IN THE SUPREME COURT
STATE OF FLORIDA

CASE NO. SC00-490

JOHN CASTILLO, a minor by and through his mother, next friend and natural guardian, Donna Castillo, DONNA CASTILLO and JUAN CASTILLO, individually,

Petitioners,

v.

E.I. DuPONT DE NEMOURS & COMPANY, INC.
and
PINE ISLAND FARMS, INC.,

Respondents.

RESPONDENT DUPONT'S CORRECTED ANSWER BRIEF ON THE MERITS

ON DISCRETIONARY REVIEW FROM THE
THIRD DISTRICT COURT OF APPEAL

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INTRODUCTION

As humbling as it is, particularly in this, the "Information Age," scientists simply do not know what causes most birth defects. With more breakthroughs in the Human Genome Project (the public-private partnership to map the human genetic code), it may be possible one day to pinpoint the source of all birth defects. But that point has not yet been reached, and until it has, American courts will be forced to review carefully the expert testimony in tort cases, like this one, which involve birth defects. "The ignorance that prevails as to the etiology of most birth defects . . . mean[s] that there is a large *terra incognita* where gossip and guesswork abound, so that courts must carefully control the basis for testimony pointing to a particular cause." *Lynch v. Merrell-National Labs.*, 830 F.2d 1190, 1194 (1st Cir. 1987). That need for close scrutiny of scientific evidence bearing on causation issues is even more magnified in birth-defects cases, where "[t]he sight of a helpless mutilated youngster may evoke emotion along with the corresponding wish to make somebody pay for his or her plight." *Id.* at 1196.

In this case involving a birth defect and complex scientific causation evidence, the Court of Appeal performed that job admirably, producing a thoughtful, scholarly, and detailed opinion applying *Frye v. United States*, 293 F. 1013, 1014 (D.C. Cir. 1923), to exclude the plaintiffs' expert testimony on causation because it was conceded by those very experts to be new and not generally accepted science. Indeed, the Court of Appeal did not allow the significant (though ultimately conceded) *Frye* questions presented to distract it from evenhandedly applying the more routine rules of law that were also at issue. Thus, the appellate court ruled in favor of the defense on issues that have nothing to do with the admissibility of expert scientific testimony. Unfortunately, the trial court did not acquit itself so well. At nearly every key juncture, the trial judge below went astray, and went astray badly.

As a result, plaintiffs were allowed to present a case to the jury based on violations of some of the most bedrock guarantees in our jurisprudence for ensuring

the fairness of trials. In particular, the trial court refused to grant a directed verdict motion or judgment as a matter of law when the only possible basis for a plaintiffs' verdict was inadmissible hearsay evidence the trial court itself had properly excluded *in limine*. The trial court also allowed the jury to be prejudiced by erroneous information suggesting that defendants had caused "clusters" of children in Britain to be born with the very same birth defect.

After making errors of that magnitude, it is not surprising that the trial court also allowed the worst sort of "junk science" testimony to go to the jury. Accordingly, the jury verdict in favor of the plaintiffs below was also not unexpected. People have a natural tendency to look for someone to blame for a tragedy, and a large corporate defendant makes an attractive target. Florida's tort system, however, should not be permitted to become the means for outright wealth redistribution. "Although [the plaintiff's misfortune] elicits deep compassion and regret, this Court cannot allow a human tragedy to beget a legal one." *Raynor v. Richardson-Merrell, Inc.*, C.A. No. 83-3506, 1993 WL 484200, at *3 (D.D.C. Apr. 13, 1993).

STATEMENT OF THE CASE AND FACTS

A. Background

Plaintiff Donna Castillo is a college graduate with a degree in secondary education, and has worked as a teacher and teacher's aide. *See* Tr. 884-87, 971. In June of 1990, Mrs. Castillo (then 33 years old) gave birth to a son, plaintiff John Castillo. *See id.* at 883, 891-92. John was born with microphthalmia, a rare birth defect involving severely underdeveloped eyes. *See id.* at 3004. The birth defect microphthalmia (and the closely related condition, anophthalmia – the complete absence of eyes) has been known since "ancient times." *Id.* at 4619. Other than this condition, which causes blindness, John has no birth defects. *See id.* at 889.

Almost three years after John's birth, Mrs. Castillo was contacted by John

Ashton, a British reporter preparing a story on microphthalmic children in England. *See id*. at 917-20, 1584. Ashton suggested that John's condition might have been caused by the family's proximity to an agricultural area – and in particular by a fungicide manufactured by DuPont called Benlate. *See id*. at 1585-86, 1618-19.

Plaintiffs filed this action against both DuPont and Pine Island Farms (the "Farm") later that year, alleging that Mrs. Castillo had been exposed to Benlate from a nearby "u-pick" agricultural field during her pregnancy, and that this exposure had caused John's microphthalmia. *See* R. (7/29/93) 2-14 . At a deposition in October 1993, Mrs. Castillo testified that, while pregnant with John, she would occasionally take walks around her neighborhood with her baby daughter, Adriana. *See* R. (10/21/93) 8576-77. On these walks, she said she would often visit a small shopping center located across from the "u-pick" field. *See id*. at 8578-94. On "three or four" occasions, she asserted, she observed spraying in the field, and saw a "huge foggy mist" surrounding "a farm tractor on the field." *See id*. at 8594-96. When asked whether any of that mist got on her skin, Mrs. Castillo stated: "*I don't know. I can't say for sure. I don't know*." *Id*. at 8652 (emphasis added). When asked whether she remembered "being sprayed physically so that you felt it on your skin," Mrs. Castillo replied, "*No, I don't remember that.*" *Id*. at 8653 (emphasis added).

At the core of plaintiffs' case was the theory that the mist to which Mrs. Castillo had allegedly been exposed contained Benlate, and that benomyl (the active ingredient in Benlate) had entered her body and caused John's condition. In support of that theory, plaintiffs proffered the expert testimony of Dr. Charles Vyvyan Howard, the equivalent of an associate professor at the University of Liverpool, England. In depositions, Howard testified that he believed that human fetal exposure to benomyl at a concentration of only 20 parts per billion ("ppb") in the maternal bloodstream would cause microphthalmia. *See* R. (3/12/96) 6157-58, 6203-07, 6214.

Dr. Howard based his conclusion on: (1) rat gavage studies (in which rats were

fed massive quantities of the chemical by means of tubes inserted directly down their throats to their stomachs); and (2) laboratory experiments on human and mouse cells done in petri dishes. *See id*. at 6023-25, 6061, 6157-58, 6203-07, 6214; *see also* R. (9/20/95) 4455-63. Before trial, Howard insisted that "the *principal* component of Mrs. Castillo's acute benomyl exposure was inhalational," and that she could have *inhaled* sufficient Benlate to achieve at least 20 ppb of benomyl in her blood. *See* R. (3/12/96) 6157-58, 6203-07, 6214; Tr. 3081-85, 3088 (emphasis added).

Defendants moved before trial to exclude Howard's testimony on the ground that his methodology for determining whether and at what level Benlate could cause birth defects in humans was not "generally accepted" in the scientific community and thus inadmissible. *See* R. (4/22/96) 2494-531. At a hearing on that motion, the testimony was unequivocal that Howard's methodology was not "generally accepted." Indeed, plaintiffs' own expert, Dr. Dick van Velzen, testified that the methodology on which he and Howard had relied to determine the cause of John's microphthalmia was "new," Tr. (4/30/96) 275, and that he was aware of no scientific study that had ever before purported to determine the causation of a birth defect in this manner, *see id*. at 252-53, 257, 263-65, 273; *see also* Tr. 3300.

The trial court, however, effectively refused to apply the "general acceptance" standard. Instead, the court declared that the expert testimony would be admitted "if *I believe* the science is reliable and . . . would assist the trier of fact." Tr. (5/1/96) 51-52 (emphasis added). Although DuPont reiterated its motion to exclude Howard's testimony at several other junctures before and during trial, *see* R. (5/6/96) 3160-280; R. (5/6/96) 3281-540; R. (5/6/96) 3541-43; R. (5/8/96) 3577-87; Tr. 2934, the trial court denied the pivotal motion in an untranscribed telephone conference call with the parties on May 11, 1996 (which is why the Third District had to rely on the trial judge's statements at the *Frye* hearing to infer the basis for the trial court's ruling).

A few weeks before trial (and after his deposition), Dr. Howard realized that his

exposure calculations had exaggerated the amount of spray that Mrs. Castillo could have inhaled by a factor of ten. *See* Tr. 3075-88. Plaintiffs' primary exposure theory, accordingly, shifted from exposure through the lungs ("inhalational" exposure) to exposure through the skin ("dermal" exposure).[1] At her final deposition on April 24, 1996 (some three weeks before the beginning of trial), Mrs. Castillo for the first time claimed to have been drenched by the spray. According to Mrs. Castillo's testimony at her final deposition and at trial, during a walk on November 1 or 2, 1989, she saw a tractor "bucking and jerking" at the very edge of the Farm's property immediately adjacent to a busy street. *See* R. (4/24/96) 7630; Tr. 905, 907-09, 993, 998-99. The tractor, she asserted, was not spraying the crops but instead spewing "tons" of a "cloudy, misty foggy spray" directly across the busy road toward the shopping center. *See* R. (4/24/96) 7630, 7677, 7680; Tr. 908, 989.

Mrs. Castillo testified that she walked toward the tractor *nonetheless* until she was "almost on top of [the spray area]," *see* R. (4/24/96) 7620, 7623-24; Tr. 914, 984-85, 1003-04, because she was "mesmerized" by the spray, Tr. 999-1001. She stated that she then stood directly in the spray for some two to three minutes. *See* R. (4/24/96) 7664, 7690; Tr. 995-996. While standing there "staring at the spray" coming across the street, Tr. 998, Mrs. Castillo asserted that she got drenched "*like when you would stand out in the rain*." *Id.* at 997 (emphasis added). Mrs. Castillo further testified that she was accompanied at the time by her 8-month old daughter, Adriana,

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[1] As noted above, Dr. Howard opined in a pretrial deposition that Mrs. Castillo's principal exposure to benomyl was inhalational. When Dr. Howard began emphasizing dermal exposure, of course, DuPont objected to this switch on a central issue in the case. *See* R. (6/17/96) 8829. The trial court, however, allowed Howard to present his new theory of exposure to the jury, notwithstanding a pretrial ruling forbidding experts from "sandbagging" the opposing party by altering the opinions they had expressed before trial. *See* R. (4/26/96) 2746-47; Tr. (4/29/96) 118-22.

see R. (4/24/96) 7627, 7630; Tr. 888, 901, 912, 976-77, 996-97, and knew that she was pregnant with John, *see* R. (4/24/96) 7657-59; Tr. 996.

After the alleged "drenching," Mrs. Castillo allegedly told her husband, Juan Castillo, about the incident that evening, and expressed concern about the possible effects on their unborn child, *see* R. (4/24/96) 7631-32; Tr. 4092, 4105, but did not mention the incident to her obstetrician during a regularly scheduled appointment several days later, *see* Tr. 1006, 4105.

B. The Trial Proceedings

The trial began on May 13, 1996, and lasted for 17 days. At trial, plaintiffs expressly limited their case to the single "drenching" incident, and did not contend that Mrs. Castillo had otherwise been exposed to Benlate. *See id.* at 747, 762, 5299, 5304. They also limited their case to the theory that benomyl had entered Mrs. Castillo's blood through her skin. *See id.* at 3075, 3099. Based on the rat studies, *see id.* at 2972-74, 3174, 3184, and *in vitro* experiments showing effects by benomyl on cells at a 20 ppb concentration, *see id.* at 3039-47, 3159-60, 3177-82, 3186-89, 3191-93, Howard provided the plaintiffs' critical causation evidence by declaring that, pursuant to various assumptions, Mrs. Castillo could have absorbed enough benomyl through her skin to achieve a blood level of at least 20 ppb. *See id.* at 3134-36.

At the close of the evidence, DuPont moved for a directed verdict on the ground that plaintiffs had failed to prove the core elements of their case: (1) that Benlate is defective, and (2) that any such defect proximately caused John Castillo's condition. *See* R. (5/31/96) 3854-66; Tr. 4464-79; *see also* R. (3/29/96) 1385-89; R.(4/22/96) 2494-531. On the defect issue, DuPont stressed that plaintiffs' entire case was based on an alleged *misuse* of Benlate by the Farm that was inconsistent with any finding of defect, *see* R. (5/31/96) 3860-61; Tr. 4477-79. DuPont also stressed, renewing a pre-trial motion that was only partially granted, that the Federal Insecticide,

Fungicide, and Rodenticide Act ("FIFRA") preempted the entirety of plaintiffs' case. *See* R. (3/29/96) 1390-1406; R. (5/6/96) 3116-26; R. (5/31/96) 3850. The directed-verdict motion was denied. *See* Tr. 4506-07, 5060.

The jury returned its verdict on June 7, 1996, holding DuPont liable on a strict-liability theory, and both DuPont and the Farm liable on a negligence theory. The jury awarded a total of $4 million in damages to John Castillo (his parents took nothing), of which it allocated 99.5% ($3.98 million) against DuPont, and .5% ($20,000) against the Farm. DuPont moved to set aside the verdict and/or for a new trial pursuant to Fla. R. Civ. P. 1.480 and 1.530. *See* R. (6/17/96) 8814-977. The trial court denied the motion. *See* R. (8/7/96) 8813.

C. Proceedings in the Third District Court of Appeal

DuPont and the Farm appealed from the trial court to the Third District Court of Appeal ("Third District"). The Third District held oral argument on December 15, 1997. After reviewing the case for over a year, the Third District issued an opinion on February 17, 1999 reversing the trial court and overturning the jury verdict. *See* R. (2/17/99) 8884-8912. Thereupon, plaintiffs moved for rehearing. *See* R. (3/29/99) 8913-45. After studying the case for nearly another year, the Third District denied the motion for rehearing and issued an opinion on February 9, 2000, modified in only very modest respects from its earlier decision. *See* R. (2/9/00) 8959-87. The opinion on rehearing reversed the judgment on the basis of the trial court's errors in applying *Frye* and its wrongful refusal to grant a directed verdict for DuPont once it correctly excluded hearsay evidence against DuPont. As a result, the Third District remanded for the entry of judgment for the defense. *See id.*

SUMMARY OF ARGUMENT

Below, DuPont presents four distinct arguments for this Court to dismiss or affirm in this case (or alternatively, to remand the case or order a new trial). *First*, the

Court lacks jurisdiction over this case. It is evident now that none of the purported conflicts in authority plaintiffs pointed to have borne fruit; and, in addition, the plaintiffs failed to advise the Court just how record-intensive and nuanced the appellate decision below was, and therefore how inappropriate a vehicle this case would be to resolve the issues (even assuming they were real) set forth in the petition.

Second, the Third District correctly identified two routine, but far-reaching evidentiary errors at trial: (1) refusing to grant DuPont a directed verdict after excluding inadmissible hearsay evidence on which the plaintiffs' entire causation case hinged; and (2) allowing the jury to be prejudiced by irrelevant references to non-existent clusters of the same birth defects allegedly caused elsewhere by Benlate. *See 3d DCA Op.*, 748 So. 2d at 1111-13 & n.1. The first of these errors warrants reversal of the trial court and the second warrants a new trial. Most importantly, these errors are entirely independent and sufficient grounds for granting DuPont relief from the trial court's judgment, wholly apart from the *Frye* issues on which plaintiffs focus.

Third, the Third District's *Frye* ruling was inarguably correct. The Third District properly held that the testing performed by plaintiffs' experts did not comport with *Frye* because those experts had "*conceded* at the *Frye* hearing that the direct extrapolation method they used in their study was new and that they were unaware of any scientific study that has ever purported to determine a human teratogenic exposure level in this manner." *3d DCA Op.*, 748 So. 2d at 1120 (emphasis added). Nowhere did the trial judge ever attempt to explain how, in light of this concession or otherwise, the novel methodology of plaintiffs' experts satisfied *Frye*. Moreover, numerous other facets of the work of plaintiffs' experts were not generally accepted.

Fourth, plaintiffs' entire theory of exposure to Benlate was based on a misuse of the product. That alleged misuse violated not only federal pesticide law, but also precludes a finding of defect under Florida law. Even putting aside the misuse issue, however, plaintiffs presented no evidence that Benlate was a defective product. At

best they presented evidence that the product should have been accompanied by additional warnings or been tested more extensively. But both failure-to-warn and negligent-testing liability are preempted by federal law. While the Third District found it unnecessary to address this last set of arguments because it found resolving the *Frye* and hearsay-related errors to be dispositive, *see id.* at 1113, those arguments also form another *independent* basis for affirming that court's judgment.

ARGUMENT

I. THE COURT LACKS JURISDICTION OVER THIS CASE.

In this case, the Third District needed to do little more than simply apply the settled *Frye* rule in an obvious way to bar a type of testimony that the plaintiffs' experts admitted was not generally accepted in the scientific community. *See 3d DCA Op.*, 748 So. 2d at 1120. That uncontroversial application of *Frye* created no "direct" conflicts with the decisions of other district courts. Accordingly, this Court lacks "conflict review" jurisdiction over this case under the Florida Constitution.

The reasons DuPont maintains jurisdiction does not exist are amply described in its jurisdictional brief, which DuPont urges the Court to reconsider. Indeed, after reviewing the full merits briefing in this case, the Court should easily come to the conclusion that the decision below does not conflict with *Berry v. CSX Transportation, Inc.*, 709 So. 2d 552 (Fla. 1st DCA), *review denied*, 718 So. 2d 167 (Fla. 1998), *Mills v. State*, 476 So. 2d 172 (Fla. 1985) (overruled on other ground by Fla. Stat. § 775.021(4)), *cert. denied*, 475 U.S. 1031 (1986); or *Gooding v. University Hospital Building., Inc.*, 445 So. 2d 1015 (Fla. 1984). Indeed, those cases strongly support the Third District's decision. Accordingly, this Court should dismiss its review of this case as improvidently granted. *See, e.g., Reaves v. State*, 485 So. 2d 829, 830 (Fla. 1986); *see also DiPietro v. Griefer*, 732 So. 2d 323 (Fla. 1999); *St. Mary's Hosp., Inc. v. Brinson*, 709 So. 2d 105 (Fla. 1998).

Perhaps the most important reason review should be dismissed as improvidently granted, however, stems not from what plaintiffs alleged in order to secure review and ultimately failed to prove, but from what plaintiffs entirely glossed over in their jurisdictional brief. This case involves numerous fact-laden grounds and elementary evidentiary rulings on which DuPont prevailed in the Third District, and which have nothing to do with the issues plaintiffs present. Because several adequate and independent grounds for affirmance of the judgment below exist, this case is a poor vehicle to resolve even the phantom conflicts plaintiffs conjure.

II. THE THIRD DISTRICT CORRECTLY IDENTIFIED TWO ERRONEOUS EVIDENTIARY RULINGS BY THE TRIAL COURT, WHICH REQUIRED EITHER REVERSING THE JURY VERDICT OR GRANTING A NEW TRIAL.

The trial court made two fundamental legal errors – the first of which was to deny DuPont's motion for a directed verdict once the trial court made the decision that causation evidence the plaintiffs needed to establish their case was inadmissible against DuPont, and the second of which denied DuPont a fair trial. The Third District agreed with these two evidentiary errors identified by DuPont. *First*, the Third District concluded that the testimony of the Farm's agent, Lynn Chaffin, regarding the central issue of *when* Benlate would have first been used in the 1989 growing season, was inadmissible hearsay against DuPont – which was "fatal" to plaintiffs' attempt to prove Benlate was present in the spray to which Mrs. Castillo claims to have been exposed, and thus "obligated" the trial court to grant DuPont's motion for a directed verdict. *3d DCA Op.*, 748 So. 2d at 1113. "In that light, there is *insufficient* evidence, as against DuPont, to establish that Mrs. Castillo was sprayed with Benlate." *Id*. (emphasis added). *Second*, the Third District stated the trial court should not have "allowed plaintiffs to refer at trial to an alleged link between Benlate and unspecified 'clusters' of children born without eyes in Great Britain." *Id.* at 1111 & n.1. The

"plain vanilla" and unassailable nature of these two errors underscores why this Court should dismiss the Petition as improvidently granted, or at the very least, affirm.

A. There Is Insufficient Admissible Evidence Against DuPont That Mrs. Castillo Was Sprayed With Benlate.

1. The Chaffin "Admission" Was Not Competent Evidence Against DuPont.

In telephoning Mrs. Castillo, Ashton was seeking to establish a link between Benlate and microphthalmia based on the now-discredited assertion that there were "clusters" of children with this birth defect born near agricultural areas in Britain. *See* Tr. 1598; *see also* Section II.B. below (explaining that no such "clusters" exist). When Ashton initially asked Mrs. Castillo whether she lived in an agricultural area where she might have been exposed to Benlate during her pregnancy, she answered that she did not. *See* Tr. 918. Only later did Mrs. Castillo remember the "u-pick" field in her Miami neighborhood. *See id.* at 1046, 1367, 1614, 2594. Indeed, in four separate conversations with Ashton, Mrs. Castillo utterly failed to remember the supposed drenching incident. *See id.* at 1052, 1703-04.

Once Mrs. Castillo remembered the "u-pick" field near the family's apartment, Ashton followed up with inquiries to Farm personnel. Ashton testified that Lynn Chaffin, the Farm's field manager, had told him in a phone call in May 1993 that the Farm had used Benlate in the "fall of 1989" and in "November of 1989." *Id.* at 1598, 1600. Ashton acknowledged, however, that despite standard journalistic practice *no such statements* were recorded in his notes of the conversation. *See id.* at 1588-90, 1598-1600, 1611-12.

Ashton's testimony about what Chaffin allegedly told him was classic hearsay. *See* Fla. Stat. § 90.801(c). Thus, such "hearsay evidence is inadmissible" "[e]xcept as provided *by statute*." *Id.* at § 90.802 (emphasis added); *see also Pickard v. Miggins*, 311 So. 2d 686, 688 (Fla. 3d DCA 1975) (inadmissible testimony cannot be relied on to create a genuine issue of material fact). And there simply are no statutory exceptions to § 90.802 applicable to DuPont in the circumstances of this case.

Indeed, the fact that Ashton's testimony in this regard could not be admitted against DuPont was so obvious that the trial court even ruled *in limine* that such evidence was not admissible against DuPont. *See* Tr. (4/29/96) 95-98; *see also* Tr. 1565, 1627; *compare id.* 4482 (lines 2-11) (improperly allowing plaintiffs' counsel to suggest during closing argument that the "Chaffin admission" rebutted a *DuPont* argument concerning the lack of available Benlate during November 1st or 2nd, the only two days that Mrs. Castillo testified the "drenching" incident could have occurred).

Thus, all the plaintiffs can offer by way of rebuttal is the mere assertion, unsupported by any citation, that the courts should create a common-law exception to this clear rule in circumstances where "DuPont[] reli[ed] upon the remainder of Chaffin's testimony." P.'s Br. 46. Even if Florida's courts could create such an exception, contrary to the Legislature's plain directive in § 90.802, plaintiffs frame the exception they are seeking to establish in misleading terms. This is not a case involving "the remainder of *Chaffin's* testimony" – i.e., DuPont picking and choosing what it wanted to rely on from Chaffin's testimony. Rather, what is at issue is the reliability of *Ashton's* testimony. Most importantly, there is already clear precedent precluding the use of the party-admission exception (Fla. Stat. § 90.803(18)(d)) against a different party. *See Dinter v. Brewer*, 420 So. 2d 932, 935-36 (Fla. 3d DCA 1982) (hearsay statements made by one defendant inadmissible against another).

The Third District's ruling on the Chaffin "admission" was plainly right. And the import of that simple ruling is that the Court can stop right there in its process of adjudicating the case against DuPont. For, as the District Court recognized, "[w]ithout his admission, there is insufficient evidence in this record to establish that Benlate was sprayed on the farm on the dates in question." *3d DCA Op.*, 748 So. 2d at 1113. "[T]he trial court granted DuPont's motion *in limine* to preclude the use of Chaffin's hearsay testimony against DuPont. This ruling was eminently correct and *fatal* to the plaintiffs' case against DuPont." *Id.* (emphasis added).

2. Other Circumstantial Evidence of Benlate Exposure Is Insufficient.

Plaintiffs then invite this Court to second guess the District Court's thorough record review, conducted over a more than two-year period. *See* P.'s Br. 17-18 ("detailed facts from the trial record . . . confirms exactly the opposite of the Third District's conclusion."). But this Court exercises its review powers to resolve important *questions of law and policy* about which there are differences of judicial opinion in the inferior courts, not to sift through the facts a *third* time. *See Greater Miami Dev. Corp. v. Pender*, 194 So. 867, 868 (Fla. 1940). Thus, the direction plaintiffs' argument must take again points out why this case should be dismissed.

Additionally, it is simply not true that there is ample evidence in the record other than the Ashton testimony to support the conclusion by a preponderance of the evidence that Mrs. Castillo was sprayed with Benlate on November 1st or 2nd, 1989. Plaintiffs' assertions begin with the basically correct statement that Chaffin's testimony can be interpreted to say that tomatoes were planted on the "u-pick" field between October 25 and October 27, 1989, five to eight days before November 1-2, 1989. *See* Tr. 1380. But from there the plaintiffs venture into the realm of insupportable, and indeed illogical, inference.

Plaintiffs note that Farm spray manager Eddie Sanders, Farm owner Jack Wishart, and expert witness Dr. Robert McMillan, all testified that fungicides can be used on tomatoes within a week to ten days of planting. *See* P.'s Br. 18. Indeed, plaintiffs cite Jack Wishart as confirming that "fungicides are used prophylactically." *Id.* But these literally true assertions are misleading and disingenuous because: (1) they give the wrong impression that there were concessions that *Benlate in particular* would be used so soon after the tomatoes were first planted; and (2) they ignore the undisputed record evidence that *only fungicides other than Benlate* were used in this fashion; and most importantly, (3) they ignore the undisputed record evidence that

Benlate was *not* used as a prophylactic fungicide.[2] *See* Tr. 1304, 3557-60, 3596-97, 3610. Indeed, to spray Benlate prophylactically would have been a violation of label conditions. *See* Section IV.A. below (misuse of the product is an independent and complete defense for DuPont); Def. Ex. C at 4 (product label); Tr. 4288.

The evidence was uncontradicted that the Farm used Benlate only on its tomatoes, *see* Tr. 1301, 1333, 1345, 2836, 3477, 3563-65, 3610-11, that the Farm did not plant its tomatoes before its strawberries, *see id.* at 2845, and that the Farm's strawberry plants did not arrive from California until October 25, 1989, *see id.* at 1125, 1189, 1197-98, 1320-23, 2683-86, 2821. Thus, even if the Farm had planted the tomatoes later that same day, it would not have used Benlate (in accord with label instructions) until at least three weeks later, when the first signs of disease could have appeared. *See id.* at 1304, 1333, 1368-69, 2857, 3475, 3557-60, 3566-67, 3609-10.

In reality, the plaintiffs' claimed mountain of circumstantial evidence reduces to nothing more than the molehill of claiming that the Farm denied the use of all other fungicides than Benlate in requests for admissions read to the jury, and therefore that Benlate, being the only remaining alternative, must have been used. *See* P.'s Br. 19. But this highly misleading argument is enabled only by another far-reaching evidentiary error made by the trial judge.

In the plaintiffs' requests for admissions, the Farm denied the use of *any and all* fungicides during November 1st or 2nd 1989. The trial court improperly allowed

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[2] Apparently, Benlate was used improperly by the Farm as a prophylactic in the mid-1970s. But such improper use was discontinued before 1989, and thus the trial court properly excluded an attempt by plaintiffs to use that irrelevant evidence to bridge the gap between vague testimony that some fungicides were used prophylactically by the Farm and the conclusion that Benlate was such a fungicide. *See* Tr. 2606; *Sims v. Brown*, 574 So. 2d 131, 133 (Fla. 1991) ("To be relevant, particularly if remote in time, a . . . negligent cause of conduct must be shown to continue uncorrected up to the time of the act sued upon.").

the plaintiffs to read all of the Farm's fungicide denials, on a fungicide-by-fungicide basis, to the jury – all but one, that is: the Benlate denial. *See* Tr. 1573-79, 5038, 5311. The Farm was thus expressly denied its right under the completeness statute in Florida evidence law to read the Benlate denial to the jury. *See* Fla. St. § 90.108(1).[3] Additionally, this error created the misimpression that before trial the Farm denied the use of all fungicides except the precise one at issue, and thus that its denials of using Benlate at trial were recent fabrications. Indeed the plaintiffs were permitted explicitly to draw exactly such an inference during closing argument.[4]

3. Plaintiffs' Speculative Inferences

The Farm's answer brief goes into greater detail about the many speculative and false inferences on which the plaintiffs' exposure case is based other than the Chaffin "admission," and DuPont adopts that argumentation as its own. The key point for present purposes, however, is that the plaintiffs cannot succeed in proving their case by stacking such speculative inferences one atop another. *See, e.g., Voelker v. Combined Ins. Co.*, 73 So. 2d 403, 404-07 (Fla. 1954); *Asplundh Tree Experts, Inc. v. Mason*, 693 So. 2d 44, 45 (Fla. 1st DCA), *rev. denied*, 699 So. 2d 1374 (Fla. 1997).

The plaintiffs first respond to this by claiming that "[s]ince the first fact necessary to establish that Mrs. Castillo was sprayed by the fungicide Benlate was

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[3] "When a writing or recorded statement or part thereof is introduced by a party, an adverse party may require him or her at that time to introduce any other part or any other writing or recorded statement that in fairness ought to be considered contemporaneously . . . ."

[4] *See* Tr. 5311 ("Benlate is the only possible fungicide before November 10th, 1989 . . . . [In their admissions, the Farm] said on each and every fungicide that we would not have been using them before November 10th of 1989. Now, of course, they're in court now saying we didn't use Benlate until December 19th of 1989.").

established by direct evidence [i.e. Mrs. Castillo's own testimony], the *Voelker* 'inference upon inference' rule does not apply. Long before *Voelker*, this Court recognized the propriety of relying upon circumstantial evidence in civil cases." P.'s Br. 47. This response is unavailing. *First*, Mrs. Castillo's own direct testimony establishes virtually nothing. She could only testify that she was sprayed with "tons of cloudy, misty foggy spray," Tr. 989; she could not say whether such spray was Benlate, some other agricultural chemical, or even water.

Second, the notions: (1) that the presence of *some* direct testimony makes *Voelker totally* inapposite as applied to circumstantial evidence, or (2) that *Voelker* is somehow inconsistent with the law in Florida authorizing reliance on circumstantial evidence in appropriate instances, are unsupportable. *Voelker* applies *only when* circumstantial evidence is present. *See Nielsen v. City of Sarasota*, 117 So. 2d 731, 733 (Fla. 1960). DuPont does not challenge that circumstantial evidence is admissible evidence; it merely relies on the limitations *Voelker* and its progeny place on using such evidence.

Accordingly, plaintiffs seem to allow that *Voelker* applies. To cast aspersions on that case, however, they cite a law review article criticizing *Voelker*. *See* P.'s Br. 47. That article is not legal authority, however, and is certainly not sufficient authority to overcome this rule, which is a mainstay of Florida jurisprudence. *See, e.g., Autrey v. Carroll*, 240 So. 2d 474, 477 (Fla. 1970); *Food Fair Stores, Inc. v. Trusell*, 131 So. 2d 730, 733 (Fla. 1961); *Wong v. Crown Equip. Corp.*, 676 So. 2d 981, 983 (Fla. 3d DCA), *rev. dismissed*, 683 So. 2d 486 (Fla. 1996).

Ultimately forced to take a more conventional approach, plaintiffs fall back on trying to distinguish *Voelker*. The principal case they cite in this regard is *Teate v. Winn-Dixie Stores*, 524 So. 2d 1060 (Fla. 3d DCA), *rev. denied*, 534 So. 2d 402 (Fla. 1988). In *Teate* the plaintiff needed to prove that a bag of frozen peas the plaintiff had slipped on had been on the floor long enough that a supermarket defendant could be

charged with constructive knowledge of such a dangerous condition. There was testimony that the peas were surrounded by a pool of water before the plaintiff slipped. The two competing inferences were that the peas had gradually thawed, supported by direct evidence that the floor had not been cleaned for 15 minutes, versus the supermarket's assertion that the ice crystals on the peas would have melted instantly when the peas hit the floor. *See id*. at 1061. Despite *Voelker*, the *Teate* court reversed a directed verdict in favor of the supermarket.

Teate, which distinguished *Voelker* on the formal ground that *Voelker* applies only when multiple inferences are involved, *see id.*, is inapplicable to the Castillos' case for two reasons. *See also Fritts v. Collins*, 144 So. 2d 850 (Fla. 2d DCA 1962) (also distinguishing *Voelker* because only one inference was involved).[5] *First*, multiple, stacked inferences are clearly involved in this case. *See generally* Pine Island Farms' Br. (identifying numerous speculative inferences on which the conclusion that Mrs. Castillo was sprayed with Benlate depends).

Second, even if *Teate* were potentially applicable and this case involved only one inference – i.e., going from Chaffin's alleged admission that Benlate was used in "November of 1989" to the conclusion that Benlate was used on November 1st or 2d of 1989 *in particular* – *Teate* would remain distinguishable. Some inferences are simply too speculative. *See Vecta Contract, Inc. v. Lynch*, 444 So. 2d 1093 (Fla. 4th DCA), *rev. denied*, 453 So. 2d 44 (Fla. 1984); *Victoria Hosp. v. Perez*, 395 So. 2d 1165 (Fla. 1st DCA 1981). For instance, going from an admission involving 30

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[5] Another case plaintiffs rest on is *C.R. Bard, Inc. v. Mason*, 247 So. 2d 471 (Fla. 2d DCA), *cert. denied*, 251 So. 2d 878 (Fla. 1971). There, an inference was accepted by the court notwithstanding *Voelker* because "all of the other possibilities are excluded by direct evidence." *Id*. at 471-72. Plaintiffs attempt to contend that all other reasonable causes for John's microphthalmia have been excluded. But this is simply *not* true, as is explained in Section III.B. below.

possible days to the conclusion that Benlate was sprayed on the two initial days involved in that 30-day period requires leaping a virtual chasm of speculation.[6]

At its core, *Teate* reached the result it did because to defend itself, the supermarket could only offer the strained theory that when frozen peas fall a few inches to the floor, they create instantaneous water puddles. The plaintiffs' desired inference from Chaffin's hearsay statement here is simply far more speculative than drawing the superior inference the plaintiff in *Teate* sought that frozen peas leaving a puddle of water on a supermarket floor, which had gone uncleaned for 15 minutes, must have been lying around for a while. *Compare Wilson v. Winn-Dixie Stores, Inc.*, 559 So. 2d 263, 263-64 (2d DCA) (refusing to allow speculation that spilled laundry detergent on which plaintiff had slipped resulted from a "slow leak" and thus that supermarket knew of that condition), *rev. denied*, 574 So. 2d 145 (Fla. 1990).

B. The Trial Court Erred By Allowing Plaintiffs To Link Benlate To Alleged "Clusters" of Children Born Without Eyes.

A prominent part – indeed the genesis – of the plaintiffs' story is that Mrs. Castillo was contacted by reporter John Ashton, who was investigating on his own

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[6] There are 30 days in November. Of course, assuming Benlate spraying occurred on only one day in early November (and there is no evidence to the contrary), there is thus only a 6.7% random chance (2 divided by 30) that spraying occurred on one of the two relevant days, November 1st or 2nd. This falls far short of satisfying the 51% preponderance standard of *Rivet v. Perez*, 655 So. 2d 1169, 1171 (Fla. 3d DCA1995). *See Reaves v. Armstrong World Indus., Inc.*, 569 So. 2d 1307 (Fla. 4th DCA 1990) (evidence that *eighteen* companies had manufactured asbestos used in the plaintiff's plant was insufficient under *Voelker* to demonstrate, as plaintiff was required to, that the particular asbestos that injured the plaintiff was manufactured by at least one of *three* out of the *eighteen* companies). And, of course, it goes without saying that this analysis is generous to plaintiffs because it assumes away *arguendo* all of the direct evidence (which is analyzed in the Farm's brief) indicating that spraying did not occur until *late* November at the earliest.

initiative whether the alleged phenomenon of "clusters" (of children born without eyes) also existed in the United States. *See* Tr. 1582-84. But, as the Third District accurately recognized, there were no such clusters, even in Ashton's British homeland. The British government investigated and confirmed this fact in 1998. *See 3d DCA Op.*, 748 So. 2d at 1111 n.1.

As the Third District noted with regard to the "clusters" issue: "We agree that this was error. We find that this evidence was vague and indefinite. Whatever relevance it may have had was greatly influenced by its potential to unfairly prejudice the jury. *See* § 90.403, Fla. Stat. (1995)." *3d DCA Op.*, 748 So. 2d at 1111 n.1. Thus, even if this Court were to conclude that there was sufficient admissible evidence against DuPont that Mrs. Castillo was sprayed with Benlate, the "clusters" error, which biased the jury to think that a non-existent epidemic of birth defects caused by Benlate existed elsewhere in the world, plainly warrants a new trial.

Because the trial court's error is so obvious and so difficult to explain away, the plaintiffs are forced to contend the error was harmless – references to "clusters" do not occur on every page of the voluminous transcripts for the 17-day trial, or do not occur by name. *See* P.'s Br. 49. But in fact the prejudicial nature of references to "clusters" *or to similar concepts* is so great, and the references often occurred at such pivotal junctures in the trial, that the notion that the improper references to clusters here were harmless should be rejected out of hand.

For instance, during his *opening statement*, plaintiffs' trial counsel, immediately after the trial court had overruled DuPont's objection to cluster references, referred to "the fact that the London Observer was investigating the allegation of Benlate use in Great Britain and *clusters* of children that were born without eyes." Tr. 799 (emphasis added). Plaintiffs also read to the jury portions of the depositions of Ashton, including a reference to the alleged link between Benlate and "a series of birth defects, primarily anophthalmia" in agricultural areas in Great Britain." *Id.* at 1582-83. And in

closing argument, plaintiffs' counsel reiterated that "John Ashton from The London Observer . . . was doing an investigation between [*sic*] the link between Benlate and microphthalmia." *Id*. at 5307. Plaintiffs also elicited references to the alleged "clusters" from their witnesses, bringing forth motions for mistrial, which were denied. *See id*. at 918; 2275; 2331-32; 3755, 4808.[7]

Plaintiffs advance a number of weak legal points in opposition to this conclusion, but to no avail. *First*, they claim that they have shown the harmlessness of the error here under *McCarthy v. Zdenek*, 508 So. 2d 408 (Fla. 2d DCA 1987). *See* P.'s Br. 49. But it is well-established that the erroneous admission of evidence regarding other alleged injuries without a threshold showing of substantial similarity is highly prejudicial and accordingly warrants virtually *per se* reversal. *See, e.g., Frazier v. Otis Elevator Co.*, 645 So. 2d 100, 101 (Fla. 3d DCA 1994). *McCarthy* is not a harmless-error case involving references to other alleged accidents.

Second, plaintiffs claim that the District Court contravened *Sims v. Brown*, 574 So. 2d 131 (Fla. 1991), by ruling that it was error for the trial court to allow the clusters references, because *Sims* expresses a preference for trial courts to do the initial balancing of probativity vs. prejudice concerning the admission of evidence. *See* P.'s Br. 49. But it is plain that when a trial court abuses its discretion in performing that balance, it has committed error, and the appellate courts are free to order an appropriate remedy, as made clear by *Short v. Allen*, 254 So. 2d 34, 36 (Fla. 3d DCA

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[7] Plaintiffs made no attempt below, *see* R. (5/7/96) 3546-48, or in their merits brief to this Court to demonstrate that there is "substantial similarity" between what allegedly occurred in John Castillo's case and the clusters in Britain (nor could they because such clusters did not exist). As such, it is beyond question that the trial court, in permitting such references, fundamentally undermined the fairness of the trial. *See* Fla. Stat. § 90.402, 90.403; *Cummins Ala., Inc. v. Allbritten*, 548 So. 2d 258, 266 (Fla. 1st DCA), *rev. denied*, 553 So. 2d 1164 (Fla. 1989); *Ashby Div. of Consol. Aluminum Corp. v. Dobkin*, 458 So. 2d 335, 337 (Fla. 3d DCA 1984).

1971) (court cannot deem the admission of such evidence harmless because "there is no way to know whether the prior accident testimony did or did not have a material bearing upon the verdict rendered by the jury").

III. THE THIRD DISTRICT'S *FRYE* ANALYSIS IS PLAINLY CORRECT AND DOES NOT CLOSE THE COURTS TO TORT VICTIMS WHO HAVE TRULY CARRIED THEIR BURDEN OF PROVING CAUSATION.

Even though federal courts have switched to the standard in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 587 (1993), the Florida courts continue to follow *Frye*, 293 F. at 1014, which permits an expert to testify pursuant to a novel methodology only where that methodology is "generally accepted" in the relevant expert community. *See, e.g.*, *Hadden v. State*, 690 So. 2d 573, 576 (Fla. 1997); *Ramirez v. State*, 651 So. 2d 1164, 1167-68 (Fla. 1995). Accordingly, a litigant seeking to introduce expert testimony bears the burden of establishing by a preponderance of the evidence the "'scientific acceptance and reliability'" of the expert's methodology. *Ramirez*, 651 So. 2d at 1167 (quoting Ehrhardt, *Florida Evidence* § 702.2 (1992 ed.) (footnotes omitted)). Also, whether the *Frye* test has been satisfied is a matter of law subject to *de novo* appellate review. *See, e.g., Brim v. State*, 695 So. 2d 268, 274 (Fla. 1997); *Hadden*, 690 So. 2d at 579. In resolving the *Frye* issue and the underlying facts, this Court therefore owes no deference either to the trial judge or to the jury.

Finally, it is important to note that while this brief focuses on the inability of plaintiffs' *methodologies* to meet the demands of *Frye*, DuPont still emphasizes that Dr. Howard is *unqualified* as an expert in the field of teratology. Dr. Howard is an expert in stereology (an irrelevant field) and pathology. Though pathology is one discipline from which teratologists "come," Dr. Howard has not completed the journey and achieved the requisite level of expertise required to function as a teratologist. By

holding that Dr. Howard was qualified merely because he was an expert in *one of* the fields involved in the *multidisciplinary* field of teratology, the courts below erred by illogically making experts qualified *in any single part* of a such a multidisciplinary field qualified in the *entire* multidisciplinary field itself. *See 3d DCA Op.*, 748 So. 2d at 1115-16. That holding makes no sense and also ignores the distinctiveness of teratology as its own field of scientific inquiry.

A. As Plaintiffs' Own Expert Conceded, And The Third District Recognized, *in Vitro* Tests Are Not A "Generally Accepted" Method of Establishing The Level At Which A Substance Causes Birth Defects In Humans.

Plaintiffs' central argument is that the Third District made the same error as the trial court in *Berry v. CSX Transportation* – in other words, it required the conclusions (and not just the methodologies) of the challenged experts to satisfy *Frye*. *See* P.'s Br. 39. But the Third District neither said it was doing this (as the trial court explicitly did in *Berry*), nor actually did it. Instead, on the basis of dispositive concessions by the experts themselves, it merely recognized that a key methodology employed by the plaintiffs' two key experts was not generally accepted.

Plaintiffs cannot escape the fact that their own expert, van Velzen, *expressly conceded* at the *Frye* hearing that the direct extrapolation method upon which he and Howard had relied was "new," Tr. (4/30/96) 275, and that he was aware of *no scientific study* that had ever before purported to determine a human teratogenic exposure level in this manner, *see id.* at 252-53, 257, 263-65, 273; Tr. 3300. Van Velzen also conceded at the *Frye* hearing that he did not know of "a single authoritative peer reviewed work in which a scientist has used this [direct extrapolation] technique" to determine the level at which a substance is teratogenic in

humans. *See* Tr. (4/30/96) 257.[8] And both Howard and van Velzen testified at trial that *no* scientific publication, governmental agency, or academic group had ever before relied on direct extrapolation from *in vitro* test results to determine a teratogenic exposure level in a living being. *See* Tr. 3186-88, 3304-06. Plaintiffs' citation to a letter from an EPA official, *see* P.'s Br. 22, does nothing to counter these admissions because that letter never indicates that *in vitro* tests are generally accepted *for the purpose* of determining the chemical levels that cause teratogenesis.

Even if plaintiffs had not conceded that their tests failed to comport with *Frye*, it is self-evident that one methodology cannot be applied to answer all scientific questions. Different methodologies have different purposes, and can properly be used only for such purposes. *See Brim*, 695 So. 2d at 270 (both the biological/chemical step of DNA test and the second, statistical step of DNA test were different methodologies that needed to be separately *Frye*-tested); *see also Hadden*, 690 So. 2d at 580 n.5. Take the methodological device of a barometer, for example. It is a generally accepted device for measuring air pressure. But a barometer is not generally accepted for measuring temperature. That is what thermometers are for. In everyday weather patterns, there are relationships between air pressure and temperature, but they are imperfect. No court applying the *Frye* test would allow an expert to offer an opinion as to the temperature, and especially not the precise temperature, based on barometer readings. This is not because the court would be quarreling with the conclusions the expert drew from the barometer, but basically because barometers are not the type of device that can be used for the purpose of making the conclusion the expert wished to draw. Were it otherwise, once a methodology were blessed as

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[8] DuPont presented uncontradicted expert affidavits to the same effect at the *Frye* hearing. *See* R. (5/6/96) 3096 (Dr. Brent); *id*. at 3098-99 (Dr. Lamb). Plaintiffs contrast these affidavits with their own lengthy presentation at the *Frye* hearing. *See* P.'s Br. 19-20. But this is an example where quality counts for more than quantity.

"generally accepted," it could be used to draw *any* conclusion. That absurd proposition simply cannot be, and is not, the law of Florida.

The most significant problem the plaintiffs faced in bringing this case is that even by making the most strained assumptions possible about how much Benlate Mrs. Castillo was exposed to (*see* Section III.B. below), as well as how much could have crossed the placenta into John Castillo's bloodstream, data generated by using generally accepted methods indicated that the dose Mrs. Castillo received was about *1,000 times less* than the lowest levels that were shown to cause *microphthalmic birth defects* in rat studies. *See* Tr. 4694. Thus, even assuming that one could fully transfer the results and dose levels shown in rats to humans (which also is not generally accepted – a point also explained in Section III.B), any attempt to demonstrate that John Castillo's condition was caused by Benlate would inevitably fail.

Plaintiffs' experts' *in vitro* tests were contrived to bridge that gap. Drs. van Velzen and Howard took human and rat cells, put them into the artificial medium of a glass or plastic dish (without any attempt to account for the effects of metabolism), and literally *"soaked"* the cells, for 24 hours, in varying concentrations of Benlate. *See* Tr. (4/30/96) 259; Tr. 3263-64, 3306-07. These experiments, conducted for this litigation and financed by plaintiffs' attorneys, *see* Tr. (4/30/96) 212-14; Tr. 3271-72, were crude at best. Howard and his colleague van Velzen merely applied various concentrations of benomyl to the cells in their test dishes, and recorded the lowest possible concentration levels at which effects of two certain types occurred: (1) micronuclei formation (which Howard and van Velzen wrongly thought was a proxy for cell death), or (2) inhibition of neurite growth. Not surprisingly, this analysis showed effects at levels below where microphthalmic birth defects were observed in the live rat (or *in vivo*) studies. The final step in the process was to offer the opinion that Benlate would more likely than not cause microphthalmia at the same level in humans at which it first caused these types of effects in the petri dishes.

In vitro studies are generally accepted for some purposes by practicing teratologists (most commonly, identifying the biological mechanism of action for a chemical agent on a living body[9]), just like a barometer is generally accepted to measure air pressure. But *in vitro* studies are not generally accepted for determining *the level* at which a potentially harmful agent could cause birth defects.

Plaintiffs attempt to muddy the waters by citing those parts of the record where Drs. Howard and van Velzen (particularly van Velzen) were trying to obfuscate what they were doing and purported only to be using *in vitro* tests as screening mechanisms. *See* P.'s Br. 35 (*in vitro* testing is "a tool in assessing substances' potential for toxicity") (citing Tr. (4/30/96) 121-23). Indeed, plaintiffs' experts' statements were often such doublespeak that they even protested at times not to be doing precisely what they clearly were doing. *See, e.g.*, P.'s Br. 29 (citing Tr. (4/30/96) 284 (van Velzen) ("You cannot calculate a low effect level in the mother simply from the [*in vitro*] micronucleus test for exactly the reasons you give.")); *see also* Tr. 3192, 3253. There are several responses to this attempt at misdirection.

First, plaintiffs cannot have it both ways. Either Dr. Howard's and Dr. van Velzen's *in vitro* experiments were used here to establish the levels at which Benlate causes human microphthalmia or they were not. If they were not, then there is no competent evidence in the record that Mrs. Castillo was exposed to the requisite levels of Benlate necessary to cause microphthalmia (plaintiffs would be left with the levels in the rat studies, which never were, nor even could have been achieved in Mrs. Castillo's blood). In fact, plaintiffs concede the importance of establishing the correct

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[9] "Positive results from *in vitro* studies may provide a clue signaling the need for further research, but alone do not provide a satisfactory basis for opining about causation in the human context." *Richardson by Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 830 (D.C. Cir. 1988), *cert. denied*, 493 U.S. 882 (1989).

dosage threshold.[10] Assuming Benlate were a human teratogen, plaintiffs would not satisfy the requirement of causation merely by showing that Mrs. Castillo had been exposed to *some* Benlate. They needed to establish: (1) the level at which Benlate would cause human microphthalmia, and (2) that Mrs. Castillo was exposed to such levels.

Second, plaintiffs recognized that they needed to meet these burdens and indeed that was the function of the *in vitro* tests Drs. Howard and van Velzen performed.[11] Without the effect levels obtained from the petri dish experiment, plaintiffs' causation case would collapse. Dr. Howard testified that Mrs. Castillo's blood achieved levels of 100 ppb of Benlate. *See* Tr. 3035-37, 3042; P.'s Br. 16. But animal tests showed effects only at a gavage dosage of the much higher level of 31,200 ppb (even putting

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[10] *See* P.'s Br. 9 ("As with any suspected link between a chemical and a birth defect, it must be determined whether the chemical could *reach* the fetus in an amount sufficient to cause the adverse effect."); *see also id.* at 12 ("necessary" to determine how much benomyl reached the fetus and how much was needed to affect eye development); *id.* at 36 (conclusions about teratogenicity "always depend[] upon dose"); *see also Mitchell v. Gencorp., Inc.*, 165 F.3d 778, 781 (10th Cir. 1999) ("plaintiff must demonstrate 'the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure.'") (quoting *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996)).

[11] Plaintiffs suggest that there is significance in defendants moving to exclude Dr. Howard, but not Dr. van Velzen. *See* P.'s Br. 13. But Dr. Howard was the only expert designated to testify at trial as to the ultimate issues of causation in the case. Dr. van Velzen's experiments were merely to be the foundational basis for Dr. Howard's conclusions. At trial, however, the court allowed Dr. van Velzen to testify directly to his own opinion regarding causation. *See* Tr. 3265-70. That erroneous about-face by the trial court obviously cannot be permitted to prejudice DuPont's rights on appeal. *See Nelson v. State*, 748 So. 2d 237, 240-41 (Fla. 1999) (proponent of expert evidence had burden to demonstrate general acceptance, even with regard to foundational evidence supplied by non-testifying expert).

aside the effects of metabolism, of which more below). *See* Kavlock, et al., *Teratogenic Effects of Benomyl in the Wistar Rat and CD-1 Mouse, with Emphasis on the Route of Administration*, 62 Toxicology and Applied Pharmacology 44 (1982) [hereafter "*Teratogenic Effects*, 62 Toxicology and Applied Pharmacology at __"].[12] Thus, without the levels established *in vitro*, plaintiffs' proof would have fallen short *by a factor of at least 312* (31,200 ppb divided by 100 ppb)! (Indeed, given the lack of precision in the measurements taken by and the assumptions made by Dr. Howard (see below) – the problem of "significant digits" – scientists would say that the gap plaintiffs needed to surmount here was one of the approximate size of three orders of magnitude, or 1,000 times. *See* Tr. 4694.)

Third, as the three excerpts set forth below indicate, Drs. Howard and van Velzen were, without doubt, using the *in vitro* experiments they conducted to predict the levels at which Benlate would cause the human birth defect of microphthalmia:

> (1) "The only information, to my knowledge, that we have of the effect of benomyl on human fetal cells is the study done by van Velzen, and that appears to give a lower [*sic*] effect level [LOEL] at 20 parts per billion. Therefore, I would assume that that could have a teratogenic effect." R. (3/12/96) 6158 (Dr. Howard).
>
> (2) "Q. . . . . Are you of the opinion or are you of the view that 20 parts per billion in the fetal embryo at the period of vulnerability is the lowest observable effect level [LOEL] for human [*sic*] microphthalmia in humans? A. Yes. I think we have to take the experimental information that has come from that investigation and interpret it in that way." R. (3/12/96) 6214 (Dr. Howard).

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[12] 31.2 mg/kg/day is actually the no-effect level or NOEL in the Kavlock study. And since the lowest, statistically significant, observed effect level (LOEL) in this study was 62.5 mg/kg/day, the true level at which effects begin in rats *could be significantly higher than* 31.2 mg/kg/day, or the rough equivalent of 31,200 ppb.

(3) "Q. . . . . The question is is it your opinion that if 20 parts per billion is the low effect level [LOEL] in the dish, it is also likely that that is the low effect level in the developing fetus? A. My opinion is that that is yes, the most probable fact." Tr. (4/30/96) 257 (Dr. van Velzen) (quoted in P.'s Br. 31).

See also R. (3/12/96) 6157-58, 6203-07, 6214; Tr. 3039-47, 3159-60, 3177-82, 3186-89, 3191-93.

Such direct extrapolation of *in vitro* test results to a living fetus makes no sense. A petri dish is an artificial environment quite unlike a human body. Unlike such a dish, the body has a metabolic process that quickly acts to remove toxic substances. Thus, whereas a toxic substance can linger indefinitely in a dish, such a substance would be quickly detoxified and expelled from a living body by the body's metabolism. *See* Tr. 3142-45, 3185-86, 4661-62, 4678-79, 4689-92.

It is therefore no surprise that scientists do not "generally accept" – to put it mildly – the proposition that *in vitro* test results can be directly extrapolated to a living body. "*[I]n vitro* animal test data are *not* relied upon by experts in the field of teratology for extrapolating the results found directly to the human experience." *Wade-Greaux v. Whitehall Labs., Inc.*, 874 F. Supp. 1441, 1484 (D.V.I.), *aff'd*, 46 F.3d 1120 (3d Cir. 1994) (emphasis added); *see also* Linda A. Bailey et al., *Reference Guide on Epidemiology*, *in* Federal Judicial Center, Reference Manual on Scientific Evidence 130-31 (1994) (noting that the problem with *in vitro* studies is extrapolating the findings "from tissues in laboratories to whole human beings"); Tr. 4695.

B. The Methodologies of Plaintiffs' Experts Fail To Comport With *Frye* In Numerous Other Respects.

1. The "Quantum Leap" from Animal to Human Teratogenicity Without Human Epidemiological Evidence.

To support his threshold conclusion that benomyl causes birth defects in humans, Howard relied upon studies showing that the chemical can cause birth defects

in rats by gavage. *See* Tr. 2972-75, 3038-39, 3054-55, 3171-74, 3193, 3199-200; *see also* R. (3/29/96) 6211. Howard's reliance on these rat studies to support his testimony in this case was fundamentally flawed for at least three reasons.

First, the rats in the studies upon which Howard relied were exposed to benomyl through the "gavage" method, *i.e.*, artificially feeding a "bolus" dose through test tubes directly into the stomach. *See, e.g.*, Tr. 3054, 4935-36. It is hard to imagine a more obvious contrast to the dermal exposure alleged in this case. In determining whether a substance causes birth defects, the method of exposure is critical. As scientists and courts have recognized, different methods of exposure can lead to different results. *See id.* at 4935-36, 4938, 4942, 4947. "[T]he phenomenon that different routes of administration affect the teratogenic impact of an agent has been repeatedly tested and confirmed." *Wade-Greaux*, 874 F. Supp. at 1480; *see also* Roth-Nelson & Verdeal, *Risk Evidence in Toxic Torts*, 2 Envtl. Law. 405, 420 (1996) (discounting the usefulness of "exotic" routes of exposure).

Second, the rats in the studies upon which Howard relied were exposed to far greater quantities of benomyl than the 20 ppb concentration level on which Howard based his conclusion in this case. This difference in dosage utterly undermines Howard's reliance on the gavage studies. To obtain the same dosage as a gavage, a person would have to *rapidly* drink *two to four gallons* of Benlate spray mix. *See* Tr. 3906-07. As a matter of both science and common sense, it is obvious that virtually any substance can cause injury at *some* dose.[13] An enormous dose of virtually any

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[13] As the famed Swiss physician Paracelsus explained in the sixteenth century: "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy." Doull & Bruce, *Origin and Scope of Toxicology, in Casarett & Doull's Toxicology: The Basic Science of Poisons* (3d ed. 1986). In modern times, this self-evident proposition has become known, in the (continued...)

substance can overwhelm the body's normal detoxification processes. Accordingly, courts have refused to allow "experts" to testify that a particular substance causes birth defects based on an experiment conducted at a materially larger dosage.[14]

Third, the rats in the studies upon which Howard relied are obviously of a different species than humans. Contrary to the trial court's cavalier "quantum leap," Tr. (5/1/96) 69-70, it is well-established that "substances which are teratogens in animals are not necessarily, or even likely to be, teratogens in humans." *DePyper v. Navarro*, No. 83-303467, 1995 WL 788828, at *30 (Mich. Cir. Ct. Nov. 27, 1995). Thus, although "there are approximately 2,000 agents that have been shown to be teratogenic in some animal species, . . . only about 25-30 [of those are considered to be] human teratogens." *Wade-Greaux*, 874 F. Supp. at 1480. Accordingly, courts have often emphasized that animal studies alone do not typically provide a scientifically reasonable basis for concluding that a particular substance causes human birth defects. *See, e.g.*, *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 313 (5th Cir. 1989); *Richardson by Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 830 (D.C. Cir. 1988), *cert. denied*, 493 U.S. 882 (1989); *Lynch*, 830 F.2d at 1194; *Mascarenas v.*

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[13] (...continued)
context of teratology, as "Karnofsky's Law." *See Wade-Greaux*, 874 F. Supp. at 1480 ("Another principle, Karnofsky's Law (*i.e.*, 'sledgehammer teratology') demonstrates that, at some dosage, virtually any substance is teratogenic in an animal species."); *see also* Tr. 4939-40; *Berry v. CSX Transportation*, 709 So. 2d at 559. This basic proposition is all that DuPont's trial counsel "conceded" at the *Frye* hearing. *See* P.'s Br. 7, 26 & n.6. DuPont's trial counsel *never* conceded that Benlate was a human teratogen at a *non*-sledgehammer dose.

[14] *See, e.g.*, *Viterbo v. Dow Chem. Co.*, 826 F.2d 420, 424 (5th Cir. 1987); *cf. Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 314-15 (5th Cir. 1989) (rejecting utility of gavage doses 100-500 times the drug dose normally given to humans), *cert. denied*, 494 U.S. 1046 (1990); Tr. 4943-47.

Miles, Inc., 986 F. Supp. 582, 592 (W.D. Mo. 1997).

In sharp contrast to Dr. Howard's extreme willingness to rely on positive animal studies involving a single species, his errant methodology permitted him to refuse to consider altogether the *negative* epidemiological studies that have been carried out in Italy, Norway, and Britain.[15] The trial court also ignored the two studies available at that time, breezily asserting that it is impossible to study the effects of benomyl (or any other potentially dangerous substance) on humans. "[O]bviously we can't give it to people to find out." Tr. (5/1/96) 67. Contrary to that assertion, however, it *is possible* to study the effects of benomyl on humans using statistical analysis because the chemical has been widely used around the world for decades. Data compiled by the Centers for Disease Control, which monitors a million pregnancies a year, reveal *no* increase in the incidence of microphthalmia since Benlate was put on the market in 1970. *See* Tr. 4657-58. The Italian, Norwegian, and British epidemiological studies – the only such studies to investigate the possibility of a link between exposure to benomyl and an increased risk of microphthalmia or other birth defects – have found *no such link*. *See* Tr. (4/30/96) 57, 87, 93, 99; Tr. 4242-43, 4653-56, 4854-55, 4929-

[15] An epidemiological study is one designed to "observe the effect of exposure to a single factor upon the incidence of disease in two otherwise identical populations." Black & Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 Fordham L. Rev. 732, 755 (1984); *see also* Tr. 4928-29. In the present context, the British, Norwegian, and Italian epidemiological studies examined whether there was a statistically significant relationship between potential benomyl exposure and microphthalmia. *See* Dolk, et al., *Geographical Variation in Anophthalmia and Microphthalmia in England, 1988-94*, 317 British Med. J. 905-09 (1998); Kristensen, et al., *Birth Defects among Offspring of Norwegian Farmers*, 8 Epidemiology 537-44 (1997); Spagnolo, et al., *Anophthalmia and Benomyl in Italy: A Multicenter Study Based on 940,615 Newborns*, 8 Reproductive Toxicology 397-403 (1994).

31.[16]

Such uncontradicted epidemiological evidence is far more probative as a matter of both science and law than the amateurish experiments conducted by Howard and van Velzen. "The overriding significance of epidemiological studies (human data) in determining human teratogenicity has been accepted judicially and scientifically." *Oxendine v. Merrell Dow Pharm., Inc.*, Civ. No. 82-1245, 1996 WL 680992, at *7 (D.C. Super. Ct. Oct. 24, 1996). As courts have recognized, animal and *in vitro* tests contradicted by epidemiological evidence are "nothing more than unproven medical speculation lacking any sort of consensus." *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 314-15 (5th Cir. 1989), *cert. denied*, 494 U.S. 1046 (1990).

2. Dr. Howard's Other Methodological Errors

The linchpin of plaintiffs' scientific causation case was thus Dr. Howard's conclusion that the dermal exposure alleged by Mrs. Castillo caused her son's microphthalmia. *See* Tr. 3039-47, 3177-82, 3186-89, 3191-93. But the calculations underlying Howard's conclusion that John Castillo could have been exposed *in utero* to benomyl at a concentration of 20 ppb in his mother's bloodstream do not even begin to satisfy established norms of accuracy and reliability. Rather, those

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[16] At the *Frye* hearing and at trial, an epidemiologist called by plaintiffs quarreled with the weight that should be given epidemiological studies of the specific type conducted in Italy and Norway, *see* Tr. (4/30/96) 56-62, 67-68, 72-78; Tr. 5182-200, 5207-18, but conceded that there was (i) *no* epidemiological study that established any link between benomyl and microphthalmia in humans, and (ii) no evidence of any increase in the incidence of microphthalmia since the introduction of benomyl, *see* Tr. (4/30/96) 87, 92-93; *see also* P.'s Br. 21 n.5. Notably, this expert expressly declined to ratify Dr. Howard's conclusions or methodology. *See* Tr. (4/30/96) 83. Finally, plaintiffs offered *no* criticisms on rehearing to the Third District of the British study. *See Hadden*, 690 So. 2d at 579 (state of science at the time a decision is to be rendered, even if this requires taking account of new developments, is the foundation for applying *Frye*); *see also Brim*, 695 So. 2d at 275 (same).

calculations were based on a *series* of back-of-the-envelope assumptions wholly lacking any empirical foundation. *See Ramirez*, 651 So. 2d at 1168 ("In utilizing the *Frye* test, the burden is on the proponent of the evidence to prove the general acceptance of both the underlying scientific principle *and the testing procedures used to apply that principle to the facts of the case at hand*.") (emphasis added); *Smith v. Virginia Commonwealth Univ.*, 84 F.3d 672, 687 (4th Cir. 1996) (en banc) ("[A]n expert's opinion is inadmissible when it is based on assumptions that are speculative and not supported by the record.").

First, Howard assumed that Mrs. Castillo was exposed to Benlate spray over 50% of her skin surface. *See* Tr. 3029-31, 3100. He made *no* attempt, however, to measure Mrs. Castillo, to examine the clothing she was wearing at the time, or to determine what parts of her body were allegedly exposed. *See id.* at 3100-01.

Second, Howard assumed that absolutely all of the benomyl allegedly deposited on Mrs. Castillo's skin surface remained there until she showered, and thus "continued to enter into her bloodstream for some ten hours." *Id.* at 3121; *see also id.* at 3033, 3101, 3121-29, 3135. (Compare, as well, the far longer, *24-hour period* in which the cells were "soaking" in the *in vitro* tests.) Howard thus failed to account for the obvious fact that a fair amount of the chemical would rub off in the course of normal daily activities or sleep. *See id.* at 3101-02. He even assumed that she did not wash her hands or face during this period. *See id.* at 3101. Indeed, he made no attempt to determine how easily benomyl would rub off the skin. *See id.* at 3102.

Third, Howard effectively assumed that Mrs. Castillo's skin remained *wet* over this ten-hour absorption period by failing to account for any drying. *See id.* at 3100, 3111-22. This premise was inconsistent with Mrs. Castillo's own testimony that she neither felt nor saw anything on her skin by the time she reached home. *See id.* at 1045-46. This illogical assumption is significant because – as Howard himself conceded at trial – a wet material will absorb more quickly and readily into the skin

than a dry material. *See id.* at 3112-14.

Fourth, Howard assumed for his calculations that the absorption rate of Mrs. Castillo's skin was identical to the absorption rate of an isolated layer of skin removed from a dead human body. *See id.* at 3103-11; P.'s Br. 12. As Howard himself conceded, however, an *isolated outer layer* of skin *in a petri dish* absorbs far more quickly than the *full skin thickness* covering a *live body*. *See id.* at 3107-11.

Fifth, Howard assumed that all of the benomyl absorbed through Mrs. Castillo's skin would have continued to accumulate in her bloodstream at a steady rate. *See id.* at 3030-35, 3101. That assumption, however, utterly ignores metabolic and other processes through which the body detoxifies and eliminates foreign substances. *See id.* at 3142-43. Benomyl in particular is quickly broken down and detoxified by the body. *See id.* at 4661-62, 4678-79, 4690.

These errors were compounded when the trial court wrongly precluded DuPont from presenting the testimony of Dr. Mark Hurtt to rebut the unscientific assumptions underlying Howard's exposure calculations. *See* Tr. 5062-63. Such testimony, the trial court asserted, would have been cumulative of the testimony of DuPont's teratology expert, Dr. Brent. *See id.* at 4865-66, 4875, 4891, 5044-47, 5062-63. That assertion was baseless. *See id.* at 4868-69, 5058-59, 5063. Dr. Brent did not testify regarding the specifics of Howard's exposure calculations. The exclusion of Dr. Hurtt independently warrants a new trial. *See Ramirez*, 651 So. 2d at 1168 (reversing for a new trial because "[w]ithout the testimony of experts presented by both parties, the trial judge is denied a full presentation of relevant evidence").

Furthermore, defendants' experts, Dr. Lewis B. Holmes, Dr. Robert L. Brent, and Dr. Elias Traboulsi, explained at trial that Benlate could not have caused John

Castillo's microphthalmia.[17,18,19] All three of these eminent scientists flatly rejected the notion that John Castillo's microphthalmia could have been caused by his mother's alleged dermal exposure to Benlate. In particular, they emphasized that exposure to a teratogen during the sixth or seventh week of pregnancy would not have caused only a *single* isolated birth defect. *See* Tr. 4446-47, 4645-46, 4912-13. Because many organs in addition to the eyes are being formed during that critical period of fetal development, a teratogenic exposure would almost invariably cause *multiple* birth defects. *See id.* at 4667-76, 4678; *see also id.* at 4383-84, 4852-54, 4927-28, 4945, 4953-54. "[A]ll syndromes of malformation that involve cytotoxicity [toxicity to cells] where you have general toxicity to cells of the embryo produce multiple malformation. They don't produce an isolated malformation like this." *Id.* at 4668. Indeed, Dr. Traboulsi noted that John Castillo's specific condition, microphthalmia with cysts, is particularly inconsistent with exposure to a toxic chemical because it involves growing cells. *See id.* at 4383-84, 4393-98.

In any event, the notion that a *dermal* exposure like the one alleged here could have caused even a single birth defect is highly strained, according to current scientific knowledge. The skin is a remarkably effective barrier. Absorption through the skin

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[17] Dr. Holmes is Dean of Genetics and Teratology at the Harvard University Medical School, and a longtime member and past president of the Teratology Society, the premier association of American teratologists. *See* Tr. 4904.

[18] Dr. Brent is Distinguished Professor at Jefferson Medical College, Thomas Jefferson University, and 30-year Chairman of its Pediatrics Department. *See* Tr. 4594-96. He is also past president of the Teratology Society. *See id.* at 4611-12.

[19] At the time of trial, Dr. Traboulsi was Chairman of the Ophthalmology Department at the Bayview Medical Center of Johns Hopkins University. He is a renowned specialist in the specific field of pediatric microphthalmia and a board-certified geneticist. *See id.* at 4361-63. He is currently the Director of Pediatric Ophthalmology and Adult Strabismus at the Cleveland Clinic Eye Foundation Institute.

is "slow and way b[e]low the ability to overwhelm the organism. It's easily detoxified." *Id.* at 4663. Thus, even *known* human teratogens such as vitamin A are safely applied on the skin by pregnant women. *See id.* at 4663, 4674-75, 4680-83, 4949-50. Indeed, science has yet to identify *any* substance that *dermally* causes birth defects in humans. *See id.* at 4950-51. That is why Dr. Holmes tells his nursing students every year that "to date *no* dermal exposure has been shown to be harmful in the human fetus." *Id.* at 4951 (emphasis added). *None* of the studies cited by plaintiffs' at pp. 7-8 in the lengthy note 3 contained in their brief are dermal studies.

Despite the fact that plaintiffs lay as a cornerstone of their brief to this Court the claim that their experts ruled out genetic causes for John Castillo's condition, *see, e.g*, P.'s Br. 5, plaintiffs failed to establish that the alleged exposure to Benlate is a more likely cause of John's condition than genetics. *See, e.g.*, Tr. 4377-78, 4383-84, 4398-4400, 4731-33, 4748-52, 4801-03, 4913-26, 4960-61. Indeed, plaintiffs are careful when discussing the genetic causes purportedly ruled out to apply the adjective "known," thereby suggesting (incorrectly) that the size of the category of "unknown" birth defects is trivial. Plaintiff's genetic expert, Dr. Boris Kousseff, said that he could not rule out *three* possible types of genetic causation for John Castillo's microphthalmia: (1) X-linked recessive; (2) autosomal dominant; and (3) an unfavorable mutation. *See* Tr. 2306, 2285, 2287, 2289, 2294.

In connection with their genetics argument, plaintiffs also emphasize the case law in the federal courts on "differential diagnosis" (the sequential process of exclusion by which a medical doctor arrives at his opinion about the cause of some malady for the purpose of treating it). In addition to undermining the plaintiffs' assertions that the Third DCA somehow erred by consulting federal *Daubert* case law, *see* P.'s Br. 42, the "differential diagnosis" case law is inapposite because the plaintiffs' genetics experts were not able to rule out genetics as a cause. *See also Raynor v. Merrell Pharms., Inc.*, 104 F.3d 1371, 1374-76 (D.C. Cir. 1997) (cannot

apply differential diagnosis where it is contradicted by a body of epidemiological evidence); *see also Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 156 (3d Cir. 1999) (relied on by plaintiffs) ("We repeat that all of these reliable methods for making a diagnosis cannot sanitize an otherwise untrustworthy conclusion.").[20] Finally, the expert challenged here was clearly not John Castillo's treating physician.

C. Plaintiffs' Policy Arguments Provide No Basis For Disregarding The Flaws In Plaintiffs' Causation Case.

Plaintiffs present two exaggerated policy-based arguments to support their bid to overturn the Third District's ruling:

> (1) "in the case of toxic chemicals, humans and their unborn children must stand without recourse as the guinea pigs, or rats, for the DuPonts of the world [because] DuPont and its ilk may use their animal and in vitro studies to get EPA approval . . . but the victims of toxic products may not use those same – obviously generally accepted – studies to prove their claims in court," P.'s Br. 37; and
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> (2) "the Third District has in essence told the Plaintiffs that even though science considers Benlate to be too dangerous to conduct studies of its

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[20] Plaintiffs' citation to *Heller* also vitiates the word games they attempt to play with "extrapolation" by equating it with a "conclusion." *See* P.'s Br. 43. In *Heller*, the plaintiff's expert needed to a bridge a gap of a magnitude similar to that here between the levels of the plaintiff's exposure and the levels of concern in the literature. To do so, that expert offered two dubious extrapolation methods. The Third Circuit struck those methods as being unscientific without hesitating over the *non sequitur* of whether it was merely quibbling with the expert's conclusions. *See Heller*, 167 F.3d at 163. As the definition in *Black's Law Dictionary* 587 (6th ed. 1990) recognizes, "extrapolation" is a "*process*" for drawing a conclusion, not the conclusion itself. *See also Schudel v. General Elec. Co.*, 120 F.3d 991, 997 (9th Cir. 1997) ("Extrapolation was necessary to make the studies relevant, and there was no showing that the necessary extrapolation was scientifically acceptable."), *abrogated on other ground*, *Weisgram v. Marley Co.*, 528 U.S. 440 (2000).

> effects on pregnant women, the resulting lack of positive proof of its harmful effect in the form of human epidemiological research . . . results in a liability windfall for DuPont and effectively bars the Castillos' access to the courts" *id.* at 44.

Both of these arguments ignore that the plaintiffs failed here to present generally accepted science for determining the *levels* that can cause human birth defects *and* to which Mrs. Castillo was allegedly exposed. Even on their own terms, however, both policy arguments are specters scared up by the plaintiffs, and lacking any basis in what the Third District said (*see 3d DCA Op.*, 748 So. 2d at 1120-21), or in what its holding would truly mean for other cases.

Plaintiffs' first policy argument basically alleges that it is unfair that DuPont can rely on animal studies to market a product, but that animal studies may not be relied on by an injured plaintiff to bolster his or her case. This is flawed reasoning. *First*, animal studies can be and are used in both contexts, but they must be used appropriately, and in conformity with accepted methodology in the field of teratology.[21] The animal evidence that exists in this case, for all the reasons previously stated, could never serve as accepted proof that Benlate causes human birth defects. Plaintiffs' problem is not with any judicial limit on the use of animal studies, but simply that the studies are an insufficient grounding to support their case.

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[21] *See* Frankos, *FDA Perspectives on the Use of Teratology Data for Human Risk Assessment*, 5 Fundamental and Applied Toxicology 615, 617 (1985) (noting that studies on a single species often tend not to be teratogenic in humans, but that virtually all substances that are known to be human teratogens are teratogenic in at least two animal species) [hereafter "Frankos, *FDA Perspectives*"]. Here, even gavage studies involving benomyl or its breakdown product carbendazim showed *no micro- or anophthalmia* in rabbits and mice. *See* Kavlock, *Teratogenic Effects*, 62 Toxicology and Applied Pharmacology at 51; Argus Research Laboratories, Inc., *Developmental Toxicity Study of H-15647 Administered via Gavage to New Zealand White Rabbits*, Project No. 104-008 (July 3, 1985).

Second, the regulatory system of FIFRA and Florida's tort system serve different purposes. Out of an abundance of caution, federal agencies, in approving chemicals and drugs for market, assume that harmful effects in (even one species of) animals will occur in humans as well and on that basis they calculate safety margins and devise label warnings. In other words, agencies such as the EPA regulate based on a risk-assessment approach that implicitly uses a standard of causation that is *far less* than a preponderance of the evidence.[22] The tort system, however, is not designed to regulate mere risk, but to determine causation in a definitive fashion and to allocate blame. In *Gooding v. University Hosp. Bldg.*, a case on which plaintiffs prominently relied to secure review in this forum, this Court declared: "Relaxing the causation requirement might correct a perceived unfairness to some plaintiffs who could prove the *possibility* that the medical malpractice caused an injury but could not prove the *probability* of causation." 445 So. 2d 1015, 1019 (1984) (emphasis added). Nevertheless, the Court refused to lower the tort causation standard.

Thus, plaintiffs have it backwards. It is not unfair that DuPont "may use their animal and in vitro studies to get EPA approval . . . but the victims of toxic products may not use those same [studies] to prove their claims in court." P.'s Br. 37. Rather, it *would be unfair* if the same, minimal quantum of evidence that permits regulators to impose reasonable marketing conditions on *plainly beneficial*, but *only potentially* hazardous chemicals, would suffice under the tort system to justify a pronouncement that a manufacturer was liable for in fact causing a *specific* condition.

The Castillos' second policy argument is essentially that the Third District insisted plaintiffs in birth-defect cases must present epidemiological evidence of

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[22] *See, e.g.*, Frankos, *FDA Perspectives*, at 616 ("FDA *does not* assume that responses in animal models reflect exactly what will occur *qualitatively or quantitatively* in humans. What is assumed is the ability of well-designed animal studies to provide an indication of *potential risk to humans.*") (emphasis added).

causation and that it was impossible for them to do so, because it is unethical to experiment on human beings.[23] This argument has a number of obvious flaws. *First*, the Third District emphatically *did not* require plaintiffs to present epidemiologic evidence (particularly not in cases involving harmful chemical agents as opposed to therapeutic drugs), as plaintiffs themselves acknowledge elsewhere in their brief. *See* P.'s Br. 43. Thus, the very starting premise for plaintiffs' closing-the-courthouse doors argument is erroneous.

Second, despite plaintiffs' protestations that epidemiological evidence is not available here (*see* P.'s Br. 44 ("it is undisputed that epidemiological tests are not available")), they are completely wrong, as pointed out above. *See* pages 30-32. And, because non-clinical epidemiology is potentially available for plaintiffs to prove tort cases involving non-drug chemical agents, it is clear that *even if* the Third District had required such studies to prove human causation, they would not have closed the courthouse doors to plaintiffs. Also, because non-clinical epidemiology is generally available, Dr. Brent's methodological requirement of "positive epidemiology," R. (5/6/96) 3095, is not "impossible," as plaintiffs contend. *See* P.'s Br. 21.

The Third District's approach to considering epidemiologic evidence is neither

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[23] Despite the fact that Dr. van Velzen frequently voiced admonitions below about medical ethics, especially regarding human experimentation, *see* Tr. (4/30/96) 134-35, it is ironic that Dr. van Velzen has actually become embroiled in *two* ethical/criminal scandals of his own. *See, e.g.*, Charlie Gillis, *Organ Find Linked to British Scandal: Illegal Removals: DNA Analysis Might Determine Organs' Origins*, Nat'l Post (Oct. 2, 2000) A10, 2000 WL 26901394 ("Investigators have issued a Canada-wide warrant for Dr. Van Velzen's arrest on charges of offering an indignity to a human body, while Crown prosecutors look into having him extradited from his home in the Netherlands. He could face a maximum of five years in prison if convicted. The macabre find is significant because Dr. Van Velzen is at the centre of a scandal in Liverpool, England, where it emerged that organs had been removed from more than 800 children without their parents' permission.").

pro-plaintiff nor pro-defendant. No case better illustrates that than the principal case on which the plaintiffs rely – *Berry v. CSX Transportation*. In *Berry*, the First District *reversed* a grant of summary judgment for the defense because the trial court had misapplied *Frye* by excluding the plaintiffs' experts and preventing the case from going before a jury. And *Berry*, just like this case, involved *non-drug chemical agents* (specifically, solvents used in an industrial setting). The trial court granted summary judgment to the defense because it concluded that the results from certain non-clinical, observational epidemiological studies were unreliable. By reversing the trial court, then, the First District in *Berry* was holding that it was permissible for an expert to rely on the very type of study that the plaintiffs want to ignore. *See Berry*, 709 So. 2d at 557. *Berry* is thus the perfect case not only for (1) illustrating that the studies plaintiffs pretend are non-existent do in fact exist and they have been deemed reliable by the very District Court of Appeals' opinion plaintiffs stake most of their appeal upon, but also for (2) illustrating that an approach that allows the consideration of such epidemiologic evidence favors neither defendants nor plaintiffs. The rule of *Berry* regarding epidemiology is the same as the rule of *Castillo* and that rule is both a facially *and pragmatically neutral rule*.

In contrast to *Berry*, however, this Court has no obligation to ensure plaintiff victory in situations like this case where: (1) there is no generally accepted evidence of Benlate causing human birth defects; (2) unrealistic assumptions were made to calculate the exposure level of the human being involved; (3) even under such far-fetched assumptions, the levels of chemical the plaintiff could have been exposed to are orders of magnitude less than even the risk levels identified in unrealistic exposure single-species animal studies; and (4) to bridge that gaping dosage chasm, plaintiffs must rely on *in vitro* studies for an invalid purpose that is not generally accepted by science. What plaintiffs really seek are not fair and level-headed judicial doctrines designed to keep the courthouse doors open, but rather one-way rules that would

sweep away the jurisprudential house with a flood of litigation.

IV. PLAINTIFFS FAILED TO DEMONSTRATE THAT BENLATE WAS A DEFECTIVE PRODUCT UNDER FLORIDA TORT LAW OR TO DO SO IN A MANNER CONSISTENT WITH FEDERAL LAW.

Plaintiffs presented the jury with only two theories of liability: strict liability and negligent testing. *See* Tr. 5353-63, 5483-84. Both of these theories involve the same threshold elements of defect and causation. *See Adams v. G.D. Searle & Co.*, 576 So. 2d 728, 730, 731 (Fla. 2d DCA), *rev. denied*, 589 So. 2d 290 (Fla. 1991); *Mello v. K-Mart Corp.*, 792 F.2d 1228, 1233 (1st Cir. 1986). Thus, in order to prevail on either theory, plaintiffs were required to prove by a preponderance of the evidence *both* (1) that Benlate was defective (the focus of this section of the brief) *and* (2) that such defect proximately caused John Castillo's microphthalmia (see above).

Plaintiffs presented no evidence at trial to prove that Benlate is an "unreasonably dangerous," and hence defective, product. Rather, they tried to equate defect with causation by suggesting that Benlate must be deemed *per se* defective if it caused John Castillo's condition. *See* Tr. 5361-63. That is not the law. To the contrary, it is well-established that defect and causation are separate and independent requirements. *See, e.g., West v. Caterpillar Tractor Co.*, 336 So. 2d 80, 86-87 (Fla. 1976). A manufacturer, thus, cannot be held liable for an injury caused by its product unless that product "proves to have a *defect*." *id.* at 86 (emphasis added). Were the law otherwise, manufacturers would be transformed into insurers against injury. *See, e.g., Clark v. Boeing Co.*, 395 So. 2d 1226, 1229 (Fla. 3d DCA 1981).

The absence of proof of defect was no mere oversight on plaintiffs' part. Rather, their theory of the case was fundamentally incompatible with a showing of defect because the exposure alleged by plaintiffs was premised upon an alleged *misuse* of the product by the Farm in direct violation of the federally mandated and approved labeling. Plaintiffs attempt to surmount the obstacle presented by their failure to put in affirmative evidence of the relative risks and benefits of the product by hurling

allegations at DuPont of improper conduct between federal and California regulatory authorities. Those allegations are false, impossible to square chronologically with the federal administrative record, and (in the case of California) irrelevant. Most importantly, however, even if such allegations were true, they go to whether the federally mandated warnings on the Benlate label are adequate or not. But liability for inadequate warnings and inadequate product testing that would have led to improved warnings are matters unambiguously preempted by federal law.

A. Plaintiffs' Exposure Theory Was Based On An Unlawful Misuse Of Benlate Inconsistent With A Product Defect.

Plaintiffs' allegation of defect fails because their entire exposure theory is premised upon an alleged *misuse* of Benlate in direct violation of its federally mandated and approved labeling. The injury alleged here *could not have occurred* if the pesticide had been used in a lawful manner. *See* P.'s Br. 1 ("Pine Island Farms . . .negligently spray[ed] Benlate on its fields during periods of strong wind currents, thus allowing the chemical to blow into nearby residential and shopping areas where people could be exposed to its harmful properties."). A product is not defective, however, simply because it can cause injury when used in an unlawful manner.

Plaintiffs contend that John Castillo's microphthalmia was caused by Benlate absorbed through his mother's skin during an alleged incident in which a Farm tractor spewed a steady stream of the pesticide directly across a busy thoroughfare, "mesmerizing" Mrs. Castillo and thereby causing her to stand in the spray for several minutes until drenched. *See* Tr. 907-09, 993, 997-1000. At best, this theory of exposure would establish a gross and unlawful *misuse* of Benlate by the Farm – as indeed plaintiffs specifically alleged, *see id.* at 5327-28; *see also* R. (1/8/96) 12.

The conditions for the safe use of a pesticide are set forth in its federally approved labeling. Pesticide labeling is not mere cereal-box fluff, but rather *the label is the law*; it is federal law. *See* 7 U.S.C. §§ 136(ee), 136j(a)(2)(G); 40 C.F.R.

§ 170.9(a); Def. Ex. C at 1 (Benlate labeling) (attached as App. D). The Benlate labeling forbids the use of the product "in such a manner as to directly *or through drift* expose workers, or other persons." Def. Ex. C at 1 (emphasis added); *see also id*. ("Keep all unprotected persons, children, livestock and pets away from treated area *or where there is danger of drift*.") (emphasis added); *id.* ("Do not apply when weather conditions favor drift from areas treated."). Under basic tort law, a product is not defective simply because it is capable of causing injury when used unlawfully. *See, e.g., Talquin Elec. Coop., Inc. v. Amchem Prods., Inc.*, 427 So. 2d 1032, 1033 (Fla. 1st DCA 1983) (no liability based on misuse of herbicide); *Helene Curtis Indus., Inc. v. Pruitt*, 385 F.2d 841, 856 (5th Cir. 1967) ("[F]ailure to follow directions cannot support a finding of a defect."), *cert. denied*, 391 U.S. 913 (1968).

Virtually any product – and certainly any pesticide – is capable of causing injury if thus misused. But that does not render the product "unreasonably dangerous." *See, e.g., Husky Indus., Inc. v. Black*, 434 So. 2d 988, 990-91 (Fla. 4th DCA 1983).[24] Thus, for example, "[o]rdinary sugar is a deadly poison to diabetics, and castor oil found use under Mussolini as an instrument of torture. That is not what is meant by 'unreasonably dangerous.'" Restatement (Second) of Torts § 402A, cmt. i (1976). As long as a product is "reasonably safe for its *intended use*, as manufactured and designed," it is not, as a matter of law, defective. *West*, 336 So. 2d at 86 (emphasis

[24] Indeed, Dr. Howard conceded that many substances known to be actual or potential human teratogens if misused are nonetheless widely used. *See* Tr. 3157-58. He further conceded that even everyday substances such as sugar, caffeine, and aspirin could, at a high enough dose, kill human lung cells *in vitro*, *see id.* at 3183-84 – and thus, under plaintiffs' skewed vision of the law, would qualify as "defective." Indeed, if Benlate can be deemed defective, then almost any product (and virtually every pesticide) can also be deemed defective.

added).[25] By the same token, a product is not defective if injury results from a misuse against which the manufacturer provided adequate warnings. *See, e.g.*, Restatement (Second) of Torts § 402A, cmt. j (1976); *see also* Restatement (Third) of Torts § 2 cmt. p (1997).

The federally approved Benlate label provided ample warnings against using the pesticide where there was danger of drift. Those warnings are necessarily adequate: FIFRA precludes any state-law challenge to the adequacy of the warnings on a federally approved pesticide label. *See* 7 U.S.C. § 136v(b); *see also, e.g., Grenier v. Vermont Log Bldgs., Inc.*, 96 F.3d 559, 564 (1st Cir. 1996) and n.29 below. Because plaintiffs produced no evidence that Benlate is not "reasonably safe for its intended use," they failed as a matter of law to prove a defect.

B. At Best Plaintiffs Presented Evidence That Benlate Should Have Been Marketed With Pregnancy Warnings Or Subjected To Additional Testing, But Those Theories Of Liability Were Preempted By Federal Law.

As suggested by the plaintiffs' silence on this point in their opening brief to this Court, they presented absolutely no evidence showing Benlate was defective – in other words no evidence that its risks outweighed its benefits. There is no evidence of other designs in the record or discussions of the utility of Benlate as a fungicide compared

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[25] The approach in the new Restatement (Third) of Torts § 2 (1997) is essentially the same. *See, e.g., id.* illus. 20 ("The ABC Chair Co. manufactures and sells oak chairs. The backs of the chairs have five horizontal wooden bars shaped to the contour of the human back. John, a college student, climbed up to the top bar of an ABC chair to reach the top shelf of a bookcase. The chair tipped and John fell, suffering serious harm. John brings an action against ABC, alleging that the chair should either have had the stability to support him when standing on the top bar or have had a differently designed back so that he could not use the bars for that purpose. The ABC chair is not defectively designed. John's misuse of the product is so unreasonable that the risks it entails need not be designed against.").

to the harm it allegedly causes. *Compare Perry v. Red Wing Shoe Co.*, 597 So. 2d 821, 822 (Fla. 3d DCA 1992) (testimony in record regarding alternative work boot design). Indeed, all the evidence below was that Benlate is uniquely effective, and even plaintiffs' expert Bill Hunt described it as "a very, very stable product" that is a "cure-all" and a fungicide "of choice." Tr. 1879-80; *see also* 47 Fed. Reg. 46,747, 46,749-50 (1982) (benefits of benomyl are "significant").

Instead of evidence relevant to banning Benlate or altering its design, plaintiffs focused their proofs on attempting to show that DuPont had somehow pulled the wool over the eyes of EPA regulators. That claim, which plaintiffs advanced unsuccessfully to the Third District as well, simply does not hold up to scrutiny.

According to *plaintiffs' version* of events:

- Dr. Staples performed his first study in 1980, which showed birth defects in rats at LOELs of 10 milligrams per kilogram of body weight per day. *See* P.'s Br. 12-13.
- As a result, the EPA "decided that warnings should be issued to pregnant women advising of the potential teratogenic properties of Benlate." *Id.* at 13.
- Then, DuPont improperly told Dr. Staples to redo his study in 1982. After seeing the results of that study, the EPA "pulled" its warning. And finally, DuPont changed the results of Dr. Staples' first study when it was later reported to the State of California. *See id.* at 13, 33.

The *reality* of the regulatory process is that the plaintiffs' allegations as summarized above are a chronological impossibility. The Agency considered the need for but *never* proposed a pregnancy warning for dermal exposure of the sort alleged here. And DuPont's Dr. Staples did not conclude his first study in 1980 until *after* the EPA had proposed in 1979, and the Scientific Advisory Panel that EPA must consult with, *see* 7 U.S.C. § 136w(d); 40 C.F.R. § 154.31(b)(3), had rejected an inhalational pregnancy warning later that same year. *See* 44 Fed. Reg. 51,166 (1979); 42 Fed. Reg. 61,788 (1977); Tr. 4281, 4294-99. The Agency was fully aware of the results of the

first Staples study when it *closed* its special review of benomyl in 1982, but dismissed those results as statistically insignificant. *See* Tr. 2504-05, 3717-18.[26] And the second Staples study in 1982 had nothing to do with that decision; indeed, the final EPA position document closing the special review of benomyl does not even *mention* the second Staples study except to note that "DuPont is *currently* performing a follow-up study" to the first Staples study. *Id*. at 3718 (emphasis added). Plaintiffs' bizarre accusation that DuPont relied upon the second Staples study to "manipulate[] its test results for the sake of obtaining unwarranted EPA approval for [the company] to distribute Benlate without warnings to pregnant women," P.'s Br. 33, thus goes well beyond the bounds of professional advocacy.[27]

Apparently not content with the outrageous argument that DuPont hijacked one regulatory process (the federal government's), as they claimed in the Third District

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[26] When the Agency finalized its special review of benomyl, it agreed with the Scientific Advisory Panel that Benlate did not pose any significant risk of birth defects *even to pregnant women who inhaled the pesticide on a routine basis in the course of their employment. See id*. at 46,750; Tr. 4298-304. And, of course, while the risks of a dermal exposure for pregnant women had been evaluated, the Agency never once proposed the need for such a dermal pregnancy warning – a conclusion in which the Scientific Advisory Panel concurred. "We concur with the agency's position that it would not be possible to obtain a level of benomyl in the blood through *dermal* absorption which would pose a significant risk." 47 Fed. Reg. 46,747, 46,751 (1982) (emphasis added); Tr. 4299. Thus, in October 1982, the EPA issued a final Notice of Determination concluding that the risk of birth defects caused by *any* form of exposure to Benlate was so insubstantial that *no pregnancy warning of any kind was warranted*. *See* 47 Fed. Reg. at 46,750; Tr. 4304.

[27] Plaintiffs also attempt to cast a sinister pall over the fact that a DuPont colleague once referred to Dr. Staples as a "lone ranger," Tr. 2430. But that is nothing more than a diversionary tactic. Dr. Staples clearly testified that no one at DuPont expressed "dissatisfaction" with his 1980 study results, and that "[n]obody came down and said, 'You made a mistake,' and all that kind of stuff." *Id*.

below, plaintiffs return in desperation to allegations advanced only in the trial court that DuPont also coopted the regulatory processes of California's so-called "Prop 65" system (the California Safe Drinking Water and Toxic Enforcement Act). *See* P.'s Br. 33, 41-42. The short answer to these allegations is that DuPont moved *in limine* to exclude evidence of and preclude reference to other state regulatory actions regarding Benlate, including California's Prop 65. *See* R. (4/22/96) 2563-2569. That motion was granted by the trial court in response to the plaintiffs' concession that it would not talk about the warnings that California requires or does not require regarding Benlate. *See* Tr. 2052-53; Tr. (5/09/96) 532; *see also* Tr. 2342-44.

Even if the plaintiffs' "evidence" of DuPont's "manipulation" of the regulatory process was credited, however, at best it bears on two *preempted* forms of state tort liability under FIFRA – failure-to-warn, and negligent-testing liability. In the EPA context, plaintiffs contend that (1) DuPont misled the Agency into deleting a proposed pregnancy warning, or (2) perhaps that if more tests had been performed, more results like the first Staples study would have been generated. They made no serious attempt to show that somehow the first Staples study or more studies of its ilk would have led EPA to ban Benlate. And in the California context, since benomyl is *already* listed under Prop 65, it is unclear how the alleged manipulation of the California regulatory process could have any material significance to this case (even putting aside the threshold issue that *whatever* happens in California is legally irrelevant here). Thus, all that plaintiffs are left with are their inaccurate claims that Benlate is defective because it should have included additional product warnings or been subjected to further testing.

But these remaining theories of liability are preempted by federal law, pursuant to FIFRA. "State[s] shall not impose or continue in effect any requirements for labeling or packaging in addition to or different from" those required under FIFRA. 7 U.S.C. § 136v(b). Because DuPont could reasonably predict where the plaintiffs'

proofs were headed, it moved at the outset of the case to have this entire case declared preempted under FIFRA. *See* R. (3/29/96) 1390-1406. The trial court granted that motion with respect to failure-to-warn liability, but erroneously denied it with regard to negligent-testing liability. *See* Tr. (5/10/96) 697. In addition, once the proofs were concluded, the trial court erred in not granting the renewed preemption argument in DuPont's directed-verdict motion or motion for judgment notwithstanding the verdict. By that point it was clear that plaintiffs had failed to prove a product defect directly, but were instead relying on failure-to-warn- and negligent-testing sorts of theories exclusively.[28] Applying the United States Supreme Court's analysis in *Cipollone v. Liggett Group*, 505 U.S. 504 (1992) and other preemption decisions, eight federal circuit courts of appeals and ten state supreme courts have held state failure-to-warn liability preempted because allowing such liability would interfere with FIFRA's exclusive system for regulating pesticide labeling.[29]

Negligent testing claims have also been found preempted. The logic of finding such claims preempted is also obvious: A product cannot be found defective without first analyzing its instructions for use and accompanying warnings. "[A] negligent

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[28] Because DuPont prevailed on appeal below, obtaining a favorable decision, it is wholly within its rights under Florida's so-called "tipsy coachman" rule in urging FIFRA preemption as a basis for this Court to affirm the Third District. *See Dade County Sch. Bd. v. Radio Station WQBA*, 731 So. 2d 638, 645 (Fla. 1999). In any event, preemption is a defense that may be raised at *any time* in the proceedings, as long as there is no need for additional factual development. *See Zuliana de Aviacion v. Herrera*, 763 So.2d 499 (Fla. 3d DCA 2000); *Florida Auto. Dealers Indus. Benefit Trust v. Small*, 592 So. 2d 1179, 1183-84 (Fla. 1st DCA 1992).

[29] *See, e.g., Hawkins v. Leslie's Pool Mart*, 184 F.3d 244, 248-49 (3d Cir. 1999); *Goeb v. Tharaldson*, 615 N.W.2d 800, 817 (Minn. 2000); *see also ISK Biotech Corp. v. Douberly*, 640 So. 2d 85, 89 (Fla. 1st DCA 1994), *rev. denied*, 651 So. 2d 1194 (Fla. 1995).

testing claim can only survive a preemption challenge if adequate testing would have caused the manufacturer to alter the product. If the manufacturer, upon further testing, would have altered only the label, then any claim based on inadequate testing is preempted by FIFRA." *Wright v. American Cyanamid Co.*, 599 N.W.2d 668, 674 (Iowa 1999). Given FIFRA preemption, in the absence of a preponderance of evidence supporting the conclusion that a product should be physically altered (and this record is devoid of *any* such evidence), courts must assume that the outcome of any additional testing that was allegedly not performed would have been improved product labeling and warnings – modifications that fall wholly within the preemptive scope of FIFRA.

CONCLUSION

This Court should dismiss the Petition as improvidently granted or affirm the judgment under review in its entirety. Alternatively, the Court should either remand the case to the Third District Court of Appeal for consideration in the first instance of DuPont's misuse defense, or remand the case for a new trial on the basis: (1) of the references to non-existent "clusters" of microphthalmic children, or (2) of excluding the testimony of Dr. Hurtt – both of which are errors that affected the fundamental fairness of the trial.

Respectfully submitted,

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