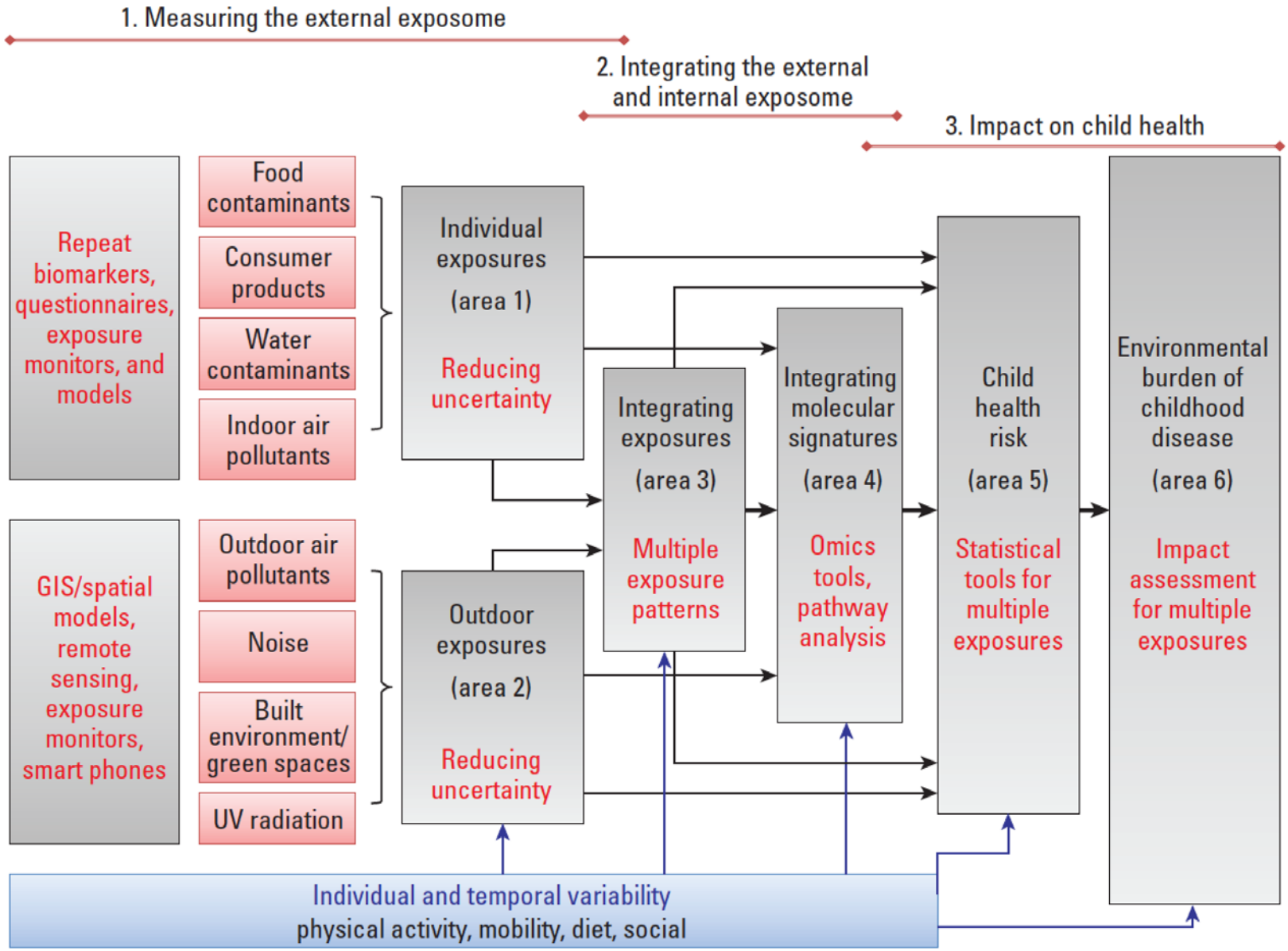




Ghantous et al., Mutage, 2015

Helix Study Conceptul Framework

Vrijheid et al., EHP, 2014



Helix Study Conceptual Framework

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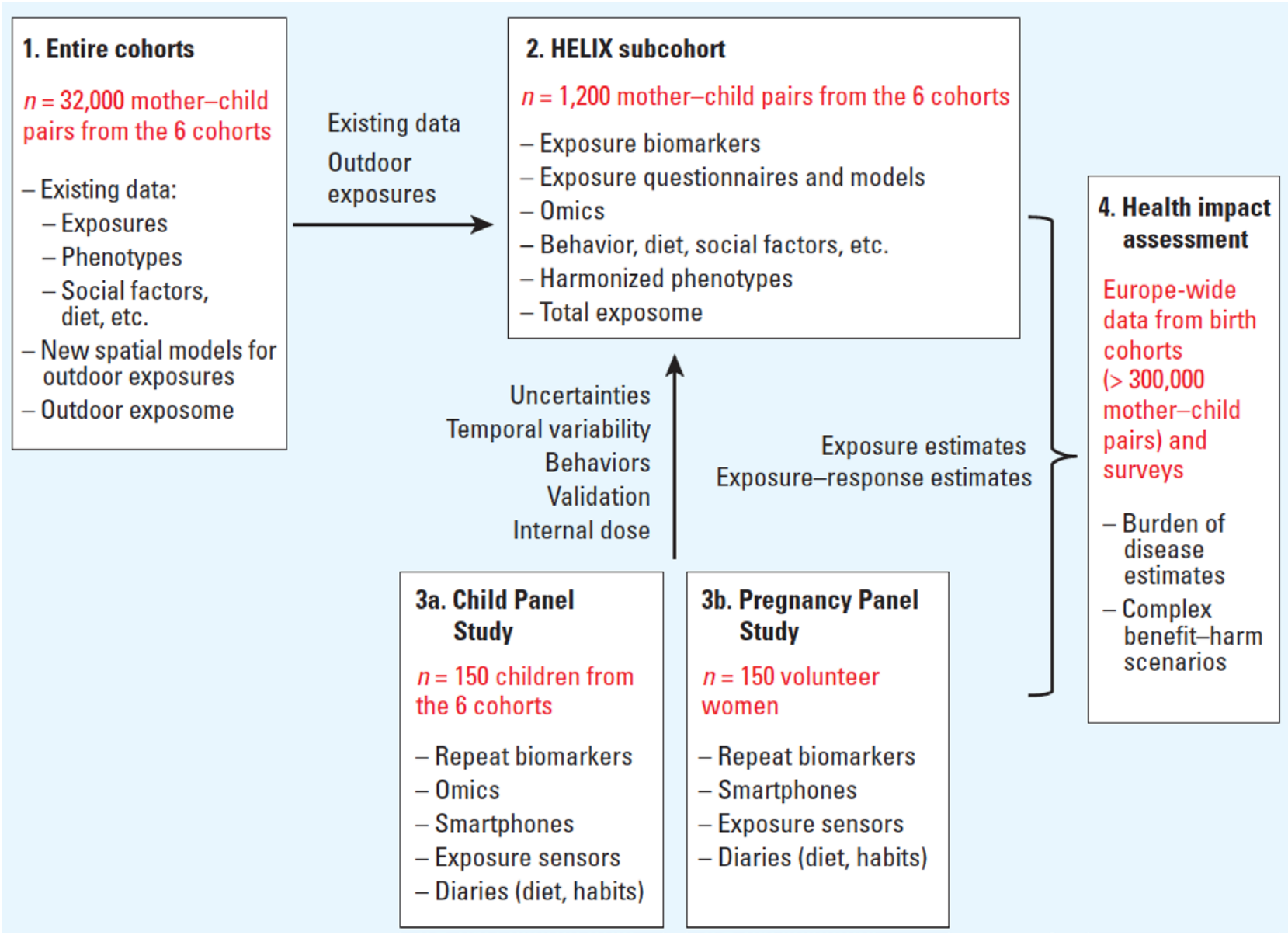


Helix Study – Outdoor Exposures

Vrijheid et al., EHP, 2014

Table 2. Outdoor exposures.

Exposure group	Entire cohort (n = 32,000), for pre- and postnatal exposure periods	Subcohort (n = 1,200)	Child Panel Study (1 week in 2 seasons) (n = 150)	Pregnancy Panel Study (1 week in 2 seasons) (n = 150)
Ambient air pollutants	LUR model for NO_2, $PM_{2.5}$, PM_{10}, PM_{coarse}, $PM_{2.5}$ absorbance, PM elemental analyses. Routine monitoring and OMI satellite data for temporal variability.	LUR model for NO_2, $PM_{2.5}$, PM_{10}, PM_{coarse}, $PM_{2.5}$ absorbance, PM elemental analyses. Routine monitoring and OMI satellite data for temporal variability.	Inhalation rates and mobility (GPS) data from smartphones. Personal monitoring (24 hr) of $PM_{2.5}$ (and black carbon.	Inhalation rates and mobility (GPS) data from smartphones. Personal monitoring (24 hr) of $PM_{2.5}$ and black carbon.
Noise	Existing municipal noise maps to obtain spatial estimates. Address-based modeling of noise at the most and least exposed facade.	New questionnaires in children on bedroom position, noise perception, etc. Noise estimates based on maps and questions.	Time–activity and mobility (GPS) data from smartphones.	Time–activity and mobility (GPS) data from smartphones.
UV	Remote sensing (satellite) UV radiation maps.	New questionnaires in children on traveling, use of sunscreens, clothes, skin color. UV radiation estimates based on maps and questions.	Time–activity and mobility (GPS) data from smartphones and questionnaires. Personal monitoring using electronic UV dosimeters.	Time–activity and mobility (GPS) data from smartphones and questionnaires. Personal monitoring using electronic UV dosimeters.
Temperature	Remote sensing (satellite) temperature maps (from thermal infrared band) and data from local meteorological stations.	New questionnaires in children on heating and air conditioning. Temperature estimates based on maps and questions.	Time–activity and mobility (GPS) data from smartphones and questionnaires. Personal monitoring of temperature using electronic dosimeters.	Time–activity and mobility (GPS) data from smartphones and questionnaires. Personal monitoring of temperature using electronic dosimeters.
Built environment/ green spaces	Normalized Difference Vegetation Index from satellite. Building density, walkability score, accessibility, bike lanes, etc., derived from GIS data.	New questionnaires in children on use of green spaces, public spaces, active transportation.	Time–activity and mobility (GPS) data from smartphones and questionnaires.	Time–activity and mobility (GPS) data from smartphones and questionnaires.

Abbreviations: GIS, geographic information system; GPS, global positioning system; LUR, land use regression; NO_2, nitrogen dioxide; NO_X, nitrous oxides; OMI, ozone monitoring instrument; $PM_{2.5}$, particles ≤ 2.5 μm in size; $PM_{2.5}$ absorbance, measurement of the blackness of $PM_{2.5}$ filters—a proxy for elemental carbon, which is the dominant light-absorbing substance; PM_{coarse}, particles between 2.5 and 10 μm in size; PM_{10}, particles ≤ 10 μm in size.

Helix Study – Individual Exposures

Table 1. Individual exposures.

Exposure group	Entire cohorts (n = 32,000)	HELIX subcohort (n = 1,200)	Child Panel Study (1 week in 2 seasons) (n = 150)	Pregnancy Panel Study (1 week in 2 seasons) (n = 150)
PCB-153, DDE, HCB, PBDE-47	—	Biomarkers: in stored pregnancy blood samples[a] and in newly collected child blood samples.	—	—
PFAS (PFOS, PFOA, PFBS, PFHxS, PFNA)	—	Biomarkers: in stored pregnancy blood samples[a] and in newly collected child blood samples. PBPK models for pregnancy and childhood.	—	
Metals (Hg, Pb, and TMS)	—	Biomarkers: in stored pregnancy samples[a] and in newly collected child samples: blood (Pb), urine (TMS), and hair (Hg).	—	—
Phthalates (13 metabolites)	—	Biomarkers: in stored pregnancy urine samples[b] and in newly collected child urine samples (last night and first morning void).	Biomarkers: in daily repeat urine samples. Daily data on diet, cosmetics. PBPK model for DEHP.	Biomarkers: in daily repeat urine samples. Daily data on diet, cosmetics. PBPK model for DEHP.
Phenols (BPA, parabens, TCS, BP3)		Biomarkers: in stored pregnancy urine samples[b] and in newly collected child urine samples (last night and first morning void).	Biomarkers: in daily repeat urine samples. Daily data on diet, cosmetics.	Biomarkers: in daily repeat urine samples over whole week. Daily data on diet, cosmetics.
OP pesticides	—	Biomarkers: in stored pregnancy urine samples[b] and in newly collected child urine samples (last night and first morning void).	Biomarkers: in daily repeat urine samples in two seasons. Daily data on diet and repellent use.	Biomarkers: in daily repeat urine samples in two seasons. Daily data on diet and repellent use.
Water DBPs	Estimates available from previous HiWATE project during and after pregnancy.	New questionnaire in children on water consumption and swimming combined with water company data.	Water consumption diaries.	Water consumption diaries.
Indoor air: BTEX, NO_2, $PM_{2.5}$	Existing questionnaire data on indoor sources during and after pregnancy.	New questionnaire in children on cooking, heating, cleaning, and ventilation.	Passive BTEX and NO_2 sampling in the home. Active $PM_{2.5}$ sampling. Questionnaire on cooking, heating, cleaning, and ventilation.	Passive BTEX and NO_2 sampling in the home. Active $PM_{2.5}$ sampling. Questionnaire on cooking, heating, cleaning, and ventilation.
ETS	Existing questionnaire and cotinine data during and after pregnancy.	New questionnaire in children. Biomarkers: cotinine measurement in newly collected child urine and/or hair samples.	Questionnaire on ETS.	Questionnaire on ETS.

Abbreviations: BP3, benzophenone-3; BPA, bisphenol A; BTEX, benzene, toluene, ethylbenzene, xylene; DBPs, disinfection by-products; DDE, dichlorodiphenyldichloroethylene; DEHP, di(2-ethylhexyl) phthalate; ETS, environmental tobacco smoke; HCB, hexachlorobenzene; Hg, mercury; NO_2, nitrogen dioxide; OP, organophospate pesticides; Pb, lead; PBDE-47, polybrominated diphenyl ether–47; PCB-153, polychlorinated biphenyl–153; PFAS, perfluoroalkyl substances; PFBS, perfluorobutanesulfonic acid; PFHxS, perfluorohexane sulfonic acid; PFNA, perfluorononanoic acid; PFOA, perfluorooctanoic acid; PFOS, perfluorooctane sulfonic acid; TCS, triclosan; TMS, total metal spectrum.
[a]Where measurements are available from previous studies, these will be used. [b]Pooling of ≥ 2 urine samples when available.

Helix Study – Omics Analyses

Vrijheid et al., EHP, 2014

Table 3. Omics analyses.[a]

Omics technique	Entire cohort (n = 32,000)	Subcohort (n = 1,200 mother–child pairs)	Child Panel Study (1 week in 2 seasons) (n = 150)[b]
Metabolomics	—	Untargeted ^{1}H NMR spectroscopy and semitargeted UPLC-MS analysis in urine; targeted analysis in serum (using Biocrates Absolute IDQ p180 Kit) in newly collected child samples.	Further analysis of daily urine samples and single serum sample at the end of each week (in winter and summer seasons) to evaluate sources of variation and short-term exposure–omics associations.
Proteomics	—	Targeted analysis in newly collected child plasma samples depending on results of analysis in the Child Panel Study.	Initial iTRAQ and MRM (or similar) analyses in plasma samples collected at end of each week (in winter and summer seasons) to evaluate sources of variation and short-term exposure–omics associations.
Transcriptomics	—	Next-generation sequencing (Illumina Hiseq2000) or microarray analysis of both mRNAs and miRNAs in newly collected child whole blood samples. In addition, plasma will be collected to analyze miRNAs in the future.	Analysis of blood samples at the end of each week (in winter and summer seasons) to evaluate sources of variation and short-term exposure–omics associations. In addition, plasma will be collected to analyze miRNAs in the future.
DNA methylation	—	Infinium Human Methylation 450 BeadChip for genome-wide methylation analysis of DNA extracted from newly collected child whole blood samples.	Analysis of blood samples at the end of each week (in winter and summer seasons) to evaluate sources of variation and short-term exposure–omics associations.

Abbreviations: ^{1}H NMR, proton nuclear magnetic resonance; iTRAQ, isobaric tags for relative and absolute quantitation; MRM, mass spectrometry–based multiple reaction monitoring; miRNA, microRNA; mRNA, messengerRNA; UPLC-MS, ultra performance liquid chromatography–mass spectrometry.

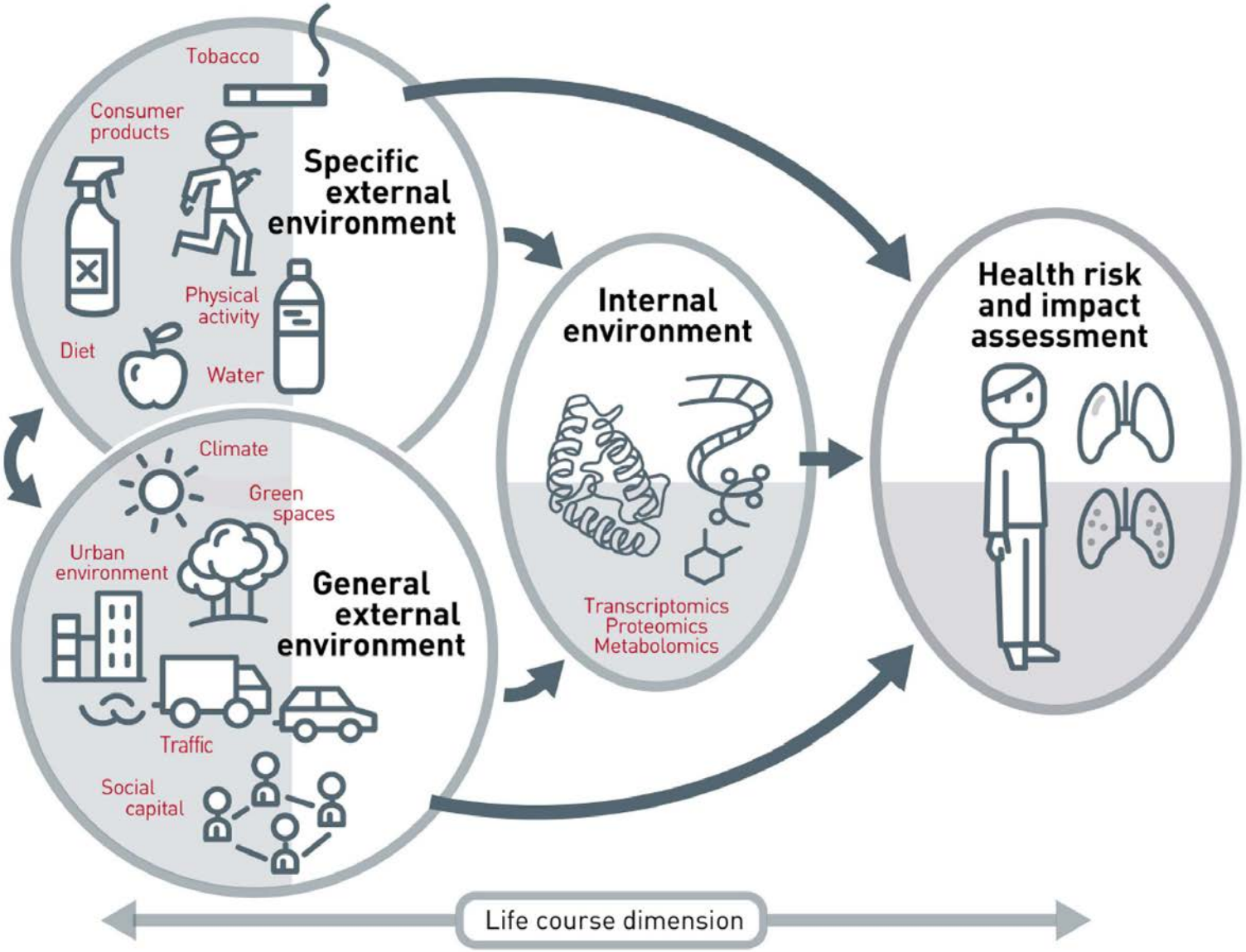
[a]Details of the techniques are described in Supplemental Material, Detailed description of omics techniques to be used in HELIX, pp. 4–6. [b]The Pregnancy Panel Study will collect biological samples similar to those of the Child Panel Study. Omics analyses are currently not foreseen in the pregnant women, but samples will be stored for future analysis, e.g., to evaluate whether specific omics findings from the children are replicated in the pregnant women.

“Omics”

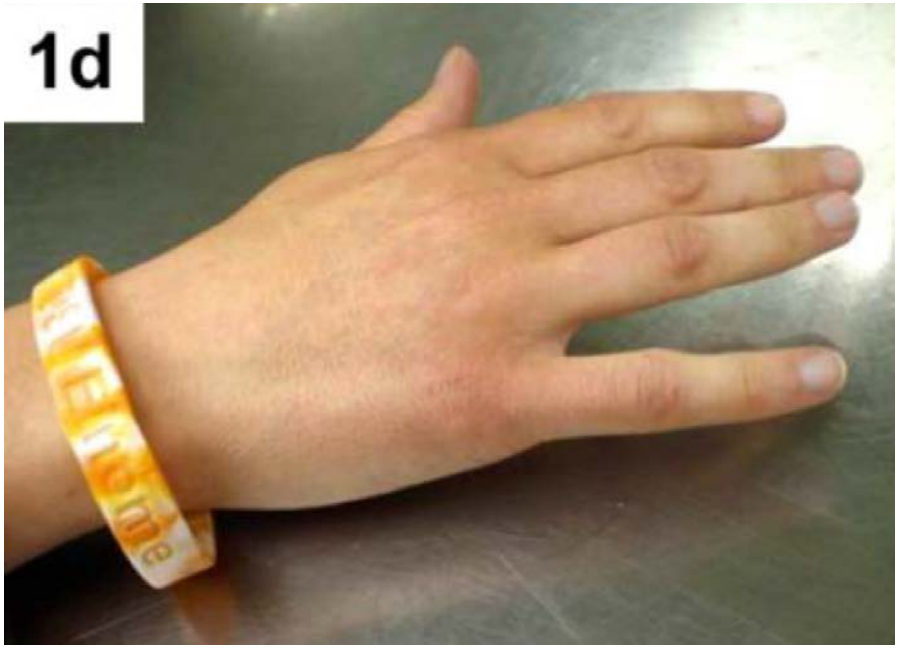
- Advantages
  - Biomarkers of exposure and effect
  - Indirectly assess all sources of exposure (chem, drugs, nutrient, etc.)
  - Unbiased
- Disadvantages
  - Short- versus long-term exposures
  - Expensive
  - Clarity of interpretation

The Exposome

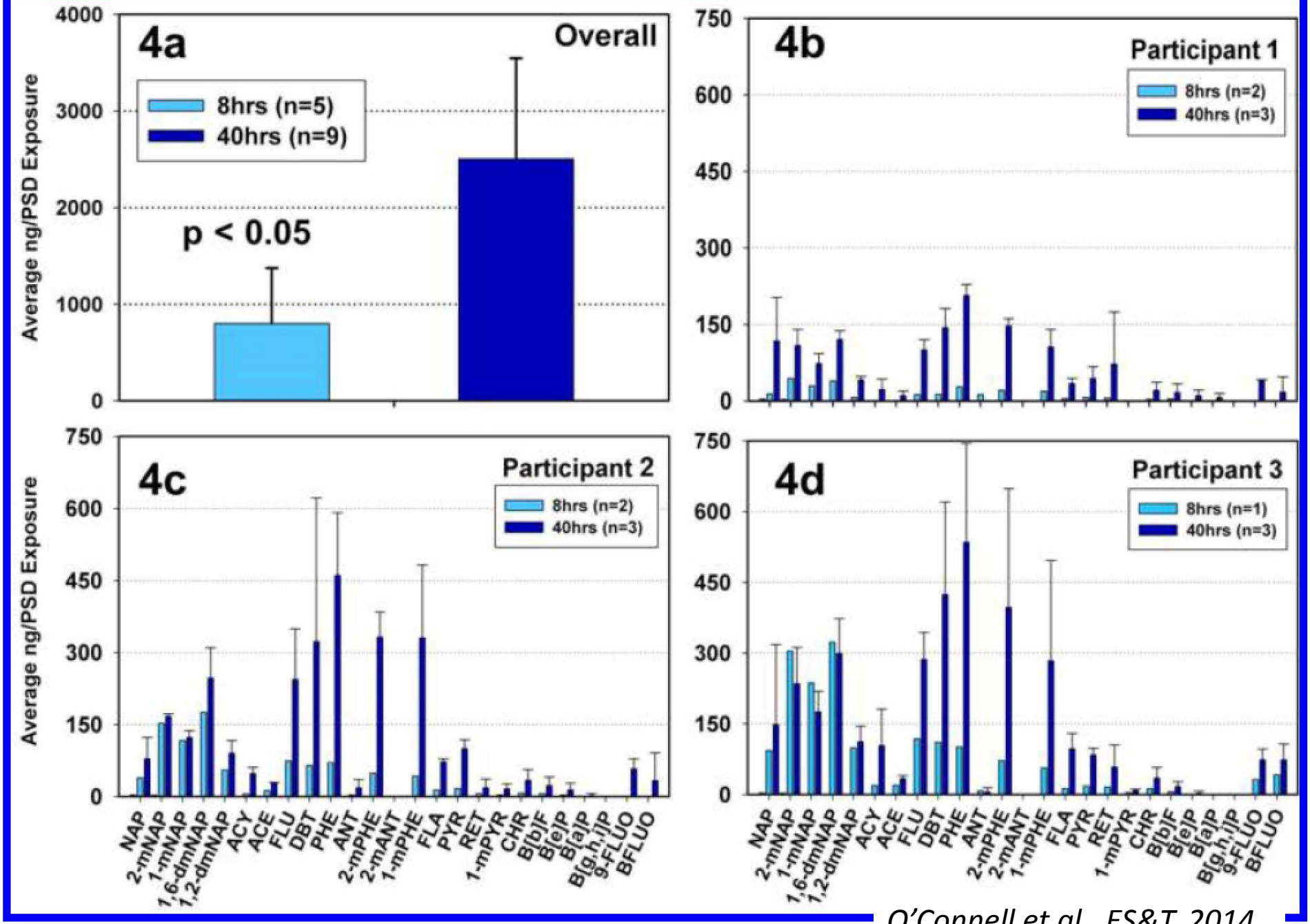
Vrijheid, BMJ, 2014



Silicone Wristbands



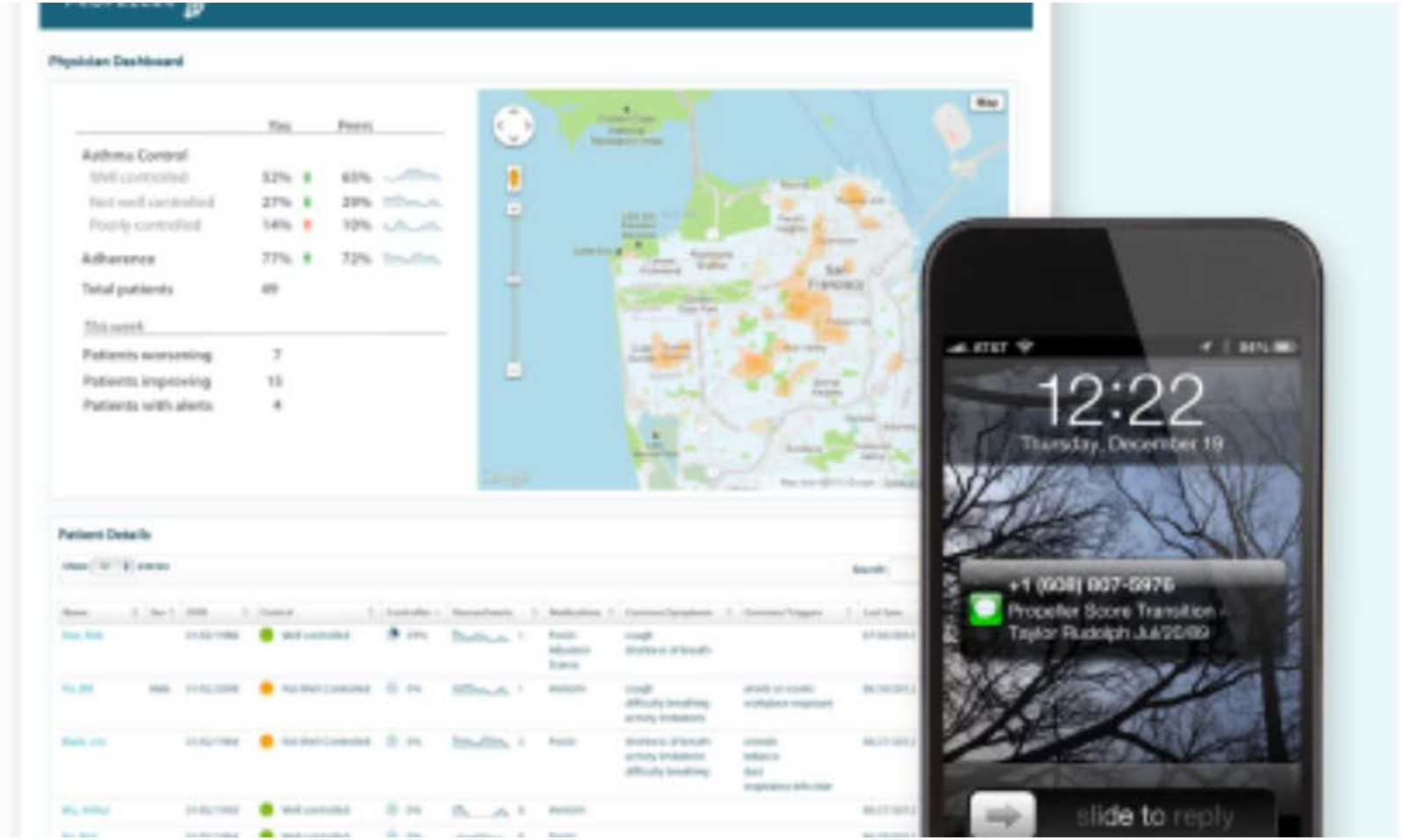
O'Connell et al., ES&T, 2014



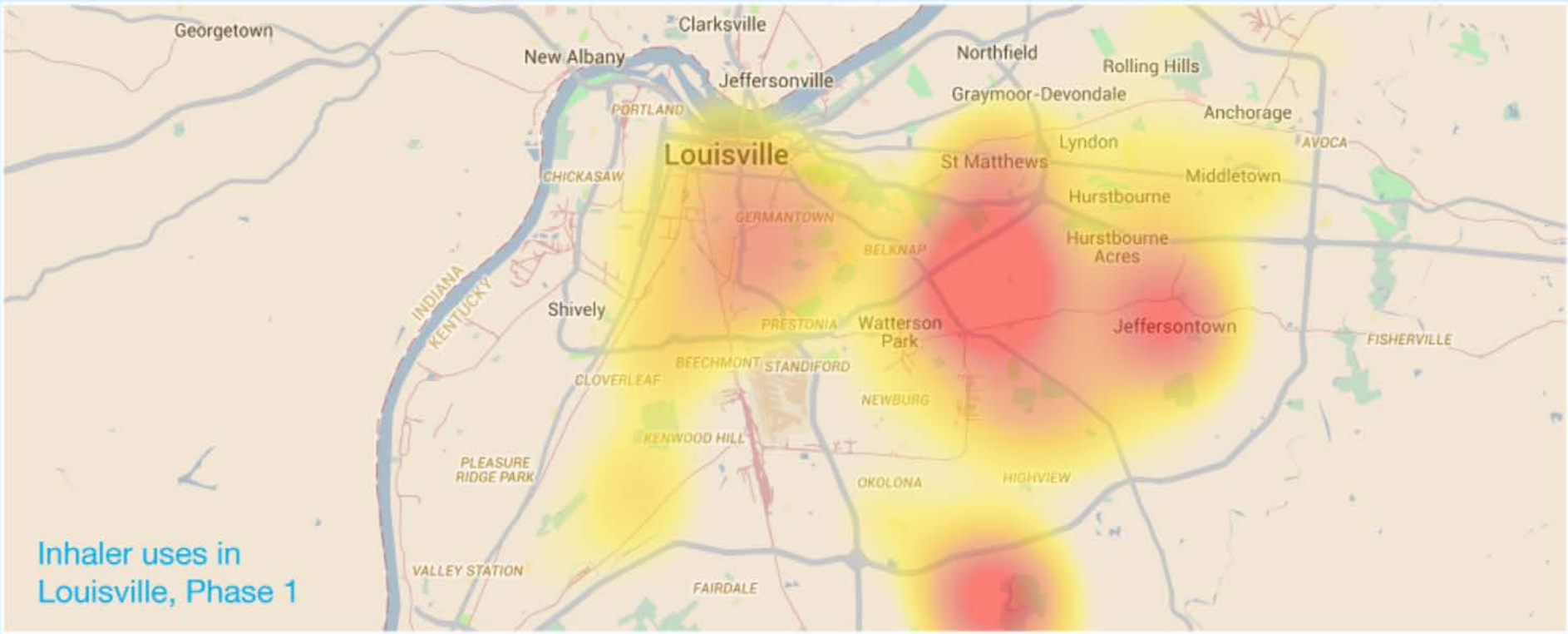
O'Connell et al., ES&T, 2014

Propeller Health Asthma/COPD Tracking

Apps and Feedback



Asthma Inhaler Tracking

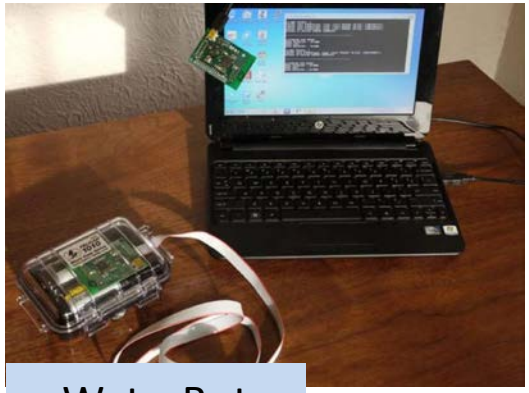


Crowd-Source Sensors

AirBot
particulates

Sensordrone
gasses

Lapka
Radiation, nitrates

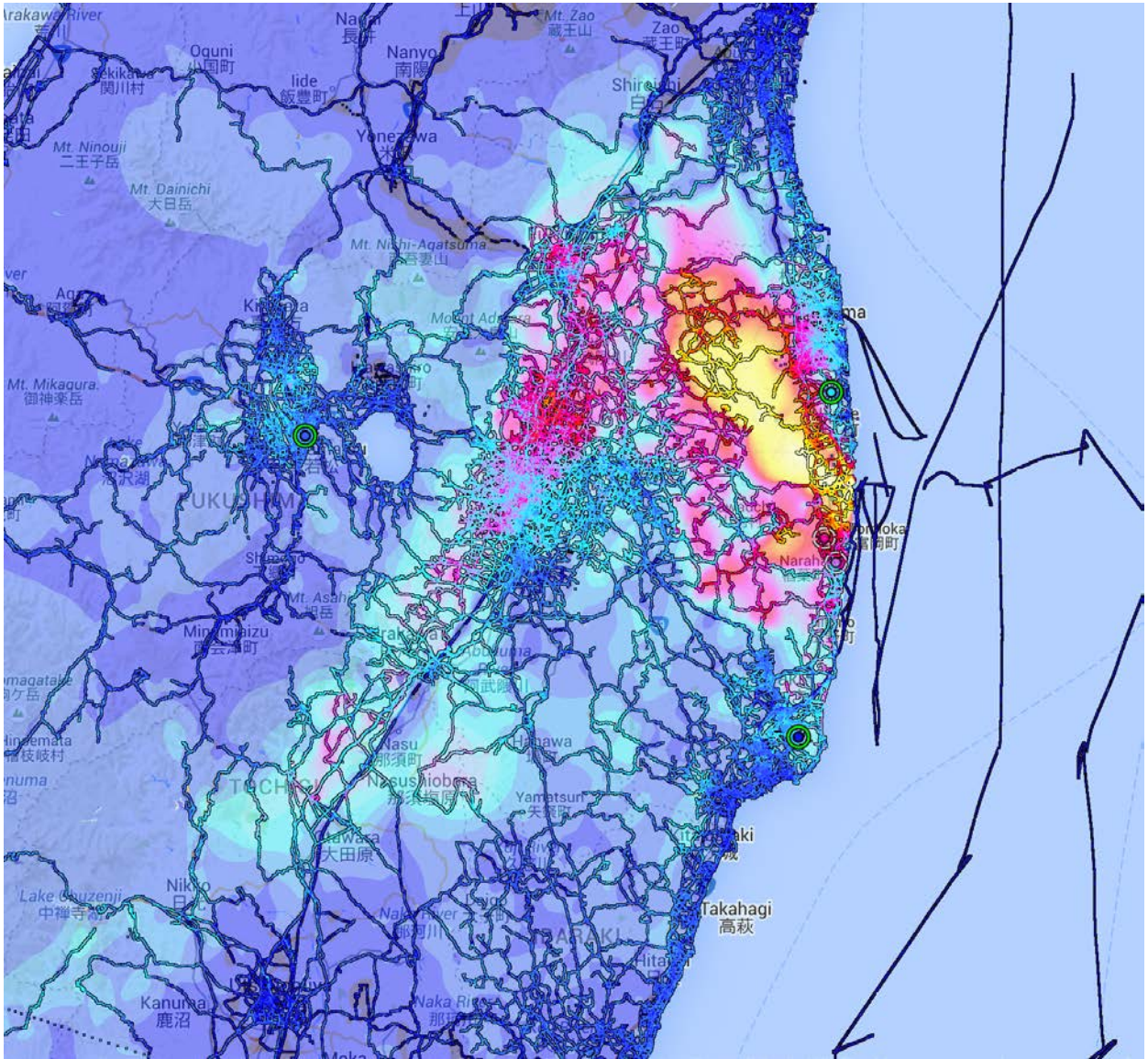


WaterBot
water quality

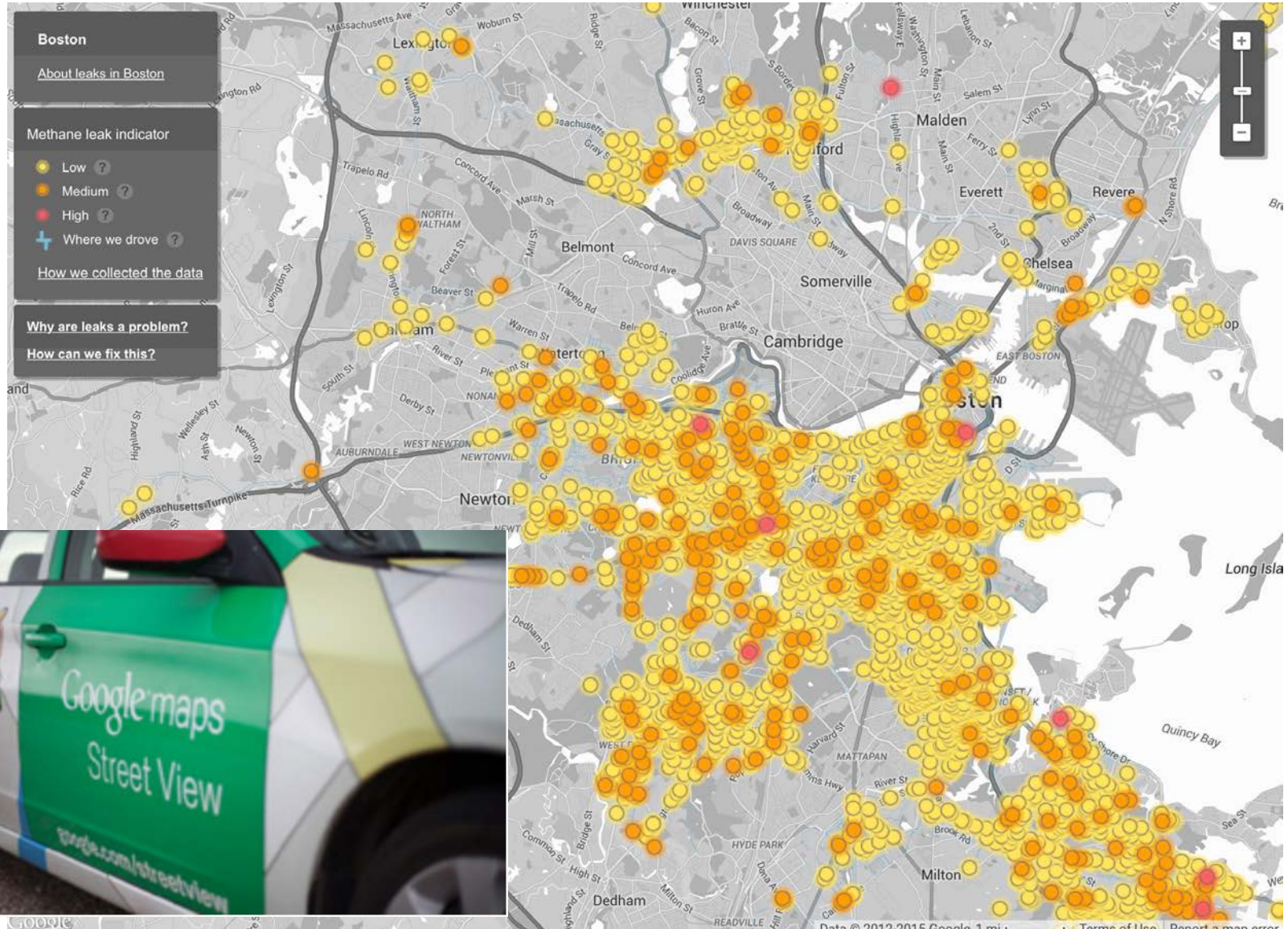
AirBeam
PM

Air Quality Egg
CO, NO

Safecast – Radiation Monitor



EDF and Google Mapping Methane



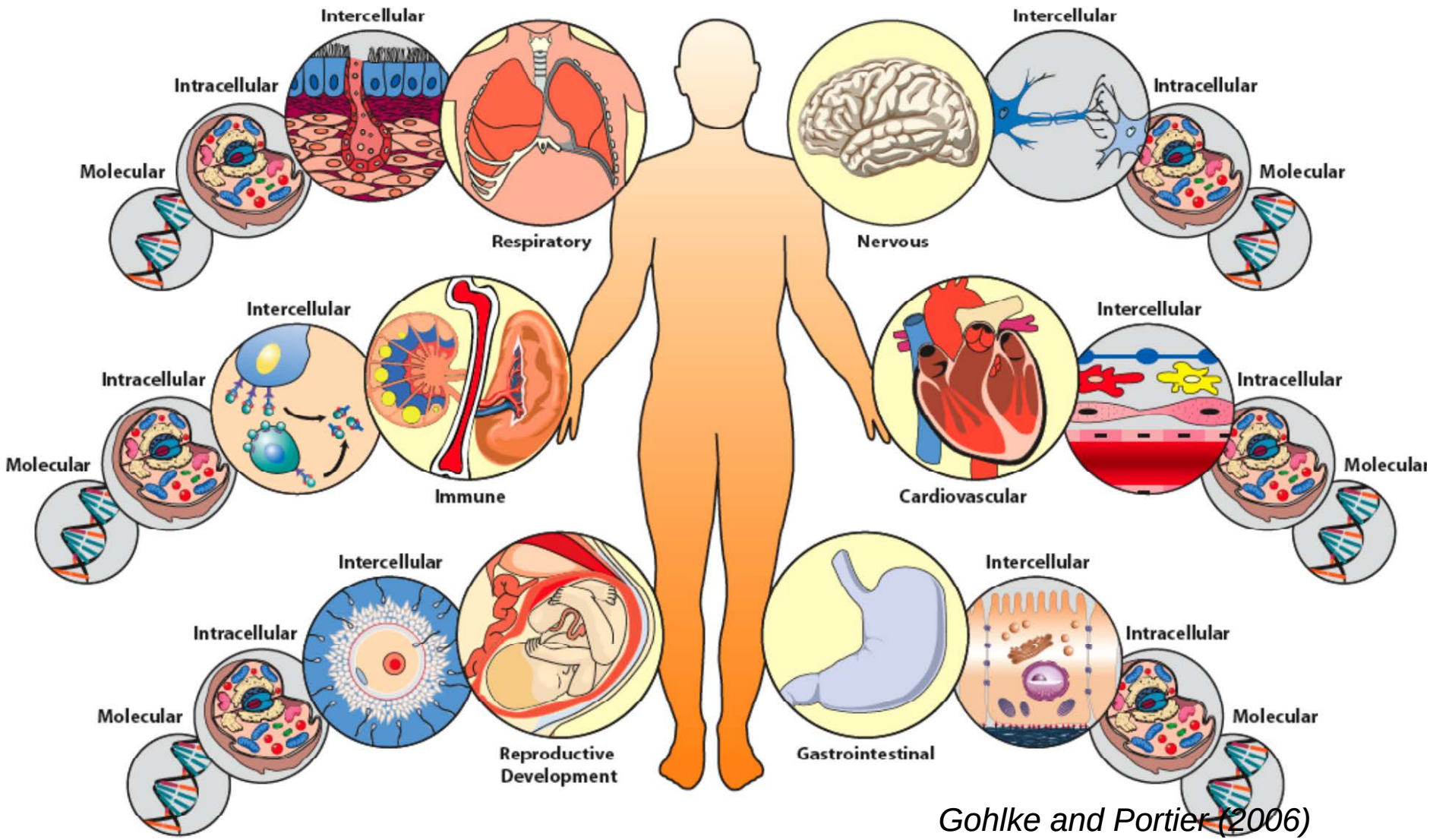
Sensor Revolution

- Advantages
  - Costs
  - External exposures can be measured over an extended timeframe
  - Crowd-sourcing
  - In a fixed network, greater density of coverage
  - As a personal monitor, direct measurement of individual contact with environment
  - As a mobile monitor, ability to map large areas, possibly with high quality instruments

Sensor Revolution

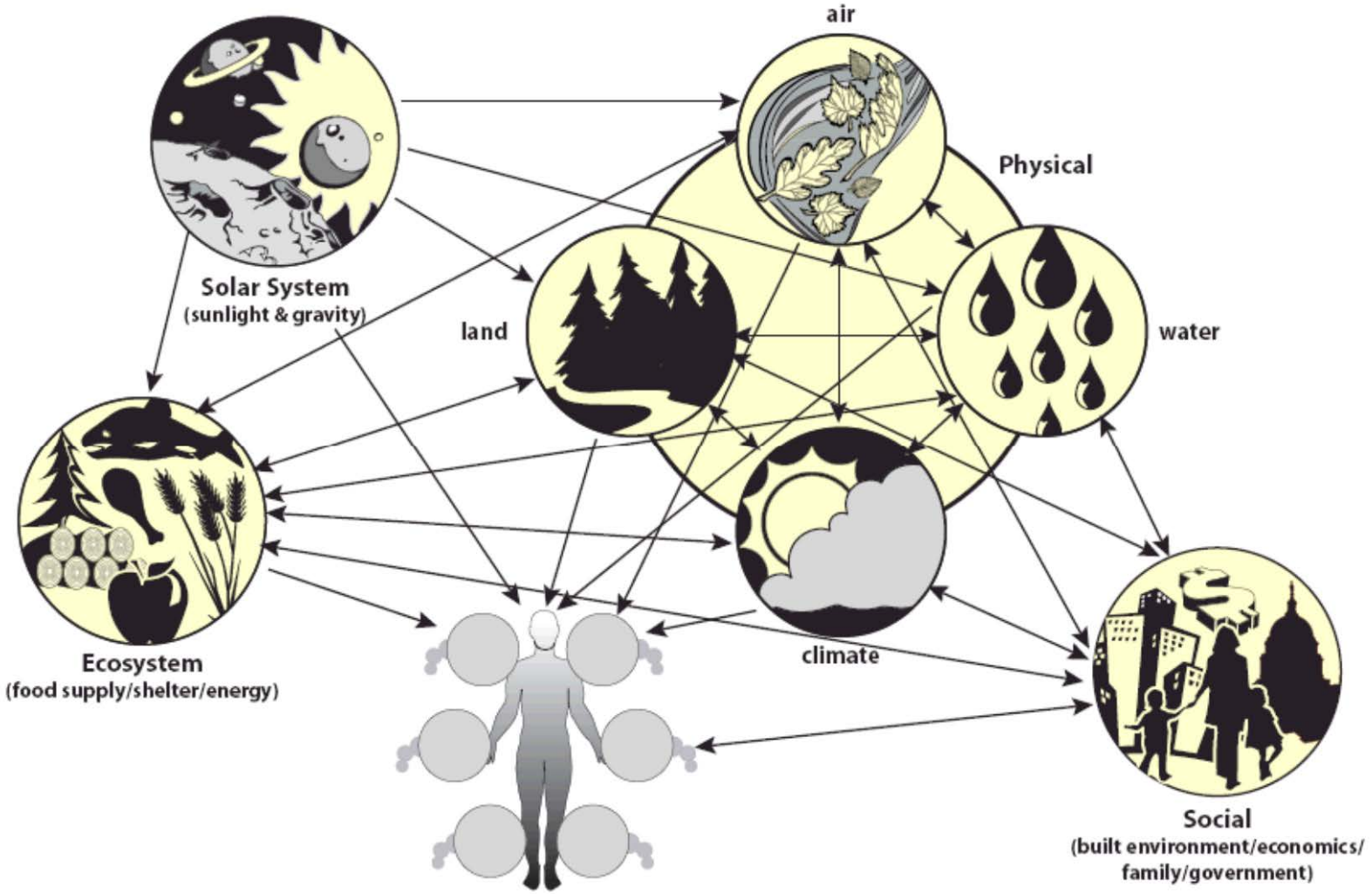
- Disadvantages
  - Limited number of exposures
  - Questionable reliability, accuracy, etc.
  - As a personal monitor, interpretation of actual exposure is difficult
  - As a mobile monitor, impossible to accurately correct for space-time variations

Systems Biology for the Individual



Gohlke and Portier (2006)

Interaction Network: Our Environment and Our Health



Gohlke and Portier (2007)

8:30 am – 10:00 am **Concurrent Sessions**

The Role of the Exposome in Predicting Disease

Room 124

Moderator: Sanwat Chaudhuri, PhD, Utah Public Health Laboratory

The Exposome — A Systems Approach for Discovery in Environmental Health
Yuxia Cui, PhD, National Institute of Environmental Health Sciences

The Exposome: Implications for Occupational Health
D. Gayle DeBord, PhD, Centers for Disease Control and Prevention

Developing Non-targeted Measurement Methods to Characterize the Human Exposome
Jon Sobus, PhD, U.S. Environmental Protection Agency