Sheley v Shiffman
2014 NY Slip Op 33470(U)
February 14, 2014
Supreme Court, Kings County
Docket Number: 44138/07
Judge: Gloria M. Dabiri

At an IAS Term, Part 2 of the Supreme Court of the State of New York, held in and for the County of Kings, at the Courthouse, at Civic Center, Brooklyn, New York, on the 20th day of January 2014.

P R E S E N T:

HON. GLORIA M. DABIRI,

Justice.

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DEKHARI SHELEY, an infant, by his mother and natural guardian, DELINDA SHELEY,

Plaintiff(s),

DECISION AND ORDER

- against -

Index No.: 44138/07

REBECCA L. SHIFFMAN, M.D., MATVEY PINKUSOVICH, M.D., DONNA A. FELDMAN, M.D., RONALD A. KESTER, M.D., and NEW YORK METHODIST HOSPITAL,

Defendant(s).

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The following papers numbered 1 to 11 read on this motion:

	Papers Numbered
Notice of Motion/Order to Show Cause/ Petition/Cross-Motion and Affidavits (Affirmations) Annexed	1–4
Opposing Affidavits (Affirmations)	5–8
Reply Affidavits (Affirmations)	9–11
Affidavit (Affirmation)	
Other Papers	

By Order to Show Cause, dated May 9, 2013, plaintiff Dekhari Sheley, by his guardian Delinda Sheley, seeks an order (1) granting renewal and/or reargument of the defendants' March 7, 2012 Order to Show Cause which resulted in this court's decision and orders of July 20, 2012 directing a Frye hearing and (2) if renewal and reargument are denied, or if the July 20, 2012 order is adhered to, granting plaintiff leave to seek a stay pending appeal.

BACKGROUND

On December 3, 2007 plaintiff, by his mother Delinda Sheley, commenced this action for medical malpractice in connection with the mother's prenatal care, labor, and the delivery of the infant plaintiff Dekhari Sheley on May 12, 2005 at New York Methodist Hospital. Issue was joined by the defendant Ronald A. Kester, M.D. on July 21, 2008 and by the remaining defendants on January 8, 2008. A preliminary conference was held on April 2, 2008, depositions were conducted, and plaintiff filed a Note of Issue on December 23, 2009.

By motion, filed January 11, 2010, the defendants sought an order, pursuant to 22 NYCRR §§ 202.21(e) and 208.17(c), vacating the Note of Issue due to outstanding discovery, including production of the plaintiff mother's medical records. In support of this aspect of their discovery demand the defendants supplied (in reply) the affidavit of a professor of psychiatry and pediatrics who opined that the infant, Dekhari Sheley, suffers from autism, a chronic neurodevelopmental and heritable disorder in which 90 percent of the risk is genetically based and the other 10 percent of the variance has not been demonstrated to be causally related and may be mediated by genetic mechanisms. Finding, *inter alia,* that the defendants' expert failed to articulate how records of the mother's prior pregnancies, terminations or gynecological treatment would be relevant to a determination as to the cause of the infant's injury, the court denied this discovery, — provided that the mother did not intend to put her prior medical treatment in issue at the trial. The Note of Issue was vacated (see Order of July 12, 2010).

Additional discovery was directed by Order of September 17, 2010. Plaintiff filed a new Note of Issue and jury demand on October 1, 2010. By Order of August 16, 2011 the plaintiff was

directed to exchange CPLR 3101(d) statements by November 17, 2011 and the defendants by December 21, 2011.

In an Order to Show Cause dated March 7, 2012 the defendants moved, in the Medical Malpractice Trial Readiness Part, to preclude plaintiff from presenting expert testimony on the issue of the causal connection between the defendants' alleged negligence and the infant's autistic disorder or, in the alternative, for a *Frye* hearing (*Frye v United States,* 293 F. 1013 [1923]). On March 12, 2012 the case was referred from the Medical Malpractice Trial Readiness Part to IAS Part 2 for trial. By Order of March 15, 2012, the defendants' Order to Show Cause was referred to IAS Part 2 to be heard on May 18, 2012. The plaintiff opposed the Order to Show Cause, by affirmation dated June 18, 2012, and the defendants submitted a reply on or about June 27, 2012. Following argument of the motion on July 20, 2012, the court granted the defendants' motion to the extent of setting the matter down for a *Frye* hearing on October 5, 2012 and directing that, by September 30, 2012, the parties exchange the medical and scientific literature upon which their experts would rely.

By letter of September 13, 2012 plaintiff requested an adjournment of the *Frye* hearing to permit plaintiff's experts to complete their review of literature relating to the etiology of autistic and pervasive developmental disorders. Defendants' counsel consented to the adjournment but opposed the plaintiff calling any expert or offering literature not previously relied upon in opposition to the defendants' Order to Show Cause. The court adjourned the hearing to November 7, 2012, directing that documents be exchanged on or before October 18, 2012. In a letter of September 27, 2012, counsel for the defendants requested an adjournment of the hearing due to the unavailability of their expert. Upon consent, the *Frye* hearing was rescheduled for November 28, 2012, and thereafter for

January 18, 2013.

By letter of January 15, 2013 counsel for plaintiff requested an adjournment of the hearing in order to allow the infant to undergo an MRI of his brain. In support, plaintiff provided the affirmation of Dr. Chon Ken Chen who averred that an MRI would assist in resolving the question of "the etiology of the infant plaintiff Dekhari Sheley's current condition." Counsel for the parties appeared in court on January 18, 2013 at which time the court granted plaintiff's application. Under cover of letter dated March 14, 2013, counsel for plaintiff exchanged Dr. Gregor J. Lawler's March 10, 2013 report of a February 13, 2013 MRI study of the infant's brain. Dr. Lawler reported "[a]bnormal increased T2 signal within the right parietal lobe periventricular white matter, likely due to prior insult (hypoxic or ischemic) to the brain parenchyma," and "[n]o acute intracrenial abnormality."

Meanwhile, by correspondence of January 29, 2013 and February 6, 2013, counsel for the defendants sought an order directing that the infant, Dekhari Sheley, submit to genetic testing. The affirmation of Dr. Kwane Anyane-Yeboa, board certified in Pediatrics and Clinical Genetics, was supplied in support of this application. Dr. Anyane-Yeboa averred that the reason for such testing was the May 2012 report of pediatric neurologist Sandra Forem who observed that the infant has "macrocephaly, multiple pigmented and hypopigmented skin lesions ... findings ... highly suggestive of incontinentia pigmenti or other phakomatosis." Dr. Anyane-Yeboa explained that the term "phakomatosis" describes "a heterogeneous group of disorders, generally hereditary, that affect skin, brain, and other organs ...." Type 1, incontinentia pigmenti acromians (or Hypomelanosis of Ito) presents with "variable degrees of mental retardation ...." According to Dr. Anyane-Yeboa, "[m]any

patients with this condition also have autism .... The majority of cases have been demonstrated to be due to chromosome mosaicism ...." The doctor opined that the "best way to demonstrate chromosome mosaicism is by biopsying the skin ... [this is] because the life cycle of the cells with abnormal chromosomes is quite short in blood, but survive better in skin cells." Dr. Anyane-Yeboa averred that "genetic testing is warranted because Dekhari Sheley has classic features of incontinentia pigmenti Type 1, a genetic disorder," and that testing should be done on blood and skin fibroblast cultures to increase the probability of demonstrating mosaicism associated with incontinentia pigmenti Type 1.

By letter of February 15, 2013 counsel for plaintiff advised the court that "[p]laintiff does not object to providing a blood sample .... However, plaintiff does object to the proposed skin biopsy." Plaintiff offered the affirmation of Dr. Chon Ken Chen, board certified in Pediatrics and Neurology, who opined that "the proposed skin biopsy test is unnecessary because a properly performed blood test can accurately test for the presence of incontinentia pigmenti Type 1 .... Moreover, the proposed skin biopsy procedure is extremely painful ... [as it] requires a deep circular cut in the skin 3-4 millimeters in diameter to remove a sample of the dermis ... leaves a wound that will require at least five days to properly heal and will almost certainly leave a permanent scar. In addition, there is the possibility that the wound may become infected." In reply, defendants supplied the February 25, 2013 affirmation of Dr. Anyane-Yeboa who opined that Dr. Chen is not qualified to opine on genetic testing, and that a biopsy of the skin is the best way to test for chromosome mosaicism.

By order, dated March 5, 2013, this court granted defendants' application to the extent of

directing genetic blood testing of the infant plaintiff for chromosome mosaicism, without prejudice to the defendants' renewed application for a skin biopsy upon a showing of good cause.

THE DEFENDANTS' ORDER TO SHOW CAUSE

As noted, the defendant's Order to Show Cause, dated March 7, 2012, sought an order precluding plaintiff from presenting expert testimony at trial as to causation between the defendants' alleged malpractice and the infant plaintiff's claimed brain damage. Defendants pointed out that while the plaintiff had not, as yet, served an expert witness disclosure (CPLR 3101[d]), the Bill of Particulars and Supplemental Bill of Particulars reveal that the plaintiff claims, *inter alia,* that the defendants' failure to appreciate and/or treat premature rupture of membranes, maternal infection, chorioamnionitis, abnormal labor, fetal distress and/or hypoxia and failure to timely deliver the infant by Cesarean section resulted in the infant's "brain damage." Defendants argued that the infant has been diagnosed with Pervasive Developmental Disorder/Autistic Disorder (autism)[1] which is primarily a genetic condition. Defendants maintained that although risk factors, such as the number of pre, peri and post natal complications, have been identified as being associated with autism, the medical literature does not support the conclusion that any of these risk factors, either individually

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[1]"The terms 'autism' and 'autism spectrum disorder' (ASD) have been used to describe a set of developmental disorders characterized by impairments in social interaction, impairments in verbal and nonverbal communication, and stereotypical restricted or repetitive patterns of behavior and interests .... Those terms are essentially synonymous with the term pervasive developmental disorder ('PDD'), commonly used in medical diagnosis .... The PDD category is further subdivided into five subcategories: autistic disorder, childhood disintegrative disorder ('CDD'), Asperger's Syndrome, Rett's Syndrome, and pervasive developmental disorder not otherwise specified ('PDD-NOS')" (*Cedillo v HHS,*2009 WL 331968 [Cl. Ct. 2009] reconsd den. 2009 WL 996299, affd 89 Fed. Cl. 1588 [2009], citing 617 F.3d 1328 [Fed. Cir. 2010], citing Institute of Medicine, Immunization Safety Review: Vaccines and Autism [The National Academies Press 2004]).

or in combination, are a cause of autism.

In support of the motion defendants supplied a copy of the reports of psychological evaluations of the infant Dekhari, performed in 2007 and 2009, which diagnosed him with Pervasive Developmental Disorder (PDD)/autistic disorder/autism [DSM IV-299-00]). The defendants also provided the affirmation of psychiatrist Alexander Kolevzon, Clinical Director of the Seaver Autism Center for Research and Treatment, and Associate Director of Residency Training in the Division of Child and Adolescent Psychiatry at Mount Sinai School of Medicine. Dr. Kolevzon affirmed that he had reviewed, *inter alia,* records of the plaintiff's prenatal care, labor and delivery, newborn admission and pediatric clinic care, the records of Dr. Mark Lew, Thursday's Child, Kidz Consulting Service, Netcare, N.Y. Early Intervention, The League Treatment Center, and Dr. Eliezer Friedman, as well as transcripts of the deposition testimony of Dekhari's mother and grandmother. Dr. Kolevzon opined that these records indicate that Dekhari has been diagnosed with autistic disorder (autism) and has not been diagnosed with any other condition. He avers that autistic disorder (autism) is a developmental condition "where the majority of risk is genetically based," and that while "various factors have been associated with autistic disorder (autism), there are no environmental risk factors that are accepted by the medical or scientific community as a cause of autistic disorder."

According to Dr. Kolevzon, risk factors that have been associated with autistic disorder include advanced parental age, low birth weight, hypoxia and various other pre, peri and/or postnatal complications. However, an association does not imply that there is a causal relationship between them. Dr. Kolevzon averred that there are "no studies or literature that establish that the above described risk factors *cause* autistic disorder (autism) and any claim that one or more of [these]

factors is a cause of autistic disorder (autism) is not accepted in the medical community." The doctor concluded, to a reasonable degree of medical certainty, that "the cause of Dekhari Sheley's autistic disorder (autism) is primarily genetic in origin."

Defendants also supplied the May 12, 2012 report of Dr. Sandra L. Forem, a pediatric neurologist who examined the infant and offered her impression that "Dekhari is an autistic five-year old boy noted to have macrocephaly, multiple pigmented and hypopigmented skin lesions, ... findings ... highly suggestive of incontinentia pigmenti or other phakomatosis."

Finally, the defendants pointed out that the infant's mother testified that he had been diagnosed with autism and had not been diagnosed with any condition other than PDD/autism; and that his grandmother, with whom he has resided since birth, also testified to the diagnosis of PDD/autism. Thus, argued defendants, the plaintiff's anticipated claim that the infant's PDD/autistic disorder was caused by the defendants' malpractice during their prenatal care or the labor and delivery — resulting in a perinatal hypoxic-ischemic insult — is not generally accepted as reliable within the scientific or medical community (*Frye v United States,* 293 F. 1013 [D.C. Cir. 1923]; *People v Wesley,* 83 NY2d 417 [1994]).

In **opposition** the plaintiff argued, *inter alia,* that brain damage is the source of the cognitive disorders and maladaptive behaviors that constitute autistic disorder and other PDD's, and that pre, peri and post natal hypoxic-ischemic insults, which are generally accepted causes of brain injury, are thus also generally accepted as contributors to the etiology of autism spectrum disorders. Plaintiff argued that the scientific and medical literature rarely furnishes an explicit pronouncement as to causation and that courts, therefore, must do what the scientific community does, which is to

consider the strength of the association reported in the literature as the test of whether a theory of causation has obtained general acceptance.

Plaintiff submitted the June 18, 2012 affirmation of Dr. Chon Ken Chen, a pediatric neurologist; the affidavit of Susan Shott, Ph.D., a biostatistician; and the Functional Assessment Report of psychologist Vicki Sudhalter, Ph.D., sworn to on June 8, 2012. Plaintiff also provided, as exhibits, publications relating to autism, its causation and risk factors.

Dr. Chen averred that following a review of Ms. Sheley's and Dekhari's medical records and the defendants' Order to Show Cause with exhibits, he conducted a neurological examination of Dekhari on May 26, 2012. Dr. Chen indicated that he does not disagree with Dr. Kolevzon's diagnosis of autistic disorder (autism) [DSM-IV 299.00], which is exclusively behaviorally defined and diagnosed, and independent of any etiological diagnosis. However, Dr. Chen pointed out that his examination of the infant also revealed toe-walking and severe dyspraxia (fine motor incoordination) which are not features of PDD disorders, but are signs of motor deficits frequently associated with peripartum hypoxic-ischemic insult.

Dr. Chen argued that Dr. Kolevzon did not consider changes in gene expression that result from environmental influences, such as inflammation and other immunological reactions to maternal infection, for example chorioamnionitis with which Ms. Sheley was diagnosed. According to Dr. Chen, oxidative stress from hypoxic-ischemic insult during labor, as alleged in this case, has been identified as a particularly significant cause of prenatal brain injury. Dr. Chen opined that it is generally accepted in the medical research community that hypoxic-ischemic brain injury during labor and delivery is one of the causes of autistic disorder.

Dr. Chen maintained that Dr. Kolevzon's own writings ("Prenatal and Perinatal Risk Factors for Autism" and "Parental and Perinatal Risk Factors for Autism," Chapter 20 of the Textbook of Autism Spectrum Disorders [2011]) demonstrate no qualitatively stronger evidence of a genetic etiology for autistic disorder than for an environmental one. In fact, argued Dr. Chen, recent research suggests that environmental factors play a greater part in autistic spectrum etiology than genetic factors (citing Hallmayer, "Genetic Heritability and Shared Environmental Factors Among Twin Pairs with Autism," Arch Gen. Psychiatry [2011]). Dr. Chen averred that it is generally accepted in pediatric neurology that hypoxic-ischemic insult in utero, by itself or potentially in concert with enhanced vulnerability to oxidative stress, increases the likelihood of autistic disorder or other PDD.

Susan Shott, Ph.D., a biostatistician with 30 years of experience, in opposition to the defendants' motion, affirmed that she had reviewed the articles submitted by the defendants as well as the current literature on autism spectrum disorder etiology, and that her research disclosed that "the current consensus of the medical and scientific community is ... [that] [a]lthough no specific causes of autism or autism spectrum disorder (whether genetic or environmental) have been definitely established ... [i]t is far more likely than not that autism and autism spectrum disorder are multifactorial, with different cases ... often having different causes .... It is far more likely than not that the causes of autism and autism spectrum disorder include both genetic and environmental factors." Dr. Shott relied, in part, on *Autism Spectrum Disorders,* (Amaral DG, Dawson G. Gesschwind DH, eds., Oxford: Oxford Univ. Press, 2011, p 825-892), a textbook on autism spectrum disorders, quoting:

> Despite modern technology and advanced research, only approximately 6% to 15% of individuals with autism will be found to have an identifiable genetic diagnosis .... Numerous genes have been investigated as possible candidate genes, but replicated findings are lacking. Current epidemiological studies ... strongly suggest multifactorial inheritance, including genetic heterogeneity with multiple major gene effects, possible contributing environmental effects, and physiologically linked processes with multiple genes.

Dr. Shott averred that a recent study which investigated genetic heritability and shared environmental factors among twin pairs with autism, found that:

> . . .
> susceptibility to [autism spectrum disorder] has moderate genetic heritability and a substantial shared twin environmental component.
> . . .
> The shared environment component ... was estimated to be larger than the genetic heritability component [contributing to autism] ... The results suggest that environmental factors common to twins explains about 55% of the liability to autism. Although genetic factors also play an important role, they are of substantially lower magnitude than estimates from prior twin studies of autism.
> . . .
> [Hallmayer J. et al Genetic Heritability and Shared Environmental Factors Among Twin Pairs with Autism, *Arch Gen. Psychiatry* 2011; 68:1095-1102].

Dr. Shott noted that the *Autism Spectrum Disorders* textbook (*supra*) describes the medical and scientific literature concerning obstetric factors as a cause of autism, as follows:

> Fetal/Infant characteristics such as low APGAR scores, breech presentation, and fetal distress have been observed in autism. Current thinking regarding mechanistic implications of [fetal] stress markers is that prenatal or birth hypoxia, the former being inducible by infection and the latter by C-section, may alter structural or functional features of CNS development, with permanent consequences through mechanisms such as delayed neuronal mitigation, degeneration and chromatolysis in the striatum, reduced myelination, increased dopamine DI receptor binding, astrocytic fibroblast growth factor, and binding of

insulin-like growth factors I and II.

Dr. Schott opined that it is widely accepted in the medical and scientific community that environmental factors — which include antepartum or intrapartum hypoxic fetal distress — are at least as causally related to autism and autism spectrum disorders as are genetic factors. In this regard, Dr. Shott also relied upon Gardener H, Spiegelman D, Buka SL., *Perinatal and Neonatal Risk Factors for Autism: A comprehensive meta-analysis*. *Pediatrics* 2011; 128, 344-355.

> "The obstetrical complications that have emerged as significant risk factors for autism in the current meta-analysis suggest a possible role of fetal and neonatal hypoxia. In particular, growth retardation, fetal distress, umbilical-cord wrapping around the neck, low APGAR score, respiratory distress, resuscitation, meconium aspiration, and Cesarean delivery are all potential risk factors that also may be associated within increased risk of hypoxia. Although some brain abnormalities observed in individuals with autism may reflect a potential role of oxygen deprivation during development, this possibility requires additional examination. Hypoxia also has been shown to increase dopaminergic activity, and there is evidence for dopamine overactivation in autism."

Finally, plaintiff supplied the affirmed report of Vicki Sudhalter, Ph.D., a psychologist who evaluated Dekhari on September 11, 2010 and concluded that he meets "the behavioral criteria for a diagnosis of Autistic Disorder. Dr. Sudhalter found Dekhari's medical history to be "remarkable for a difficult delivery, characterized by prolonged labor, maternal fever, and a non-reassuring fetal heart rate pattern suggestive of fetal distress" and opined that "these factors placed him at risk for significant injury to his central nervous system during a critical period of brain development." It was her opinion that based upon the infant's neurobehavioral and neuropsychological deficits, his medical history and the current medical literature, the infant's "brain damage (consistent with perinatal hypoxic brain injury), significantly contributed to his cognitive,

social and behavioral impairments."

In **reply,** defendants argued that none of the plaintiff's experts offered their personal opinion as to the role of hypoxia in the causation of autism, and that a fair reading of the medical literature upon which they relied reveals that there has been much research devoted to the role of many possible causes of autism (including perinatal hypoxic-ischemic insult) but no clear statement that any of these causes have been determined to be a cause of the condition.

THE PLAINTIFF'S ORDER TO SHOW CAUSE

Presently before the court is the plaintiff's Order to Show Cause which seeks leave to renew or reargue the defendants' March 7, 2012 motion for an order precluding plaintiff's expert testimony at trial as to a causal link between the defendants' alleged malpractice and the infant's brain damage or for a *Frye* hearing, which resulted in this court's order of July 20, 2012,[2] directing a *Frye* hearing on the issue of whether it is generally accepted in the relevant medical community that hypoxic-ischemic insult to an infant during labor and/or delivery can be a competent producing cause of autism. Upon renewal and/or reargument plaintiff seeks an order vacating the court's July 20, 2012 order and setting the matter down for a trial. Alternatively, plaintiff seeks consideration of the additional submissions and a written order explaining the scope of the *Frye* hearing. Finally, should the court deny renewal and reargument, plaintiff seeks leave to move for a stay of the July 20, 2012 order pending appeal.

In support of her Order to Show Cause, plaintiff argues that the February 13, 2013 MRI of the infant's brain reveals "that the white matter damage seen in the MRI is secondary to a

[2]The record of the July 20, 2012 proceedings cannot be located.

hypoxic-ischemic insult (HIE) at or around the time of birth." Thus, plaintiff contends, it "matters not whether the plaintiff has ... PDD/Autistic Disorder/Autism ... and ... it does not matter whether the [PDD/Autistic Disorder/Autism] was 'caused' by the HIE or exists independently and is caused by something other than the HIE." Plaintiff argues that she does not dispute the infant's diagnosis of autistic disorder, but maintains that this diagnosis "simply characterizes some of his behavior patterns" and that plaintiff is prepared to prove at trial that the infant *also* has brain damage caused by hypoxic-ischemic encephalopathy (HIE) resulting in cognitive and motor deficits and mental retardation. Plaintiff agrees to stipulate that plaintiff will *not* attempt to prove that Dekhari's PDD/Autistic Disorder/Autism was caused by hypoxic-ischemic encephalopathy. Plaintiff also offers to stipulate that plaintiff shall not mention PDD/Autistic Disorder/Autism before a jury *except* in response to claims raised by the defendants.[3]

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[3]Plaintiff also points out that Dr. Kolevzon, the defendants' expert, merely opined that Autistic Disorder/Autism is a "developmental condition" where the "majority of risk" is "genetically based," and that Dekhari's Autistic Disorder/Autism is "primarily genetic in origin." Thus, argues plaintiff, the defendants cannot "prove" that hypoxic-ischemic encephalopathy, or other environmental risk factors, was *not* a cause of, or did not aggravate, the infant's Autistic Disorder/Autism.

Citing cases decided under the Childhood Vaccine Injury Act (42 USC §300aa -10 *et seq.* [which compensates vaccine-related injury or death]), plaintiff argues that recovery has been granted where it has been found that the vaccine did not *directly* cause the autism or autistic symptomology, but instead caused, or exacerbated, a condition which, in turn, resulted in autistic symptomology

See, Poling v HHS (2008 WL 1883059 [2008] [vaccination "significantly aggravated an underlying mitochondrial disorder, which predisposed [child] to deficits in cellular energy metabolism and manifested as a regressive encephalopathy with features of autism spectrum disorder].

See, however, Cedillo v HHS (2009 WL 331968 [Fed. Ct., Feb 12, 2009], affd 89 Fed. Cl. 158 [Fed. Cl., Aug. 6, 2009], affd 617 F.3d 1328 [Fed. Cir., Aug 27. 2010] [holding no showing that it was more probably than not that vaccine caused autism]).

Plaintiff submits, *inter alia,* the April 22, 2013 affirmation of radiologist Gregory J. Lawler, M.D., the April 13, 2013 affirmation of pediatric neurologist Chon Ken Chen, M.D., and the April 18, 2013 affirmation of Bruce L. Halbridge, M.D.

Dr. Lawler affirms that upon his review of the above-referenced affirmations of Doctors Chen and Halbridge, and the defendant Dr. Matvey Pinkusovich's May 12, 2002 operative report, that it is his "opinion to a reasonable degree of medical certainty, that the observations of abnormality referenced to in [his March 12, 2002] report are the result of hypoxic-ischemic insult suffered by Dekhari Sheley during labor and delivery."

Dr. Chen opines that an early C-section would have avoided the trauma, inflammation, cytokine exposure, cord compression, hypoxia and cerebral edema which occurred during the peripartum period. According to Dr. Chen, failure to perform an early C-section caused Dekhari to suffer hypoxic-ischemic insult, beginning intrapartum and continuing through the first days of life. This injury resulted in neuronal cell death in the parietal lobe, which is depicted on the brain MRI as white matter damage. Dr. Chen indicates that the infant has spasticity in his calf muscles as a result of upper motor neuron injury which is the cause of his toe walking, and that his dyxpraxia (fine motor incoordination) is also secondary to an upper motor neuron injury. Dr. Chen opines that Dekhari's mental retardation, and cognitive and motor deficits, are the result of brain injury during and immediately after birth, secondary to hypoxic-ischemic insult.

Dr. Bruce L. Halbridge avers that he has reviewed the prenatal care records, the labor and delivery chart, the newborn record, and the fetal heart monitoring strips. Dr. Halbridge opines that the "fetus was subjected to an ongoing hypoxic-ischemic insult for more than six hours (after

chorioamnionitis and fetal tachycardia were unmistakable) before [a] belated C-section delivery for more than eight hours, based upon the repetitive late and variable decelerations and arrest of descent and dilatation." "The cause of the variable decelerations (which began by 1:59 on May 12 and continued up until delivery at 8:57 on May 12) was umbilical cord compression, as uterine contractions exerted pressure on the cord wrapped twice around the fetus." The expert opines that this hypoxic-ischemic insult was sufficient to cause brain damage.

In **opposition** to plaintiff's Order to Show Cause the defendants point out that the affirmation of Dr. Chon Ken Chen does not comply with the requirements of CPLR 2106 and, therefore, should be disregarded (*see Offman v Singh,* 27 AD3d 284 [2006]). Defendants next argue that the plaintiff's attempt to ascribe the infant's condition to hypoxic-ischemic insult must fail for two reasons. First, "neonatal neurological syndrome" at birth or within the first hours or days of life is the *sine qua non* of a causal connection between brain injury and hypoxic-ischemic insult during labor. Defendants argue that, here, the infant did not experience a neonatal neurological syndrome at, or within, the first hours or days of life, but in fact had excellent Apgar scores of 9 and 9, had an uneventful course in the nursery, and developed normally until approximately eighteen months of age. Second, the defendants contend, the infant's condition — described by Dr. Chen as mental retardation, cognition deficits, dyspraxia and spasticity manifested in toe walking — are findings consistent with autism or could be due to numerous other causes other than upper motor neuron injury. Thus, defendants argue, the plaintiff cannot establish that the infant's condition was caused by a hypoxic-ischemic insult.

Defendants submit the affirmations of Dr. Andrew M. Steele who is board certified in

Pediatrics and Neonatal-Perinatal Medicine; Dr. Sandra Forem who is board certified in Pediatrics and Neurology; and Dr. Caren Jahre who is board certified in Radiology and Neuroradiology.

Dr. Steele avers that as a neonatologist he is familiar with the signs and symptoms of neonatal hypoxic-ischemic encephalopathy and that he has had extensive experience in diagnosing and treating newborn infants with this condition. Dr. Steele indicates that he reviewed, *inter alia,* the prenatal, emergency department, labor and delivery, newborn and pediatric clinic records and the fetal heart monitor tracings. Dr. Steele opines that Dekhari "did *not* sustain a hypoxic-ischemic insult sufficient to cause 'mental retardation and cognitive and motor deficits.'" Dr. Steel cites the American College of Obstetricians and Gynecologist and the American Academy of Pediatrics in *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenisis and Pathophysiology*, which states, in part:

> "It can be stated with certainty ... that the pathway from an intrapartum hypoxic-ischemic injury to subsequent cerebral palsy must progress through neonatal encephalopathy.
>
> . . .
>
> Research supports that spastic quadriplegia, especially with a movement disorder, is the only type of cerebral palsy associated with an acute interruption of blood supply. Purely dyskinetic or atoxic cerebral palsy, especially where there is an associated learning difficulty, commonly has a genetic origin and is not caused by intrapartum or peripartum asphyxia ....

Furthermore, notes Dr. Steele, the "essential criteria" for an intrapartum event sufficient to cause cerebral palsy are: (1) evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery; (2) early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks gestation; (3) cerebral palsy of the spastic quadriplegic or dyskinetic type; and (4)

exclusion of other identifiable etiologies such as trauma, coagulation disorder, infectious conditions, or genetic disorders. Dr. Steele opines that Dekhari did not suffer a neonatal encephalopathy, let alone severe or moderate encephalopathy, as required to ascribe his present neurodevelopmental impairments to intrapartum asphyxia.

Dr. Steele avers that the occurrence of neonatal neurological syndrome is a *sine qua non* "for attributing subsequent brain injury to intrapartum insult" and that three features must exist before concluding that intrapartum insult was the likely cause of neonatal brain injury. They are: (1) evidence of fetal distress,(2) depression at birth and (3) an overt neonatal neurological syndrome in the first hours and days of life. Dr. Steel notes that, here, while there is evidence of fetal distress, — *to wit,* meconium-stained amniotic fluid, — Dekhari was not depressed at birth and did not suffer overt neonatal neurologic syndrome. Rather, his Apgar scores were 9 and 9 at one and five minutes and, other than a single weak cry during a NICU examination, his neurologic examinations were repeatedly normal up to, and including, his discharge at three days. Thus, Dr. Steele concludes, there is no evidence that the infant suffered intrauterine asphyxia or a hypoxic-ischemic insult.

In her affirmation of June 27, 2013, pediatric neurologist Sandra L. Forem avers that at her examination of Dekhari, on May 12, 2010, he presented with "autism, macrocephaly, multiple skin lesions consistent with possible phakomatosis and a normal neuromuscular examination. There were no findings in [her] examination to suggest a hypoxic-ischemic etiology." Dr. Forem indicates that she performed a general physical exam (skin, heart, lungs, abdomen etc.) and a detailed neurological exam which included mental status, cranial nerves, sensory, motor reflexes and gait. Dekhari was noted to have complex motor stereotypes, absence of purposeful eye contact or social

referencing, dysarthric speech, echolalia, jargon and rare intermittent toe strike in the absence of spasticity. She noted that the extensive areas and patches of hypopigmented and hyperpigmented skin were suggestive of incontinentia pigmenti (phakomatosis), and that phakomatoses are a group of cogenital hereditary developmental anomalies having selective involvement of tissue of ectodermal origin, which include both skin and brain.

Dr. Forem avers that she has reviewed the report and affirmation of Dr. Chen, and that she agrees with Dr. Chen that "Dekhari is autistic, has pervasive developmental disorder and is mentally retarded." However, Dr. Forem found no evidence of spasticity on her examination, and opines that toe walking is common in autistics. Dr. Forem indicates that there "are various theories as to why this is so, including tactile hypersensitivity due to sensory dysfunction, a proprioceptive disorder and/or a vestibular-visual dysfunction."

Further, Dr. Forem indicates, her findings are inconsistent with a hypoxic-ischemic insult as the etiology of Dekhari's condition. These findings include: (1) a normal fundoscopic exam (no optic pallor); (2) normal neuromuscular exam, including intact strength, normal tone (lack of spasticity, dystonia or hypotonia) and normal bulk; (3) history of normal gross motor development; (4) normal deep tendon reflexes; and (5) macrocephaly with head circumference above the 98 percentile and greater than two standard deviations above the mean for a child of his age. Dr. Forem explains that macrocephaly, in the absence of hydrocephalius, is of particular significance because it is the opposite of what one would expect to find in the case of a significant hypoxic-ischemic encephalopathy, and that one would expect a child who has suffered a significant hypoxic-ischemic insult to develop microcephaly.

Neuroradiologist, Dr. Caren Jahre avers that she reviewed a CD disc containing the MRI of Dekhari's brain, performed on February 13, 2013. She opines that "the MRI reveals a solitary small focus of linear hyperintense signal on T2 Flair and T2-weighted images in the right parietal centrum semiovale white matter, which is likely a small focus of gilosis (scarring) which can occur surrounding a dilated perivascular space, perhaps from fluid leakage. This finding is not consistent with a hypoxic-ischemic insult to the brain sustained during labor and/or delivery." Dr. Caren agrees that there is enlargement of the perivascular spaces, which are normal fluid-containing spaces surrounding normal vessels, and which are usually *not* apparent on MRI except when enlarged. According to Dr. Caren, "[e]nlarged perivascular spaces increase in prevalence with age and are uncommon in children. However, their prevalence has also been reported to be increased in children with autism. They are not due to a hypoxic-ischemic insult during labor or delivery." Finally, Dr. Caren notes that she was not provided with the report of the radiologist who supervised the MRI study.

In **reply** plaintiff argues that the issue in this case, — *to wit,* whether the infant has brain injury and whether he was exposed to a hypoxic-ischemic insult capable of causing brain injury, — are not issues that are properly resolved by a *Frye* hearing. Plaintiff argues that the defendants are free to challenge, at trial, the testimony of plaintiff's neuroradiologist, Dr. Lawler, that the "asymmetric abnormal increased linear signal within the right parietal lobe periatrial/periventricular white matter, on the MRI of the infant's brain, was likely due to prior insult to the brain parenchyma." The plaintiff argues that plaintiff will only seek to establish that the infant plaintiff suffered brain damage as a result of perinatal hypoxic-ischemic insult.

DISCUSSION

"The requirement that a motion for leave to renew be based upon newly-discovered facts is a flexible one, and a court, in its discretion, may grant renewal upon facts known [or which should have been known] to the moving party at the time of the original motion [*see Daniel Perla Assocs. v Ginsberg,* 256 A.D.2d 30 ...]" (*Gadson v New York City Housing Authority,* 263 AD2d 464 [1999]). Under the circumstances herein, renewal is granted.

In opposition to the defendants' Order to Show Cause which requested, *inter alia,* a *Frye* hearing, the plaintiff argued that the neurological disorder which is the source of the infant's autistic disorder is the result of pre, peri and/or post natal hypoxic-ischemic insult. More specifically, plaintiff's expert pediatrician, Dr. Chen, opined that it is generally accepted in the medical research community that hypoxic-ischemic brain injury during labor and delivery is one of the causes of autistic disorder. Dr. Chen stated: "It is my opinion, to a reasonable degree of medical certainty, that Dekhari Sheley suffered a hypoxic-ischemic insult in utero, and that insult substantially contributed to Dekhari Sheley's autistic disorder."

However, in support of the instant Order to Show Cause, plaintiff now maintains that she will argue at trial that rather than, or in addition to, autism the infant has brain damage caused by hypoxic-ischemic encephalopathy, caused by the defendants' negligence, and resulting in cognitive and motor deficits, and mental retardation. In this regard, counsel for plaintiff avers that "[p]laintiff will stipulate *not* to attempt to prove that what the defendants characterize as Dekhari's PDD/Autistic Disorder/Autism was caused by hypoxic-ischemic encephalopathy ("HIE"); but instead plaintiffs will prove (i) that the infant has brain damage caused by HIE, and (ii) he has

cognitive and motor deficits and mental retardation that are the result of brain damage. Counsel avers that an MRI of the infant's brain confirms that he suffered brain damage secondary to ... hypoxic-ischemic insult" and that "[i]t therefore matters not whether the plaintiff has ... PDD/Autism/Autistic Disorder ... and further, it does not matter whether the PDD/Autism/Autistic Disorder was caused by HIE or exists independently and is caused by something other than HIE." Because the plaintiff has abandoned her claim of a causal link between perinatal hypoxic-ischemic insult and the PDD/Autistic Disorder/Autism, with which the infant has been diagnosed, there is no need to apply the *Frye* test (*see Lugo v New York City Health and Hosp. Corp.*, 89 AD3d 42, [2011]; *Ratner v McNeil-PPC, Inc.*, 91 AD3d 63 [2011]).

The parties do not dispute that the existence of a causal link between perinatal hypoxic-ischemic insult and brain injury has broad acceptance in the medical and research community (see e.g., Jeffrey M. Perlman, *Summary Proceedings From the Neurology Group on Hypoxic-Ischemic Encephalopathy,* 117 Pediatrics 528-533 [2006]; Robert Vannucci, *Hypoxic-Ischemic Encephalopathy,* 17 American Journal of Perinatology, 11-120 [2002]; Joseph J. Volpe, *Neurology of the Newborn,* 401 [5th ed, 2008]; American College of Obstetricians and Gynecologist, Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology [2002]; Joseph J. Volpe, M.D. Neurology of the Newborn, 331-332 [4th ed. 2001]; *see also Metsaris v 73rd Corp.*, 105 AD2d 67, 83 [1984]; *Brucato v Pennington,* 128 AD2d 823 [1987]; *Scheuerman v Health and Hospitals Corp. of City of New York,* 243 AD2d 553 [1997]; *Bermeo v Atakent,* 241 AD2d 235 [1998]; *Gong v Gjoni,* 294 AD2d 648 [2002]; *James v Corwin,* 19 AD3d 336 [2005]; *Fernandez v Moskowitz,* 85 AD3d 566 [2011]; *Lugo v New York City Health and Hospitals Corp.*, 89 AD3d

42, 62 [2011]). Rather, the defendants argue that plaintiff cannot establish specific causation – *to wit,* that the infant suffered an hypoxic-ischemic insult or brain damage caused by hypoxic-ischemic insult. However, these are now questions to be properly placed before a jury, the defendants having failed to raise these issues in a timely motion for summary judgment (CPLR 3212; *Fernandez v Moscowitz, M.D.,* 85 AD3d 566, 567-568 [2011]; *Fritz v Burman,* 107 AD3d 936 [2013]).

Moreover, the fact that the infant's condition may have causes other than perinatal hypoxic/ischemic insult does not preclude a finding of injury by such other cause(s). A plaintiff is not obligated to eliminate all possibility that the injuries resulted from causes other than a defendant's negligence (*Oakes v Patel,* 20 NY3d 633, 647 [2013]; *Ledogar v Giordano,* 122 AD2d 834, 837 [1986]). Accordingly, it is

ORDERED, that plaintiff's Order to Show Cause (#5) is granted to the extent that renewal is granted and upon renewal the court's order of July 20, 2012, which granted a *Frye* hearing, is vacated and the plaintiff's Order to Show Cause is otherwise denied; and it is further

ORDERED, that the defendants' Order to Show Cause (#4) is denied; and it is further

ORDERED, that the attorneys for the parties appear in Part 2 of this Court on February 24, 2014 at 9:30 A.M. for jury selection and trial.

E N T E R,

J.S.C. HON GLORIA DABIRI

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