Lung Disease from Microwave Popcorn Flavoring?

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A review of the scientific evidence pertaining to respiratory conditions linked to diacetyl and other flavoring agents, related litigation and the regulatory environment.

Some recent news articles have reported lung disease in consumers exposed to microwaved popcorn. For example, one series in 2007 described a report by a prominent pulmonologist, Dr. Cecile Rose of the National Jewish Hospital in Denver who diagnosed bronchiolitis obliterans, an insidious and disabling lung disease, in a patient who had allegedly contracted the disease from inhaling the fumes of microwave popcorn. The news shocked the medical community and prompted major manufacturers of microwave popcorn to announce that they were discontinuing the use of the flavorings containing the principle suspected agent—a chemical known as diacetyl.

While potential adverse health effects from exposure to diacetyl in microwave popcorn may be a new consumer concern, diacetyl has been on the radar screen of some industrial hygienists, government regulators and litigators for many years. Over the past decade there has been much scientific and regulatory scrutiny of diacetyl and its alleged role in lung disease in exposed plant workers. A number of lawsuits have alleged that workers exposed to diacetyl in butter-flavor compounds have suffered a variety of pulmonary injuries, including asthma, bronchiectasis, bronchiolitis obliterans, chronic bronchiolitis, chronic obstructive pulmonary disease, emphysema and severe lung impairment. In these cases, workers have alleged that butter flavor manufacturers failed to warn of the dangers in using butter flavor compounds in the production of microwave popcorn and other food products. Defendants in these cases include raw material suppliers—such as diacetyl suppliers—butter flavor manufacturers, including some of the largest flavor manufacturers, and microwave popcorn producers, including some large food companies.

Certain individuals in the medical, scientific, government and regulatory community have suggested a causal link between respiratory exposure to diacetyl and other flavorings and pulmonary injury. While rigorous scientific evidence supporting a causal
connection between diacetyl and any specific lung injury is extremely limited, and it might be argued that evidence is absent, the term “popcorn worker’s lung,” has nevertheless been applied to the putative health effect of exposure to diacetyl and other flavoring agents. Until recently, claims involving lung injury were strictly limited to the workplace setting.

This article reviews the scientific and medical evidence pertaining to the respiratory conditions allegedly linked to exposure to diacetyl and other flavoring agents, related litigation, the regulatory environment and implications for future flavoring agent cases.

**Scientific and Medical Overview**

**Diacetyl Exposure is Linked to the Lung Disease Bronchiolitis Obliterans**

Diacetyl is a natural byproduct of fermentation, and is found in beer, wine and butter. At low levels it imparts a smooth, slippery taste while at high levels, it imparts a butterscotch flavor. Diacetyl can also be produced chemically as a volatile ketone and has been used as a flavoring agent in a wide variety of products ranging from margarine to candy to bread for nearly 50 years. It has been widely used as a flavoring agent in microwave popcorn since the mid-1980s to provide the “buttery flavor” popular among consumers.

Earlier in this decade, researchers first reported cases of bronchiolitis obliterans and other lung diseases in a cohort of popcorn plant workers in Missouri. Strikingly, the workers were relatively young, in good health and had surprisingly short occupational exposures to diacetyl.

Following the reporting of this association, case reports, cross-sectional occupational epidemiological studies, and animal studies have led many scientists and government regulatory authorities to conclude that excess exposure to some constituents of butter flavoring can lead to an obstructive pulmonary condition. However, neither the necessary level of exposure nor the precise clinical characteristics of the condition are well-established.

**Obstructive Lung Disease: An Overview**

Lung diseases are commonly divided into two broad categories: restrictive lung diseases and obstructive lung diseases. Restrictive lung diseases are characterized by reduced expansion of the pulmonary tissues, with subsequent decreased total lung capacity. Restrictive diseases include the dust diseases, or pneumoconiosis, and most of the infiltrative lung conditions, specifically environmental diseases, sarcoidosis, idiopathic pulmonary fibrosis, and the collagen diseases. A. Husain and V. Kumar, “The Lung,” in Pathologic Basis of Disease, at 711–72, 729 (7th ed. 2005).

In contrast, the type of lung disease thought to be associated with constituents of butter flavorings is referred to as an obstructive lung disorder, which refers to the constellation of diseases characterized by limited expiratory air flow caused by a number of factors, and increased air flow resistance owing to partial or complete obstruction from the trachea and larger bronchi to the terminal and respiratory bronchioles. Individuals with obstructive lung disease have limited minimal air flow rates during forced expiration, usually measured by forced expiratory volume at 1 second (FEV1). Common obstructive disorders include emphysema, chronic bronchitis, bronchiectasis and asthma. A. Husain and V. Kumar, “The Lung” in Pathologic Basis of Disease, at 711–72, 716 (7th ed. 2005).

One specific disease diagnosis that has been linked by many researchers to butter-flavoring vapors is known as bronchiolitis obliterans (ICD9 code 466). This is a relatively uncommon disease characterized by both inflammatory processes as well as fibrosis (scarring) at the level of the small bronchioles of the small airways. Bronchiolitis obliterans fibrosis occurs in the lung’s small airways, as a result of damage to the bronchiolar epithelium. A.F. Hubbs et al., “Respiratory Toxicologic Pathology of Inhalated Diacetyl in Sprague-Dawley Rats.” 36 Toxicol. Pathol. 330, 330, (2008). This disease is characterized by plugs of loose fibrous connective tissue that obliterate the lumens of the bronchioles. A. Husain and V. Kumar, “The Lung” in Pathologic Basis of Disease at 711–72, 722 (7th ed. 2005). With bronchiolitis obliterans, air becomes entrapped, the small airways hyperinflated, which, in turn, results in the inability to rapidly empty the distal lung units during expiration. In short, air is trapped in the lungs because it cannot exit adequately due to obstruction. In addition to obstructed airways, patients with bronchiolitis obliterans have progressive shortness of breath on exertion, chronic cough and wheezing. Richard Kanwal, “Bronchiolitis Obiterans in Workers Exposed to Flavoring Chemicals,” 14 Curr. Opin. Pulm. Med. 141, 144 (2008).

The age range of workers afflicted with bronchiolitis obliterans ranges from the second to the fifth decade. While symptoms typically have a gradual onset, with increasing shortness of breath over months or years of continued exposure, some individuals have acute manifestation of disease. These workers may slightly improve temporarily, then progressively become worse. After exposure ceases, workers usually experience disease stabilization, although some experience worsening lung function for up to two years. The potential severity of the disease is evidenced by five sentinel workers who were placed on lung transplant lists. Of note, medical treatments for bronchiolitis obliterans have been ineffective to date. Richard Kanwal, “Bronchiolitis Obiterans in Workers Exposed to Flavoring Chemicals,” 14 Curr. Opin. Pulm. Med. 141, 144 (2008).

A critical feature of bronchiolitis obliterans is that an individual’s obstructed airflow is not reversible when he or she is given bronchodilators. This is in contrast to other obstructive lung disorders such as chronic obstructive lung disease (COPD), where air flow obstruction is accompanied by reduction in lung capacity to diffuse carbon dioxide when obstruction is severe. COPD, unlike bronchiolitis obliterans, often responds favorably to bronchodilators. This distinction provides an important way to distinguish bronchiolitis obliterans from other obstructive pulmonary diseases, and as such, determining
whether elements of butter flavoring are implicated in a bronchiolitis obliterans-related claim.

Scientific Studies Support a Link between Diacetyl and Obstructive Lung Disease

Animal Studies

Animal studies conducted by the National Institute of Occupational Safety and Health (NIOSH) showed that diacetyl in popcorn butter flavoring was highly toxic to the airways of rats. A.F. Hubbs et al. “Necrosis of Nasal and Airway Epithelium in Rats Inhaling Vapors of Artificial Butter Flavoring.” 185 Toxicol. Appl. Pharmacol. 128, 134 (2002). In 2008, two published toxicology studies from one research group alone have demonstrated that acute, chronic high doses of diacetyl impaired respiratory function in both rats and mice. A.F. Hubbs et al. “Respiratory Toxicologic Pathology of Inhaled Diacetyl in Sprague-Dawley Rats.” 36 Toxicol. Pathol. 330, 330 (2008); D.L. Morgan et al. “Respiratory Toxicity of Diacetyl in C57BL/6 Mice.” 103 Toxicol. Sci. 169, 172 (2008). In addition, at least one study in guinea pigs showed diacetyl exposure initially targeted and damaged the respiratory epithelium and compromised epithelial barrier function. J.S. Fedan et al. “Popcorn Worker’s Lung: In Vitro Exposure to Diacetyl, an Ingredient in Microwave Popcorn Butter Flavoring, Increases Reactivity to Methacholine.” 215 Toxicol. Appl. Pharmacol. 17, 22 (2006). Although the animals were exposed to diacetyl in solution rather than in gaseous form, this study demonstrated the toxic effects of diacetyl on the respiratory lining. Id. A more recent study demonstrated cellular degeneration and death in the lining of the nose, larynx, trachea and intrapulmonary airways of rats inhaling diacetyl vapors as a single agent exposure. These data suggest that the no observable adverse effect level (NOAEL) is less than 93 ppm, but that exact value is still unknown. A.F. Hubbs et al. “Respiratory Toxicologic Pathology of Inhaled Diacetyl in Sprague-Dawley Rats.” 36 Toxicol. Pathol. 330, 337 (2008). In addition, these data demonstrate that even brief, high diacetyl exposures can damage epithelium, exposure characteristics that can occur in workplaces where workers mix flavorings.


However, these data must be interpreted with caution. Rats may experience greater nasal epithelial damage than humans from vapor exposures, since the rat nose absorbs gases much more efficiently than the human nose. Of note, the smaller diameter of the rat respiratory tract would have increased resistance, decreased airflow and increased mucosal deposition of vapors when compared to the corresponding human respiratory structures. Therefore, it is probable that rats are less likely than humans to have lung damage resulting from vapors. A.F. Hubbs et al. “Respiratory Toxicologic Pathology of Inhaled Diacetyl in Sprague-Dawley Rats.” 36 Toxicol. Pathol. 330, 330 (2008); K. et al. “Evaluation of Flavorings-related Lung Disease Risk at Six Microwave Popcorn Plants.” 48(2) J. Occup. Environ. Med. 149, 155 (2006).

Human Studies


For The Defense
respectively, and had no history of pulmonary disease. Each mixed diacetyl powder routinely without exposure control methods, such as local exhaust ventilation. Since these reports emerged, several other flavor compounds have been diagnosed with bronchiolitis obliterans. In response, CDHS disseminated a diacetyl hazard alert and description of sentinel cases to flavor manufacturers, health care providers and worker organizations. Available at http://ww2.cdph.ca.gov/programs/ohb/Pages/flavorings.aspx. As a result of these cases, in January 2007, the CDHS with assistance from NIOSH, began performing quality checks of submitted screening spirometry from companies that manufacture flavoring chemicals. B. Materna et al. “Fixed Obstructive Lung Disease among Workers in the Flavor-Manufacturing Industry—California, 2004–2007.” 56(16) MMMR Weekly 389, 392 (2007). Accordingly, the government scientists involved in these studies have generally concluded that diacetyl has some causal link to the adverse pulmonary events observed. The CDC also recently published a Health Hazard Evaluation Report suggesting an association between severe obstructive lung disease in workers at a manufacturing plant and diacetyl exposure. R. Kanwal and G. Kullman. Report on Severe Fixed Obstructive Lung Disease in Workers at a Flavoring Manufacturing Plant; Health Hazard Evaluation Report HETA 2006-0303-3043 Carmi Flavor and Fragrance Company, Inc. (2007). However, it should be noted that these studies are cross sectional and descriptive in nature, and no clear disease definitions or criteria have been proposed.

Moreover, a recent study analyzed 175 workers in a chemical production plant that produced diacetyl between 1960 and 2003. This study supported the conclusion that an agent in the diacetyl production process caused bronchiolitis obliterans syndrome in process operators. Process operators involved in the study had the highest levels of diacetyl exposure in the internal reference group. Importantly, this is the first study where cases of bronchiolitis obliterans were identified in a plant manufacturing diacetyl. Frits G.B.G. van Rooy et al. “Bronchiolitis Obliterans Syndrome in Chemical Workers Producing Diacetyl for Food Flavorings.” 176 Am. J. Respir. Crit. Care Med. 498, 503 (2007). Thus, these studies set forth seminal evidence demonstrating a link between occupational exposure to flavorings containing diacetyl and the development of obstructive lung disease in workers exposed to significant levels of the chemicals in flavors. Further studies are necessary to determine whether diacetyl alone can cause bronchiolitis obliterans, or whether this disease arises in conjunction with exposure to other chemicals. In addition, safe levels of exposure are still unknown.

The available scientific evidence, therefore, provides a reasonable basis for developing the hypothesis that high dose inhalation of diacetyl or some ingredient in microwave butter flavoring in the occupational settings might be linked to some sort of chronic lung disease. However, the evidence to date is hardly definitive. Placing a causal link between exposure to these flavor compounds and chronic lung disease in consumers is even more speculative and clearly without scientific support.

A Concern for Consumers?

A flurry of news reports followed the public dissemination of a July 18, 2007 letter to the FDA’s Deputy Director by a noted investigator, Dr. Cecile Rose of the National Jewish Medical and Research Center, highlights the complexity of establishing whether chemical exposures cause, or are capable of causing, chronic diseases. Indeed, in the letter to the FDA, Dr. Rose explicitly stated that “it is difficult to make a causal connection based on a single case report” and that her team “cannot be sure that this patient’s exposure to butter flavored microwave popcorn from daily heavy preparation has caused his lung disease.” Nevertheless, rushing to judgment, the plaintiff’s bar has abandoned prudence and used case reports such as Dr. Rose’s as “evidence” that exposure to popcorn butter-flavoring vapors causes adverse pulmonary events in consumers. A simple Google search of the phrase, “popcorn worker lung,” or “diacetyl lung injury,” will generate links to several plaintiff’s firms citing Dr. Rose’s July 18, 2007 letter as “proof” that consumers are at risk of lung disease from microwave popcorn.

Why is it scientifically unsound to rely upon a single case report or even a number of case studies to establish that inhalation of popcorn butter-flavoring vapor causes lung disease? Although this is a complex question well beyond the scope of this article, in general, it is impossible to determine whether a particular disease, such as bronchiolitis obliterans, simply occurs as part of the general background or if its rate was truly increased as a result of the exposure. Epidemiology, which studies how, when and where diseases occur, is devoted to unearthing this answer in public health departments and university research programs that devote hundreds of millions of dollars to it every year. The only time it is possible to causally link an exposure to a disease without addressing the potential that the disease was part of the background is when the disease in question is truly a “signature disease,” which means it is a truly diagnostically unique entity that only occurs in the presence of the hazardous exposure, such as the case with asbestosis.

When confronted with a single case such as that noted in Dr. Rose’s July 18, 2007 letter to the FDA, physicians perform a differential diagnosis, which, through process of elimination, excludes all but the best diagnosis, to determine, to the extent possible, the illness. However, a differential diagnosis rarely, if ever, identifies the underlying cause of the illness. According to their letter to the FDA, the physicians assert two things: (1) the patient’s symptoms are very similar to symptoms of occupational workers with lung disease who were exposed to diacetyl in specific manufacturing environments; and (2) the researchers have no other plausible explanation for the illness in this individual. Thus, according to these investigators, bronchiolitis obliterans is such a rare condition that it is unexpected independent of some unusual event, such as an exposure to a toxic substance.

However, a differential diagnosis suggesting some sort of link between an exposure to diacetyl and bronchiolitis obliterans is problematic because it does not take into account a number of factors, including the following:

- There is no well-accepted definition of diacetyl-induced lung disease
- There is a background rate of obstructive lung disease in the general population
- There are other known causes of obstructive lung disease

The bottom line is that without a reliable, controlled epidemiological assess-
ment looking at the rate of a well-defined lung condition in consumers, both exposed and unexposed, to popcorn butter-flavoring vapors, it is impossible to determine if the disease diagnosed in Dr. Rose’s July 18, 2007, letter to the FDA was causally related to the individual’s exposure to diacetyl vapors, or if it would have occurred independent of exposure.

Advocates of a specific position, such as plaintiff’s lawyers, are often reluctant to precisely define a disease entity.

The Importance of Disease Definition
In linking a disease entity to an exogenous exposure, the importance of a precise disease definition is often overlooked. Indeed, without a precise disease definition, it is impossible to properly assess the potential causal link. The more specific and unusual the disease, the rarer it is in the general population, reducing, to some extent, the need for controlled epidemiological studies. Consider a hypothetical disease, Blue Fingernail Syndrome, defined as the fingernails of the left hand turning a distinct shade of cobalt blue. In this case, controlled epidemiological studies would not be needed to link an exposure to this disease; a few cases would be sufficient to link the exposure to the disease. In reality, disease entities are not normally so unique or well-defined. When there is even a small background rate of a disease in the general population, controlled studies are required to assess reliably whether an exposure truly increases the rate of that disease entity.

Advocates of a specific position, such as plaintiffs’ lawyers, are often reluctant to precisely define a disease entity because then individual cases can fall outside the realm of potential causation; if an individual case does not meet the disease criteria, it can’t be considered an occurrence of that disease. Therefore, imprecise disease definitions are proposed. For example, someone might posit that a particular exposure causes headaches. From a scientific standpoint, such a loose disease definition is acceptable, but without more precision, such as specifying the types of headaches and how long the headaches must occur, the background rate of the disease entity will be extremely high. A high background rate of disease in the general population will diminish association between the exposure and the disease because both exposed and unexposed individuals will present with the disease.

When a disease entity has a very precise disease definition—for example, a truly, narrowly defined bronchiolitis obliterans syndrome—it will present rarely. Fewer cases are categorized as that disease, reducing, to some degree, the need for controlled scientific studies to definitively establish association. Some controlled study would still, however, be necessary.

When the disease is more common or imprecisely defined, such as “any lung disorder,” the number of cases increases dramatically, in both the exposed and unexposed populations, and if a causal link is posited, it becomes critically important to determine if exposure increases the risk of the disease.

Notably, the World Health Organization’s International Classification of Diseases (ICD-10) lists a disease entity called “Obliterative bronchiolitis” (J68.4). This is a rare, chronic respiratory condition that, according to the ICD definition, is linked to “chemicals, gasses, fumes, and vapors.” Thus, a physician could confer this diagnosis on an individual exposed to diacetyl suffering from lung abnormalities. Nevertheless, the absence of scientific evidence linking this condition to microwave butter flavoring specifically, begs the question of whether the diagnosis is accurate in the first place.

Background Rate of Obstructive Lung Disease
Dr. Rose’s July 18, 2007, letter to the FDA fails to acknowledge the background rate of bronchiolitis obliterans in the general population or specify other toxic vapors to which the diagnosed individual might have been exposed that could explain his illness. The prevalence in the U.S. of chronic obstructive lung disease (COPD), an obstructive pulmonary disease, has been estimated to be as high as 24 million. Indeed, according to a recent New York Times article, a 500-year-old mummy unearthed in 1999 was “diagnosed” with bronchiolitis obliterans, possibly as the result of an infection. Thus, lung disease resembling the disease experienced by food flavoring workers has been around far longer than diacetyl-containing food flavorings.

Other Causes of Obstructive Lung Disease
With respect to other causes of his lung condition, it is our understanding that the patient described by Dr. Rose was overweight and that when he lost weight, his FEV-1 went from 50 percent of normal to 75 percent of normal. Additionally, he was a furniture salesman, and he may have performed some furniture stripping with chemical products, exposures that very well might be causally linked to adverse pulmonary functioning.

Regulatory Prudence vs. Evidence for Causation
To be sure, decisions about public health are separate from the science involved in determining whether a chemical actually causes an illness. The former operates from the principle of “abundance of caution,” while the latter operates from precise, time-tested rules. FDA officials have rightfully responded vigorously to Dr. Rose’s letter of July 18, 2007, to determine if there is any potential cause for public health concern. But a serious, well-considered regulatory assessment is quite different from the wide public outcry and hysteria posing a demonstrated link between butter-flavoring vapors and adverse respiratory health based on a single, anecdotal case report. Everyone from the news media to the plaintiffs’ bar jumped on the bandwagon, suggesting that the link is now well-established. Using anecdotal reports to draw unscientific conclusions about cause and effect is irrational, at best.

It is unfortunate that a party can take a legitimate communication from a conscientious doctor out of context, and thereby engender public fear. Analogous dramatic mistakes have been made in the past based on anecdotal evidence. Previous erroneous claims grounded in anecdotes held that breast implants caused systemic autoimmune disease and that cellular telephones
caused brain cancer. Only by responding to these irresponsible allegations with well-considered and rigorous science were they effectively dispelled.

**Litigation Landscape**

For the last several years the plaintiff’s bar has been slowly and methodically building their case against diacetyl in an ongoing effort to find the next asbestos. Approximately 13 cases are currently pending in Illinois, Indiana and elsewhere, mostly throughout the Midwest. Plaintiffs found early success with substantial verdicts, which have only served to raise the profile of this litigation, encouraging the plaintiff’s bar to pursue recovery against new, diverse defendants. By effectively using the media, complex medical issues with multiple causal links have been blindly linked together as “popcorn lung,” adding unfounded credibility to the plaintiff’s argument that there is a direct and causal link between a wide array of lung impairments and exposure to diacetyl at undefined levels, to the exclusion of other known causes.

From the plaintiff’s perspective, the search for “the next asbestos” requires that every case follow a predictable plot line: sympathetic plaintiff is injured by evil, greedy industry, which put profit ahead of safety by ignoring, or worse, suppressing, clear, scientific evidence about the product’s harm. Many toxic tort cases not only follow the same plot line, they often involve the same players, including the same veteran lawyers, experts, and in the case of silica, even some of the same plaintiffs as the asbestos litigation. Claims including diacetyl are starting to follow the same pattern.

At the early stages of the diacetyl litigation, litigants pursued claims against the food flavoring trade associations and manufacturers under various theories, including conspiracy and fraudulent concealment. Many of these cases did not survive the motion to dismiss stage, due to the heightened pleading requirements in fraud claims, which forced plaintiffs to focus on more traditional product liability theories. Plaintiffs also faced difficulties in securing jurisdiction and establishing liability against the trade associations that played no role in the manufacture, distribution or sale of diacetyl, forcing plaintiffs to change their focus from the trade associations to manufacturers and suppliers.

Many of the fraud claims pursued in the early diacetyl actions alleged that the defendants participated in a “conspiracy” to withhold information concerning the potential health hazards associated with diacetyl. In *Remmes v. International Flavors & Fragrances, Inc.*, 389 F. Supp. 2d 1080 (N.D. Iowa 2005), a worker in a popcorn processing plant sued multiple defendants, including the suppliers and a trade association, alleging that he had been injured through exposure to diacetyl in food flavoring. The plaintiff’s fraud claim alleged that the defendants conspired with at least one manufacturer to suppress the health risks of butter flavorings. The court declined to accept the plaintiff’s attempt to group the fraud allegations against multiple defendants and dismissed the fraud claims with leave to amend on the basis that the complaint failed to attribute specific fraudulent statements to specific defendants. The plaintiff eventually served an amended complaint outlining additional allegations in support of the fraudulent concealment claim, but failed to allege justifiable reliance, a necessary element to maintain such a claim. *Remmes v. International Flavors & Fragrances, Inc.*, 453 F. Supp. 2d 1058 (N.D. Iowa 2006). The plaintiff’s amended claim was eventually dismissed.

Although plaintiffs found limited success in pleading fraudulent concealment claims, the diacetyl litigation still threatens to expand the number and identity of litigants, and the theories of liability. Plaintiffs have begun to bring traditional product liability claims against suppliers and manufacturers and others in the direct stream of commerce, focusing on duty to warn theories and an analysis of what the manufacturers knew, and when they became knowledgeable.

In other toxic tort matters, defendants have often relied, at least in part, on the government’s research and reaction to potential hazards associated with exposure as a benchmark to measure their own culpability. Diacetyl has long been listed on the governments list of food additives that are Generally Regarded as Safe (GRAS). Not surprisingly, the GRAS system has come under withering attack by plaintiffs and their experts and even by some members of Congress. Defendants will have an increasingly difficult time relying solely on the government’s GRAS listing and regulatory agency reaction as a benchmark for their own behavior because the government has been widely criticized for its slow and arguably inadequate response to the diacetyl issue.

As in other toxic tort matters, defendants, with the assistance of industrial hygienists and other experts, will focus their defenses on the level of exposure experienced by a plaintiff and the level of exposure that causes illness. Understanding both levels will be critical to defending exposure claims arising from workers in the food flavoring industry and claims arising from the general public’s purchase of food products containing diacetyl. The success of the defendants’ and plaintiffs’ battle over the scientific validity of these claims, and the levels of exposure necessary to cause illness, will be critical in determining whether the diacetyl litigation will continue to grow.

**Defense Strategies Moving Forward**

As with most toxic torts involving complex scientific issues, successful defense of diacetyl-related claims will be based on the following four broad strategies, separately and in combination:

- **General Causation Defense:** Attack the scientific reliability of linking diacetyl exposure in the workplace and/or in consumer settings to lung disease in general. Just because an exposure is plausibly related to an injury, this is not evidence that the exposure did cause that injury.
- **Exposure Defense:** Focus on the specifics of each individual plaintiff’s exposures. Attempt to make the case that while diacetyl is capable of causing pul-

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monary events under certain occupational circumstances, it does not cause them at the exposure levels seen by the overwhelming majority of claimants.

- **Specific Causation Defense:** Identify all potential causes of plaintiff’s condition. In almost every case the diagnosis will have been made by “differential diagnosis,” which will have supposedly “ruled out” all other potential causes of the alleged condition. Thorough review and close scrutiny of a plaintiff’s past medical records and occupational exposure history will often yield many other potential causes missed by the diagnosing doctor.

- **State of the Art Defense:** Depending on when the exposure took place and what precautions were used in the workplace, the state of scientific knowledge may not have developed to a point where it could have reasonably guided any different action by the defendant.

Regarding the general causation defense, there is considerable debate among scientific experts regarding which compounds—in addition to or apart from diacetyl—could be potential etiological agents in this type of pulmonary injury. In fact, NIOSH stated outright that the etiological agent responsible for these reported respiratory symptoms is, as yet, undetermined. Only further in-depth evaluation of potential respiratory exposure routes and the available toxicological evidence can shed more light on the issue of the responsible agent(s). Further, the potential effect of workers’ smoking behavior on propensity to sustain pulmonary injury needs to be fully explored. An effective defense to diacetyl claims must be based on understanding the science surrounding these claims.

To effectively explore the specific causation defense, it will be necessary to develop a comprehensive understanding of the exposure characteristics in plant environments as well as a deeper understanding than is currently available of other potentially toxic agents to which production workers are exposed. For example, if airborne volatile organics adhere to the surface of other airborne particulates resulting in increased pulmonary exposure, the cause of adverse pulmonary events could theoretically involve more than diacetyl exposure alone. If so, particulate control and good housekeeping are important measures to reduce exposure. The suppliers of butter flavoring to microwave popcorn plants have no control over airborne particulate loads in microwave popcorn-producing plants, and yet this particulate load may play a role in users’ exposures to a product’s compounds. An effective defense must be based on an overall understanding of the details of each plaintiff’s alleged exposure.

For purposes of the state of the art defense, counsel working with clients will need to develop a thorough understanding of their client’s knowledge of the potential hazards of and practices concerning diacetyl exposure, including client review of product material safety data sheets (MSDS), warnings given to and received by suppliers, and client history of past workers compensation claims.

The attorney will also need to work with his or her client to develop a timeline charting when the client knew about any potential diacetyl toxicity and when the client could have or should have reasonably known that diacetyl exposure could cause injury. The timeline needs to take into account what was generally available in the medical and scientific literature. Once again, do not be surprised to learn that reasonable minds will differ on the scope of the “problem.” For example, the initial 2000 NIOSH industrial hygiene survey published in the *Journal of Occupational and Environmental Hygiene* in 2005 concluded that there were no exposures that were thought to be hazardous.

As with all toxic tort claims, the best defense will be a well-coordinated one based on sound science and well-developed, case-specific facts.