Cigarettes and Coffee: Do Newly-Discovered Sources of Diacetyl Spell the End of Popcorn Lung Litigation or the Beginning of a New Phase?

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DIACETYL seemed destined to be the next big thing in toxic tort litigation. With a scary sounding scientific name, an incurable signature disease, and deep-pocketed defendants, it appeared to be the perfect foil. However, after some initial successes over a decade ago, the litigation has failed to deliver as the plaintiffs' tort bar had hoped. Recently, though, there have been several developments, including further testing that indicates diacetyl exposure in excess of proposed OSHA standards is not limited to those working in popcorn flavoring plants. It remains to be seen if these new findings will either: (i) increase the focus and viability of diacetyl litigation; or (ii) further weaken the shaky science behind injurious diacetyl exposure by introducing some logic to the

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analysis (to wit, how can diacetyl be dangerous at low levels when people are exposed at high levels on a daily basis?).

I. Cigarettes and Coffee: Everyday Diacetyl Exposure

Otis Redding glamorized the guilty pleasure of smoking cigarettes while drinking coffee in his song "Cigarettes and Coffee." "I'm sittin' here talkin' with my baby over cigarettes and coffee . . . ."[2] He also foretold two activities that can cause exposure to large doses of diacetyl. Traditional cigarette smoke contains over 3,000 organic compounds, including a large amount of diacetyl.[3] One study suggests that the mean diacetyl concentrations in cigarette smoke ranges from 250 to 361 parts per million ("ppm").[4]

With a touch of clairvoyance, Redding may have mentioned smokeless e-cigarettes too. Even those that now partake in that hipper version of smoking are exposed to large concentrations of diacetyl. Diacetyl has been detected in many types of flavored e-cigarettes.[5] One study found that the concentration of diacetyl in e-cigarette smoke ranged from below the detection limit to as high as 239 ppm.[6]

Coffee is also an apparent source of significant diacetyl exposure. Recent studies have found that coffee workers are also subject to diacetyl exposure.[7] For example, long-term concentrations for coffee shop baristas ranged from 0.013-0.016 ppm, with long-term concentrations for the customers ranged from 0.010-0.014 ppm, while the mean estimated time weighted average ("TWA") exposures for the baristas ranged from 0.007-0.013 ppm.[8] Another study measured the concentration

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[2] Otis Redding, *Cigarettes and Coffee, on* The Soul Album (Volt/Atco 1966).

[3] Kazutoshi Fujioka and Takayuki Shibamoto, *Determination of Toxic Carbonyl Compounds in Cigarette Smoke*, 21 ENVIRON. TOXICOL. 47 (2006).

[4] Jennifer S. Pierce, Anders Abelmann, Lauren J. Spicer, Rebecca E. Adams and Brent L. Finley, *Diacetyl and 2,3-pentanedione exposures associated with cigarette smoking: implications for risk assessment of food and flavoring workers*, 44 CRITICAL REVIEWS IN TOXICOLOGY 450 (2014).

[5] J. Allen, S. Flanigan, M. LeBlanc, J. Vallarino, P. MacNaughton, J. Stewart, and D. Christiani, *Flavoring Chemicals in E-Cigarettes: Diacetyl, 2,3-Pentanedione, and Acetoin in a Sample of 51 Products, Including Fruit-, Candy-, and Cocktail-Flavored E-Cigarettes*, 124 ENVIRON HEALTH PERSPECT. 733 (2016).

[6] *Id.*

[7] Rachel L. Bailey, Ryan F. LeBouf, and Kristin J. Cummings, *Coffee Workers at Risk for Lung Disease*. Available at https://blogs.cdc.gov/niosh-science-blog/2016/01/25/coffee-workers/ (January 25, 2016).

[8] Jennifer S. Pierce, Anders Abelmann, Jason T. Lotter, Chris Comerford, Kara Keeton and Brent L. Finley, *Characterization of naturally occurring airborne diacetyl concentrations associated with the preparation and consumption of unflavored coffee*, 2 TOXICOLOGY REPORTS 1200 (2015).

of naturally occurring diacetyl in the headspace of an open cup of unflavored coffee to be 7 ppm.[9]

Notwithstanding these exposures, there is no credible study linking either cigarette smoke or coffee to an increase in popcorn lung.

II. Background

A. What is Diacetyl?

Diacetyl (2,3-butanedione) is a very volatile naturally occurring organic compound. It is a natural byproduct of beer and wine making. It has a characteristic buttery flavor and has been produced industrially as a food-flavoring additive. Most notably, it has been used as a flavoring agent in microwave popcorn.

B. What is Popcorn Lung?

"Popcorn Lung" is a term used to describe bronchiolitis obliterans, which is an irreversible obstructive lung disease characterized by an inflammatory blockage of the bronchioles– the tiniest and narrowest airways in the lungs. Bronchioles are found in the respiratory system where air is fed to the alveoli (air sacs) and ultimately the blood stream. Bronchiolitis obliterans can be caused by trauma to the bronchioles after organ transplant, as well as by exposure to irritant gases like chlorine, ammonia, nitrogen dioxide, sulfur dioxide, and phosgene (collectively "irritant gases").[10] It is thought that exposure to irritant gases at high concentrations causes injury to the epithelial lining of the bronchioles.[11]

Toxicokinetics affecting concentrations at the bronchiolar epithelium substantially drive the risk of bronchiolitis obliterans from irritant gases. Highly soluble irritant gases like ammonia generally follow a threshold-dependent cytotoxic mechanism of action, so that at sufficiently high doses, exposure results in severe inflammation of the upper respiratory tract and the bronchiolar epithelium concurrently. This is followed by acute respiratory distress, pulmonary edema, and post inflammatory concentric fibrosis that become clinically obvious within a few months of exposure. In contrast, irritant gases with lower solubility like phosgene also follow a threshold-dependent mechanism of

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[9] Chahan Yeretzian, Alfons Jordan, and Werner Lindinger, *Analysing the Headspace of Coffee by Proton-Transfer-Reaction Mass Spectrometry*, 223-224 INT J. MASS SPECTROMETRY 115 (2003).

[10] National Jewish Health, Bronchiolitis Obliterans, available at https://www.nationaljewish.org/healthinfo/conditions/bronchiolitis/.

[11] Brent D. Kerger and M. Joseph Fedoruk, *Pathology, toxicology, and latency of irritant gases known to cause bronchiolotos obliterans disease: Does diacetyl fit the pattern?*, 2 TOXICOLOGY REPORTS 1463 (2015).

cytotoxic action, but can exhibit more insidious and isolated bronchiolar tissue damage with a latency similar to fibrosis.

To date, animal and human studies on diacetyl, a highly soluble gas, have not identified a coherent pattern of pathology and latency similar to that which would be expected based on studies of other known causes of bronchiolitis obliterans disease.

III. Regulatory Status

A. FDA

The FDA has long classified diacetyl as GRAS ("generally recognized as safe"). It is not considered a food additive and therefore does not require approval for use by the FDA.[12] Note that the FDA considers diacetyl as GRAS only for consumption of small quantities and not for inhalation. However, on May 5, 2016, the FDA announced that it is assuming regulatory authority over e-cigarettes.[13] It remains to be seen if this authority will impact the diacetyl component of e-cigarettes.

B. OSHA

OSHA has not provided any specific standard regulating occupational diacetyl exposure,[14] and"[m]any current MSDSs [material safety data sheets] do not reflect any new information regarding health effects and respiratory hazards associated with diacetyl."[15] OSHA has published guidance for diacetyl and food flavorings containing diacetyl, recommending that employers update their MSDSs and labels to provide information regarding the possible health hazards from prolonged exposure to diacetyl.[16] OSHA's guidance is unmistakably aimed at clearer identification of health risks beyond skin, eye, and airway irritation, noting that "updated health hazard information . . . must be reported in the health effects section" and that chemical manufacturers and importers of any food flavoring containing more than one percent diacetyl "must convey information that animals exposed to diacetyl experienced

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[12] 21 C.F.R. 184.1278 (Diacetyl), available at https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?fr=184.1278 (last accessed November 24, 2017).

[13] http://www.fda.gov/Tobacco Products/Labeling/ProductsIngredientsComponents/ucm456610.htm (last accessed November 24, 2017).

[14] Occupational Safety and Health Administration, *Flavorings-Related Lung Disease*, available at https://www.osha.gov/SLTC/flavoringlung/ (last accessed November 24, 2017).

[15] OSHA, *Hazard Communication Guidance for Diacetyl and Food Flavorings Containing Diacetyl,* available at https://archive.fo/jZwex_(captured May 4, 2016, last accessed November 24, 2017).

[16] *Id.*

damage to the nose and upper airways, including severe damage to cells lining the respiratory tract" in their MSDSs.[17]

Currently, OSHA does not mandate any particular respiratory protection, but recommends that "[b]ased on the NIOSH investigations of microwave popcorn plants, a NIOSH-certified air-purifying respirator equipped with organic vapor cartridges in combination with particulate filters would provide the minimum level of protection" and also leaves open that supplied air respirators are an option.[18]

C. NIOSH

As of guidance published in October 2016, NIOSH recommends a permissible exposure limit ("PEL") of 0.005 ppm as a time weighted average ("TWA") during a 40-hour work week. NIOSH recommends a short-term exposure limit ("STEL") for diacetyl of 0.025 ppm for a 15-minute time period.[19] According to NIOSH, all "employees who work in or enter areas where diacetyl" is "used or produced" should be included in a medical monitoring program that includes "periodic exposure and medical evaluation and monitoring exposure controls and appropriate employee training on potential health effects, respiratory protection, and use of controls."[20] NIOSH's 2016 recommendations for managing occupational exposure call for a combination of workspace ventilation, administrative limitation of worker exposure, and an appropriate respirator approved for use by NIOSH under 42 C.F.R. Part 84—the type of which would vary according to exposure time and concentration.[21] OSHA is expected to adopt the NIOSH recommendations shortly.

IV. Epidemiology Studies of Industrial Exposures to Diacetyl

Notwithstanding its seemingly harmless ability to impart buttery goodness to popcorn, chardonnay, and other delicious foods, regulators have long been concerned about the possible link between diacetyl and Popcorn Lung, an extremely serious illness. This issue received widespread notoriety in the early 2000's. In 2002, *USA Today* reported on a study conducted by the U.S. Centers for Disease Control

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[17] *Id.*

[18] *Id.*

[19] National Institute of Occupational Safety and Health, Publication No. 2016-111, *Criteria for a recommended standard: occupational exposure to diacetyl and 2,3-pentanedione*, available at https://www.cdc.gov/niosh/docs/2016-111/default.html (last accessed November 24, 2017).

[20] *Id.*

[21] *Id.* at 205.

and Prevention ("CDC"), which found several cases of bronchiolitis obliterans in workers at the Gilster-Mary Lee ("GML") microwave popcorn manufacturing plant in Jasper, Missouri.[22]

The CDC reported that eight patients out of the 425 former employees at this factory "had a respiratory illness resembling bronchiolitis obliterans;" four were mixers and four were microwave-packaging workers, with no cases reported in workers in other areas of the factory.[23] Extrapolating and using crude statistics, the CDC concluded that the evidence of illness was 31% among mixers and 1% among microwave-packaging workers.[24]

The first published study of industrial exposure to diacetyl was conducted by Kreiss et al ("Kreiss") at the same Jasper, Missouri plant.[25] At the time of the study in November 2000, the plant had 135 current workers, of whom 117 completed questionnaires and 116 took spirometric tests. The study concluded that compared to national averages these workers had: (i) 2.6 times the expected rates of chronic cough and shortness of breath; (ii) 2 times the expected rate of physician-diagnosed asthma and chronic bronchitis; and (iii) 3.3 times the expected rate of airway obstruction.[26] Industrial hygiene sampling detected diacetyl as 32.27 ppm in the mixing room, 1.88 ppm in the microwave-packaging area, and 0.56 ppm in the quality control/maintenance area.[27]

The Kreiss study is not without its discrepancies. For example, it concluded that the odds of being affected with airway obstruction were much higher for quality control ("QC") workers (five out of six workers) than for workers elsewhere in the GML plant.[28] This seems inconsistent with a dose response relationship, as the airborne diacetyl levels in the QC area were much lower than elsewhere in the plant.[29] Further, the authors at least tacitly recognize that there could be other causes aside from diacetyl, since the GML workers were exposed to: (i) more than 100 volatile organic compounds inside the plant;[30] and (ii) other possible causes of lung disease outside the plant.[31]

Subsequent studies do not definitely demonstrate a connection

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[22] Centers for Disease Control and Prevention, Weekly, *Fixed Obstructive Lung Disease in Workers at a Microwave Popcorn Factory – Missouri*, 345-347 (April 26, 2002).
[23] *Id.*
[24] *Id.*
[25] Kathleen Kreiss, Ahmed Gomaa, Greg Kullman, Kathleen Fedan, Eduardo Simoes, and Paul Enright, *Clinical Bronchiolitis Obliterans in Workers at a Microwave-Popcorn Plant*, 347 N. ENGL. J. MED. 330 (2002).
[26] *Id.* at 332.
[27] *Id.* at 333.
[28] *Id.*
[29] *Id.*
[30] *Id.* at 330-331.
[31] *Id.* at 332.

between diacetyl exposure and respiratory disease. For example, there was another study of the GML plant performed in 2001 and published in 2006 by Akipinar-Elci, et al. ("Akipinar-Elci").[32] Akipinar-Elci studied some of the same workers as Kreiss. But the approach was different in that in addition to spirometry data, Akipinar-Elci also looked at the levels of exhaled nitric oxide ("NO"). The authors theorized that the level of exhaled NO could be a marker for bronchiolitis obliterans. Akipinar-Elci studied: (i) 107 workers in the mixing, microwave packaging, and QC areas, which the study classified as a "high-exposure group;" and (ii) 28 workers in the office, packaging, and warehouse area, which the study classified as a "low-exposure group."[33] At the time of the study the diacetyl air concentrations were reportedly: 2.24 ppm in mixing room; 0.426 ppm in packaging; 0.401 in QC; and 0.15 in the office low exposure areas.[34] Even though the high-exposure group was exposed to diacetyl in levels much higher than the low-exposure group, the study found no significant differences in spirometry results between the two groups.[35]

Perhaps the most comprehensive study was conducted by R. Kanwal et al. ("Kanwal").[36] Kanwal studied six microwave popcorn plants (including GML, which served as the index plant) which collectively employed 708 people.[37] Of the 708 then-current employees, 537 (76%) participated in the study.[38] The study stratified the employees into several groups:

A. Ever mixers:
    i. mixers who worked as mixers for more than 12 months;
    ii. mixers who worked as mixers for less than 12 months;
B. Never mixers;
C. Packaging area workers (who never worked as mixers):
    i. workers who worked in packaging areas with isolated tanks;

[32] Muge Akpinar-Elci, Kimberly Stemple, Omur Elci, Raed Dweik, Kathleen Kreiss and Paul Enright, *Exhaled nitric oxide measurement in workers in a microwave popcorn production plant*, 12 INT. J. OCCUP. ENV. HEALTH 106 (2006).
[33] *Id.*
[34] *Id.*
[35] *Id.*
[36] Richard Kanwal, Greg Kullman, Chris Piacitelli, Randy Boylstein, Nancy Sahakian, Stephen Martin, Kathleen Fedan, Kathleen Kreiss, *Evaluation of Flavorings-Related Lung Disease Risk at Six Microwave Popcorn Plants*, 48 J. OCCUP. ENVIRON. MED. 149 (2006).
[37] *Id.* at 149, 152.
[38] *Id.*

ii. workers who worked in packaging packaging areas with non-isolated tanks; and,

D. Smokers.[39]

The study included a medical records review as well as a medical survey.[40] The medical survey included: (i) shortness of breath when hurrying on level ground or walking up a slight hill ("SOB1"); (ii) shortness of breath when walking with people one's own age on level ground ("SOB2"); (iii) chronic cough; (iv) wheezing; and (v) airways obstruction as measured on a spirometer.[41]

Kanwal found that in the five study plants (i.e., not including the GML index plant) the mean diacetyl area measurements ranged from 0.2 to 1.2 ppm in the mixing areas and 0.004 to 0.7 ppm in the packaging area.[42] His review of the medical records of the study plants' employees indicated that one mixer at three of the plants and three packaging area employees at another had fixed airways obstruction.[43] The study concluded that among all of the stratified groups, there was no statistically significant difference in airways obstruction.[44] It further found that there were no differences in the occurrence of the majority of self-reporting symptoms (e.g., SOB1, etc.).[45]

Similarly, other studies of diacetyl-exposed workers also fail to clearly link diacetyl exposure to bronchiolitis obliterans. Some fail to consider the impact of the hundreds of other volatile organic compounds present at the subject workplaces.[46] One study even reached the conclusion that "[a] cumulative diacetyl exposure-response relationship could not be demonstrated or did not exist."[47] Moreover, that study found that respiratory performance actually improved as a function of diacetyl exposure.[48]

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[39] *Id.* at 150.
[40] *Id.* at 152.
[41] *Id.* at 150.
[42] *Id.* at 151.
[43] *Id.* at 152.
[44] *Id.* at 152-156.
[45] *Id.*
[46] J.E. Lockey, T.J. Hilbert, L.P. Levin, P.H. Ryan, K.L. White, E.K. Borton, C.H. Rice, R.T. McKay, and G.K. LeMasters, *Airway obstruction related to diacetyl exposure at microwave popcorn production facilities*, 34 EUR. RESPIR. J. 63 (2009).
[47] Frits G. van Rooy, Lidwien A. Smit, Remko Houba, Vanessa A. Zaat, Jos M. Rooyackers, Dick J. Heederik, *A cross-sectional study of lung function and respiratory symptoms among chemical workers producing diacetyl for food flavourings*, 66 OCCUP. ENVIRON. MED. 105 (1999) (The study concluded, however, that "[t]he excess of respiratory symptoms in this retrospective cohort supports the occupational hazard in diacetyl production . . . .").
[48] *Id.*

V. Animal Toxicology Studies

There have been several studies that evaluated the toxicity of both artificial butter flavoring vapors ("BFV") and diacetyl. Hubbs et al ("Hubbs 2002") exposed laboratory rats to heated BFV for six hours at four different exposure levels of diacetyl: (i) low exposure, defined as 203 ppm; (ii) middle exposure, defined as 285 ppm; (iii) high exposure, defined as 352 ppm; and (iv) high pulsing exposure, defined as 371 ppm with a range of 720 to 940 ppm.[49] It should be noted that the BFV did not just contain diacetyl but in fact included numerous other chemicals such as acetic acid, butyric acid, 2-nonanone, and other volatile organic compounds.[50] The rats exposed to middle, high, and high pulsing exposures experienced pulmonary airway necrosis.[51] Rats in all four exposure groups experienced some necrosis of the nasal cavity.[52] The authors also determined that the alveoli of the rats in all groups "were unaffected."[53]

About six years later the authors repeated this study, this time using vapors of pure diacetyl ("Hubbs 2008").[54] However, the 2008 study had a different design utilizing two different inhalation experiments at 99, 198, and 295 ppm: (i) continuous six- hour diacetyl inhalation; and (ii) continuous six-hour diacetyl inhalation plus fifteen-minute pulsed diacetyl inhalation.[55] The study found necrosis of the nasal epithelium in all rats exposed at 198 and 295 ppm, but not in rats exposed at 99 ppm.[56] It also found that effects on the upper respiratory epithelium were limited to two of the six rats exposed to a continuous 295 ppm diacetyl for six hours.[57] Further, none of the diacetyl exposures were reported to have caused injury or damage to the alveoli.

Another 2008 study was conducted by Morgan et al ("Morgan").[58] In 2008, Morgan

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[49] Ann F. Hubbs, Lori A. Battelli, William T. Goldsmith, D.W. Porter, David Frazer, Sherri Friend, Diane Schwegler-Berry, Robert R. Mercer, J. S. Reynolds, A. Grote, Vincent Castranova, Gregory Kullman, Jeffrey S. Fedan, J. Dowdy, and W. G. Jones, *Necrosis of Nasal and Airway Epithelium in Rats Inhaling Vapors of Artificial Butter Flavoring*, 185 TOXICOL. APPL. PHARMACOL. 128 (2002).

[50] *Id.*

[51] *Id.* at 133.

[52] *Id.*

[53] *Id.* at 128.

[54] Ann F. Hubbs, William T. Goldsmith, Michael L. Kashon, David Frazer, Robert R. Mercer, Lori A. Battelli, Gregory J. Kullman, Diane Schwegler-Berry, Sherri Friend and Vincent Castranova, *Respiratory toxicologic pathology of inhaled diacetyl in Sprague-Dawley Rats*, 26 TOXICOLOGIC PATHOL. 330-344 (2008).

[55] *Id.* at 331-332.

[56] *Id.* at 334

[57] *Id.* at 334-335.

[58] Daniel L. Morgan, Gordon P. Flake, Patrick J. Kirby and Scott M. Palmer, *Respiratory toxicity of diacetyl in C57BI/6 mice*, 103 TOXICOL. SCI. 169 (2008).

exposed mice to diacetyl at levels from 25 ppm to 1200 ppm.[59] Morgan used exposure durations much longer that Hubbs, and Morgan found that exposures of 200 or 400 ppm for five days caused necrotizing rhinitis, necrotizing laryngitis, and bronchiolitis; while exposures of 100, 200, or 400 ppm for one hour per day over four weeks caused fewer nasal and laryngeal injuries.[60] The study also exposed mice to diacetyl at 1200 ppm in fifteen-minute exposure intervals twice a day over a four-week period, with similar results.[61] None of the exposures utilized by Morgan caused lesions or injuries to the bronchioles of the study animals.[62]

Hubbs et al conducted another study in 2012 ("Hubbs 2012") this time exposing one group of rats to diacetyl and another group to 2,3-pentanedione. [63] Similar to diacetyl, 2,3-penanedione is a diketone used for, among other things, synthetic flavoring to impart a buttery or cheesy note.[64] Hubbs 2012 studied the effects of 2,3-pentanedione on airways using a six-hour exposure of: (i) 2,3-pentanedione at 112, 241, 318, or 354 ppm; and (ii) diacetyl at 240 ppm.[65] Also, to study delayed toxicity, Hubbs 2012 exposed additional rats to 2,3-pentanedione for six hours at 318 ppm and then sacrificed the rats at intervals of: (i) 0 to 2 hours; (ii) 12 to 14 hours; or (iii) 18 to 20 hours after exposure.[66] Hubbs observed necrotizing rhinitis and necrotizing tracheitis in the rats exposed to both diacetyl and 2,3-pentanedione, with increasing upper airways necrosis at increasing levels of 2,3-pentanedione.[67] Evidence of neither significant effects in the posterior nasal epithelium nor significant lesions in the bronchus was observed in the diacetyl exposed rats.[68] An additional group of rats inhaled 270 ppm 2,3-pentanedione to study if the exposure could, among other things, cause an alteration of gene expression.[69] Hubbs ultimately concluded "that 2,3-pentanedione is a respiratory hazard that can also alter gene expression in the brain."[70]

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[59] *Id.* at 170.
[60] *Id.* at 173.
[61] *Id.*
[62] *Id.* at 173, 177.
[63] Ann F. Hubbs, Amy M. Cumpston, William T. Goldsmith, Lori A. Battelli, Michael L. Kashon, Mark C. Jackson, David Frazer, Jeffrey S. Fedan, Madhusudan P. Goravanahally, Vincent Castranova, Kathleen Kreiss, Patsy A. Willard, Sherri Friend, Diane Schwegler-Berry, Kara L. Fluharty and Krishnan Sriram, *Respiratory and Olfactory Cytotoxicity of Inhaled 2,3-pentanedione in Sprague-Dawley Rats*, 181 AM. J. PATHOL. 829 (2012).
[64] 2,3-PENTANEDIONE, ChemicalLand21.com, available at http://www.chemicalland21.com/lifescience/foco/2,3-PENTANEDIONE.htm (last accessed November 24, 2017).
[65] Hubbs 2012, *supra* note 63, at 829.
[66] *Id.*
[67] *Id.*
[68] *Id.*
[69] *Id.*
[70] *Id.*

As briefly discussed above, the studies have some fundamental shortcomings. Most notable is that all the studies use inhalation exposures far beyond those experienced in actual work places where diacetyl is utilized. Also, the injuries sustained by the study animals endured are not consistent with bronchiolitis obliterans in humans given that the animal injuries are mostly of the nasal and upper respiratory track rather than the alveoli or bronchioles.

VI. Industrial Lawsuits

It did not take long for the industrial exposure lawsuits to commence. In 2001, the first diacetyl lawsuit was filed on behalf of workers at the Jasper, Missouri microwave popcorn plant. The Jasper lawsuits were followed by others elsewhere among the country. In 2010, a Chicago case involving industrial exposure to diacetyl resulted in a verdict of over $30 million.

In one case, a plaintiffs' inability to point to a standard of care governing diacetyl exposure was found as a basis for dismissal of the plaintiff's negligence claim in an industrial exposure suit, even where denial of a motion for a new trial on other claims was reversed on appeal. In *Velasquez v. Centrome, Inc.*,[71] the plaintiff worked at food flavoring manufacturer Gold Coast, and had responsibilities transporting diacetyl in both closed and open containers throughout the company's facility. As a result of his work, he allegedly breathed diacetyl particles in the ambient air while pouring diacetyl and while using a sprayer to mix diacetyl into batches of both liquid and dry flavorings.[72] The diacetyl was supplied by defendant Centrome, Inc., d/b/a Advanced Biotech (Advanced).[73] A jury hearing the case reached a special verdict, finding, in part that: (1) Advanced had been negligent; (2) Advanced's negligence was not a substantial factor in causing harm to Velasquez; (3) Advanced had violated one or more of the provisions of the Hazard Communication Standard (see 29 C.F.R. § 1910.1200); (4) Advanced's violation of the Hazard Communication Standard was not a substantial factor in causing harm to Velasquez; (5) the design of Advanced's diacetyl was not a substantial factor in causing harm to Velasquez; (6) Advanced's diacetyl did not fail to perform as safely as an ordinary person would have expected when used or misused in an intended or reasonably foreseeable way; and (7) ordinary persons would have recognized the potential risks of diacetyl. At the close of trial, the Court granted Advanced's motion for nonsuit

[71] 233 Cal. App. 4th 1191, 1194, 183 Cal. Rptr. 3d 150, 154 (Cal. Ct. App. 2015).

[72] *Id.*

[73] *Id.* at 1193.

based on Velasquez's common law negligence theory. On appeal of the denial of a motion for new trial, with respect to the claims of negligence per se and strict products liability, the Court found that the nonsuit ruling was proper because plaintiff Velasquez failed to present any evidence during trial on the standard of care in the food flavoring industry at the time Velasquez was exposed to Advanced's diacetyl, a required element for a cause of action for common law negligence.[74]

In addition to the legal impediments, given that there are only a limited number of plaintiffs who have actually worked with diacetyl, the plaintiffs' bar had to cast a wider net to increase the pool of possible plaintiffs—as a result, consumers of buttery-tasting products have made their way to the courthouse doors.

VII. Consumer Litigation

In 2008, plaintiff Wayne Watson sued several corporate defendants in the District of Colorado, including Gilster-Mary Lee Corp. alleging that he voraciously consumed two to three bags of microwave popcorn per day for ten years. Watson claimed his remarkable appetite for popcorn caused him to develop lung ailments. A federal jury sided with him and awarded him a $7 million verdict, which the Court thereafter reduced to just over $3 million. Interestingly, in its opinion reviewing (and therein reducing) damages, the Court noted that "the use of diacetyl in butter-flavored microwave popcorn, did not continue during the pendency of the case"—and actually "ceased nearly a year before this action was even commenced."[75]

In another case venued in the Northern District of Iowa, plaintiff David Stults alleged that he too ate between one and three bags of microwave popcorn every day for twenty years—and, as the Eighth Circuit observed "practiced 'a ritual of slowly opening the freshly-popped bag as he breathed the aroma in through his nose.'"[76] Ultimately, a jury heard Stults' breach-of-implied-warranty claim against International Flavors & Fragrances ("IFF").[77] Several expert witnesses agreed that the plaintiff had bronchiolitis obliterans, but disagreed on whether it was caused by diacetyl exposure or an

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[74] In the same opinion, the California appellate court in *Velasquez* reversed the judgment (including the verdict) with respect to the claims (i.e., other than negligence) because of the prejudicial effect of the jury learning that the plaintiff had been an undocumented resident of the United States. *Velasquez*, 233 Cal. App. 4th at 1193.

[75] Watson v. Dillon Companies, Inc., No. 08-CV-00091-WYD-CBS, 2013 WL 4547477, at *6 (D. Colo. Aug. 28, 2013).

[76] Stults v. Am. Pop Corn Co., 815 F.3d 409, 412 (8th Cir. 2016).

[77] *Id.*

autoimmune disease.[78] The jury found in favor of defendant IFF, and Stults appealed the denial of his motions for judgment as a matter of law and for a new trial on the grounds that certain experts' testimony had been stricken and with regard to certain limiting instructions.[79] The Eighth Circuit affirmed, concluding among other things that: (1) a new trial was not warranted based on a defense expert witness's alleged failure to adequately rule out diacetyl as cause of consumer's bronchiolitis obliterans (Stults contended that the expert did not sufficiently rule it out in testifying that plaintiff's "disease state occurred at a time when he was not eating much popcorn . . . per his deposition;" and (2) issues of whether diacetyl fumes were hazardous to breathe, whether diacetyl-free butter flavorings were available, and whether butter flavorings were in popcorn consumer ate were appropriately for the jury to decide."[80]

We can also expect to see diacetyl litigation relating to exposure to products such as coffee and e-cigarettes. In January 2017, NIOSH published a blog indicating that five people who had worked in a coffee roasting facility had developed bronchiolitis obliterans. NIOSH found that the source of diacetyl came from a flavoring chemical added to coffee as well as from the grinding, processing, and storing of flavored coffee.[81]

Diacetyl is also found in many types of e-cigarettes, particularly those with fruit and candy flavors, and it is conceivable that resourceful plaintiffs' attorneys will venture into these new product lines by way of consumer litigation targeting diacetyl.

VIII. Future of Diacetyl Litigation

We can expect to see industrial and heavy consumer exposure cases continue. These cases are likely to continue proceed on theories of strict products liability and negligence. The consumer cases where the consumer has not manifested any personal injury present a different challenge for the plaintiffs' bar. Since strict products liability requires an actual personal injury, plaintiffs are likely to add false advertising and breach of warranty claims. Turning to consumer fraud causes of action and statutory schemes without an identifiable injury is torn from the aggressive plaintiff counsel's playbook, but with mixed success. These cases have been met with some skepticism,[82] with courts

[78] *Id.* at 412-413.
[79] *Id.* at 414.
[80] *Id.* at 417.
[81] Bailey et al., *supra* note 7.
[82] *See, e.g.,* Parker v. Brush Wellman, Inc., 377 F. Supp. 2d 1290, 1296-1299 (N.D. Ga. 2005) (expressing doubt that non-detectable, "sub-clinical" conditions can

frequently dismissing such claims,[83] although some have entertained them. The risk of future harm has likewise been held inadequate as an actual compensable injury in many cases,[84] although it may be sufficient to confer standing.[85] Whether plaintiffs adopt this somewhat unpromising course remains to be seen.

IX. Summary and Conclusion

Diacetyl litigation has not multiplied for various reasons, mostly centering on the considerable hurdles in proving both general causation and specific causation. With OSHA observing that "[n]o firm causative relationship between diacetyl exposure and bronchi obliterans has

constitute an injury); Henry v. Dow Chem. Co., 701 N.W.2d 684, 686 (Mich. 2005) (determining that extensive factual determinations surrounding possible future harm is a question for the legislature, not the courts); Burns v. Jaquays Mining Corp., 752 P.2d 28, 32 (Ariz. Ct. App. 1987) (rejecting claims of residents on land adjacent to asbestos-producing mill for risk of cancer and emotional distress based on transitory, nonrecurring physical conditions, such as headaches, indigestion, weeping, muscle spasms, depression, and insomnia, on the grounds that these did not constitute "substantial bodily harm"); O'Neil v. Simplicity, Inc., 553 F. Supp. 2d 1110, 1112–1120 (D. Minn. 2008) (discussing how class action on behalf of buyers of recalled cribs failed to state cognizable claims for breach of warranty, violation of various state statutes, and unjust enrichment where they did not allege actual manifestation of a defect resulting in some injury); Harrison v. Leviton Mfg. Co., No. 05–CV–0491, 2006 WL 2990524, at *4–7 (N.D. Okla. Oct. 19, 2006) (concerning defective electrical outlets that allegedly could overheat and cause fires; "Courts do not allow consumers to bring claims against manufacturers for products that are perceived to be harmful, but that have not actually cause[d] an identifiable injury").

[83] *See, e.g.,* Myers-Armstrong v. Actavis Totowa, LLC, 2009 U.S. Dist. LEXIS 38112, *4 (N.D. Cal. April 22, 2009) (concluding plaintiff who sued for economic damages after consuming a product recalled due to contamination in the manufacturing process lacked standing because she had consumed the pills and obtained their benefit with no downside); *In re* McNeil Consumer Healthcare Mktg. & Sales Practices Litig., MDL No. 2190, 2011 U.S. Dist. LEXIS 76800, 2011 WL 2802854, at *14-15 (E.D. Pa. July 15, 2011) (finding mere purchase of defective recalled products not sufficient to establish economic injury where plaintiffs did not suffer any ill effects from consuming the products).

[84] Boyd v. Orkin Exterminating Co., 381 S.E.2d 295, 296, 298 (Ga. Ct. App. 1989) (holding children with elevated levels of pesticide in their blood could not recover for "increased risk of cancer" because they had to prove to a "'reasonable medical certainty' that such consequences would occur" (quoting Phillip E. Hassman, *Annotation, Admissibility of Expert Medical Testimony as to Future Consequences of Injury as Affected by Expression in Terms of Probability or Possibility*, 75 A.L.R.3d 9 (1977))).

[85] Cent. Delta Water Agency v. United States, 306 F.3d 938, 947, 949-950 (9th Cir. 2002) (agreeing "with those circuits that have recognized that a credible threat of harm is sufficient to constitute actual injury for standing purposes, whether or not a statutory violation has occurred" in suit by downstream farmers to prevent release of water they alleged would cause salinity adversely affecting their irrigated crops).

been established." (OSHA 2014c), and NIOSH concluding that "[o]verall, current evidence points to diacetyl as one agent that can cause flavorings-related lung disease, other flavoring ingredients may also pay a role,"[86] support from the usual relevant agencies has been scant for establishing general causation.[87]

Further, animal toxicological studies are inconclusive, failing to report significant effects on the deep lung portion (i.e., the area of the bronchiole) in the test animals (Holebs) despite exposure to very high diacetyl concentrations. Several epidemiological studies found no relationships between diacetyl exposure and lung impairment. Reports of bronchiolitis obliterans in workers with no known diacetyl exposure is not surprising considering the large number of substances that can cause bronchiolitis obliterans such as sulfur dioxide, nitrogen dioxide, ammonia, chlorine, hydrogen fluoride, etc. and those studies concluding that there is an association between diacetyl exposure and bronchiolitis obliterans ignore whether other workplace chemicals might have caused the bronchiolitis obliterans.

Specific causation[88] has likewise been difficult to show, as it is usually established through differential diagnosis and exposure assessments. Bronchiolitis obliterans is fairly uncommon, difficult to diagnose, and often misdiagnosed, with asthma, chronic bronchitis, emphysema, and pneumonia among its various symptoms.[89] To ensure accuracy,[90] diagnosis must be confirmed through lung biopsy, a procedure fraught with risk of complications. Showing specific causation is no less a challenge than showing general causation. In short, while a suit

[86] National Institute for Occupational Safety and Health, *Flavorings-Related Lung Disease*, available at https://www.cdc.gov/niosh/topics/flavorings/exposure.html (last accessed November 24, 2017).

[87] General causation is that the agent in question is capable of producing a particular disorder (i.e., asbestos – mesothelioma). General causation is usually established through toxicology and/or epidemiology.

[88] Specific causation requires that the plaintiff was exposed to the agent in sufficient quantity to cause the particular disorder and said that the exposure did in fact cause the disorder in the plaintiff.

[89] Centers for Disease Control and Prevention, *Flavorings – Related Lung Disease – NIOSH Workplace Safety and Health Topics*, available at https://www.cdc.gov/niosh/topics/flavorings/ (last accessed November 24, 2017).

[90] *See, e.g.,* Newkirk v. ConAgra Foods, Inc., 727 F. Supp.2d 1006, 1011 (E.D. Wash. 2010), *aff'd,* 438 Fed. Appx 607 (9th Cir. 2011) ("A conclusive diagnosis of bronchiolitis obliterans may be made only through a lung biopsy.").

defeated by science is a wonderful outcome after a jury verdict, it is even better before a case is ever filed and litigated. It remains to be seen whether non-popcorn uses for diacetyl will spark new litigation, or whether the plaintiff bar will learn from these lessons.