A Section 508–conformant HTML version of this article is available at https://doi.org/10.1289/EHP11760.

Effects of Behavioral, Clinical, and Policy Interventions in Reducing Human Exposure to Bisphenols and Phthalates: A Scoping Review

Nicole E. Sieck,[1] Meg Bruening,[2] Irene van Woerden,[3] Corrie Whisner,[4] and Devon C. Payne-Sturges[1]

[1]Maryland Institute for Applied Environmental Health, School of Public Health, University of Maryland, College Park, Maryland, USA
[2]Department of Nutritional Sciences, The Pennsylvania State University, University Park, Pennsylvania, USA
[3]Department of Community and Public Health, Idaho State University, Pocatello, Idaho, USA
[4]College of Health Solutions, Arizona State University, Phoenix, Arizona, USA

Background: There is growing interest in evidence-based interventions, programs, and policies to mitigate exposures to bisphenols and phthalates and in using implementation science frameworks to evaluate hypotheses regarding the importance of specific approaches to individual or household behavior change or institutions adopting interventions.

Objectives: This scoping review aimed to identify, categorize, and summarize the effects of behavioral, clinical, and policy interventions focused on exposure to the most widely used and studied bisphenols [bisphenol A (BPA), bisphenol S (BPS), and bisphenol F (BPF)] and phthalates with an implementation science lens.

Methods: A comprehensive search of all individual behavior, clinical, and policy interventions to reduce exposure to bisphenols and phthalates was conducted using PubMed, Web of Science, Cumulative Index to Nursing and Allied Health Literature (CINAHL), and Google Scholar. We included studies published between January 2000 and November 2022. Two reviewers screened references in CADIMA, then extracted data (population characteristics, intervention design, chemicals assessed, and outcomes) for studies meeting inclusion criteria for the present review.

Results: A total of 58 interventions met the inclusion criteria. We classified interventions as dietary ($n=27$), clinical ($n=13$), policy ($n=14$), and those falling outside of these three categories as "other" ($n=4$). Most interventions (81%, 47/58) demonstrated a decrease in exposure to bisphenols and/or phthalates, with policy level interventions having the largest magnitude of effect.

Discussion: Studies evaluating policy interventions that targeted the reduction of phthalates and BPA in goods and packaging showed widespread, long-term impact on decreasing exposure to bisphenols and phthalates. Clinical interventions removing bisphenol and phthalate materials from medical devices and equipment showed overall reductions in exposure biomarkers. Dietary interventions tended to lower exposure with the greatest magnitude of effect in trials where fresh foods were provided to participants. The lower exposure reductions observed in pragmatic nutrition education trials and the lack of diversity (sociodemographic backgrounds) present limitations for generalizability to all populations. https://doi.org/10.1289/EHP11760

Introduction

Bisphenols and phthalates are endocrine disrupting chemicals (EDCs)[1–3] associated with numerous adverse health outcomes, such as reproductive and developmental dysfunction,[4,5] metabolic disease,[6–9] neurobehavioral development,[10–14] and immune dysfunction,[15,16] most notably when exposure occurs during critical windows of susceptibility.[17,18] Bisphenol A (BPA) is used in epoxy resins for consumer and food product packaging,[19] such as canned foods,[20] in polycarbonate water bottles,[21] dental sealants,[22] and thermal receipts.[23] Humans are exposed to BPA via oral, inhalation, and dermal exposure, although oral exposure from dietary sources is the most common.[24–26] As restrictions on the use of BPA in consumer products have been put into place, the structural analogues bisphenol S (BPS) and bisphenol F (BPF) have emerged as two of the most widely used substitutes. BPS can be found in thermal paper[27] and to produce epoxy resin and polycarbonate plastic.[28,29] Likewise, BPF is also used in epoxy linings for canned food and other types of coatings.[29–31] Both BPS and BPF have been detected in personal care products[32] as well as in a variety of foods, including dairy products, seafood, meats, and cereal products.[33] Phthalates are highly abundant plastic additives used primarily as plasticizers to soften materials and make them flexible.[34,35] Human biomonitoring shows widespread exposure to phthalates[36,37] from a variety of sources including food due to contamination from its packaging as well as other food contact materials used in food processing.[38–40] Personal care products, building materials, and medical devices also contribute to human exposure to phthalates.[41,42]

Potential health effects and widespread exposure to bisphenols (especially BPA) and phthalates have prompted regulators (described below), researchers,[43] public health advocates,[44–46] and professional medical societies[47–49] to recommend that consumers take actions to reduce exposures. Risk evaluations have led to the restriction of some uses of specific phthalates and bisphenols in many countries. For example, in 2008, the United States Congress banned the use of di(2-ethylhexyl) phthalate (DEHP), butyl benzyl phthalate (BBzP), and dibutyl phthalate (DBP) in children's toys and childcare articles,[50] and in 2017, the US Consumer Protection Safety Commission[51] increased the list of prohibited phthalates to eight. BPA is prohibited from use in baby bottles in the US[52] and Canada.[53] Likewise, the EU has listed DEHP and DBP in its authorization list under Registration, Evaluation, Authorization, and Restriction of Chemicals (REACH), and more than a dozen phthalates are included in the candidate list for authorization.[54] BPA and BPF are listed on the candidate list of substances under REACH, which limits use of these chemicals in a variety of consumer products. These regulatory restrictions have been credited with decreases in exposure to these EDCs.[55,56] However, continued human exposure raises concerns about co-exposures to mixtures of phthalates and bisphenols, social disparities in these exposures, and cumulative health risk.[24,36,37,57–68] Meanwhile various groups continue to educate the public on individual actions they can take as consumers to avoid exposures to phthalates and BPA.[69–71]

Address correspondence to Devon C. Payne-Sturges, 4200 Valley Dr., Suite 2242, College Park, MD 20742-2611 USA. Email: dps1@umd.edu

Supplemental Material is available online (https://doi.org/10.1289/EHP11760).
The authors declare they have no conflicts of interest to disclose.
Conclusions and opinions are those of the individual authors and do not necessarily reflect the policies or views of EHP Publishing or the National Institute of Environmental Health Sciences.
Received 23 June 2022; Revised 9 December 2023; Accepted 29 January 2024; Published 13 March 2024; Corrected 16 April 2024.

Note to readers with disabilities: *EHP* strives to ensure that all journal content is accessible to all readers. However, some figures and Supplemental Material published in *EHP* articles may not conform to 508 standards due to the complexity of the information being presented. If you need assistance accessing journal content, please contact ehpsubmissions@niehs.nih.gov. Our staff will work with you to assess and meet your accessibility needs within 3 working days.

There is growing interest in using implementation science frameworks[72–74] to evaluate hypotheses regarding the importance of specific approaches to individual or household behavior change or adoption of an intervention by institutions. As an interdisciplinary team of environmental health and nutritional science researchers with expertise in behavioral and policy interventions, we contribute to these emerging discussions by conducting and reporting results from a scoping review of interventions to reduce exposure to the most widely used and studied bisphenols (BPA, BPS, BPF) and phthalates. In addition to behavioral interventions to reduce exposures to bisphenols and phthalates, we were also interested in the effects that policies have had on reducing exposure to these chemicals. To that end, our research question was, "How effective are behavioral, clinical, and policy interventions in reducing human exposure to BPA, BPS, BPF, and/or phthalates, as measured using biomarkers?" We also considered whether differences in effects were present dependent upon the intervention type and setting, as well as if there were differences in effects across the life course. Results will provide insights on the types of interventions that work as well as characteristics to consider when designing studies to identify methods and strategies that enable more widespread uptake into practice.

Methods

This scoping review followed the PRISMA extension for scoping reviews (PRISMA-ScR)[75] and JBI[76] guidelines.[75,76] We conducted a scoping review as we sought to systematically review the published literature to examine and synthesize the effectiveness of interventions across a variety of settings to inform future intervention strategies, including individual/family behaviors, clinical care, and policy change.[77,78]

Inclusion/Exclusion Criteria

The research question was developed using the PICO (population, intervention, comparator, outcome) framework.[79] Our PICO framework is shown in Table S1. Briefly, we included original research conducted in humans that included an intervention to examine changes in exposure to bisphenols and/or phthalates through a control group or repeated measures, along with measured biomarkers to assess for changes in exposure. We included studies published in 2000 or later, as this was when epidemiologic studies started being published around bisphenol and phthalate exposures, as well as studies investigating disparities in vulnerable populations. We excluded animal studies, studies that did not include biomarkers of exposure, conference abstracts, and studies not published in English. Additional details on our inclusion and exclusion criteria are shown in Table S2.

Search Strategy

We searched four databases: PubMed, Web of Science, Cumulative Index to Nursing and Allied Health Literature (CINAHL), and Google Scholar. Search terms were discussed by all authors using the PICO statement as a guide. We included search terms for the chemicals of interest, including abbreviations, e.g., BPA, and variations in spelling. Search terms for interventions, including terms for different types, e.g., crossover, behavioral, policy, etc., and variations in names of interventions, e.g., randomized clinical trial, randomized control trial, RCT, etc., were included to capture the range of intervention types included in the review. Biomarker-related terms, e.g., biological marker, urine, serum, etc., were included in all searches. In PubMed, Web of Science, and CINAHL, titles and abstracts were searched. The searches were conducted through November 2022. N.E.S. consulted with University of Maryland librarians prior to developing the search strategy. Database search strategies are listed in Table S3 and include the number of results per search for each time the searches were completed. Google Scholar searches were divided into three searches due to character limitations, and each individual search was stopped after N.E.S. did not observe any potentially relevant results for 10 pages of search results. All searches were filtered to include studies published in 2000 or later.

Title and Abstract Screening

Following initial duplicate article and animal study removal, references were uploaded to CADIMA (online tool), which was used to facilitate the review.[80] An additional duplicate check was carried out by CADIMA software prior to reviewing titles and abstracts.

For the title/abstract screening, all references were screened by two reviewers using the criteria in Table S2. N.E.S. screened 100% of references, while the other four team members evenly divided the second screen of each reference. If disagreements arose, the reference was discussed between N.E.S. and the second reviewer. If an agreement was not reached, other team members were consulted to resolve the disagreement.

Full Text Review

References not excluded at the title/abstract screening stage were then moved to full-text review and were independently screened by either N.E.S. or D.C.P-S. along with one additional team member. The detailed inclusion and exclusion criteria used at the full-text stage are shown in Table S4. If a discrepancy arose, the two team members discussed the issue(s), and any remaining discrepancies were then discussed among all authors so a consensus could be reached. We additionally cross-checked the reference lists of all included papers and related reviews[81–83] to ensure that all relevant studies were captured in our review. Any identified articles were entered into CADIMA and underwent the same process of screening using the selection criteria discussed above.

Data Extraction and Analysis

The data extracted included authors, publication year, journal, country of origin, year(s) of data collection, details on the study population [age(s), demographics], intervention characteristics (setting, design, duration, and type), the biomarker measured and if it was adjusted (e.g., for creatinine), statistical analyses, effect of the intervention including mean and median biomarker concentrations used to calculate percent change in biomarkers, and secondary outcomes assessed. When possible, we calculated and reported the percent change for urinary biomarker data that was adjusted for urinary dilution. References were organized by the chemical(s) included in the intervention and by the setting in which the intervention took place.

Results

A total of 14,495 records were identified. After duplicates were deleted ($n = 4{,}026$), 10,469 records were title- and abstract-screened. Of these, 152 full-text articles underwent full review, and 58 met the inclusion criteria to be included in the present scoping review (Figure S1). Of the 58 studies, 27 contained interventions for bisphenols only, 20 for phthalates only, and 11 included interventions for both bisphenols and phthalates. We classified interventions as dietary (including broader educational and lifestyle interventions; $n = 27$ publications from 25 unique studies) (Table 1), clinical exposures ($n = 13$ publications from 12 unique studies) (Table 2), behavioral interventions falling

Table 1. Characteristics of dietary intervention studies ($n = 27$).

Author(s) (year)	Study design	Participants	Intervention characteristics	Study duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
INTERVENTIONS WITH FOOD PROVIDED								
Peng et al. (2019)[84]	Randomized crossover	$n = 20$ (10 females); age range: 21–32 years	2 × 2 crossover, where groups consumed sandwiches made with fresh ingredients for one phase and sandwiches made with canned ingredients for the other phase; all participants were instructed to not consume coffee and food in canned or plastic containers during washout days	4 days (1 day washout before and between intervention arms)	Urinary BPA	Morning void after 10–12 hour overnight fast collected in the morning before intervention and at 2-, 4-, and 6-hours postintervention for both arms; unadjusted and creatinine-adjusted values presented	Urinary BPA concentrations were significantly lower after consuming fresh soup**	↓ 41–71% BPA concentration following fresh food consumption
Carwile et al. (2011)[85]	Randomized crossover	$n = 75$ (38 females); mean age	2 × 2 crossover, where groups consumed a 12-ounce serving of canned soup daily for one phase and a 12-ounce serving of fresh soup for the other phase; participants did not alter consumption of other foods	12 days; each phase last 5 days with 2-day washout period between	Urinary BPA	Spot samples collected between 1500 and 1800 hours on 4th and 5th days of each phase; corrected for specific gravity	Adjusted geometric mean concentration of BPA was significantly higher after canned soup compared to fresh soup phase***	↓ 95% BPA concentration after fresh soup phase
Rudel et al. (2011)[86] and Ackerman et al. (2014)[87]	Pre-post	$n = 5$ families (10 adults, 10 children); median age: adults: 40.5 years, children: 7 years	Families were provided with fresh foods prepared with little to no contact with plastic	8 days (2 days preintervention, 2 days intervention, 3 days postintervention)	Urinary BPA and urinary metabolites for DEHP, DBP, BBzP, DEP, and DMP	End of day spot samples collected on days 1, 2, 4, 5, 7, and 8; unadjusted and creatinine-adjusted values presented	Urinary levels of BPA and DEHP metabolites decreased significantly during the fresh food intervention*, then significantly increased postintervention	↓ 66% BPA; ↓ 53–56% DEHP metabolites; ↓ 12% BBzP metabolites; ↑ 23% DEP metabolites (pre- vs. during intervention)
Barrett et al. (2015)[88]	Pre-post	$n = 10$ (low-income pregnant women); mean age (SD): 26.4 (5.0) years	Mostly fresh and organic foods prepared using stainless steel cookware were provided to participants, who were also asked to not use water bottles	7 days (1 day preintervention, 3 days intervention, 3 days postintervention)	Urinary metabolites of DEHP, including ΣDEHP, as well as DBP, DEP, DiBP, BBzP, DnOP, DiDP, and DiNP	End of day spot samples collected on days 1, 4, and 7; specific-gravity metabolite concentrations reported	No significant change in DEHP or other phthalate metabolites; no consistent trend in metabolite levels was observed; participants noted dissatisfaction with the diet	No change in phthalate metabolites
Sathyanarayana et al. (2013)[89]	RCT	$n = 10$ families (19 adults and 21 children; $n = 5$ families per group); mean age: adults: not reported, children: 6 and 5 years in Arms 1 and 2, respectively	Arm 1: catered fresh and organic foods prepared without plastic provided to half of families; families instructed to use ceramic dishes and metal utensils; Arm 2: educational handouts on strategies to reduce exposure to BPA and phthalates provided to families	16 days (5 days preintervention, 5 days intervention, 6 days postintervention)	Urinary BPA and metabolites of DEHP, including ΣDEHP, DBP, DEP, and BBzP	Evening spot samples collected on day 5 (preintervention), days 10 and 11 (intervention days, which were combined), and day 16 (postintervention). Reported findings unadjusted for creatinine, which reportedly did not differ from adjusted	BPA* and DEHP metabolites*** significantly increased in Arm 1, which was related to contamination in the provided food; no significant changes in BPA or phthalate metabolites observed in Arm 2	↑ 2,377% ΣDEHP metabolites; ↑ 100% BPA concentration (due to contamination)

Table 1. (*Continued.*)

Author(s) (year)	Study design	Participants	Intervention characteristics	Study duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Sessa et al. (2021)[90]	Nonrandomized trial	$n=130$ (66 females); mean age (SD): intervention ($n=65$): 9.09 (0.8) years; control ($n=65$): 9.27 (0.8) years	Intervention group received one meal daily at school using a plastic-free service; no changes for the control group; parents/guardians of children in both groups did not microwave plastics during the study period	6 months	Urinary BPA	Morning void collected after overnight fast before the start of the intervention and at 3 and 6 months; unadjusted and creatinine-adjusted values presented	Significant reductions observed in adjusted and unadjusted urinary BPA concentrations in intervention group at 3 and 6 months*, while there was no significant change in the control group over the study period	↓ 9% BPA between control intervention group at 6 months
BEVERAGE CONTAINER INTERVENTIONS								
Carwile et al. (2009)[39]	Nonrandomized crossover	$n=77$ (36 female); median age: 19 years	Comparison of BPA levels when using stainless steel bottles for cold beverages and avoiding drinking water from polycarbonate water dispensers to exclusive use of consuming cold beverages from polycarbonate bottles	2 weeks (1 week using stainless steel bottle, 1 week using polycarbonate bottle)	Urinary BPA	Samples collected during the evening on 2 of the last 3 days of each week; creatinine-adjusted values reported	Compared to stainless steel bottles, use of polycarbonate bottle significantly increased urinary BPA concentrations***	↓ 20% BPA with stainless steel compared to polycarbonate bottle use
Li et al. (2013)[91]	Nonrandomized crossover	$n=12$ (6 females); age: all participants were 25 years of age	Comparison of BPA concentrations after using ceramic versus polycarbonate cups to drink boiled and bottled water	3 weeks (1st: use of ceramic cups to drink bottled water; 2nd: use of ceramic cups to drink water boiled in stainless steel; 3rd: use of poly-carbonate cups to drink boiled water)	Urinary BPA	Morning voids collected on days 8, 15, and 22; creatinine-adjusted values reported	Geometric mean concentrations of BPA were significantly lower when boiled water was consumed from ceramic cups compared to drinking bottled water in ceramic cups*; consumption of boiled water from polycarbonate cups significantly increased BPA concentrations*	↓ 27–51% BPA concentration with ceramic compared to polycarbonate cup use
Bae et al. (2015)[92]	Randomized crossover	$n=60$ (56 females); mean age (SD): 73.1 (4.2) years	Participants consumed two soymilk beverages per visit from glass bottles, cans, or a combination of a glass and canned beverage	Varied somewhat by participant, 3 visits with ≥1 week interval between visits	Urinary BPA	Samples collected 2 hours after consumption of soy milk; unadjusted and creatinine-adjusted values reported	BPA concentrations were significantly lower after consuming 2 beverages from glass containers comparted to 2 canned and 1 glass and 1 canned beverage ***	↓ 94% BPA when two beverages were consumed from glass compared to 2 from cans
Sakaki et al. (2022)[93]	Randomized crossover	$n=30$ (22 females); mean age (SD): 24.8 (5.5) years	Comparison of exposure biomarkers after consuming capsule coffee compared to French press coffee from ceramic mug; participants instructed to avoid all other coffee	15 days (7 days run-in, 1 day intervention, 6 days washout, 1 day intervention)	Urinary BPA, BPS, BPF, DBP, and DEHP metabolites	Samples collected at three time points (prior to coffee consumption and at 6 and 18 hours postconsumption); did not specify metabolites measured or correction for urinary dilution	No association between the type of coffee consumed and biomarker levels; BPS was not detected in any sample; BPF and DBP were each detected in only one sample after consuming French press; BPA and DEHP metabolites detected in multiple samples but there did not appear to be any pattern	No change in bisphenol concentration or phthalate metabolites

Table 1. (*Continued.*)

Author(s) (year)	Study design	Participants	Intervention characteristics	Study duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
EDUCATIONAL AND LIFESTYLE INTERVENTIONS								
Ji et al. (2010)[94]	Pre-post	$n = 25$ (9 females); mean age (SD): 37.7 (13.3) years	Participants instructed to follow a vegetarian diet; no information provided about plastic packaging use	5 days	Urinary metabolites of DEP, DnBP, DiBP, and DEHP	Spot samples collected within 1 hour of start of intervention and within 2 hours of program completion; unadjusted and creatinine-adjusted values presented	Creatinine-adjusted metabolites of DEP, DnBP, DiBP, and select DEHP levels in urine samples significantly decreased during the program*	↓ 61–90% DEP; ↓ 63–73% DnBP; ↓ 60–71% DiBP; ↓ 48–56% select DEHP metabolites
Correia-Sá et al. (2017)[96] and Correia-Sá et al. (2018)[95]	Nonrandomized trial	$n = 112$ [57 females; $n = 69$ in healthy diet group (2 samples dropped from BPA analysis due to contamination concerns), $n = 43$ in regular diet group]; mean age (SD): 10.4 (3.3) years; range: 4–18 years	Participants assigned to groups based on BMI, with those considered overweight/obese assigned to the healthy diet group and those considered normal weight/underweight assigned to maintaining their regular diet; the healthy diet group was assigned to a nutritionist, who counseled on balanced nutrition and consuming less packaged and processed foods	3 months	Urinary BPA and metabolites of DMP, DEP, BBzP, DCHP, DnPEP, DiBP, DnBP, DEHP, DiNP, DiDP, DnOP, including ΣDiBP, ΣDEHP, ΣDiNP, ΣDiDP	First morning urine samples collected after 3 months of the intervention; two samples dropped from BPA analyses due to possible contamination; unadjusted and creatinine-adjusted values presented	There were no significant differences in BPA concentrations between the two groups, although BPA concentrations were ∼ 18% lower in the healthy diet group; median concentrations of phthalates were non-significantly lower in the healthy diet group (15–33%), except DEP, with only ΣDEHP reaching statistical significance**	↓ 28% ΣDEHP metabolites in healthy diet group
Dallio et al. (2018)[97]	Pre-post	$n = 120$ (29 females in group with NAFLD ($n = 60$); 32 females in healthy control group); mean age (SD): NAFLD: 57 (11) years, control: 54 (13) years	Participants provided guidance on following a BPA-free diet, including avoiding canned beverages and food, choosing glass containers instead of plastic, avoiding plastics numbered 3 and 7, and limiting microwave cooking, especially with plastic	1 month	Plasmatic and urinary BPA	Blood and 24-hour voids collected before and after intervention; creatinine-adjusted values presented	There was a significant reduction in BPA plasma levels* but not in urinary BPA levels in NAFLD participants; one participant reported not following the BPA-free dietary guidelines properly	↓ 62% plasmatic BPA; no change in urinary BPA concentrations
Galloway et al. (2018)[98]	Pre-post	$n = 94$ (53 female); age range: 17–19 years	Participants asked to minimize their intake of processed and packaged foods to reduce exposure to BPA	7 days	Urinary BPA	One morning void collected before start of intervention, second spot sample collected around same time of day 7 days later; unadjusted and creatine-adjusted values presented	No significant change in BPA, but those with the highest BPA concentrations at the start of the trial were more likely to have a decrease in BPA**; participants reported that it would be difficult to follow diet long-term	No change in BPA concentrations

Table 1. (*Continued.*)

Author(s) (year)	Study design	Participants	Intervention characteristics	Study duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Hutter et al. (2016)[99]	Case study	$n = 1$ family; ages: mother: 38, father: 44, daughter: 10, sons: 7 and 13 years	Family actively took steps to reduce their exposure to plastics through lifestyle changes, by replacing household items with nonplastic alternatives, avoiding foods packaged in plastic, and more frequent wet mopping	~3 months	Urinary BPA, metabolites of DEHP, DEP, BBzP, DiBP, DnBP, DnOP, DnPeP, DCHP, and DiDP	Morning voids collected 15 days after family started the transitions and 2 months later; creatinine-adjusted values presented	BPA was detected in only the father's first sample; most phthalate metabolites were detectable but there were differences in detectable phthalates between family members, and there was not a specific pattern of increases or decreases over the case study period	↓ 150% DEHP, DnBP, DiBP, DiNP, BBzP metabolites compared to German Human-Biomonitoring Commission reference values
Chen et al. (2015)[100]	Pre-post	$n = 30$ (all female and previously had been exposed to high levels of phthalates), median age (range): 9.7 (4–13) years	Multifaceted intervention strategies developed, including: frequent handwashing, avoidance of cosmetics and personal care products, nonprescribed medications, plastic containers and food packaging, microwaved food, and nutrition supplements	1 week	Urinary metabolites of DEHP, BBzP, DEP, DMP, and DPB	First morning voids collected pre- and postintervention; unadjusted and creatinine-adjusted values are presented	23 participants were compliant; measured phthalate metabolites were significantly lower postintervention for the compliant group***; limiting plastic cup use lowered metabolites of DBP and DEHP; increased handwashing reduced DMP and DMBP metabolites; low shower gel and shampoo reduced DEP and DBP metabolites, respectively, avoiding fragrances reduced DBP and DEP metabolites	↓ 31–72% depending on phthalate metabolite in compliant group
Kim et al. (2020)[101]	Pre-post	$n = 37$ families (20 mothers with 1 child, 15 mothers with 2 children, and 2 mothers with 3 children); age range: mothers: 30–40 years ($n = 16$), 40–50 years ($n = 21$); children: 4–6 years ($n = 36$), 7–12 years ($n = 20$)	Participants asked to avoid canned, instant, and delivery food as well as plastic containers, notably those labeled #7	8 days (2 days preintervention, 3 days intervention, 3 days postintervention)	Urinary BPA and BPS	Evening spot samples collected on days 1 and 2 (preintervention), days 4 and 5 (intervention), and 7 and 8 (postintervention); controlled for specific gravity	BPA concentrations were significantly reduced in both mothers and children**; BPS concentrations significantly decreased in mothers* but not in children	Mothers: ↓ 53.1% BPA, ↓ 63.9% BPS; Children: ↓ 47.5% BPA, ↓ 20% BPA
Park and Chung (2021)[102]	Pre-post	$n = 30$ (females with menstrual pain); mean age (SD): 22.1 (1.5) years	Three component intervention to decrease fast and processed food consumption: (1) small-group education, (2) follow-up monitoring, and (3) peer support via social network communication	4 weeks intervention (3 menstrual cycles, maximum of 22 weeks for participants with 6-week cycles)	Urinary BPA	First morning midstream voids collected at baseline and three additional times postintervention, measured at intervals of 4–6 weeks, depending on length of menstrual cycle; creatinine-adjusted values presented	BPA concentrations were significantly reduced at first and second*, but not third, menstrual cycle; BPA concentrations were not associated with dietary intervention compliance (43.3% of participants were considered to have high compliance)	↓ 59% BPA between baseline and first menstrual cycle; ↓ 27% BPA between baseline and third menstrual cycle

Table 1. (*Continued.*)

Author(s) (year)	Study design	Participants	Intervention characteristics	Study duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Wu et al. (2021)[103]	Pre-post	$n = 35$ (pregnant women, 27 completed the intervention)	Intervention consisted of three components: nutrition and diet (e.g., avoiding fast food), lifestyle changes (e.g., restricting personal care product use), and environment (e.g., minimizing transportation by car); participants also educated on negative health effects of phthalate exposure	8 months (beginning before 8 weeks gestation until 9 months gestation)	Urinary metabolites of DMP, DEP, DnBP, DnOP, BBzP, and DEHP, including ΣDEHP	Monthly spot samples collected at antenatal appointments; adjusted for specific gravity	All urinary concentrations of the phthalate metabolites decreased* except for BBzP and one DEHP metabolite	↓ 40% sum of molar concentration of all metabolites
Rutkowska et al. (2020)[104]	Pre-post	$n = 26$ (12 females); mean age not reported	Participants received information about reducing exposure to bisphenols and phthalates and were provided healthier alternatives to household cleaning products, cosmetics, food packaging, etc.	~6 months	Urinary BPA, BPS and metabolites of DEP, DiBP, and DEHP	Midstream morning void collected in a glass jar with metal lid; did not control urinary dilution	Urinary concentrations significantly decreased for all bisphenols and phthalate metabolites except DiBP	↓ ~15–35% in bisphenol concentrations and phthalate metabolites
Pirard and Charlier (2022)[105]	Pre-post	$n = 92$ (49 females); mean age (SD): 49.0 (10.2) years	Education campaign combined public conferences on endocrine disrupting chemicals, health effects, and general information on how to reduce exposures coupled with individual reports of biomonitoring results	2 years	Urinary BPA, BPS, BPF, and phthalate metabolites for DEHP, DEP, and BBzP	Spot samples collected between February and May 2015 and June and September 2018; adjusted for creatinine, but only unadjusted values are presented	BPS** and BPF*** concentrations significantly increased over time, but remained lower than BPA concentrations; BPA concentrations decreased nonsignificantly; phthalate metabolites for DnBP* and BBzP** decreased significantly over time, 2/3 DEHP metabolites* decreased, and DEP decreased nonsignificantly	↓ 37% DEHP metabolites; ↓ 42% BBzP metabolites
Kim et al. (2021)[106]	RCT	$n = 51$ (mothers of young children; $n = 26$ in intervention group, $n = 25$ in control group); mean age (SD): intervention: 35.8 (3.9) years, control: 35.1 (2.9) years	Intervention group received interactive, web-based program on limiting exposure to bisphenols and phthalates through diet, personal care products, and heath behaviors (10 criteria); the control group received information about endocrine disruptors in the mail, including methods of preventing exposure	~6 weeks	Urinary BPA and metabolites of DEHP	Samples collected at baseline, at 1 week (intervention), and at 1 month (postintervention); presented values adjusted for creatine; samples considered very dilute or concentrated were excluded from the analysis	Concentrations of BPA and DEHP metabolites decreased significantly in the intervention group but not in the control group*	↓ 54% BPA concentration in intervention group; ↓ 11–22% DEHP metabolites in intervention group

Table 1. (*Continued.*)

Author(s) (year)	Study design	Participants	Intervention characteristics	Study duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Hagobian et al. (2017)[107]	RCT	$n = 24$ (all female); mean age (SD): 20.9 (1.5) years	Intervention group provided with BPA-free products (cosmetics, hygiene products, and food/beverage containers), had weekly in-person meetings on negative health consequences of and how to avoid BPA, and received a weekly email with general information about BPA, healthy eating, and physical activity; control group received the weekly email only	3 weeks	Urinary BPA	Samples collected midstream after fast at study entry and at the end of the intervention; adjusted for creatinine	BPA concentrations significantly decreased in the treatment group and significantly increased in the control group*	↓ 45% BPA concentrations in intervention group
Hagobian et al. (2021)[108]	RCT	$n = 30$ (premenopausal women with obesity, $n = 15$ in both intervention and control groups); mean age (SD): intervention: 21.5 (3.3) years, control: 21.5 (3.1) years	Behavioral intervention that encouraged organic food consumption, avoidance of foods packaged in plastic or canned, storage of food in glass (containers provided), and replacement personal care products were provided for study duration	3 weeks	Urinary BPA, BPS, and BPF	Samples were collected midstream after an overnight fast at study entry and after 3 weeks at study completion; adjusted for creatinine	BPA and BPF concentrations did not vary between groups at the end of the trial; BPS concentrations were higher in the intervention group at the start of the trial, but had significantly decreased by the end of the trial **	↓ 55% BPS concentrations in intervention group; no change in BPA and BPF concentrations
El Ouazzani et al. (2022)[109]	RCT	$n = 230$ ($n = 78$ control group; $n = 152$ intervention group; part of PREVED cohort of pregnant women); mean age (SD): intervention: 32.8 (4.0) years, control: 33.1 (4.3) years	Environmental health workshops provided on nutrition, personal care product use, and indoor air quality; participants had to participate in at least 2 of the workshops to be included in analyses	~16 months	Urinary and colostrum concentration of BPA	Samples collected at study entry (first trimester), 2 month later, at childbirth, and 14 months postchildbirth; authors did not specify time of day when collection occurred; creatinine-adjusted values presented for urinary measures; colostrum collected at childbirth	There were no significant differences between BPA concentrations in either urine or colostrum between the intervention and control groups	No change in BPA concentrations

Note: BBzP, butyl benzyl phthalate; BMI, body mass index; BPA, bisphenol A; BPF, bisphenol F; BPS, bisphenol S; DBP, dibutyl phthalate; DCHP, di-cyclohexyl phthalate; DEHP, di(2-ethylhexyl) phthalate; ΣDEHP, molar sum of DEHP metabolites; DEP, diethyl phthalate; DiBP, di-isobutyl phthalate; ΣDiBP, molar sum of DiBP; DiDP, di-isodecyl phthalate; ΣDiDP, molar sum of DiDP; DiNP, di-isononyl phthalate; ΣDiNP, molar sum of DiNP; DMP, dimethyl phthalate; DnBP, di-n-butyl phthalate; DnOP, di-n-octyl phthalate; DnPeP, di-n-pentyl phthalate; NAFLD, nonalcoholic fatty liver disease; PCP, personal care product; RCT, randomized controlled trial; SD, standard deviation. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table 2. Interventions of medical exposures ($n = 13$).

Author(s) (year)	Study design	Participants	Intervention characteristics	Duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Zimmerman-Downs et al. (2010)[112]	RCT	$n = 30$ (16 females); age range: 18–40 years	Participants divided to receive either a high dose (four occlusal sealants) or a low dose (one occlusal sealant) of BPA	<1 day	Salivary and serum BPA	Saliva and blood samples collected 1 hour prior to sealant placement and 1, 4, and 24 hours postplacement	Salivary concentrations peaked at 4 hours postplacement and were significantly higher in the high-dose group at 1 and 4 hours** but not 24 hours, postplacement; BPA was not detected in serum for either group	↓ ~63% BPA in low-dose group
Seifi et al. (2022)[122]	Group randomized trial	$n = 40$ (18 females); mean age (range): females: 21.1 (13–35) years, males: 20 (12–33) years	Comparison of two debonding methods, ultrasonic and rotary using tungsten carbide bur, to determine if one of these methods results in a lower release of BPA into a patient's saliva	1 day	Salivary BPA	Saliva samples were collected immediately after adhesive removal	Mean BPA concentration in the saliva was significantly lower in the group that received the rotary debonding method***	↓ 64% BPA after rotary debonding
Bosch-Panadero et al. (2016)[114]	Nonrandomized crossover	$n = 69$ (hemodialysis patients, sex distribution not reported); mean age (SD): hemodialysis patients: 65 (13) years	BPA concentrations compared within patients using a BPA-free (polynephron) and BPA-containing (polysulfone) dialyzer; assigned dialyzer that was different from what was used prior to the intervention	6 months (3 months per dialyzer)	Serum and intracellular BPA	Blood samples collected at baseline, 3 months, and 6 months	Use of the BPA-containing dialyzer increased serum and intracellular BPA*, while use of the BPA-free dialyzer decreased serum and intracellular BPA*	↓ 33% BPA with BPA-free dialyzer over 3 months
Mas et al. (2018)[118]	Randomized crossover	$n = 60$ (hemodialysis patients, participant sex not provided); mean age not provided [subset of this sample ($n = 18$) also participated in Bosch-Panadero et al. (2016)[114]]	Comparison of BPA concentrations when using a BPA-free (polynephron) dialyzer to BPA-containing (polysulfone) dialyzer for online hemodiafiltration; noted tubing made from polyvinyl chloride	~9 months (≥6 months using one dialyzer then switched to other type of dialyzer for 3 months)	Plasmatic BPA	Fasting blood samples collected prior to midweek dialysis session; one pre- and post-session blood sample was collected to assess changes within a session for each type of dialyzer	Plasma BPA lower using online hemodiafiltration compared to conventional hemodialysis; mean BPA levels were significantly lower for patients using polynephron compared to polysulfone dialyzer regardless of crossover order**	↓ 51–62% BPA when switched from BPA-containing to BPA-free dialyzer
Murakami et al. (2007)[116]	Nonrandomized crossover	$n = 15$ (7 females, 8 males); mean age (SD): 69.1 (8.4) years	All participants used a BPA-containing polysulfone dialyzer prior to start of intervention, then switched to a cellulose dialyzer to assess for changes in BPA	~5 months (at least 3 months with polysulfone, 1-month crossover to cellulose, 1-month crossback to polysulfone dialyzer)	Serum and intracellular BPA	Blood samples collected before and after hemodialysis treatment at the end of the three phases	Serum BPA decreased significantly when participants crossed to the cellulose dialyzer**, then increased significantly when participants crossed back to the polysulfone dialyzer**	↓ 65–78% BPA posthemodialysis using cellulose compared to polysulfone dialyzer
Shintani (2001)[117]	Nonrandomized trial	$n = 4$ (hemodialysis patients, patient sex not provided); mean age not provided	Each of the four participants was treated with a different type of dialyzer and which were sterilized using two different methods	3 months of thrice weekly dialysis treatment (4 hours/session)	Plasma BPA	Blood samples collected before and after one hemodialysis at end of 3-month trial	BPA was not detected in participant blood prior to hemodialysis; for the two participants whose dialyzers were sterilized using gamma-rays, there was no detectable BPA in plasma, whereas participants whose dialyzers were sterilized using autoclaving had BPA concentrations of 0.2 and 0.7 ppb	No increase in BPA levels following gamma-ray sterilization

Table 2. (*Continued.*)

Author(s) (year)	Study design	Participants	Intervention characteristics	Duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Quiroga et al. (2017)[119]	Nonrandomized crossover	$n = 22$ (hemodialysis patients, 3 female); mean age (SD): 73 (14) years	Examine if use of online hemodiafiltration (OL-HDF) reduces BPA concentrations compared to high-flux hemodialysis	9 weeks (3 phases, 3 weeks each; patients started with high-flux hemodialysis, switched OL-HDF, then back to high-flux hemodialysis)	Plasma BPA	Blood samples collected pre- and postdialysis in the last session of each of the three phases	Pre- and postdialysis BPA levels decreased significantly when using OL-HDF**, although BPA concentrations were not different significantly between postdialysis between high-flux hemodialysis and online hemodiafiltration; there was a greater reduction of free BPA for OL-HDF***	↓ 11% BPA concentration after online hemo-dialfiltration; ↓ 46% reduction in free BPA, specifically after OL-HDF
Takahashi et al. (2008)[110] and Takahashi et al. (2009)[111]	RCT	$n = 16$ (all male); mean age (SD): intervention (non-DEHP tubing, $n = 8$): 56 (11) years, control (DEHP tubing, $n = 8$): 62 (7.6) years	Comparison of DEHP concentrations between two groups undergoing cardiopulmonary bypass; control group had surgery performed with DEHP-coated tubing, intervention had non-DEHP tubing	<1 day	Plasma DEHP	Blood samples collected before anesthesia and at end of surgery	The level of DEHP was significantly increased at the end of surgery compared to the preoperative level in both groups**; the level of DEHP was significantly lower in the intervention group after surgery*	↓ 77.5% DEHP in intervention group
Mettang et al. (2000)[115]	Nonrandomized crossover	$n = 6$ (peritoneal dialysis patients, 4 females; one participant dropped on day 23); mean age not reported	Participants were provided with and trained on use of a DEHP-free peritoneal dialysis device	45 days (2 days preintervention, 42 days intervention, 1 day postintervention)	Serum and urinary metabolites of DEHP	24-hour urine samples were collected and adjusted for creatinine; blood samples were drawn 2 days prior to start of intervention and 42 days after start of intervention	No significant change observed in urinary metabolites, although a significant decrease in serum was observed*	No change in urinary metabolites; ↓ 9% in serum
Kambia et al. (2001)[113]	RCT	$n = 20$ (intervention: 2 females; control: 3 females); age range: intervention: 37–75 years, control: 61–86 years	Comparison of the amount of DEHP released into the blood of hemodialysis patients when dialysis tubing is plasticized with tri(2-ethylhexyl) trimellitate (TOTM) and DEHP (intervention) compared to tubing plasticized with DEHP only (control)	Each patient underwent dialysis for a 4 hour-period three times a week, although data were collected from a single dialysis session	Plasma DEHP	Blood samples collected immediately before dialysis and at minutes 5, 15, 30, 60, 120, 180, and 240 during dialysis	DEHP was detected in all blood samples, but levels increased more through the hemodialysis session in the control group compared to the intervention group**; TOTM reduced the leachability of DEHP, although TOTM was detected in the blood of the intervention group	↓ 66% DEHP metabolites
Vanhorebeek et al. (2022)[121]	Natural experiment	$n = 216$ (PICU patients, 95 female, 2004–2007) and $n = 334$ (PICU patients, 153 females, 2012–2015); median age (IQR): $n = 216$: 1.5 (0.3–5.7) and $n = 334$: 1.9 (0.4–0.6) years	Comparisons made between interventions administered 8 years apart to examine impact of phase out of DEHP on exposure biomarkers (secondary analysis of PICU patients from Tight Glucose Control and PEPaNIC studies)	12 years (2004–2007 and 2012–2015)	Plasma DEHP metabolite concentration	Blood sample collected on last day of PICU stay	Exposure to DEHP in PICU patients significantly decreased over time***	↓ 17–69% DEHP metabolites over time

Table 2. (*Continued.*)

Author(s) (year)	Study design	Participants	Intervention characteristics	Duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Leuenberger et al. (2016)[120]	RCT	$n = 15$ (all male, $n = 7$ in non-DEHP group); mean age (SD): 25.5 (4.2) years	To determine if an autologous blood transfusion (within same individual) using blood stored in a DEHP-free, n-butyryl-tri-(n-hexyl)-citrate bag reduced exposure to DEHP compared to a regular, DEHP-containing blood bag; authors noted that tubing and other medical equipment contained DEHP	~ 50 days (participants received saline solution as baseline, 2 weeks later participants donated one bag of blood, then the blood was infused back into the participant 36 days later)	Urinary metabolites of DEHP	Samples collected 4 days, 1 day, and 1–3 hours before infusion; then 3, 6, and 12 hours postinfusion; adjusted for specific-gravity	Both groups had increases in all five DEHP metabolites after the transfusion; the DEHP metabolite levels in the group with the DEHP-free bag were significantly lower than in the DEHP group***	↓ 72–84% DEHP metabolites 6 hours postinfusion, depending on metabolite, in group that had the DEHP-free bag

Note: BBzP, butyl benzyl phthalate; BPA, bisphenol A; DBP, dibutyl phthalate; DEHP, di(2-ethylhexyl) phthalate; DEP, diethyl phthalate; DiBP, di-isobutyl phthalate; DiNP, di-isononyl phthalate; DMP, dimethyl phthalate; DnBP, di-n-butyl phthalate; NAFLD, nonalcoholic fatty liver disease; OL-HDF, online hemodiafiltration; ppb, parts per billion; PEPaNIC, Early versus Late Parenteral Nutrition in the Pediatric Intensive Care Unit; PICU, pediatric intensive care unit; RCT, randomized controlled trial; SD, standard deviation; TOTM, tri(2-ethylhexyl) trimellitate. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

outside these two categories as "other" ($n = 4$) (Table 3), and policy changes ($n = 14$) (Table 4).

Dietary Interventions

A total of 25 dietary interventions were identified with strategies ranging from controlled consumption of fresh produce, reductions in canned food use, switching to nonplastic beverage containers, and receiving education on reducing exposure to plastics. The interventions were conducted in settings across the world, including China ($n = 2$), the EU ($n = 8$), South Korea ($n = 5$), Taiwan ($n = 2$), and the US ($n = 8$). The sample size for dietary intervention studies ranged from 1 family of 5 to 230 (mean = 58.8) participants. The duration of the studies ranged from 4 d to 2 years. Nine studies were conducted in female participants only, and the remaining studies ($n = 16$) in both male and female participants.

Interventions with foods provided. Peng et al.[84] conducted a crossover study where participants were provided meals for 1 day that were made with either fresh or canned ingredients. They observed significantly lower urinary BPA concentrations (41–71%) on the intervention day when fresh foods were consumed compared to canned food consumption.[84] Similarly, Carwile et al.[85] conducted a crossover study where participants consumed either canned or fresh soup for 5 days. Substantially lower BPA concentrations were found when the participants consumed fresh rather than canned soup.[85] Rudel et al.[86] provided prepared meals to the five families participating in the study and observed significant decreases in urinary BPA and DEHP metabolites during the intervention (66% and 53–56% for BPA and DEHP metabolites, respectively), whereas phthalate metabolites for DBP, BBzP, dimethyl phthalate (DMP), and diethyl phthalate (DEP) remained unchanged. These data were reanalyzed by Ackerman et al.[87] to examine temporal variability in DEHP metabolites by testing urine samples individually rather than combined urine samples within intervention phases.[86,87] The results were similar, showing a significant decrease of over 50% for each of the DEHP metabolites from the preintervention to the intervention periods and a nonsignificant increase during the post-intervention period.[87] Conversely, Barrett et al.[88] did not observe decreases in metabolites for phthalates DEHP, DBP, DEP, BBzP, di-isobutyl phthalate (DiBP), di-n-octyl phthalate (DnOP), di-isodecyl phthalate (DiDP), and di-isononyl phthalate (DiNP) following an intervention where mostly fresh and organic foods were provided, comparing pre- and post-intervention. Sathyanarayana et al.[89] conducted a randomized trial where families assigned to the treatment group were provided with prepared meals and families in the control group were provided with written recommendations. Surprisingly, there were significant increases in total BPA and phthalate metabolites in the treatment group of ~ 100% for BPA, 189% for MEP (metabolite of DEP), 56% for MBP (metabolite of DBP), 66% for MBzP (metabolite of BBzP), and for all DEHP metabolites, including 2,377% for ΣDEHP, whereas there were no statistically significant differences in the control group, findings which were due to a contamination event in the foods provided to the treatment group.[89]

One study was conducted in an institutional setting.[90] Sessa et al.[90] randomized elementary school students to either a treatment group that received one daily meal at school using a plastic-free service or to a control group. The treatment group had significant reductions in both unadjusted and creatinine-adjusted urinary BPA concentrations over the 6-month study period, whereas no change was observed in the control group, an effect that was modified by BMI.

Beverage container interventions. Three studies focused exclusively on BPA exposure from beverage containers, all

Table 3. Other intervention studies ($n = 4$).

Authors (year)	Study design	Participants	Intervention characteristics	Duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Ehrlich et al. (2014)[124]	Nonrandomized crossover trial	$n = 23$ (18 females, 5 males completed receipt handling without gloves; 9 females and 3 males completed full study); mean age (SD): 35 (12) years	Comparison of no gloves versus use of nitrile gloves when handling receipts for a continuous 2 hours	~1.5 weeks (2 one-day sessions with 1 week washout period between sessions)	Urinary BPA	Spot sample provided immediately before handling receipts and 4, 8, 12, and 24 hours after; adjusted for specific gravity values presented	BPA detected in 100% of samples following receipt handling without gloves, compared to 83% of samples at baseline; no significant increase in BPA concentrations when gloves were worn to handle receipts	No change in BPA concentration when gloves were worn
Lee et al. (2018)[123]	Nonrandomized crossover trial	$n = 52$ (all female); median age (IQR): 51 (48–53) years	Comparison of no gloves versus use of gloves during shifts as a cashier	2 weeks (1 week no glove use followed by 1 week of glove use during work)	Urinary BPA	Spot samples collected on two consecutive days during each week; unadjusted and specific gravity-adjusted values presented	During the week without gloves, there was a significant increase in urinary BPA from the preshift to the postshift***; during the week with gloves, postshift urinary BPA was significantly lower compared to the week without gloves***	↓ 49% BPA postshift during week wearing gloves
Harley et al. (2016)[125]	Pre-post	$n = 100$ (all female); age range: 14–18 years	Personal care products advertised as "low chemical" or "phthalate- and paraben-free" were provided to the participants	5 days (1 day preintervention, 3 day intervention, and 1 day postintervention)	Urinary metabolites for DEP, DnBP, DiBP, DEHP, and BBzP; urinary BPA also measured	Spot samples collected on day 1 (preintervention) and day 5 (postintervention) and were collected at same time of day; controlled for specific-gravity and creatinine	68%, 58%, and 55% of the participants showed a decrease in DEP, DnBP, and DiBP, respectively; DEP was significantly lower after the intervention* while the decreases in DnBP and DiBP were not; BPA and phthalate metabolites not typically found in personal care products (DEHP, BBzP) did not change during the intervention	↓ 27% DEP metabolites
Sears et al. (2020)[126]	RCT	$n = 288$ (intervention: $n = 143$; control: $n = 145$); age range: 1–3 years	Secondary analysis of a trial to reduce childhood exposure to lead through measures to reduce household dust which can contain phthalates	Residential intervention occurred prior to 32 weeks gestation	Urinary metabolites for DEHP, BBzP, DiNP, and DiDP; for a negative control, DEP was also examined	Annual spot samples collected at 1, 2, and 3 years of age; creatinine-adjusted values presented	Urinary metabolites for DEHP, DiNP, and DiDP were significantly lower in the intervention group*; the DEP metabolite was not significantly different; geometric means were consistent across age groups	↓ 11–12%; ΣDEHP metabolites

Note: BBzP, butyl benzyl phthalate; BPA, bisphenol A; DEHP, di(2-ethylhexyl) phthalate; ΣDEHP, molar sum of DEHP metabolites; DEP, diethyl phthalate; DiBP, di-isobutyl phthalate; DiDP, di-isodecyl phthalate; DiNP, di-isononyl phthalate; DnBP, di-n-butyl phthalate; DnOP, di-n-octyl phthalate; IQR, interquartile range; RCT, randomized controlled trial. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table 4. Policy intervention studies ($n = 14$).

Author(s) (year)	Location	Participants	Intervention characteristics	Duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Shin et al. (2020)[127]	USA	$n = 192$ (pregnant females, 205 pregnancies); mean age (range): 34.5 (20.5–49.2) years	Time trend analysis to assess impact of regulations for phthalates that occurred in the mid to late 2000s from the Consumer Product Safety Commission	6 years (2007–2013)	Urinary metabolites of DEP, DiBP, DBP, BBzP, DEHP, DOP, DiNP, and DiDP, including ΣDEHP and ΣDBP	Up to three first morning voids (taken 1 week apart) and one 24-hour urine sample in second and third trimesters; adjusted for specific gravity	Metabolites for DEP, BBzP, ΣDBP, and ΣDEHP significantly decreased over the study period*	↓ 11, 3, 3, 19% per year for DEP, BBzP, ΣDBP, and ΣDEHP metabolites, respectively
Kim et al. (2021)[128] [subset of study population from Shin et al. (2020)[127]]	USA	$n = 218$ (pregnant females, 233 pregnancies); mean age (range): 34.9 (20.5–49.2) years	Assessment of temporal trends of urinary concentrations in pregnant women in California from 2007 to 2014; to understand whether broad social forces affected exposure to the target compounds in the US, authors compared their measured urinary concentrations with those of other pregnancy cohorts	7 years	Urinary BPA, BPS, and BPF	Up to three first morning voids (taken 1 week apart) and one 24-hour urine sample in second and third trimesters; adjusted for specific gravity	BPA concentrations decreased significantly over time**; BPS and BPF were detectable in <35% of samples	↓ 46% BPA
Bastiaensen et al. (2021)[129]	Belgium	$n = 416$; age range: 14–15 years	General time trends from biomonitoring cycles, report on changes after policy restrictions were implemented for DEHP, DnBP, DiBP, BBzP, DINP, DIDP	15 years (2003–2018)	Urinary metabolites of DEP, DnBP, DiBP, BBzP, DEHP, DINP, and DiDP	Random spot samples collected during the day at school, adjusted for specific gravity and body weight	Exposure still widespread, but DEHP and DnBP metabolites decreased significantly over time***; nonsignificant decreases in DEP, DiBP, BBzP; concentrations observed in this study were similar to related studies from Germany, Canada, and the US	↓ 81% ΣDEHP metabolites; ↓ 56% DnBP metabolites
Schoeters et al. (2017)[130]	Belgium	$n = 210$ (2002–2006; 89 females); $n = 208$ (2007–2011; 113 females, age range: 14–15 years)	General time trends, decreases attributed to policy change, specifically Directive 2005/84/EC	∼9 years (2007–2015)	Urinary metabolites of DBP and DEHP, including ΣDEHP	Spot samples collected during school day; creatinine-adjusted values presented	ΣDEHP and DBP metabolites significantly decreased over time*	↓ 70% ΣDEHP metabolites; ↓ 41% DBP metabolites
Frederiksen et al. (2020)[131]	Denmark	$n = 300$ (all male, 100 participants from each of 3 years: 2009, 2013, 2017); mean age (range): 20 (18–30) years	Gradual phase out of DiBP, DnBP, BBzp, and DEHP in the EU	8 years (2009–2017)	Urinary BPA, BPS, BPF, and metabolites of DMP, DEP, DiPrP, DnPrP, DiBP, DnBP, BBzP, DnPeP, DEHP, DHxP, DCHP, DHpP, DnOP, DiNP, and DiDP	Spot morning urine, adjusted for urine density	Metabolites for DMP, DEP, BBzP, DiBP, DnBP, DEHP, DHpP, DiNP were significantly lower in 2013 and 2017 compared to 2009***; BPA significantly decreased from 2009 to 2017***, whereas BPS*** and BPF* significantly increased over time; total concentration of chemicals and their replacement decreased over time	↓ 30–73% DMP, DEP, BBzP, DiBP, DnBP, DEHP, DHpP, DiNP metabolites; ↓ 57% BPA; ↑ 64% BPS; ↑ 7% BPF
Tschersich et al. (2021)[58]	Germany	$n = 516$ (2014–2017 cycle); $n = 509$ (2003–2006 cycle); sex distribution of participants not reported; age range: 3–14 years	Comparison of nationally representative populations through biomonitoring programs before and after implementation of EU policies related to BPA	14 years (2003–2017)	Urinary BPA	First morning void; unadjusted and creatinine-adjusted values presented	BPA significantly decreased over the study period***, with the largest decrease among those 3–5 years old** followed by those 6–10 years old***; among those 11–14 years old, the decrease (8%) was not statistically significant.	↓ 26% BPA all ages; ↓ 37% BPA in children 3-5 years; ↓ 33% BPA in children 6–10 years

Table 4. (*Continued.*)

Author(s) (year)	Location	Participants	Intervention characteristics	Duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Göen et al. (2011)[132]	Germany	$n = 240$ (60 per year, 120 females); mean age (range): 23.3 (19–29) years	To investigate whether the regulatory use restriction, issued for individual phthalates is or is not successful	6 years (2002–2008)	Urinary metabolites of DnBP, DiBP, BBzP, DEHP, and DiNP	24-hour samples at each time point, creatinine-adjusted and unadjusted values presented	DEHP***, DnBP***, and BBzP* metabolites significantly decreased over time, while DiNP metabolites significantly increased***; no significant change in DiBP metabolites over time	↓ 11–33% DEHP metabolites; ↓ 57% DnBP metabolites; ↓ 42% BBzP metabolites; ↑ 47–56% DiNP metabolites
Tranfo et al. (2018)[133]	Italy	$n = 328$ [2011: $n = 157$ (83 female) 2016: $n = 171$ (111 females)]; mean age (SD): 2011: 36.5 (7.2) years for females, 40.4 (7.3) years for males; 2016: 42.4 (8.3) years for females, 41.0 (9.2) years for males	Phthalates were included in the authorization process defined in European Regulation CE 1907/2006 (REACH) starting in 2011. An earlier European directive 79/769/CEE set the maximum permitted concentrations of six phthalates in toys and children's products and remained as restrictions in 2011	5 years (2011–2016)	Urinary metabolites of DEHP, DEP, DnBP, BBzP, and DBzP, including ΣDEHP	Samples (timing not noted) limited to participants with creatinine concentrations between 0.3 and 3 g/L	Statistically significant difference for all metabolites in both males and females between the two periods*, except for DEP, an unregulated phthalate, which decreased non-significantly in females	↓ 72–73% ΣDEHP metabolites; ↓ 98% DnBP metabolites; ↓ 91–94% BBzP metabolites
Gyllenhammar et al. (2017)[134]	Sweden	$n = 178$ (all female); median age (range): 29.6 (20–41) years	Investigate if attempts to decrease production and exposure to these chemicals has resulted in decreased human exposure	5 years (2009–2014)	Urinary BPA, BPS, BPF and metabolites of DEP, DiDP, DPHP, DnBP, BBzP, DEHP, and DiNP	Morning spot urine; adjusted and unadjusted for urine density presented	Significant negative temporal trends observed for metabolites of DEP*, DnBP***, BBzP*** and for 4/5 metabolites of DEHP***; significant decreases over time were observed for BPA,* whereas BPF* increased and BPS was stable over time	↓ 10% per year BPA; ↑ 20% per year BPF; ↓ 16–17% per year DEHP metabolites; ↓ 9% per year DEP metabolites; ↓ 14% per year DnBP metabolites; ↓ 16% per year BBzP metabolites
Shu et al. (2018)[135]	Sweden	$n = 1{,}651$ (pregnant females; 271, 683, 593, and 104 in 2007, 2008, 2009, and 2010, respectively); mean age (SD): 31.0 (4.8) years	Comparison of phthalate metabolites over the four different sampling periods to encompass time points when new policies restricting phthalates were put in place	4 years (2007–2010)	Urinary metabolites of DEP, DBP, BBzP, DEHP, DiNP, DPHP, and DiDP, including ΣDEHP and ΣDiNP	First morning void collected between 3 and 27 weeks gestation; creatinine-adjusted values presented	Decreases over time for all DEHP metabolites***; increases over time for DBP**, DiDP***, DPHP**, and ΣDiNP** metabolites	↓ 16.2 ΣDEHP metabolites; ↑ 16.4 DBP metabolites
Larsson et al. (2017)[136]	Sweden	$n = 213$ (100 in 1998–2000 and 113 in 2015); sex distribution of participants not reported; mean age (range): 1998–2000: 49 (45–55) months; 2015: 50 (40–58) months	Assess trends of bisphenol and phthalate metabolites in children compared to samples taken ∼15 years prior	17 years	Urinary BPA, BPS, BPF, and metabolites of DEHP, BBzP, DnBP, DiBP, DEP, DMP, DiNP, DPHP, and DiDP	First morning void; adjusted for urine density	BPA concentrations and metabolites of regulated phthalates DEHP, DnBP, BBzP, and DEP significantly decreased over time***; there was no change in BPF over time and BPS could not be analyzed over time; metabolites for DiNP, a substitute phthalate, increased non-significantly over time	↓ 56% BPA; ↓ 83–85% DEHP metabolites; ↓ 77% DnBP metabolites; ↓ 83% BBzP metabolites; ↓ 75% DEP metabolites
Lin et al. (2020)[143]	China/USA	$n = 55$ (28 females); mean age (SD): 24.0 (7.8) years	Compare differences in BPA exposure while living in Beijing as compared to living in LA to examine effects of different policies between the two locations	∼14 weeks per year for 6 years; 10 weeks spent in Beijing over summer (2012–2017)	Urinary BPA	Multiple first morning voids after ≥8 hours fast collected from each participant, >1 week apart; creatinine-adjusted values presented	BPA concentrations were significantly higher while in Beijing compared to Los Angeles for all years except 2012; urinary concentrations of BPA indicate 2.9-fold higher exposure in Beijing***; the geometric mean of BPA while in Los Angeles decreased annually over the study period by about 25% per year***, whereas a smaller, nonsignificant decrease of 5% per year was observed in Beijing over the same time	↓ 77% BPA (in Los Angeles, 2012–2017); ↓ 76% BPA in Los Angeles compared to Beijing (2017)

Table 4. (*Continued.*)

Author(s) (year)	Location	Participants	Intervention characteristics	Duration	Exposure variable(s)	Biospecimen collection	Intervention findings	Percent change
Lu et al. (2022)[144] [subset of study population from Lin et al. (2020)[143]]	China/USA	$n = 26$ (14 females); mean age (SD): 23.8 (5.6) years	Compare differences in BPA exposure while living in Beijing as compared to living in Los Angeles to examine effects of different policies between the two locations as well as associations with lipid profiles	~14 weeks per year; 10 weeks spent in Beijing over summer (2014–2015)	Urinary BPA	Multiple first morning voids after ≥8 hours fast collected from each participants, >1 week apart; creatinine-adjusted values presented	Participants were exposed to significantly higher levels of BPA after traveling from Los Angeles to Beijing (4.0 times; 95% CI: 2.7–6.1)*** possibly due to more strict regulation policies in the US compared to China	↓ 82% BPA (Los Angeles compared to Beijing)
Lyu et al. (2022)[145]	Japan	$n = 132$ (all females); mean age (SD): 59.3 (12.4) years	Assess trends of phthalate exposure over time period when regulations and industry practices changed	~13 years (1993–2016)	Urinary metabolites of phthalates DMP, DEP, DiBP, BBzP, DnOP, DiNP, DiDP, and DEHP	Spot urine samples collected at routine health care visits; creatinine-adjusted values presented	A significant negative correlation with ΣDEHP and year of sampling was found**, but a clear decrease was observed only between 1993 and 2000; decreases in DnBP*, BBzP*, and DiBP were observed from 1993 to 2000, with no significant changes in later years	↓ 41–71% select DEHP metabolites

Note: BBzP, butyl benzyl phthalate; BPA, bisphenol A; BPF, bisphenol F; BPS, bisphenol S; CI, confidence interval; DBP, dibutyl phthalate; ΣDBP, molar sum of DBP metabolites; DBzP, dibenzyl phthalate; DEHP, di(2-ethylhexyl) phthalate; ΣDEHP, molar sum of DEHP metabolites; DEP, diethyl phthalate; DHpP, di-n-heptyl phthalate; DHxP, di-n-hexyl phthalate; DiBP, di-isobutyl phthalate; DiDP, di-isodecyl phthalate; DiNP, di-isononyl phthalate; ΣDiNP, molar sum of DiNP metabolites; DiPrP, di-iso-propyl phthalate; DMP, dimethyl phthalate; DnBP, di-n-butyl phthalate; DnOP, di-n-octyl phthalate; DnPrP, di-n-propyl phthalate; DPHP, di-2-propylheptyl phthalate; SD, standard deviation. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

finding decreased urinary BPA concentrations when participants used nonplastic and noncanned containers. Specifically, geometric mean concentrations of BPA were 20% less when stainless steel bottles were used compared to polycarbonate bottles.[39] After a week of ceramic cup use, the geometric mean BPA concentration was significantly lower by 16–51% compared to a week of polycarbonate cup use.[91] The geometric mean of urinary BPA was 94% lower after consuming two soymilk beverages from glass bottles compared to consuming two soymilk beverages from cans.[92] One additional study examined differences in BPA, BPS, BPF, DBP, and DEHP metabolites in a randomized crossover where French press and capsule coffee were consumed.[93] Although the French press did not contain plastic like the capsule machine, no association between how the coffee was prepared and urinary bisphenols and phthalates was found.[93]

Educational and lifestyle interventions. We identified 15 educational and lifestyle interventions from 16 publications, 11 which showed significant decreases in most or all urinary biomarkers. Following a 5-day vegetarian diet intervention, metabolites for all measured phthalates decreased, except for DEHP where only some of the metabolites decreased.[94] In an intervention in which overweight and obese children were assigned to a "healthy diet" group for weight control and normal and underweight children were assigned to continue their regular diet, researchers observed a significant 28% lower concentration in the molar sum of DEHP metabolites in the healthy diet group while other phthalates were not significantly different between the two groups.[95] Although statistical significance was not reached for the other phthalate metabolites, there was a trend of lower median phthalate metabolite concentrations in the healthy diet group.[95] In addition, BPA concentrations were ~18% lower in the healthy diet group, although this difference was not statistically significant.[96] Dallio et al.[97] provided male nonalcoholic fatty liver disease patients with strategies to avoid dietary sources of BPA exposure but did not observe decreases in urinary BPA concentrations, although a 62% decrease was seen in plasma. Similarly, when guidelines on reducing dietary exposure to BPA were provided to teenage participants, the intervention did not have a significant effect on reducing BPA exposure biomarkers.[98]

Eleven of the educational and lifestyle interventions used multiple approaches to reduce exposure to both bisphenols and phthalates. One study involved a single family that avoided many sources of plastic, and although significant changes in BPA and phthalate metabolites were not observed over the 2-month study, the family members' metabolite concentrations were below German Human Biomonitoring Commission reference values, both at the start and end of the follow-up period.[99] Another included more frequent handwashing and avoiding plastic dishes and packaging, personal care products, nutritional supplements, and some building materials in an intervention to reduce exposure to phthalates in Taiwanese girls.[100] There were reported issues with compliance in this study, but the participants categorized as "compliant" had a 71–96% reduction in phthalate metabolites.[100] In an intervention that recruited mothers and their children, participants were instructed to avoid canned foods, plastic containers, instant food, and delivery food. BPA concentrations were significantly reduced following the 3-d intervention in both mothers and children, but BPS concentrations decreased significantly only in the mothers.[101] In an intervention to reduce exposure to BPA, Park and Chung recruited female college students with reported menstrual pain and instructed them to decrease fast and processed food consumption.[102] This was augmented by follow-up monitoring and peer support via social network communication. There were significant decreases in BPA biomarkers between baseline and the first and second menstrual cycles, but

levels returned to baseline by the third menstrual cycle.[102] An additional intervention in pregnant women consisted of dietary changes, including restrictions on fast food, canned food, etc.; lifestyle changes, including reduction in personal care product use and avoiding plastics; and environmental changes, such as reducing exposure to secondhand smoke and exercising.[103] Significant declines were observed for phthalate metabolites MECP (from DnOP), MiBP, MMP (from DMP), MEP, mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), mono-n-butyl phthalate (MnBP), and mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP) over the intervention and the summed total for all measured phthalates decreased by 40% from baseline levels.[103] Two studies provided educational information about reducing exposures to phthalates and bisphenols.[104,105] In one, participants were provided information on reducing exposure via changes in household cleaning products, cosmetics, and food packaging, which resulted in significantly lower concentrations of BPA and phthalate biomarkers for DEHP and DEP compared to baseline in 26 adults.[104] In the other, a combination of educational campaigns and public conferences were conducted to provide information on endocrine disruption and strategies to reduce exposure.[105] Several phthalate metabolites decreased over the study period (2 years), BPA decreased nonsignificantly, and BPS and BPF concentrations significantly increased.[105]

Five randomized controlled trials were conducted using a variety of strategies to reduce exposures. One web-based intervention aimed to reduce exposure to BPA and phthalates through a variety of strategies, including consumption of fresh foods, use of glass instead of plastic containers, more frequent handwashing, etc.[106] In this study, the treatment group had significantly decreased concentrations of MEHP, MEOHP, and BPA. Hagobian et al.[107] aimed to reduce exposure to BPA, while Hagobian et al.[108] aimed to reduce exposure to BPA, BPF, and BPS by targeting dietary and personal care product sources of exposure, coupled with counselor feedback in weekly face-to-face meetings in the treatment group over a 3-wk period. In both studies, the control group received general information about healthy eating and sources of BPA exposure. Urinary BPA concentrations declined significantly[107] but remained unchanged along with BPF in the later study,[108] with only BPS decreasing significantly (1.42 μg/g creatinine) in the treatment group.[108] El Ouazzani et al.[109] assessed the efficacy of three workshops aimed at decreasing endocrine disruptors among pregnant women, in comparison to providing participants leaflets. The workshops covered air quality, nutrition, and personal care products, but there was no significant difference in urinary BPA concentrations between the control and treatment groups.[109]

Interventions of Clinical Exposures

Twelve studies in 13 publications involved intervention to reduce exposures in clinical settings (Table 2). The sample size ranged from 4 to 216 (mean = 42.8). One study was conducted in young children, one included both adults and adolescents, and the remaining studies were conducted in adults. Three studies lasted 1 day due to the nature of the intervention,[110–112] the duration of one was unclear,[113] and the remaining studies lasted 1.5 months to 12 years.[114–121] Studies were completed in multiple countries, including the EU ($n = 6$), Iran ($n = 1$), Japan ($n = 4$), Switzerland ($n = 1$), and the US ($n = 1$).

Two interventions involved dental procedures. One study assessed differences in salivary and serum BPA levels in participants randomly assigned to receive either one or four occlusal dental sealants, with those receiving fewer sealants having lower salivary BPA (peak of 3.98 ng/ml vs. 9.08 ng/ml) levels.[112] The other study compared two methods to remove the adhesive for fixed orthodontic brackets (i.e., braces), with significantly lower salivary BPA concentrations in the group that had the brackets removed using a rotary method compared to an ultrasonic method.[122]

Five studies examined differences in BPA levels via interventions related to hemodiafiltration, with reductions in exposure measured following use of non-BPA-containing materials[114,116–118] and online hemodiafiltration.[119] Overall, the use of non-BPA-containing materials for dialysis resulted in reductions in BPA exposure ranging from ∼6–50%, with one study achieving BPA levels below the detectable limit.[117]

The remaining studies aimed to reduce exposure to phthalates from medical exposures.[110,111,113,115,120] A crossover study in five patients on peritoneal dialysis to compare phthalate exposure when using plasticizer-free devices to plasticizer-containing devices observed reductions in phthalate biomarkers when plasticizer-free devices were used.[115] In two studies that assessed the same group of patients undergoing cardiopulmonary bypass surgery, researchers observed significantly lower phthalate metabolite concentrations when nonDEHP tubing was used during surgery.[110,111] Kambia et al.[113] used a randomized clinical trial design to examine DEHP concentrations between one group of patients that used tubing plasticized with DEHP compared to a group that used tubing plasticized with an alternative plasticizer, tri(2-ethylhexyl) trimellitate. Following a hemodialysis session, DEHP levels were significantly lower (68%) in the group that used the nonDEHP tubing.[113] Vanhorebeek et al.[121] compared groups of pediatric patients from two earlier interventions to examine whether the children in the later intervention had lower DEHP metabolites, as DEHP was in the process of being phased out. The authors observed significant decreases in DEHP metabolites in the later intervention, ranging from about 17–68% depending on the metabolite.[121] Lastly, Leuenberger et al. (2016)[120] conducted a randomized clinical trial and found that participants treated with nonDEHP blood bags for an autologous blood transfusion (transfusion of participant's own blood) had significantly lower levels of DEHP metabolites compared to participants treated with DEHP-containing blood bags.

Other Types of Interventions

Of the remaining nonpolicy interventions, three were conducted in the US and one in South Korea. The sample size for these studies ranged from 23 to 288 (mean = 115.8). Two studies conducted interventions to reduce exposure to BPA from receipt paper by having participants wear gloves, with significantly lower biomarkers of exposure when gloves were worn.[123,124] The geometric mean of BPA in urine increased from 1.8 μg/L [95% confidence interval (CI): 1.3, 2.4] to 5.8 μg/L (95% CI: 4.0, 8.4) after not wearing gloves, whereas there was no change when gloves were worn. One intervention provided 100 female adolescent participants with personal care products advertised as "low chemical" or "phthalate-free" and observed 0.5–27.4% reductions in urinary biomarkers for phthalates typically found in personal care products.[125] The fourth intervention implemented measures to reduce household dust accumulation, including repairing walls with water damage, paint stabilization, and creating smooth floor surfaces (e.g., refinishing wooden floors), thereby reducing exposure to phthalates from paints and building materials, which can accumulate in dust.[126] This intervention was delivered to pregnant women and resulted in an 11–12% lower concentration of urinary phthalate metabolites in their children when compared to control treatment.[126]

Policy Interventions

As shown in Table 4, seven policy intervention studies focused on phthalates, four focused on bisphenols, and three included analyses of both bisphenols and phthalates. Seven of the 14

articles included an examination of BPA with three including exposure trends in BPA analogues BPF and BPS. Most studies used a natural experiment study design to retrospectively examine whether policies promulgated to regulate phthalates and bisphenols in consumer products were associated with changes in their exposure over several years, and sometimes decades. In these policy intervention studies, the sample size ranged from 26 to 1,651 (mean = 370.3) individuals, with the majority of studies conducted among both males and females ($n = 8$; females only, $n = 5$; males only, $n = 1$) and across the life course.

One study examined phthalate exposure in pregnant women in California following enactment of the Consumer Product Safety Improvement Act (CPSIA) of 2008 in the US and recommendations made by the CPSC's Chronic Hazard Advisory Panel on Phthalates and Phthalate Alternatives Committee in 2014 to further restrict the use of phthalates and phthalate substitutes in children's toys, childcare products, and in products used by women of childbearing age.[127] This study found decreases in urinary concentrations of metabolites for the regulated phthalates but increases over time in some of the replacement phthalates. Specifically "MEP, MBP, ΣDBP, and ΣDEHP decreased over the study period [percent change per year (95% CI): −10.8% (−14.8, −6.7%); −3.4% (−7.4, −0.8%); −3.4% (−6.4, −0.2%); −18.8% (−22.0, −15.4%), respectively] while ΣDiBP, MCPP (metabolite of DnOP), and MCOP (metabolite of DnOP) increased [percent change per year (95% CI): 3.8% (0.7, 7.0%); 4.7% (0.1, 9.5%); 13.7% (8.1, 19.6%), respectively]."[127] A longitudinal study by Kim et al.[128] reported a geometric mean decrease of 5.7% per year (95% CI: −8.2%, −3.2%) in BPA exposure among 218 pregnant women in California from 2007 to 2014. The authors concluded that this downward trend may reflect "US nationwide regulation efforts and/or California advocacy campaigns since the late 2000s to mid-2010s for reducing exposure to phenols (including bisphenols), parabens, and triclocarban in women of reproductive age and infants."[128]

The remaining policy intervention studies were mostly conducted in EU countries, including Belgium,[129,130] Denmark,[131] Germany,[58,132] Italy,[133] and Sweden.[134–136] This is not surprising as the EU has banned the use of certain phthalates in toys, food-containing materials, and cosmetics with additional restrictions taking effect in 2020[54,137–140] and has banned BPA from the production of baby bottles and cosmetics.[141,142] Following the implementation of EU policies to reduce exposure, a 41% reduction in median BPA metabolites from 2009 to 2017 (2.27–1.33 ng/mL, osmolality-adjusted) was observed by Frederiksen et al.,[131] while Gyllenhammar et al.[134] reported yearly 9.8% reduction from 2009 to 2014, but also a 20% increase in BPF, over the same period. Tschersich et al.[58] reported reductions in BPA exposure among children in Germany for the period 2003–2017, but this varied by age group; among children 3–5 years of age, the decrease was significant at 37% and 33% for children 6–10 years of age; and among children 11–14 years of age, the decrease was not statistically significant (−8%).

Reductions in phthalate metabolites were observed by Bastiaensen et al.,[129] Schoeters et al.,[130] Frederiksen et al.,[131] Göen et al.,[132] Gyllenhammar et al.,[134] Larsson et al.,[136] Shu et al.,[135] and Tranfo et al.,[133] although some specific low-molecular-weight phthalates and phthalate replacements increased over the study period in Frederiksen et al.,[131] Göen et al.,[132] and Shu et al.[135] For example, both studies analyzing biomonitoring data from the Flemish Environment and Health Study (FLEHS) noted significant decreases in DEHP exposure among adolescents[129,130]; between 2008 and 2013, the geometric mean of DEHP exposure (as measured by sum of its urinary metabolites) was reduced by ∼ 70%.[130] Among young men in Denmark, Frederiksen et al.[131] observed significantly lower urinary metabolite concentrations for eight phthalates [DMP, DEP, BBzP, DiBP, DnBP, DEHP, di-n-heptyl phthalate (DHpP), DiNP] ranging from 36% for DiNP to 75% for DEP during 2009–2017. Using repeated cross-sectional analysis of biomonitoring data from 2002 to 2008, Göen et al.[132] found exposure among German college students to DnBP decreased by a factor of 7–8 and to DEHP and BBzP by a factor of 2–3. In contrast, DiNP exposure increased by a factor of 4.[132] In addition to the report on BPA reductions among Swedish women,[135] Gyllenhammar et al.[134] noted yearly decreases (percentage change in concentration) for MEP (−9.8), MBP (−14), and MBzP (−16) and four out of five metabolites of DEHP [MEHHP (−16), MEOHP (−17), MECPP (−16), and MCMHP (−17)].[134] Similarly, Larsson et al. (2017)[136] observed decreases in BPA and regulated phthalates in children by comparing levels in 2015 to those measured in 2010.

Three studies examined temporal changes in exposures to BPA and phthalates in Asian countries. These studies were motivated by changes in the design and formulation of products in response to regulations and industry practices. Lin et al.[143] and Lu et al.[144] took advantage of a natural experiment involving student participants in a summer exchange program between University of California Los Angeles (UCLA) and Peking University (PKU) in Beijing, China. The authors hypothesized that contrast between restrictions on BPA use in consumer products between the US and China would be reflected in urinary BPA measures of the exchange students before and after travel to the two countries. Among 55 US students who spent 10 wks in Beijing from 2012 to 2017, creatine adjusted urinary BPA levels increased 2.91-fold (95% CI: 2.43, 3.50) but then returned to baseline after study participants returned to Los Angeles.[143] Using banked spot urine samples donated by 132 healthy Japanese women from 1993 to 2016, Lyu et al.[145] reported significant declining trends for only MCPP and MEOHP phthalate metabolite concentrations ($r = -0.879$ for MCPP, $r = -0.831$ for MEOHP), whereas for ΣDEHP metabolites, a significant negative correlation with the year of sampling was also found ($r = -0.261$); however, a clear decrease was observed only between 1993 and 2000. Although not estimated by these authors, the percentage decrease in mean creatinine-corrected concentrations (μg/g creatinine) for MCCP and MEOHP from 1993 to 2016, based on figure plots, appear to be 55% and 62%, respectively. A Ministry of Health of Japan regulation on DEHP use in gloves adopted in 2000 was cited as a possible reason for the declines observed, but this was not directly tested by the study authors.

Discussion

Bisphenols and phthalates are associated with a wide variety of negative health effects. To our knowledge, this is the first scoping review of multiple types of interventions to reduce exposure to bisphenols and phthalates. In this review, we examined different types of interventions focused on exposure to bisphenols and phthalates using human biomonitoring data. Given the range of study types included in this review, we provide insights on the types of interventions, from individual to societal changes, that were designed to test effectiveness of reducing exposures. Taken together, these studies suggest that increased fresh food consumption and plastic-free foodware can help reduce exposure to BPA and, to a lesser extent, phthalates.[86,89] Identified studies consistently concluded that the use of materials other than plastic and cans reduced exposure to BPA in indicating that beverage and food containers may be a source of exposures and thus a potential target for lowering BPA exposures.[84,92] Trials with food and other products provided support for the efficacy of dietary interventions and imply the potential impact of both home and school-based interventions to lower exposure to bisphenols

and phthalates. The mode of information/intervention delivery and age of participants may impact compliance, and current data provide a starting point for future programs. While there is some indication of reduced bisphenol and phthalate exposures, the identified studies revealed that current dietary interventions may not be effective at broadly reducing exposure to bisphenols and phthalates. Reducing exposure to BPA and phthalates in clinical settings is feasible and effective when coated or plasticizer-free materials are used.[110,119–121] Understanding what interventions work best to reduce exposures to bisphenols and phthalates is important given the potential positive health effects with reduced exposure.

Overall, studies of policy interventions showed greater magnitude in exposure reductions over longer periods of time and were primarily based on repeated cross-sectional analyses at a population level; this provides confidence that banning bisphenols and phthalates can reduce exposure in perpetuity. In contrast to the policy interventions, the vast majority of interventions reviewed (75%, 33/44) occurred over very short time scales ($\sim$ weeks to a few months) with a lack of consistent sampling timeframes (spot urine vs. 24-h pool) and repeated sampling across the intervention. Evaluating the effectiveness of interventions in home and work settings was often challenging due to a lack of details on intervention procedures and reported noncompliance among study participants. This was especially concerning when considering if interventions can be sustainable long term. Overall, we posit the literature is in great need of longer-term intervention studies with repeated sampling, coupled with more detailed reporting on intervention delivery frequency and procedures. Whenever possible, we recommend that interventions be designed with interdisciplinary teams including behavior change, psychology, nutrition, etc. expertise, to ensure that designs represent interventions that are both feasible and sustainable in real-world settings and include behavioral measures that may help explain any deviations from intended/expected findings.

Human biomonitoring estimates internal exposures (i.e., body burdens) by measuring the respective chemicals or their metabolites in human biospecimens (e.g., urine or blood). Thus, biomonitoring represents an integrated measure of exposure from multiple sources and routes and permits an integrated exposure assessment, even when the quantity and quality of external exposures are unknown and/or if the significance of the contribution of different routes of exposure is ambiguous. This is a strength of biomonitoring but can also be a weakness when trying to assess the effectiveness of interventions to reduce exposure to chemicals, such as phthalates and bisphenols, that have multiple sources of exposure. The nonpolicy interventions reviewed partially overcame this problem by assessing exposures via biomonitoring before and after removing specific sources of exposure or comparing two groups (with and without the source of exposure). Studies assessing policy interventions relied on natural experiment/quasi experimental designs to assess general trends in biomarkers of exposure for phthalates, bisphenols, and their replacement chemicals over time.

The selection of the appropriate biomarker (i.e., concentration of parent chemical or metabolite) and the biospecimen is important as this can influence interpretation of results and impact ability to make comparisons across studies. For example, urinary markers for BPA and phthalates are commonly used because these chemicals are nonpersistent in the body as they metabolize quickly. Urine is considered the "best matrix for epidemiological assessment of exposure to BPA, phthalates, and other polar, nonpersistent chemicals to whose exposures can be episodic in nature."[146] However, urinary biomarkers are at risk of measurement error due to variability in urinary dilution. A recent comparison of correction of urinary dilution using specific gravity and creatinine adjustment suggests agreement between the methods, although both measurements differ systematically between individuals based on sociodemographic, body composition factors, and collection strategy.[147,148] Of the 58 studies reviewed, 81% (47) analyzed urinary biomarkers, and of these, 96% (45/47) adjusted for dilution. An additional limitation of using urinary metabolites to assess exposure to phthalates and bisphenols is that these concentrations primarily reflect exposure within a relatively short period preceding urine collection. As a result, there can be considerable within-person and between-person temporal variability of metabolite concentrations, especially in spot urine samples. Ideally, multiple urine samples or a 24-h sample would be collected, but this places a burden on study participants, so researchers tend to use single or spot urine samples (79%, 37/47). However, prior studies indicate that a single spot urine sample may be moderately representative of long-term exposure averages "if samples are collected from a large number of persons and randomly collected relative to meal ingestion times and bladder emptying times."[149] In 11 studies, blood samples were analyzed to assess exposures occurring directly in the bloodstream (e.g., from a change in medical tubing material).[110,111,113,115–119,121] However, there are concerns about using blood as the matrix because the metabolites of phthalates and BPA can be orders of magnitude lower than in urine[65,146,147] due to difficulty in preventing contamination introduced by sample collection and analysis. Saliva was used as a biological matrix only in studies where a dental intervention was performed.[112,122]

For the general public, the dominant exposure route for phthalates and BPA is through the diet.[24,25] Interventions that substitute dietary items and measure changes in concentration of chemicals or metabolites in urine or blood can identify sources of phthalates and bisphenols and demonstrate the efficacy of interventions. For example, interventions using nonplastic beverage containers (e.g., stainless steel water bottles) appeared to consistently reduce exposure to BPA.[39,91,92] However, individual-level dietary interventions yielded inconsistent results between studies, calling into question the effectiveness and sustainability of implementing this approach at a larger scale. A number of studies noted participant noncompliance to the dietary interventions,[88,97,100] and some participants reported dissatisfaction with and difficulty following dietary changes.[19,88,98] Most of the interventions were very short in duration, and biomarker concentrations often returned to or approached baseline following completion of the intervention.[86,87] Increased consumption of fresh and organic foods as an intervention may not be feasible for many families because of income as well as access and availability of these types of foods. Further, as demonstrated by Sathyanarayana et al.,[89] even after careful selection of food items assumed to be lower in concentrations of target chemicals, exposures can still occur, and the source can be very unclear.[89] It is important to note that data on ingredient composition of consumer products are difficult to obtain because reporting is not always required by law (in the US). For example, the phthalate DEP is frequently used as part of the fragrance added to products, but the labeling of the constituents of a fragrance is not required by the FDA, as this can be considered a "trade secret."[150] Thus, strategies targeted at the individual consumer level to avoid products containing these chemicals may not result in significant long-lasting reductions in exposure,[151] supporting the implementation of policies to reduce population-level exposure.

Studies that we reviewed demonstrate that using phthalate-free medical devices and intravenous (IV) bags reduce human exposures.[110,113,120,121,152] Yet, the US FDA has made little progress toward removing harmful phthalates and other EDCs from medical equipment in the 20 years since it issued guidance recommending health care providers "consider" alternatives to

medical devices, particularly IV bags, containing DEHP.[153] IV bags that do not contain DEHP are available, but those that do contain this phthalate can contain up to 40% DEHP by weight.[154] As recently as 2021, members of the US Congress have pushed the FDA on this subject.[155] Research could investigate the barriers to adopting EDC-free medical equipment more widely by health care providers as well as barriers at governmental policy level, although IV bags and medical equipment are not the only source of EDC exposure over which the FDA (and similar governmental agencies in the EU) has authority.

Policy interventions have great potential to reach many people from all demographic and social backgrounds and bring about lasting reductions in exposures. While substantial and persistent downward trends in exposure to phthalates and BPA were observed in US- and EU-based studies that assessed impacts of policies around these chemicals, we note a few challenges. Some of the policy studies had imprecise time periods of study relative to specific policies evaluated or did not specify a time period or a particular point in time after which exposure levels were expected to be reduced.[127,130,131] This creates difficulty for linking the biomonitoring results to specific consumer products targeted by policies. For example, phthalates considered the most toxic have successively been phased out in the EU by applying gradually stricter regulations. The use of DEHP, DnBP, and BBzP were banned in toys and childcare articles in 2007, whereas DiNP and DIDP were banned from toys intended for mouthing.[140] After 2015, DEHP, DnBP, BBzP, and DIBP, which are classified as reproduction toxic category 1B, could not be used for any application within the EU without permission.[54] Although these four phthalates are strictly regulated within the EU today, they are likely found in many products and materials which will continue to be used for a long time to come; these four phthalates will continue to contribute to human exposure as demonstrated by the studies we reviewed, albeit at lower levels than in the past. Additionally, these studies show it may be hard to pinpoint a specific policy responsible for the exposure reductions, especially when there can be voluntary phase out and substitutions by the producers which can also change the exposure patterns to phthalates and bisphenols. Indeed, Frederiksen et al.[131] noted that EDCs are being replaced with other related chemicals with similar function and with similar health concerns. Studies tracking human exposures post restrictions on BPA demonstrated decreases in exposure over time[58,131,134] but also concomitant increase in exposure to BPA analogues, BPS and BPF.[131,134] Similarly, exposures to di-iso-nonyl-cyclo-hexane-1,2-dicarboxylate (DINCH), which was introduced in 2002 to replace DEHP and other phthalates in PVC, have increased.[129,131,134] Metabolite levels of these substitute chemicals have significantly increased during the last decade in the US and EU countries.[131,134] To better inform implementation science, future policy evaluation studies should consider expanding the focus to include understanding the rules, values, and norms of the "implementers" of policy and how they respond to new knowledge (e.g., more information about replacement chemicals and behavior of industry in response to advocacy and prior piecemeal policy decisions by governmental bodies) and to explore the use of more sophisticated statistical analysis approaches, such as interrupted time series analyses to link change in policy/organizational behavior to change in exposures.[156]

Few of the studies included in the present review conducted qualitative analyses, which can be beneficial in this context to illuminate challenges with implementation of interventions, most notably for the dietary interventions. Galloway et al.[98] conducted a qualitative analysis to assess the sustainability of the diet. Half of the participants reported an increase in food costs and more time spent shopping. Additionally, most participants (91%) reported feelings of restriction around food choice. Dissatisfaction with the diet was reported by half of the participants in the controlled dietary intervention conducted by Barrett et al.[88] Participants in this study provided suggestions for improving the intervention, such as potential small changes rather than diet overhauls that could be made by individuals.

We also assessed differences in intervention effectiveness across the life course. The dietary interventions made up the largest subset of interventions and had the widest representation of age groups included in the studies. In general, interventions appeared to be most effective in adults,[84,86] but there were also examples of ineffective interventions.[88,89] In children, results were mixed, and authors tended to report more issues with participant compliance.[100] Adolescents also reported difficulty in complying with dietary changes.[98] In one study of mother-child pairs, the intervention was more successful in the mothers.[106] There did not appear to be meaningful differences across the life course for medical exposure interventions, which were completed in adults in all except one study. Policy interventions overall were effective in reducing exposure in all age groups assessed, which ranged from 3 years[58] to $\sim$ 50 years of age.[127,133]

There are remaining gaps in the literature regarding interventions to reduce exposures to phthalates and bisphenols, from study design and approach to needed research on the barriers to adopting effective interventions more widely using implementation science frameworks.[152,156–158] Humans are still exposed to phthalates and BPA and increasingly to their respective lesser-known substitutes and analogues as indicated by studies in this review. Future work would be strengthened by including measurements of replacement chemicals for the bisphenols and phthalates to address the problem known as "regrettable substitution" when a toxic chemical or category of chemicals is removed from the market, only to be substituted by similar chemicals that may pose comparable concerns or be virtually untested for toxicity, beginning the cycle of exposures and adverse effects all over again.[159–161] None of the policy intervention studies could link biomarker levels with specific consumer products; therefore, source tracking along with human biomonitoring must be included in future research. It is important to include interdisciplinary research teams so that studies are better designed to ensure broader impact of the research. For example, nutrition researchers should be engaged in dietary intervention studies to incorporate evidence-based practices in controlled dietary studies. Poor compliance with dietary interventions was noted in several studies,[100,102] which could be addressed in future studies through the use of additional study designs and implementation strategies. Additionally, future studies would be strengthened by continued measurement of biomarkers following the intervention cessation to assess the feasibility of long-term modifications to reduce chemical exposure. Intervention length has been noted in recent reviews as an area for greater focus, especially in nutrition-related interventions[82]; long-term intervention studies are needed to understand the sustainability and effectiveness of interventions to better inform policy. We recommend that larger sample sizes and testing in diverse populations and settings will strengthen findings and improve generalizability as disparities and inequities have been reported for EDC exposure and health risks.[162–164]

The adaptation, uptake, sustainment, and spread of evidence-based interventions, practices, and policies that prevent or mitigate harmful exposures are increasingly recognized as priorities in the environmental health field.[165] Determining effective ways to facilitate this translation from research to action is a goal of implementation science which is defined as "the study of methods to promote the adoption and integration of evidence-based practices, interventions, and policies into routine health care and

public health settings to improve impact on population health."[73,166] In other words, implementation science deals with the challenges of translating intentions into desired changes. Our study contributes to these emerging discussions. We reviewed the efficacy of interventions to reduce bisphenol and phthalate exposures across a range of settings, age groups, and strategies. We identify a number of barriers to advancing dietary interventions such as cost/time/persistence at the individual level which may be a future focus for diet-specific implementation research. We recommend that further research on dietary interventions strive to gather and report more robust information on intervention delivery and compliance and aim to design interventions based on potential for implementation in real-world situations. This means having structured processes that explicitly incorporate context and are monitored during implementation. Better and consistent reporting on interventions, especially dietary intervention, based on recommended guidelines[167] would also facilitate comparisons across studies. Humans are also exposed to phthalates such as DEHP through medical devices, which are subjected to regulation by the FDA in the US. Certain populations such as patients with end-stage kidney disease and high-risk newborns are particularly heavily exposed.[168] However, policy level interventions more consistently reduced exposure. As such, it is our opinion that more efforts are needed at the policy level. For example, in the US, more coordination between the FDA, US Environmental Protection Agency (EPA), and the US Department of Agriculture is needed to decrease exposure to harmful EDCs, such as phthalates and bisphenols, for the American population. A chemical class or cumulative risk approach could result in a more efficient process to reduce exposures to multiple hazardous chemicals and, ultimately, reduce health risks.[169,170]

Strengths and Limitations of the Scoping Review

We followed PRISMA and JBI guidelines for conducting scoping reviews.[75,76] There is risk of publication bias impacting our findings, with studies reporting null results less likely to be published. However, we did find several studies reported findings where the intervention did not have a significant effect on all target chemicals,[88] and one intervention led to an increase in exposure.[89] While the number of intervention studies aimed at reducing EDC exposures in general is increasing and are the topic of other recent reviews,[81–83] our study focused specifically on phthalates and the most common bisphenols and examined multiple types of interventions, not just dietary or medical. We further did not limit our review to a specific stage in the life course. We believe the review is more comprehensive in this regard and enables a broader view of the state of the science for diverse interventions to reduce bisphenol and phthalate exposures.

Conclusions

In our review, evaluation of biomonitoring data post-policy implementation on restricting the use of phthalates and BPA in certain consumer products has shown dramatic downward trends in exposures.[58,127,129,130,132,133,136] Studies evaluating policies targeting the reduction of phthalates and BPA in goods and packaging reported larger and more consistent reductions in exposures to these EDCs and were less burdensome on individuals compared to dietary interventions. Interventions involving medical exposures, glove use, and personal care products led to reduced exposures to these bisphenols and phthalates; however, these interventions did not reduce exposure from the primary route—dietary.[24,25]

Armed with this knowledge, we propose the following call to action. An environmental health implementation science agenda on phthalates and bisphenols would provide more rigorous research that tests a broad range of strategies for encouraging policy change across federal, state, local, and international levels and how to prevent regrettable substitutions. This means studying the behavior of manufacturers, governmental policy decision-makers, and their interrelationships. Future interventions and policy changes should also consider how best to reduce phthalate and bisphenol exposure across the life course, as adults have been a primary focus of previous intervention studies and younger individuals may struggle with compliance without assistance or regular reminders. Consideration of the social determinants of health and collection of qualitative data will also be important when aiming to reach all individuals and implement interventions successfully. Such a research agenda would necessitate interdisciplinary collaborations to draw upon a wealth of theoretical perspectives.

Acknowledgments

Nicole Sieck was supported by NRT-INFEWS: UMD Global STEWARDS (STEM Training at the Nexus of Energy, WAter Reuse and FooD Systems) which was awarded to the University of Maryland School of Public Health by the National Science Foundation National Research Traineeship Program, Grant number 1828910.

References

1. Rosofsky A, Janulewicz P, Thayer KA, McClean M, Wise LA, Calafat AM, et al. 2017. Exposure to multiple chemicals in a cohort of reproductive-aged Danish women. Environ Res 154:73–85, PMID: 28039828, https://doi.org/10.1016/j.envres.2016.12.011.
2. Vandenberg LN, Chahoud I, Heindel JJ, Padmanabhan V, Paumgartten FJR, Schoenfelder G. 2010. Urinary, circulating, and tissue biomonitoring studies indicate widespread exposure to bisphenol A. Environ Health Perspect 118(8):1055–1070, https://doi.org/10.1289/ehp.0901716.
3. Rochester JR. 2013. Bisphenol a and human health: a review of the literature. Reprod Toxicol 42:132–155, PMID: 23994667, https://doi.org/10.1016/j.reprotox.2013.08.008.
4. Berger K, Eskenazi B, Kogut K, Parra K, Lustig RH, Greenspan LC, et al. 2018. Association of prenatal urinary concentrations of phthalates and bisphenol A and pubertal timing in boys and girls. Environ Health Perspect 126(9):97004, PMID: 30203993, https://doi.org/10.1289/EHP3424.
5. Watkins DJ, Sánchez BN, Téllez-Rojo MM, Lee JM, Mercado-García A, Blank-Goldenberg C, et al. 2017. Phthalate and bisphenol A exposure during in utero windows of susceptibility in relation to reproductive hormones and pubertal development in girls. Environ Res 159:143–151, PMID: 28800472, https://doi.org/10.1016/j.envres.2017.07.051.
6. Tran V, Tindula G, Huen K, Bradman A, Harley K, Kogut K, et al. 2017. Prenatal phthalate exposure and 8-isoprostane among Mexican-American children with high prevalence of obesity. J Dev Orig Health Dis 8(2):196–205, PMID: 28031075, https://doi.org/10.1017/S2040174416000763.
7. Harley KG, Berger K, Rauch S, Kogut K, Claus Henn B, Calafat AM, et al. 2017. Association of prenatal urinary phthalate metabolite concentrations and childhood BMI and obesity. Pediatr Res 82(3):405–415, PMID: 28426647, https://doi.org/10.1038/pr.2017.112.
8. Hoepner LA, Whyatt RM, Widen EM, Hassoun A, Oberfield SE, Mueller NT, et al. 2016. Bisphenol A and adiposity in an inner-city birth cohort. Environ Health Perspect 124(10):1644–1650, PMID: 27187982, https://doi.org/10.1289/EHP205.
9. Heggeseth BC, Holland N, Eskenazi B, Kogut K, Harley KG. 2019. Heterogeneity in childhood body mass trajectories in relation to prenatal phthalate exposure. Environ Res 175:22–33, PMID: 31102947, https://doi.org/10.1016/j.envres.2019.04.036.
10. Hyland C, Mora AM, Kogut K, Calafat AM, Harley K, Deardorff J, et al. 2019. Prenatal exposure to phthalates and neurodevelopment in the CHAMACOS cohort. Environ Health Perspect 127(10):107010, PMID: 31652105, https://doi.org/10.1289/EHP5165.
11. Balalian AA, Whyatt RM, Liu X, Insel BJ, Rauh VA, Herbstman J, et al. 2019. Prenatal and childhood exposure to phthalates and motor skills at age 11 years. Environ Res 171:416–427, PMID: 30731329, https://doi.org/10.1016/j.envres.2019.01.046.
12. Engel SM, Patisaul HB, Brody C, Hauser R, Zota AR, Bennet DH, et al. 2021. Neurotoxicity of ortho-phthalates: recommendations for critical policy reforms to protect brain development in children. Am J Public Health 111(4):687–695, PMID: 33600256, https://doi.org/10.2105/AJPH.2020.306014.

13. Ejaredar M, Nyanza EC, Ten Eycke K, Dewey D. 2015. Phthalate exposure and childrens neurodevelopment: a systematic review. Environ Res 142:51–60, PMID: 26101203, https://doi.org/10.1016/j.envres.2015.06.014.
14. Kobrosly RW, Evans S, Miodovnik A, Barrett ES, Thurston SW, Calafat AM, et al. 2014. Prenatal phthalate exposures and neurobehavioral development scores in boys and girls at 6–10 years of age. Environ Health Perspect 122(5):521–528, PMID: 24577876, https://doi.org/10.1289/ehp.1307063.
15. Zhou A, Chang H, Huo W, Zhang B, Hu J, Xia W, et al. 2017. Prenatal exposure to bisphenol A and risk of allergic diseases in early life. Pediatr Res 81(6):851–856, PMID: 28141789, https://doi.org/10.1038/pr.2017.20.
16. Berger K, Eskenazi B, Balmes J, Kogut K, Holland N, Calafat AM, et al. 2019. Prenatal high molecular weight phthalates and bisphenol A, and childhood respiratory and allergic outcomes. Pediatr Allergy Immunol 30(1):36–46, PMID: 30338578, https://doi.org/10.1111/pai.12992.
17. Braun JM. 2017. Early-life exposure to EDCs: role in childhood obesity and neurodevelopment. Nat Rev Endocrinol 13(3):161–173, PMID: 27857130, https://doi.org/10.1038/nrendo.2016.186.
18. Khodasevich D, Holland N, Hubbard A, Harley K, Deardorff J, Eskenazi B, et al. 2023. Associations between prenatal phthalate exposure and childhood epigenetic age acceleration. Environ Res 231(pt 1):116067, PMID: 37149020, https://doi.org/10.1016/j.envres.2023.116067.
19. Kang JH, Kondo F, Katayama Y. 2006. Human exposure to bisphenol A. Toxicology 226(2–3):79–89, PMID: 16860916, https://doi.org/10.1016/j.tox.2006.06.009.
20. Noonan GO, Ackerman LK, Begley TH. 2011. Concentration of bisphenol A in highly consumed canned foods on the U.S. market. J Agric Food Chem 59(13):7178–7185, PMID: 21598963, https://doi.org/10.1021/jf201076f.
21. Park J-S, Kim S, Park M, Kim Y, Lee H, Choi H, et al. 2017. Relationship between dietary factors and bisphenol A exposure: the second Korean national environmental health survey (KoNEHS 2012–2014). Ann Occup Environ Med 29:42, PMID: 29075503, https://doi.org/10.1186/s40557-017-0200-1.
22. Löfroth M, Ghasemimehr M, Falk A, von Steyern PV. 2019. Bisphenol A in dental materials - existence, leakage and biological effects. Heliyon 5(5):e01711, PMID: 31193754, https://doi.org/10.1016/j.heliyon.2019.e01711.
23. Pacyga DC, Sathyanarayana S, Strakovsky RS. 2019. Dietary predictors of phthalate and bisphenol exposures in pregnant women. Adv Nutr 10(5):803–815, PMID: 31144713, https://doi.org/10.1093/advances/nmz029.
24. Huang RP, Liu ZH, Yin H, Dang Z, Wu PX, Zhu NW, et al. 2018. Bisphenol A concentrations in human urine, human intakes across six continents, and annual trends of average intakes in adult and child populations worldwide: a thorough literature review. Sci Total Environ 626:971–981, PMID: 29898562, https://doi.org/10.1016/j.scitotenv.2018.01.144.
25. Geens T, Aerts D, Berthot C, Bourguignon JP, Goeyens L, Lecomte P, et al. 2012. A review of dietary and non-dietary exposure to bisphenol-A. Food Chem Toxicol 50(10):3725–3740, PMID: 22889897, https://doi.org/10.1016/j.fct.2012.07.059.
26. Martínez MA, Rovira J, Prasad Sharma R, Nadal M, Schuhmacher M, Kumar V. 2018. Comparing dietary and non-dietary source contribution of BPA and DEHP to prenatal exposure: a Catalonia (Spain) case study. Environ Res 166:25–34, PMID: 29859370, https://doi.org/10.1016/j.envres.2018.05.008.
27. Liao C, Liu F, Kannan K. 2012. Bisphenol S, a new bisphenol analogue, in paper products and currency bills and its association with bisphenol a residues. Environ Sci Technol 46(12):6515–6522, PMID: 22591511, https://doi.org/10.1021/es300876n.
28. Wu L-H, Zhang X-M, Wang F, Gao C-J, Chen D, Palumbo JR, et al. 2018. Occurrence of bisphenol S in the environment and implications for human exposure: a short review. Sci Total Environ 615:87–98, PMID: 28963899, https://doi.org/10.1016/j.scitotenv.2017.09.194.
29. Rochester JR, Bolden AL. 2015. Bisphenol S and F: a systematic review and comparison of the hormonal activity of bisphenol a substitutes. Environ Health Perspect 123(7):643–650, PMID: 25775505, https://doi.org/10.1289/ehp.1408989.
30. Gallart-Ayala H, Moyano E, Galceran MT. 2011. Analysis of bisphenols in soft drinks by on-line solid phase extraction fast liquid chromatography–tandem mass spectrometry. Anal Chim Acta 683(2):227–233, PMID: 21167975, https://doi.org/10.1016/j.aca.2010.10.034.
31. Chen D, Kannan K, Tan H, Zheng Z, Feng Y-L, Wu Y, et al. 2016. Bisphenol analogues other than BPA: environmental occurrence, human exposure, and toxicity: a review. Environ Sci Technol 50(11):5438–5453, PMID: 27143250, https://doi.org/10.1021/acs.est.5b05387.
32. Liao C, Kannan K. 2014. A survey of alkylphenols, bisphenols, and triclosan in personal care products from China and the United States. Arch Environ Contam Toxicol 67(1):50–59, PMID: 24639116, https://doi.org/10.1007/s00244-014-0016-8.
33. Liao C, Kannan K. 2013. Concentrations and profiles of bisphenol A and other bisphenol analogues in foodstuffs from the United States and their implications for human exposure. J Agric Food Chem 61(19):4655–4662, PMID: 23614805, https://doi.org/10.1021/jf400445n.
34. Nerin C, Canellas E, Vera P. 2016. Plasticizer migration into foods. In: *Reference Module in Food Science*. Amsterdam, Netherlands: Elsevier Inc. https://doi.org/10.1016/B978-0-08-100596-5.21392-9.
35. Erythropel HC, Maric M, Nicell JA, Leask RL, Yargeau V. 2014. Leaching of the plasticizer di(2-ethylhexyl)phthalate (DEHP) from plastic containers and the question of human exposure. Appl Microbiol Biotechnol 98(24):9967–9981, PMID: 25376446, https://doi.org/10.1007/s00253-014-6183-8.
36. CDC (Centers for Disease Control and Prevention). 2021. *Fourth Report on Human Exposure to Environmental Chemicals, Updated Tables*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. https://www.cdc.gov/exposurereport/ [accessed 8 December 2021].
37. Health Canada. 2019. *Fifth Report on Human Biomonitoring of Environmental Chemicals*. https://www.canada.ca/en/health-canada/services/environmental-workplace-health/reports-publications/environmental-contaminants/fifth-report-human-biomonitoring.html [accessed 17 February 2022].
38. Schecter A, Lorber M, Guo Y, Wu Q, Yun SH, Kannan K, et al. 2013. Phthalate concentrations and dietary exposure from food purchased in New York state. Environ Health Perspect 121(4):473–494, PMID: 23461894, https://doi.org/10.1289/ehp.1206367.
39. Carwile JL, Luu HT, Bassett LS, Driscoll DA, Yuan C, Chang JY, et al. 2009. Polycarbonate bottle use and urinary bisphenol a concentrations. Environ Health Perspect 117(9):1368–1372, PMID: 19750099, https://doi.org/10.1289/ehp.0900604.
40. Geueke B, Muncke J. 2018. Substances of very high concern in food contact materials: migration and regulatory background. Packag Technol Sci 31(12):757–769, https://doi.org/10.1002/pts.2288.
41. Hammel SC, Levasseur JL, Hoffman K, Phillips AL, Lorenzo AM, Calafat AM, et al. 2019. Children's exposure to phthalates and non-phthalate plasticizers in the home: the TESIE study. Environ Int 132:105061, PMID: 31400598, https://doi.org/10.1016/j.envint.2019.105061.
42. Philippat C, Bennett D, Calafat AM, Picciotto IH. 2015. Exposure to select phthalates and phenols through use of personal care products among Californian adults and their children. Environ Res 140:369–376, PMID: 25929801, https://doi.org/10.1016/j.envres.2015.04.009.
43. Bennett D, Bellinger DC, Birnbaum LS, Bradman A, Chen A, Cory-Slechta DA, et al. 2016. Project TENDR: targeting environmental neuro-developmental risks the TENDR consensus statement. Environ Health Perspect 124(7):A118–A122, PMID: 27479987, https://doi.org/10.1289/EHP358.
44. Healthcare Without Harm. 2019. *Non-Toxic Healthcare: Alternatives to Phthalates and Bisphenol A in Medical Devices*. https://noharm-uscanada.org/documents/non-toxic-healthcare-alternatives-phthalates-and-bisphenol-medical-devices-2nd-edition [accessed 22 February 2023].
45. Collaborative for Health & Environment. 2016. *Bisphenol A*. https://www.healthandenvironment.org/environmental-health/environmental-risks/chemical-environment-overview/bpa [accessed 22 February 2023].
46. EWG (Environmental Working Group). 2021. *Six Tips to Avoid Phthalates After Study Highlights Health Harms, Billion-Dollar Costs*. https://www.ewg.org/news-insights/news/2021/10/six-tips-avoid-phthalates-after-study-highlights-health-harms-billion [accessed 1 August 2023].
47. Trasande L, Shaffer RM, Sathyanarayana S, Council on Environmental Health. 2018. Food additives and child health. Pediatrics 142(2):e20181410, PMID: 30037972, https://doi.org/10.1542/peds.2018-1410.
48. FIGO (The International Federation of Gynecology and Obstetrics). 2015. *Global OB-GYN Group Urges Greater Efforts to Prevent Toxic Chemical Exposure*. https://www.figo.org/news/congress-news-global-ob-gyn-group-urges-greater-efforts-prevent-toxic-chemical-exposure [accessed 31 July 2023].
49. Endocrine Society. 2022. *Endocrine-Disrupting Chemicals*. https://www.endocrine.org/advancing-research/scientific-statements/endocrine-disrupting-chemicals [accessed 1 August 2023].
50. U.S. Congress. 2008. *Consumer Product Safety Improvement Act of 2008. Pub L No. 110-314 (110th Congress, 14 August 2008)*. https://www.cpsc.gov/s3fs-public/cpsia.pdf [accessed 14 November 2021].
51. U.S. CPSC (Consumer Product Safety Commission). 2017. *Prohibition of Children's Toys and Child Care Articles Containing Specified Phthalates: Determinations Regarding Certain Plastics. 82 FR 41163*. https://www.federalregister.gov/documents/2017/08/30/2017-18387/prohibition-of-childrens-toys-and-child-care-articles-containing-specified-phthalates-determinations [accessed 8 December 2021].
52. FDA (Food and Drug Administration). 2012. *Indirect Food Additives: Polymers. 77 FR 41899*. https://www.federalregister.gov/documents/2012/07/17/2012-17366/indirect-food-additives-polymers [accessed 14 November 2021].
53. Government of Canada. 2010. *Canada Consumer Product Safety Act*. https://laws-lois.justice.gc.ca/eng/acts/C-1.68/FullText.html [accessed 14 November 2021].
54. ECHA (European Chemicals Agency). 2022. *Authorisation List*. https://echa.europa.eu/authorisation-list [accessed 9 May 2022].

55. Pollock T, Karthikeyan S, Walker M, Werry K, St-Amand A. 2021. Trends in environmental chemical concentrations in the Canadian population: biomonitoring data from the Canadian health measures survey 2007–2017. Environ Int 155:106678, PMID: 34118655, https://doi.org/10.1016/j.envint.2021.106678.
56. LaKind JS, Naiman DQ. 2015. Temporal trends in bisphenol A exposure in the United States from 2003–2012 and factors associated with BPA exposure: spot samples and urine dilution complicate data interpretation. Environ Res 142:84–95, PMID: 26121292, https://doi.org/10.1016/j.envres.2015.06.013.
57. Jung SK, Choi W, Kim SY, Hong S, Jeon HL, Joo Y, et al. 2022. Profile of environmental chemicals in the Korean population—results of the Korean national environmental health survey (KoNEHS) cycle 3, 2015–2017. Int J Environ Res Public Health 19(2):626, PMID: 35055445, https://doi.org/10.3390/ijerph19020626.
58. Tschersich C, Murawski A, Schwedler G, Rucic E, Moos RK, Kasper-Sonnenberg M, et al. 2021. Bisphenol A and six other environmental phenols in urine of children and adolescents in Germany –human biomonitoring results of the German environmental survey 2014–2017 (GerES V). Sci Total Environ 763:144615, PMID: 33383503, https://doi.org/10.1016/j.scitotenv.2020.144615.
59. Gennings C, Hauser R, Koch HM, Kortenkamp A, Lioy PJ, Mirkes PE, et al. 2014. *Report to the U.S. Consumer Products Safety Commission by the Chronic Hazard Advisory Panel on Phthalates and Phthalate Alternatives*. https://www.cpsc.gov/s3fs-public/CHAP-REPORT-With-Appendices.pdf [accessed 8 December 2021].
60. Karrer C, de Boer W, Delmaar C, Cai YP, Crepet A, Hungerbuhler K, et al. 2019. Linking probabilistic exposure and pharmacokinetic modeling to assess the cumulative risk from the bisphenols BPA, BPS, BPF, and BPAF for Europeans. Environ Sci Technol 53(15):9181–9191, PMID: 31294980, https://doi.org/10.1021/acs.est.9b01749.
61. Li JF, Zhang WX, Zhou YQ, Shi JC, Xia W, Xu SQ, et al. 2021. Cumulative health risks for bisphenols using the maximum cumulative ratio among Chinese pregnant women. Environ Pollut 270:116044, PMID: 33261967, https://doi.org/10.1016/j.envpol.2020.116044.
62. Reyes JM, Price PS. 2018. Temporal trends in exposures to six phthalates from biomonitoring data: implications for cumulative risk. Environ Sci Technol 52(21):12475–12483, PMID: 30272963, https://doi.org/10.1021/acs.est.8b03338.
63. National Research Council. 2008. *Phthalates and Cumulative Risk Assessment: The Tasks Ahead*. Washington, DC: The National Academies Press.
64. Zota AR, Calafat AM, Woodruff TJ. 2014. Temporal trends in phthalate exposures: findings from the national health and nutrition examination survey, 2001–2010. Environ Health Perspect 122(3):235–241, PMID: 24425099, https://doi.org/10.1289/ehp.1306681.
65. Calafat AM, Valentin-Blasini L, Ye X. 2015. Trends in exposure to chemicals in personal care and consumer products. Curr Environ Health Rep 2(4):348–355, PMID: 26342608, https://doi.org/10.1007/s40572-015-0065-9.
66. Nguyen VK, Kahana A, Heidt J, Polemi K, Kvasnicka J, Jolliet O, et al. 2020. A comprehensive analysis of racial disparities in chemical biomarker concentrations in United States women, 1999–2014. Environ Int 137:105496, PMID: 32113086, https://doi.org/10.1016/j.envint.2020.105496.
67. Park C, Hwang M, Baek Y, Jung S, Lee Y, Paek D, et al. 2019. Urinary phthalate metabolite and bisphenol a levels in the Korean adult population in association with sociodemographic and behavioral characteristics: Korean national environmental health survey (KoNEHS) 2012–2014. Int J Hyg Environ Health 222(5):903–910, PMID: 30773337, https://doi.org/10.1016/j.ijheh.2019.02.003.
68. Unal E, Lynn T, Neidich J, Salazar D, Goetzl L, Baatz JE, et al. 2012. Racial disparity in maternal and fetal-cord bisphenol a concentrations. J Perinatol 32(11):844–850, PMID: 22402483, https://doi.org/10.1038/jp.2012.12.
69. Endocrine Society. 2023. *What You Can Do About EDCs*. https://www.endocrine.org/topics/edc/what-you-can-do [accessed 22 February 2023].
70. Environmental Working Group. 2022. *EWG's Guide to Endocrine Disruptors*. https://www.ewg.org/consumer-guides/ewgs-guide-endocrine-disruptors [accessed 25 February 2023].
71. Zizzu A, 2016. *9 Ways to Avoid Hormone-Disrupting Chemicals*. New York, NY: Natural Resources Defense Council. https://www.nrdc.org/stories/9-ways-avoid-hormone-disrupting-chemicals [accessed 24 February 2023].
72. Rosenthal J, Arku RE, Baumgartner J, Brown J, Clasen T, Eisenberg JNS, et al. 2020. Systems science approaches for global environmental health research: enhancing intervention design and implementation for household air pollution (HAP) and water, sanitation, and hygiene (WASH) programs. Environ Health Perspect 128(10):105001, PMID: 33035121, https://doi.org/10.1289/EHP7010.
73. Bauer MS, Damschroder L, Hagedorn H, Smith J, Kilbourne AM. 2015. An introduction to implementation science for the non-specialist. BMC Psychol 3(1):32, PMID: 26376626, https://doi.org/10.1186/s40359-015-0089-9.
74. NIEHS (National Institute of Environmental Health Sciences). 2022. *Advancing Environmental Health Equity Through Implementation Science*. https://tools.niehs.nih.gov/conference/eheworkshop2022/ [accessed 17 February 2022].
75. Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Levac D, et al. 2018. PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. Ann Intern Med 169(7):467–473, PMID: 30178033, https://doi.org/10.7326/M18-0850.
76. Peters MDJ, Godfrey C, McInerney P, Munn Z, Tricco A, Khalil H. 2020. Chapter 11: scoping reviews. In: *JBI Manual for Evidence Synthesis*. Aromataris E, Munn Z, eds. Adelaide, Australia: JBI.
77. Munn Z, Peters MDJ, Stern C, Tufanaru C, McArthur A, Aromataris E. 2018. Systematic review or scoping review? Guidance for authors when choosing between a systematic or scoping review approach. BMC Med Res Methodol 18(1):143, PMID: 30453902, https://doi.org/10.1186/s12874-018-0611-x.
78. Munn Z, Pollock D, Khalil H, Alexander L, Mclnerney P, Godfrey CM, et al. 2022. What are scoping reviews? Providing a formal definition of scoping reviews as a type of evidence synthesis. JBI Evid Synth 20(4):950–952, PMID: 35249995, https://doi.org/10.11124/JBIES-21-00483.
79. Higgins JPT, Thomas J, Chandler J, Cumpston M, Li T, Page MJ, et al. 2019. *Cochrane Handbook for Systematic Reviews of Interventions Version 6.0 (updated July 2019)*. London, UK: Cochrane. www.training.cochrane.org/handbook.
80. Kohl C, McIntosh EJ, Unger S, Haddaway NR, Kecke S, Schiemann J, et al. 2018. Online tools supporting the conduct and reporting of systematic reviews and systematic maps: a case study on CADIMA and review of existing tools. Environ Evidence 7(1):8.
81. Yang TC, Jovanovic N, Chong F, Worcester M, Sakhi AK, Thomsen C, et al. 2023. Interventions to reduce exposure to synthetic phenols and phthalates from dietary intake and personal care products: a scoping review. Curr Environ Health Rep 10(2):184–214, PMID: 36988899, https://doi.org/10.1007/s40572-023-00394-8.
82. Martin L, Zhang Y, First O, Mustieles V, Dodson R, Rosa G, et al. 2022. Lifestyle interventions to reduce endocrine-disrupting phthalate and phenol exposures among reproductive age men and women: a review and future steps. Environ Int 170:107576, PMID: 36283156, https://doi.org/10.1016/j.envint.2022.107576.
83. Park J, Lee H, Lee S, Lee H. 2022. Interventions on reducing exposure to endocrine disrupting chemicals in human health care context: a scoping review. Risk Manag Healthc Policy 15:779–791, PMID: 35502443, https://doi.org/10.2147/RMHP.S358561.
84. Peng CY, Tsai EM, Kao TH, Lai TC, Liang SS, Chiu CC, et al. 2019. Canned food intake and urinary bisphenol A concentrations: a randomized crossover intervention study. Environ Sci Pollut Res Int 26(27):27999–28009, PMID: 31352597, https://doi.org/10.1007/s11356-019-05534-y.
85. Carwile JL, Ye X, Zhou X, Calafat AM, Michels KB. 2011. Canned soup consumption and urinary bisphenol A: a randomized crossover trial. JAMA 306(20):2218–2220, https://doi.org/10.1001/jama.2011.1721.
86. Rudel RA, Gray JM, Engel CL, Rawsthorne TW, Dodson RE, Ackerman JM, et al. 2011. Food packaging and bisphenol A and bis(2-ethyhexyl) phthalate exposure: findings from a dietary intervention. Environ Health Perspect 119(7):914–920, PMID: 21450549, https://doi.org/10.1289/ehp.1003170.
87. Ackerman JM, Dodson RE, Engel CL, Gray JM, Rudel RA. 2014. Temporal variability of urinary di(2-ethylhexyl) phthalate metabolites during a dietary intervention study. J Expo Sci Environ Epidemiol 24(6):595–601, PMID: 24448002, https://doi.org/10.1038/jes.2013.93.
88. Barrett ES, Velez M, Qiu X, Chen SR. 2015. Reducing prenatal phthalate exposure through maternal dietary changes: results from a pilot study. Matern Child Health J 19(9):1936–1942, PMID: 25652062, https://doi.org/10.1007/s10995-015-1707-0.
89. Sathyanarayana S, Alcedo G, Saelens BE, Zhou C, Dills RL, Yu J, et al. 2013. Unexpected results in a randomized dietary trial to reduce phthalate and bisphenol A exposures. J Expo Sci Environ Epidemiol 23(4):378–384, PMID: 23443238, https://doi.org/10.1038/jes.2013.9.
90. Sessa F, Polito R, Monda V, Scarinci A, Salerno M, Carotenuto M, et al. 2021. Effects of a plastic-free lifestyle on urinary bisphenol a levels in school-aged children of Southern Italy: a pilot study. Front Public Health 9:626070, PMID: 33598445, https://doi.org/10.3389/fpubh.2021.626070.
91. Li X, Ying GG, Zhao JL, Chen ZF, Lai HJ, Su HC. 2013. 4-Nonylphenol, bisphenol-A and triclosan levels in human urine of children and students in China, and the effects of drinking these bottled materials on the levels. Environ Int 52:81–86, PMID: 21794921, https://doi.org/10.1016/j.envint.2011.03.026.
92. Bae S, Hong YC. 2015. Exposure to bisphenol A from drinking canned beverages increases blood pressure: randomized crossover trial. Hypertension 65(2):313–319, PMID: 25489056, https://doi.org/10.1161/HYPERTENSIONAHA.114.04261.
93. Sakaki JR, Provatas AA, Perkins C, Chun OK. 2022. Urinary excretion of estrogenic chemicals following consumption of capsule coffee and French press coffee: a crossover study. Toxicol Rep 9:728–734, PMID: 36518418, https://doi.org/10.1016/j.toxrep.2022.03.024.
94. Ji K, Kho YL, Park Y, Choi K. 2010. Influence of a five-day vegetarian diet on urinary levels of antibiotics and phthalate metabolites: a pilot study with "temple stay" participants. Environ Res 110(4):375–382, PMID: 20227070, https://doi.org/10.1016/j.envres.2010.02.008.

95. Correia-Sá L, Kasper-Sonnenberg M, Pälmke C, Schütze A, Norberto S, Calhau C, et al. 2018. Obesity or diet? Levels and determinants of phthalate body burden–a case study on Portuguese children. Int J Hyg Environ Health 221(3):519–530, https://doi.org/10.1016/j.ijheh.2018.02.001.
96. Correia-Sá L, Kasper-Sonnenberg M, Schütze A, Pälmke C, Norberto S, Calhau C, et al. 2017. Exposure assessment to bisphenol A (BPA) in Portuguese children by human biomonitoring. Environ Sci Pollut Res Int 24(35):27502–27514, PMID: 28980160, https://doi.org/10.1007/s11356-017-0358-7.
97. Dallio M, Masarone M, Errico S, Gravina AG, Nicolucci C, Di Sarno R, et al. 2018. Role of bisphenol A as environmental factor in the promotion of non-alcoholic fatty liver disease: in vitro and clinical study. Aliment Pharmacol Ther 47(6):826–837, PMID: 29322544, https://doi.org/10.1111/apt.14499.
98. Galloway TS, Baglin N, Lee BP, Kocur AL, Shepherd MH, Steele AM, et al. 2018. An engaged research study to assess the effect of a 'real-world' dietary intervention on urinary bisphenol A (BPA) levels in teenagers. BMJ Open 8(2): e018742, PMID: 29431133, https://doi.org/10.1136/bmjopen-2017-018742.
99. Hutter HP, Kundi M, Hohenblum P, Scharf S, Shelton JF, Piegler K, et al. 2016. Life without plastic: a family experiment and biomonitoring study. Environ Res 150:639–644, PMID: 27235111, https://doi.org/10.1016/j.envres.2016.05.028.
100. Chen C-Y, Chou Y-Y, Lin S-J, Lee C-C. 2015. Developing an intervention strategy to reduce phthalate exposure in Taiwanese girls. Sci Total Environ 517:125–131, PMID: 25725197, https://doi.org/10.1016/j.scitotenv.2015.02.021.
101. Kim S, Lee I, Lim JE, Lee A, Moon HB, Park J, et al. 2020. Dietary contribution to body burden of bisphenol A and bisphenol S among mother-children pairs. Sci Total Environ 744:140856, PMID: 32721674, https://doi.org/10.1016/j.scitotenv.2020.140856.
102. Park S, Chung C. 2021. Effects of a dietary modification intervention on menstrual pain and urinary BPA levels: a single group clinical trial. BMC Womens Health 21(1):58, PMID: 33563271, https://doi.org/10.1186/s12905-021-01199-3.
103. Wu W, Cao L, Zheng T-T, Feng S-Y, Ma G-W, He Y-Y, et al. 2021. Prenatal phthalate exposure reduction through an integrated intervention strategy. Environ Sci Pollut Res Int 28(40):57183–57191, PMID: 34085200, https://doi.org/10.1007/s11356-021-14613-y.
104. Rutkowska A, Olsson A, Piotrowska-Szypryt M, Namieśnik J. 2020. Changes in daily life reduce indoor exposure to selected endocrine disruptors in the home environment: a pilot intervention study. Acta Biochim Pol 67(2):273–276, PMID: 32558529, https://doi.org/10.18388/abp.2020_5369.
105. Pirard C, Charlier C. 2022. Urinary levels of parabens, phthalate metabolites, bisphenol A and plasticizer alternatives in a Belgian population: time trend or impact of an awareness campaign? Environ Res 214(pt 2):113852, PMID: 35820649, https://doi.org/10.1016/j.envres.2022.113852.
106. Kim JH, Kwak JM, Kang H. 2021. Web-based behavioral intervention to reduce exposure to phthalate metabolites, bisphenol A, triclosan, and parabens in mothers with young children: a randomized controlled trial. Int J Hyg Environ Health 236:113798, PMID: 34186503, https://doi.org/10.1016/j.ijheh.2021.113798.
107. Hagobian T, Smouse A, Streeter M, Wurst C, Schaffner A, Phelan S. 2017. Randomized intervention trial to decrease bisphenol A urine concentrations in women: pilot study. J Womens Health (Larchmt) 26(2):128–132, PMID: 27726525, https://doi.org/10.1089/jwh.2016.5746.
108. Hagobian T, Delli-Bovi Z, Mercado A, Bird A, Guy M, Phelan S. 2021. Development and feasibility of randomized trial to reduce urinary bisphenols in women with obesity. Pilot Feasibility Stud 7(1):24, PMID: 33436090, https://doi.org/10.1186/s40814-020-00744-5.
109. El Ouazzani H, Fortin S, Venisse N, Dupuis A, Rouillon S, Cambien G, et al., 2022. Perinatal environmental health education intervention to reduce exposure to endocrine disruptors: the PREVED project. Int J Environ Res Public Health 19(1):70, PMID: 35010328, https://doi.org/10.3390/ijerph19010070.
110. Takahashi Y, Shibata T, Sasaki Y, Fujii H, Bito Y, Suehiro S. 2008. Di(2-ethylhexyl) phthalate exposure during cardiopulmonary bypass. Asian Cardiovasc Thorac Ann 16(1):4–6, PMID: 18245696, https://doi.org/10.1177/021849230801600102.
111. Takahashi Y, Shibata T, Sasaki Y, Fujii H, Ikuta T, Bito Y, et al. 2009. Impact of non-di-(2-ethylhexyl)phthalate cardiopulmonary bypass tubes on inflammatory cytokines and coagulation-fibrinolysis systems during cardiopulmonary bypass. J Artif Organs 12(4):226–231, PMID: 20035395, https://doi.org/10.1007/s10047-009-0477-0.
112. Zimmerman-Downs JM, Shuman D, Stull SC, Ratzlaff RE. 2010. Bisphenol a blood and saliva levels prior to and after dental sealant placement in adults. J Dent Hyg 84(3):145–150, PMID: 20579427.
113. Kambia K, Dine T, Azar R, Gressier B, Luyckx M, Brunet C. 2001. Comparative study of the leachability of di (2-ethylhexyl) phthalate and tri (2-ethylhexyl) trimellitate from haemodialysis tubing. Int J Pharm 229(1–2):139–146, PMID: 11604266, https://doi.org/10.1016/s0378-5173(01)00840-7.
114. Bosch-Panadero E, Mas S, Sanchez-Ospina D, Camarero V, Pérez-Gómez MV, Saez-Calero I, et al. 2016. The choice of hemodialysis membrane affects bisphenol a levels in blood. J Am Soc Nephrol 27(5):1566–1574, PMID: 26432902, https://doi.org/10.1681/ASN.2015030312.
115. Mettang T, Pauli-Magnus C, Alscher DM, Kirchgessner J, Wodarz R, Rettenmeier AW, et al. 2000. Influence of plasticizer-free CAPD bags and tubings on serum, urine, and dialysate levels of phthalic acid esters in CAPD patients. Perit Dial Int 20(1):80–84, PMID: 10716589.
116. Murakami K, Ohashi A, Hori H, Hibiya M, Shoji Y, Kunisaki M, et al. 2007. Accumulation of bisphenol A in hemodialysis patients. Blood Purif 25(3):290–294, PMID: 17622711, https://doi.org/10.1159/000104869.
117. Shintani H. 2001. Determination of the endocrine disrupter bisphenol-A in the blood of uremia patients treated by dialysis. Chromatographia 53(5–6):331–333, https://doi.org/10.1007/BF02490435.
118. Mas S, Bosch-Panadero E, Abaigar P, Camarero V, Mahillo I, Civantos E, et al. 2018. Influence of dialysis membrane composition on plasma bisphenol A levels during online hemodiafiltration. PLoS One 13(3):e0193288, PMID: 29529055, https://doi.org/10.1371/journal.pone.0193288.
119. Quiroga B, Bosch RJ, Fiallos RA, Sánchez-Heras M, Olea-Herrero N, López-Aparicio P, et al. 2017. Online hemodiafiltration reduces bisphenol A levels. Ther Apher Dial 21(1):96–101, PMID: 27995744, https://doi.org/10.1111/1744-9987.12475.
120. Leuenberger N, Barras L, Nicoli R, Robinson N, Baume N, Lion N, et al. 2016. Urinary di-(2-ethylhexyl) phthalate metabolites for detecting transfusion of autologous blood stored in plasticizer-free bags. Transfusion 56(3):571–578, PMID: 26581034, https://doi.org/10.1111/trf.13408.
121. Vanhorebeek I, Malarvannan G, Güiza F, Poma G, Derese I, Wouters PJ, et al. 2022. Phasing out DEHP from plastic indwelling medical devices used for intensive care: does it reduce the long-term attention deficit of critically ill children? Environ Int 158:106962, PMID: 34739923, https://doi.org/10.1016/j.envint.2021.106962.
122. Seifi S, Mirzakouchaki B, Rafighi A, Aghanejad A, Hamidi AA, Shahrbaf S. 2022. Evaluation of the bisphenol released in the saliva after residual adhesive removal in orthodontic patients by using ultrasonic scaling and rotary system: a single-center randomized clinical trial. Am J Orthod Dentofacial Orthop 163(2):148–153, PMID: 36396531, https://doi.org/10.1016/j.ajodo.2022.06.023.
123. Lee I, Kim S, Kim KT, Kim S, Park S, Lee H, et al. 2018. Bisphenol A exposure through receipt handling and its association with insulin resistance among female cashiers. Environ Int 117:268–275, PMID: 29778011, https://doi.org/10.1016/j.envint.2018.05.013.
124. Ehrlich S, Calafat AM, Humblet O, Smith T, Hauser R. 2014. Handling of thermal receipts as a source of exposure to bisphenol A. JAMA 311(8):859–860, PMID: 24570250, https://doi.org/10.1001/jama.2013.283735.
125. Harley KG, Kogut K, Madrigal DS, Cardenas M, Vera IA, Meza-Alfaro G, et al. 2016. Reducing phthalate, paraben, and phenol exposure from personal care products in adolescent girls: findings from the HERMOSA intervention study. Environ Health Perspect 124(10):1600–1607, PMID: 26947464, https://doi.org/10.1289/ehp.1510514.
126. Sears CG, Lanphear BP, Calafat AM, Chen A, Skarha J, Xu Y, et al. 2020. Lowering urinary phthalate metabolite concentrations among children by reducing contaminated dust in housing units: a randomized controlled trial and observational study. Environ Sci Technol 54(7):4327–4335, PMID: 32101426, https://doi.org/10.1021/acs.est.9b04898.
127. Shin HM, Dhar U, Calafat AM, Nguyen V, Schmidt RJ, Hertz-Picciotto I. 2020. Temporal trends of exposure to phthalates and phthalate alternatives in California pregnant women during 2007–2013: comparison with other populations. Environ Sci Technol 54(20):13157–13166, PMID: 32940456, https://doi.org/10.1021/acs.est.0c03857.
128. Kim K, Shin HM, Busgang SA, Boyd Barr E, Panuwet P, Schmidet RJ, et al. 2021. Temporal trends of phenol, paraben, and triclocarban exposure in California pregnant women during 2007–2014. Environ Sci Technol 55(16):11155–11165, PMID: 34347462, https://doi.org/10.1021/acs.est.1c01564.
129. Bastiaensen M, Gys C, Colles A, Malarvannan G, Verheyen V, Koppen G, et al. 2021. Biomarkers of phthalates and alternative plasticizers in the Flemish environment and health study (FLEHS IV): time trends and exposure assessment. Environ Pollut 276:116724, PMID: 33631684, https://doi.org/10.1016/j.envpol.2021.116724.
130. Schoeters G, Govarts E, Bruckers L, Den Hond E, Nelen V, De Henauw S, et al. 2017. Three cycles of human biomonitoring in Flanders - time trends observed in the Flemish environment and health study. Int J Hyg Environ Health 220(2 pt A):36–45, PMID: 28160993, https://doi.org/10.1016/j.ijheh.2016.11.006.
131. Frederiksen H, Nielsen O, Koch HM, Skakkebaek NE, Juul A, Jørgensen N, et al. 2020. Changes in urinary excretion of phthalates, phthalate substitutes, bisphenols and other polychlorinated and phenolic substances in young Danish men; 2009–2017. Int J Hyg Environ Health 223(1):93–105, https://doi.org/10.1016/j.ijheh.2019.10.002.
132. Göen T, Dobler L, Koschorreck J, Müller J, Wiesmüller GA, Drexler H, et al. 2011. Trends of the internal phthalate exposure of young adults in Germany—follow-up of a retrospective human biomonitoring study. Int J Hyg Environ Health 215(1):36–45, PMID: 21889907, https://doi.org/10.1016/j.ijheh.2011.07.011.

133. Tranfo G, Caporossi L, Pigini D, Capanna S, Papaleo B, Paci E. 2018. Temporal trends of urinary phthalate concentrations in two populations: effects of REACH authorization after five years. Int J Environ Res Public Health 15(9):1950, PMID: 30200671, https://doi.org/10.3390/ijerph15091950.
134. Gyllenhammar I, Glynn A, Jönsson BA, Lindh CH, Darnerud PO, Svensson K, et al. 2017. Diverging temporal trends of human exposure to bisphenols and plastizisers, such as phthalates, caused by substitution of legacy EDCs? Environ Res 153:48–54, PMID: 27898309, https://doi.org/10.1016/j.envres.2016.11.012.
135. Shu H, Jönsson BA, Gennings C, Svensson Å, Nånberg E, Lindh CH, et al. 2018. Temporal trends of phthalate exposures during 2007–2010 in Swedish pregnant women. J Expo Sci Environ Epidemiol 28(5):437–447, PMID: 29472621, https://doi.org/10.1038/s41370-018-0020-6.
136. Larsson K, Lindh CH, Jönsson BA, Giovanoulis G, Bibi M, Bottai M, et al. 2017. Phthalates, non-phthalate plasticizers and bisphenols in Swedish preschool dust in relation to children's exposure. Environ Int 102:114–124, PMID: 28274486, https://doi.org/10.1016/j.envint.2017.02.006.
137. EC (European Commission). 2004. *Commission Directive 2004/93/EC of 21 September 2004 Amending Council Directive 76/768/EEC for the Purpose of Adapting its Annexes II and III to Technical Progress*. https://eur-lex.europa.eu/legal-content/EN/ALL/?uri=CELEX:32004L0093 [accessed 19 April 2022].
138. EC. 2007. *Commission Directive 2007/19/EC of 30 March 2007 Amending Directive 2002/72/EC Relating to Plastic Materials and Articles Intended to Come Into Contact With Food and Council Directive 85/572/EEC Laying Down the List of Simulants to be used for Testing Migration of Constituents of Plastic Materials and Articles Intended to Come into Contact with Foodstuffs*. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:32007L0019 [accessed 19 April 2022].
139. EC. 2018. *Commission Regulation (EU) 2018/2005 of 17 December 2018 Amending Annex XVII to Regulation (EC) no 1907/2006 of the European Parliament and of the Council Concerning the Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) as Regards bis(2-ethylhexyl) Phthalate (DEHP), Dibutyl Phthalate (DBP), Benzyl Butyl Phthalate (BBP) and Diisobutyl Phthalate (DIBP)*. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:32018R2005 [accessed 19 April 2022].
140. EP (European Parliament). 2005. *Directive 2005/84/EC of the European Parliament and of the Council 14 December 2005 Amending for the 22nd Time Council Directive 76/769/EEC on the Approximation of the Laws, Regulations and Administrative Provisions of the Member States Relating to Restrictions on the Marketing and Use of Certain Dangerous Substances and Preparations (Phthalates in Toys and Childcare Articles)*. https://eur-lex.europa.eu/legal-content/EN/ALL/?uri=CELEX:32005L0084 [accessed 19 April 2022].
141. EC. 2011. *Commission Directive 2011/8/EU of 28 January 2011 Amending Directive 2002/72/EC as Regards the Restriction of Use of Bisphenol A in Plastic Infant Feeding Bottles*. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A32011L0008 [accessed 19 April 2022].
142. EP. 2009. *Regulation (EC) no 1223/2009 of the European Parliament and of the Council of 30 November 2009 on Cosmetic Products*. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=celex:32009R1223 [accessed 19 April 2022].
143. Lin Y, Qiu X, Liu J, Tseng C-H, Allard P, Araujo JA, et al. 2020. Different temporal trends of exposure to bisphenol A among international travelers between Los Angeles and Beijing. Environ Int 141:105758, PMID: 32402980, https://doi.org/10.1016/j.envint.2020.105758.
144. Lu X, Lin Y, Qiu X, Liu J, Zhu T, Araujo JA, et al. 2022. Triglyceride profiles are associated with subacute exposure to bisphenol A in healthy young adults. Sci Total Environ 825:153991, PMID: 35192814, https://doi.org/10.1016/j.scitotenv.2022.153991.
145. Lyu Z, Harada KH, Kim S, Fujitani T, Cao Y, Hitomi T, et al. 2022. Exposure to phthalate esters in Japanese females in Kyoto, Japan from 1993 to 2016: temporal trends and associated health risks. Environ Int 165:107288, PMID: 35588674, https://doi.org/10.1016/j.envint.2022.107288.
146. Calafat AM, Koch HM, Swan SH, Hauser R, Goldman LR, Lanphear BP, et al. 2013. Misuse of blood serum to assess exposure to bisphenol A and phthalates. Breast Cancer Res 15(5):403, https://doi.org/10.1186/bcr3494.
147. Koch HM, Calafat AM. 2009. Human body burdens of chemicals used in plastic manufacture. Philos Trans R Soc Lond B Biol Sci 364(1526):2063–2078, PMID: 19528056, https://doi.org/10.1098/rstb.2008.0208.
148. Kuiper JR, O'Brien KM, Ferguson KK, Buckley JP. 2021. Urinary specific gravity measures in the U.S. population: implications for the adjustment of non-persistent chemical urinary biomarker data. Environ Int 156:106656, PMID: 34062395, https://doi.org/10.1016/j.envint.2021.106656.
149. Ye X, Wong L-Y, Bishop AM, Calafat AM. 2011. Variability of urinary concentrations of bisphenol a in spot samples, first morning voids, and 24-hour collections. Environ Health Perspect 119(7):983–988, PMID: 21406337, https://doi.org/10.1289/ehp.1002701.
150. FDA. 2022. *Fragrances in Cosmetics*. https://www.fda.gov/cosmetics/cosmetic-ingredients/fragrances-cosmetics [accessed 4 August 2023].
151. Dodson RE, Boronow KE, Susmann H, Udesky JO, Rodgers KM, Weller D, et al. 2020. Consumer behavior and exposure to parabens, bisphenols, triclosan, dichlorophenols, and benzophenone-3: results from a crowdsourced biomonitoring study. Int J Hyg Environ Health 230:113624, PMID: 33011057, https://doi.org/10.1016/j.ijheh.2020.113624.
152. Tabak RG, Khoong EC, Chambers DA, Brownson RC. 2012. Bridging research and practice: models for dissemination and implementation research. Am J Prev Med 43(3):337–350, PMID: 22898128, https://doi.org/10.1016/j.amepre.2012.05.024.
153. FDA. 2002. *[Docket No. 02D–0325] Medical Devices; Draft Guidance; Medical Devices Made With Polyvinylchloride Using the Plasticizer di-(2–Ethylhexyl) phthalate; Availability, Food and Drug Administration*. https://www.govinfo.gov/content/pkg/FR-2002-09-06/pdf/02-22687.pdf [accessed 18 May 2022].
154. Bernard L, Decaudin B, Lecoeur M, Richard D, Bourdeaux D, Cueff R, et al. 2014. Analytical methods for the determination of DEHP plasticizer alternatives present in medical devices: a review. Talanta 129:39–54, PMID: 25127563, https://doi.org/10.1016/j.talanta.2014.04.069.
155. Porter KL, Speier J, Roybal-Allard L, Wild S, Schakowsky J, Eshoo A. 2021. *Feb 18, 2021 Letter to Acting FDA Commissioner, Janet Woodcock*. https://roar-assets-auto.rbl.ms/documents/8190/Letter%20to%20FDA%20about%20DEHP%20IV%20Bags.pdf [accessed 18 May 2022].
156. Nilsen P, Ståhl C, Roback K, Cairney P. 2013. Never the twain shall meet?—A comparison of implementation science and policy implementation research. Implement Sci 8:63, PMID: 23758952, https://doi.org/10.1186/1748-5908-8-63.
157. Bauer MS, Kirchner J. 2020. Implementation science: what is it and why should I care? Psychiatry Res 283:112376, PMID: 31036287, https://doi.org/10.1016/j.psychres.2019.04.025.
158. Damschroder LJ, Aron DC, Keith RE, Kirsh SR, Alexander JA, Lowery JC. 2009. Fostering implementation of health services research findings into practice: a consolidated framework for advancing implementation science. Implement Sci 4:50, PMID: 19664226, https://doi.org/10.1186/1748-5908-4-50.
159. Maertens A, Golden E, Hartung T. 2021. Avoiding regrettable substitutions: green toxicology for sustainable chemistry. ACS Sustain Chem Eng 9(23):7749–7758, PMID: 36051558, https://doi.org/10.1021/acssuschemeng.0c09435.
160. Committee on the Design and Evaluation of Safer Chemical Substitutions: A Framework to Inform Government and Industry Decision; Board on Chemical Sciences and Technology; Board on Environmental Studies and Toxicology; Division on Earth and Life Studies; National Research Council. 2014. *A Framework to Guide Selection of Chemical Alternatives*. Washington (DC): National Academies Press, PMID: 25473704, https://doi.org/10.17226/18872.
161. Šimunović A, Tomić S, Kranjčec K. 2022. Medical devices as a source of phthalate exposure: a review of current knowledge and alternative solutions. Arh Hig Rada Toksikol 73(3):179–190, PMID: 36226817, https://doi.org/10.2478/aiht-2022-73-3639.
162. James-Todd TM, Chiu YH, Zota AR. 2016. Racial/ethnic disparities in environmental endocrine disrupting chemicals and women's reproductive health outcomes: epidemiological examples across the life course. Curr Epidemiol Rep 3(2):161–180, PMID: 28497013, https://doi.org/10.1007/s40471-016-0073-9.
163. Ruiz D, Becerra M, Jagai JS, Ard K, Sargis RM. 2018. Disparities in environmental exposures to endocrine-disrupting chemicals and diabetes risk in vulnerable populations. Diabetes Care 41(1):193–205, PMID: 29142003, https://doi.org/10.2337/dc16-2765.
164. Santaliz Casiano A, Lee A, Teteh D, Madak Erdogan Z, Treviño L. 2022. Endocrine-disrupting chemicals and breast cancer: disparities in exposure and importance of research inclusivity. Endocrinology 163(5):bqac034, PMID: 35325096, https://doi.org/10.1210/endocr/bqac034.
165. Hering JG. 2018. Implementation science for the environment. Environ Sci Technol 52(10):5555–5560, PMID: 29693368, https://doi.org/10.1021/acs.est.8b00874.
166. NCI (National Cancer Institute). 2021. *About Implementation Science (IS)*. https://cancercontrol.cancer.gov/IS/about.html [accessed 10 May 2022].
167. Hoffmann TC, Glasziou PP, Boutron I, Milne R, Perera R, Moher D, et al. 2014. Better reporting of interventions: template for intervention description and replication (TIDieR) checklist and guide. BMJ 348:g1687, https://doi.org/10.1136/bmj.g1687.
168. Genco M, Anderson-Shaw L, Sargis RM. 2020. Unwitting accomplices: endocrine disruptors confounding clinical care. J Clin Endocrinol Metab 105(10):e3822–e3827, PMID: 32614432, https://doi.org/10.1210/clinem/dgaa358.
169. Maffini MV, Rayasam SDG, Axelrad DA, Birnbaum LS, Cooper C, Franjevic S, et al. 2023. Advancing the science on chemical classes. Environ Health 21(suppl 1):120, PMID: 36635752, https://doi.org/10.1186/s12940-022-00919-y.
170. Beronius A, Zilliacus J, Hanberg A, Luijten M, van der Voet H, van Klaveren J. 2020. Methodology for health risk assessment of combined exposures to multiple chemicals. Food Chem Toxicol 143:111520, PMID: 32640355, https://doi.org/10.1016/j.fct.2020.111520.