March 24, 2017

Submitted Electronically to CPSC Docket

Office of the Secretary
U.S. Consumer Product Safety Commission
Room 820, 4330 East West Highway,
Bethesda, MD 20814

RE: Estimated Phthalate Exposure and Risk to Pregnant Women and Women of Reproductive Age as Assessed Using 2013/2014 NHANES Biomonitoring Data, Docket Number CPSC-2014-0033

Dear Secretary:

The Vinyl Institute (VI)[1] is pleased to submit these comments to the U.S. Consumer Product Safety Commission (CPSC) on the recently available report "Estimated Phthalate Exposure and Risk to Pregnant Women and Women of Reproductive Age as Assessed Using 2013/2014 NHANES Biomonitoring Data."[2]

Some VI member companies manufacture phthalate plasticizers for flexible vinyl materials, and other VI members compound phthalates with vinyl resins to make flexible vinyl compounds for conversion into finished products. VI members, therefore, will be adversely affected by the implications associated with the conclusions of the CPSC science staff's analysis based on the Center for Disease Control's (CDC) National Human Health and Nutrition Examination Survey (NHANES) 2013/2014 biomonitoring data.

The VI agrees with the use of the most recent NHANES data from 2013/2014 to support any future phthalate rulemaking activity. However, it is important that the CPSC staff's analysis and approach interpret and apply the NHANES data correctly to ensure that the phthalates under scrutiny are properly assessed in a scientifically sound fashion. This requires that the analysis reflect the physical properties of the phthalates, their actual use in consumer products, and potential risks from such use. Otherwise, the result will certainly impose unintended consequences on manufacturers and processors of these chemicals, the downstream manufacturers they support who make consumer products, and the

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[1] The Vinyl Institute (VI) is a U.S. trade association founded in 1982 and currently represents the major vinyl resin, monomer, and additive producers. The VI serves as the collective voice for the vinyl industry, engaging industry stakeholders in shaping the future of the vinyl industry. More information about the Vinyl Institute can be found on our website: www.vinylinfo.org.

[2] CPSC, *Notice of Availability: Estimated Phthalate Exposure and Risk to Women of Reproductive Age as Assessed Using 2013/2014 NHANES Biomonitoring Data*, 82 Fed. Reg. 11,348 (February 22, 2017).

consumers who use those products. In addition to supporting the comments filed by ExxonMobil Chemical Company, American Chemistry Council, and the Flexible Vinyl Alliance on this latest staff analysis, VI is submitting these comments to urge CPSC to carefully consider changing the approach to its analysis.

I. Cumulative Risk Analysis (CRA) is an Incorrect Approach for Phthalates Hazard Determinations

Cumulative Risk Analysis (CRA) would be a reasonable approach to evaluating the risk posed by various phthalates in consumer products if 1) products simultaneously contained each of the different substances in the group, and 2) each of the different substances were close in both physical properties and potency. This is not the case for di-ethyl hexyl phthalate (DEHP) and di-iso nonyl phthalate (DINP) phthalates included in this CRA. DINP and DEHP are rarely, if ever, used together in any product. Also, very significant physical property differences exist between DEHP and DINP plasticizers, two of which are described in more detail below. The different physical property differences result in dramatically different exposure potentials, which in turn undermine any basis for applying a CRA.

a. Vapor pressure – this is a good indicator of how volatile a substance is at various temperatures. It stands to reason that the higher the vapor pressure, the greater the exposure an individual might experience. Selected relevant phthalate ester vapor pressures at 25 °C are given in the table below.[3]

Plasticizer	Vapor Pressure (Pa)
DINP	7.20×10^{-5}
DEHP	85.3×10^{-5}

This table shows that the vapor pressure difference between the two phthalates is approximately one order of magnitude at room temperature, which is evidence of their incorrect grouping together in the CRA. Moreover, even with this large vapor pressure difference between DINP and DEHP, both have extremely low vapor pressure. As a result, *neither* of these phthalate plasticizers would be expected to evaporate at normal room temperature, suggesting a very low potential for exposure from either substance in any consumer product.

b. Plasticizer retention in substrate – Certain plasticizers are more strongly bound in vinyl products than others. Differences in polarizable ring structures between plasticizers yield different electrostatic bonds between PVC chains and the plasticizer.[4] Furthermore,

[3] ExxonMobil Petroleum and Chemical B.V.B.A., "Submission of information on DINP CAS#68515-48-0, EC#271-090-9 as an alternative to DEHP Applications for Authorisation" at *10 (Jan. 7, 2014).
[4] *Id.* at *9.

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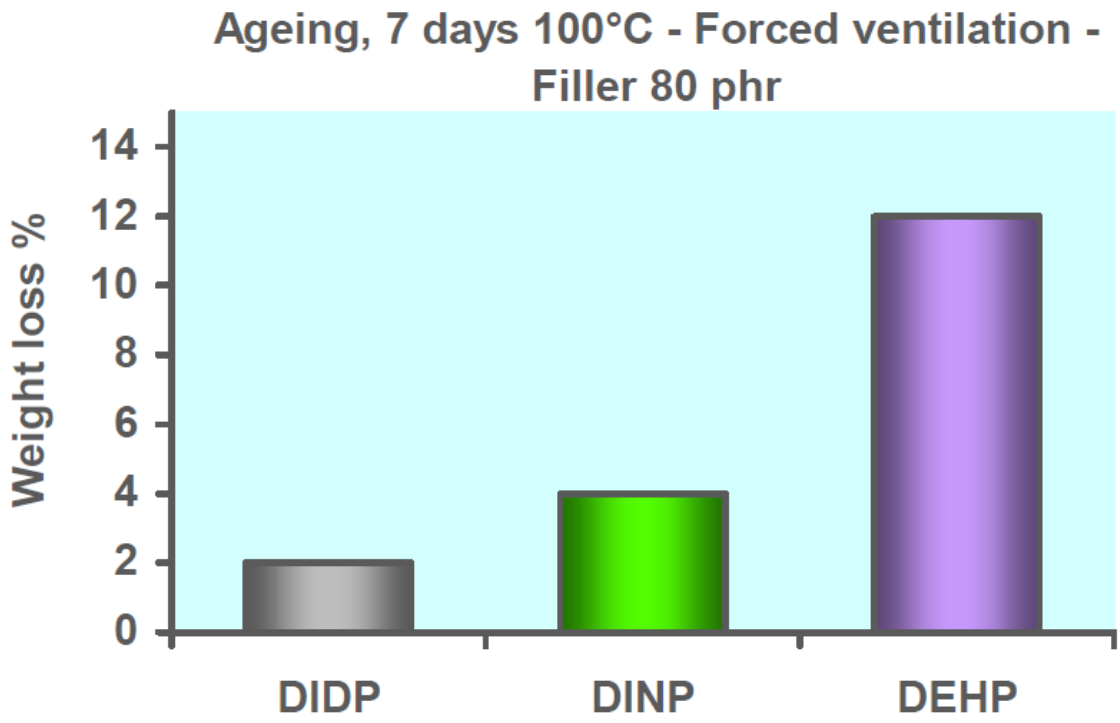
differences in molecular weight of plasticizers will influence diffusion within PVC and plasticizer retention over time, even against harsh aging conditions.[5] The following chart illustrates these properties.[6]



Accelerated aging is often used to identify differences between ingredients in a compound. Results of accelerating aging at elevated temperatures of 100° C are depicted in the graph above for three common plasticizers. Use conditions of vinyl materials typically involve the -30°C to +60°C range, so the accelerated aging scenario does not represent realistic conditions of use in a consumer product. The differences in the plasticizers in this graph illustrate why DINP and DEHP plasticizers are not correctly grouped in the CRA.

Critically, these two physical property differences between DEHP and DINP lead to a large difference in exposure from products containing these plasticizers. The two plasticizers are not typically used together, so VI recommends analyzing DINP differently and excluding it from the CRA.

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[5] *Id.*

[6] *Id.* Data generated using Internal Coppco™ computer program (PVC compound physical properties prediction). PVC formulation: S-PVC 100 phr, DEHP 50 phr, DINP 52 phr, DIDP 55 phr, CaCO3 80 phr.

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II. Case 2 Should Not be a Determinant in Any Regulatory Action Due to Its Flaws

The CHAP uses "no effect" levels for DEHP in Case 2 models as an index chemical and applies those same parameters to DINP. This gross flaw overstates the cumulative risk contribution from DINP for the CRA in Case 2. The VI advocates disregarding Case 2.

III. NHANES Data Should Not Be Used for Estimating Individual Risk

The Hazard Index computed using NHANES data is a statistically insignificant calculation indicative of single exposures at a particular point in time for the individual studied; repeated testing on the same individual is not part of the NHANES. In any event, there are too few cases to make statistically-significant percentage of the population estimates, which CPSC acknowledged in describing the data as "statistically unstable." This is a serious flaw in the approach taken in this recent analysis. In other words, just because an individual spot sample leads to a hazard index (HI) greater than one for that individual at that point in time does not mean that the individual is at risk from the considered phthalates. Risk arises from maintaining exposure above a specified level for a prolonged period. The reanalysis cannot extrapolate a single individual's risk at a single moment to a sustained risk for a percentage of the total US population.

An additional concern is the exclusion of the MINP metabolite in the calculation of Daily Intake Values for DINP. As explained in more detail in ExxonMobil's comments, the NHANES biomonitoring report provides values for phthalate metabolites detected in urine samples that can be converted to the daily intake levels that would have resulted in those metabolite levels. For several phthalates, values for more than one metabolite are reported. For example, NHANES includes data for metabolites of DEHP abbreviated as MEHP, MEHHP, MEOHP, and MECPP. For DINP, NHANES reports data for the MCOP and MINP metabolites.

The equation used to convert the metabolite data into exposure values allows all of the metabolite data to be incorporated in determining the daily intake value. The CHAP followed this method with all the DEHP metabolite data, but for some unexplained reason, the CHAP analysis excluded the MINP metabolite for DINP and only included the MCOP data. By ExxonMobil's estimate, Daily Intake Estimates of DINP for women of reproductive age would be 17% lower if MINP metabolites are included in the calculation.[7] VI advocates including all the data from the NHANES survey for the CHAP analysis.

The key takeaway from the NHANES data should be the trend of decreasing cumulative risk over time. The CPSC science staff now has three consecutive data points showing that the cumulative risk from the considered phthalates is below an HI of 1. This conclusion should provide the basis for confidently lifting the restriction on DINP.

[7] ExxonMobil Chemical Company Comments submitted to docket ID no. CPSC-2014-0033 (March 24, 2017): p. 9.

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IV. CPSC Must Consider Available Data That Indicates Humans Are Less Sensitive Than Rodents to the Considered Phthalates

The CHAP incorrectly concluded that there is insufficient evidence to believe humans are less sensitive to the subject phthalates than the rodent test species. This ignores the body of scientific studies using marmosets that indeed supports humans as being less sensitive than rodents to the effects of certain phthalates.[8]

In addition, certain effects of di(*n*-butyl) phthalate (DBP) exposure vary even among different rodent species:

> "Male rats exposed *in utero* to certain phthalate plasticizers exhibit multinucleated germ cell (MNG) induction and suppressed steroidogenic gene expression and testosterone production in the fetal testis, causing TDS-consistent effects of hypospadias and cryptorchidism. Mice exposed to phthalates *in utero* exhibit MNG induction only. This disparity in response demonstrates a species-specific sensitivity to phthalate-induced suppression of fetal Leydig cell steroidogenesis. Importantly, *ex vivo* phthalate exposure of the fetal testis does not recapitulate the species-specific endocrine disruption, demonstrating the need for a new bioassay to assess the human response to phthalates."[9]

This reference to the Brown University results would lead one to conclude that potency of certain phthalate health effects estimated from rodent studies is dependent on the type of species studied, which was not considered in the CHAP or CPSC science staff analyses.

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We thank the CPSC for its consideration of these comments. Should the Commission or CPSC staff have questions or require further information, please contact me.

Respectfully submitted,

Richard Krock
Vice President, Regulatory and Technical Affairs

cc: Kent R. Carlson, Ph.D., Toxicologist, Division of Toxicology & Risk Assessment

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[8] See, *e.g.*, McKinnell, C., et al, "Effect of fetal or neonatal exposure to monobutyl phthalate (MBP) on testicular development and function in the marmoset", Human Reproduction 24, no. 9 (2009): 2244-2254.
[9] Heger NE, et. al, "Human Fetal Testis Xenografts Are Resistant to Phthalate-Induced Endocrine Disruption", Environ. Health Perspect. 120, no. 8 (2012): :1137–1143, available at: http://dx.doi.org/10.1289/ehp.1104711.

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