.

OFFICE OF SPECIAL MASTERS

No. 93-689V

June 5, 2000

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MARIA S. ROOKS, as Legal Representative of RANDALL TYLER ROOKS	*	
Petitioner,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	
* *		

Sylvia Chin-Caplan, Boston, MA, for petitioner.
Gerard W. Fischer, Washington, DC, for respondent.

DECISION

MILLMAN, Special Master

Statement of the Case

Petitioner filed a petition on November 8, 1993 under the National Childhood Vaccine Injury Act (hereinafter, "the Vaccine Act" or "the Act"), 42 U.S.C. § 300aa-1 et seq., alleging that her son, Randall Tyler Rooks (hereinafter, "Tyler") was injured in utero when his mother received a measles, mumps, rubella vaccination while pregnant with him. Subsequently, petitioner amended her allegation to reflect that she received solely a measles vaccination while pregnant with Tyler, who

was born with cerebral dysgenesis, agenesis of the corpus callosum, hypospadias, and cleft palate, and currently suffers from severe developmental delay, visual problems, and seizures.

On August 22, 1995, the undersigned dismissed this case for failure to make a prima facie case because the undersigned held that Tyler was not a recipient of a vaccine under the Act. 42 U.S.C. § 300aa-11(c)(1)(A). On January 29, 1996, the Honorable Moody R. Tidwell issued an Order vacating the undersigned's decision and remanding the case for further proceedings consistent with his opinion. In that opinion, he held that the purpose of the Act was to compensate vaccine injuries and that petitioner should be given the opportunity to prove that Tyler had vaccine injuries.

A hearing was held on March 20, 2000. Testifying for petitioner were Dr. Paul Maertens and Maria Rooks. Testifying for respondent were Dr. John L. Sever and Dr. Robert L. Brent.

FACTS

Maria S. Rooks received rubeola (measles) vaccine on November 9, 1990. This was her second dose. P. Ex. 8, p. 2. Tyler was born on July 12, 1991. P. Ex. 8, p. 17. He had a cleft palate, questionable intersex problem, absent corpus callosum, hypospadias with an ambiguous genitalia, and a very small mandible. P. Ex. 11, p. 11.

TESTIMONY

Dr. Paul Maertens, a board-certified pediatric neurologist, testified first for petitioner. Tr. at 5-6. He initially saw Tyler in April 1992 when Tyler was nine months old. Tr. at 8. He had prominent occipital horns and seizures. Tr. at 9. Dr. Maertens started Tyler on anti-convulsants. *Id.* Mrs. Rooks had been vaccinated when she was two weeks pregnant. Tr. at 10. Dr. Maertens testified that one should avoid vaccination when pregnant especially during the first three months, when the fetus is at high risk. *Id.* Dr. Maertens stated that the risk of vaccinating a pregnant woman

is that the live virus can infect the baby by crossing the placenta, replicating in the fetus, and causing damage. Tr. at 11.

The time of vaccination is very important. Tr. at 20. Mrs. Rooks was at a very early stage in her pregnancy when she received the vaccine. Tr. at 22. Any of the malformations Tyler has occurred at this time. Tr. at 20. Organogenesis, when the organs are created, occurs during the first three months of gestation. Tr. at 21-22. It is a critical time period. Tr. at 22. Measles can cause birth defects. Tr. at 23. The only paper available, by Jesperson, concerns the disease measles, not the vaccine, and the type of birth defects resulting from it. *Id.* Nine percent of fetuses exposed to measles virus in their first three months had birth defects. *Id.* This was a retrospective study and thus underestimates the incidence. The risk is inversely proportional to the gestational age. Tr. at 24.

Dr. Maertens testified that the measles vaccine could have caused Tyler's injuries because it was administered just at the time when the corpus callosum, palate, and genital organs were formed. Tr. at 25. He stated that the vaccine did cause Tyler's injuries. *Id.* Tyler had neuromigrational defects because the grey matter did not go where it should have. Tr. at 27. Neuromigration occurs by 18 weeks. Tr. at 28. Dr. Maertens testified that measles virus got into different cells. Tr. at 28-29.

Mrs. Rooks testified briefly at this point that she remembered receiving measles vaccine from the Public Health School in order to stay in college, but did not recall feeling ill. Tr. at 30-31. This was her second measles vaccination. Tr. at 33.

Dr. Maertens stated that the second vaccination should bring immunization to 100 percent. Tr. at 36. The basis of his opinion of causation is: that measles is a live virus; in the wild, it causes birth defects similar to Tyler's; rubella (German measles) virus causes similar defects; early

exposure increases risk at the highest level; and all of the manifestations of abnormality occurred at the time of vaccination. Tr. at 37. DNA viruses work on the structure of cells and incorporate their own genome in the cells. Tr. at 40. Measles is not a mild virus. Tr. at 41. It tends to cause severe delayed problems in the brain. *Id.*

On cross-examination, Dr. Maertens conceded that there are no epidemiological studies relating measles vaccine to birth defects. Tr. at 47. The Physicians Desk Reference (PDR) does not recommend giving vaccinations to pregnant women. Tr. at 48. It would be unethical. *Id.* Therefore, such a study would never be available. *Id.*

Tyler's family does not have a history of similar birth defects. Tr. at 52. There was no gestational diabetes, no eclampsia, no intake of drugs such as Thalidomide, no chromosomal defect, and no amniotic band syndrome. *Id.* The doctors did not test Tyler for metabolic problems, but ruled out other syndromes. Tr. at 51. The only cause left is measles vaccine. Tr. at 52. The danger of malformation is quite low with measles virus. Tr. at 53. Timing is essential here. *Id.* It was extremely early in Mrs. Rooks' pregnancy, increasing the risk of defects. *Id.* Tyler's susceptibility was highest to the virus' intruding into some cells, causing damage, and interfering with organogenesis. Tr. at 54.

The number of malformed live births is underestimated in the United States. Tr. at 55. Four to eight percent of live births have some malformations. *Id.* There is no syndrome other than SSPE (subacute sclerosing panencephalitis) of which Dr. Maertens is aware that is related to measles vaccine. Tr. at 56. There is no measles virus syndrome in fetuses. *Id.*

Dr. John Sever, a board-certified pediatrician who also has a Ph.D. in microbiology and immunology, testified first for respondent. Tr. at 66. He has administered vaccines in the past. Tr. at 68. Live virus vaccines are contraindicated for early pregnancy. Tr. at 69. There is no data as

to their safety and they have the potential for adverse effects. Tr. at 70. Rubella can cause defects in the fetus in the first three months of gestation. *Id.* Doctors withhold mumps and rubeola vaccines because of the possibility of risk. *Id.*

Dr. Sever's opinion is that measles vaccine had no relationship to Tyler's birth anomalies. Tr. at 73. This was Mrs. Rooks' second dose of measles vaccine. *Id.* A large body of data regarding measles vaccines shows that 95 to 97 percent of vaccinees receive good antibody response and are protected from measles. *Id.* A second dose would not produce anything. It would only boost antibody levels already present. Tr. at 74. Only the occasional person has no immunity. *Id.*

Dr. Sever looked at medical literature on measles' effect on pregnant woman, his own experience with measles vaccine, 330 VAERS reports on measles vaccine, and the virology of measles virus itself (i.e., the ability of the virus to cross the placenta) in forming his opinion in this case. Tr. at 74-75.

There are several case reports of different abnormalities among pregnant women who gave birth after having the measles disease: hairlip, protruding eye, heart defects. Tr. at 75. In Oklahoma, 24 pregnant women had no children with abnormalities. Tr. at 75-76. In Greenland, there was no pattern of abnormality, but one child had atypical leukodystrophy. Tr. at 76. In South Australia, out of 18 women, one had a child with partial deafness and another had one with Down's syndrome. *Id.* In New York, among 60 women, one had a deaf child. *Id.* In Israel, one child was stillborn, and one had hydrocephalus where the mother contracted measles in the third trimester. *Id.* There is no consistent pattern of abnormalities here. Tr. at 77. There is no medical literature dealing with measles vaccine's effects on fetuses. Tr. at 77.

Dr. Sever has published 100 papers on rubella as an infection in pregnancy. Tr. at 78. Both rubella (German measles) and rubeola (measles) are paramyxoviruses. *Id.* They come from the same general group of viruses but differ in genetic make-up and the symptoms they produce. *Id.* Rubella virus has an effect on the fetus. Tr. at 79. It causes severe damage if contracted in the first few months of gestation: central nervous system damage, cataracts, heart malformation, and deafness. *Id.*

There is no similar congenital measles syndrome in fetuses.[1] Tr. at 80. There is no consistent pattern of damage, increased rate of abortions, or stillbirths with measles as there is with German measles. Tr. at 80-81. There are only sporadic, unrelated instances of abnormality. Tr. at 82. One needs to have a syndrome in order to opine that there is causation. Tr. at 83.

About three to four percent of babies born have a birth defect, in which the cause is known for about 50 percent or less. Tr. at 84. There are 330 VAERS reports, of which five allege fetal injury from measles vaccine (one of them being the Rooks case). *Id.*

The virus needs to cross the placenta in order for the vaccine to damage the fetus. Tr. at 87. Moiri studied the placenta of a dead baby. Tr. at 87-88. This is the only example of measles virus exposed to the fetus we have. Tr. at 89. There was antigen to the measles virus in the placenta but not in the fetal organs, which indicates that the virus did not cross the placenta. Tr. at 87-88. Rubella, on the other hand, crosses the placenta. Tr. at 88.

Measles vaccine is specifically weakened and is less likely to produce damage. Tr. at 94. Because this was Mrs. Rooks' second dose of measles vaccine, she was likely already immune to measles. Tr. at 95. The vaccine is not expected to cause infection in Mrs. Rooks because it has little

[1] A syndrome means a group of clinical findings occurring with this infection.

replication. *Id.* She might get a little boost in her immunity, but there is little likelihood that the measles vaccine would get to the placenta. Tr. at 95-96. It would just go to her lymph nodes and out of her body. Tr. at 96.

There is no basis to associate measles vaccine or measles to agenesis of the corpus callosum, hypoplasia of the cerebellar vermis, and neuromigrational defects. Tr. at 99-100. The medical literature describes quite different abnormalities. Tr. at 114. Therefore, they are not dominant etiologic factors. *Id.*

The period of gestation when a fetus encounters a teratogen is important and determines the type of defect he will have. Tr. at 115. When babies are born premature or have low birth weights, something is wrong with the placenta. Tr. at 117. Tyler's delivery had no effect on his congenital defects. *Id.* If Tyler were Dr. Sever's patient and Tyler presented with a history of 17-day gestation measles vaccine, Dr. Sever would not rule out measles vaccine as a theoretical problem, but he would not rule it in either. Tr. at 126. He would have reported Tyler's case to the Centers for Disease Control. *Id.* Dr. Sever stated there is a possible, but remote, relationship between the measles vaccine and Tyler's problems. Tr. at 127. This is a live virus vaccine and we do not know what its effects are and we do not recommend administering it in pregnancy. Tr. at 127-28.

Dr. Robert L. Brent, a pediatrician, pathologist, and radiologist, testified next for respondent. Tr. at 135. He has been chairman of the department of pediatrics at the duPont Hospital for Children for 30 years. Tr. at 136. He has spent the last 45 years doing research to prevent and treat causes of congenital malformation and has a doctorate in embryology. *Id.* He was the first fellow in the March of Dimes in birth defects. *Id.* He founded the Teratology Society in 1959-60 with Dr. Sever. *Id.* He was editor of the *Birth Defect Journal* and is now associate editor. Tr. at 137. He is board-certified in pediatrics. Tr. at 138.

Dr. Brent stated that measles vaccine did not cause Tyler's congenital malformations. Tr. at 139. There are five requirements in teratology to link a virus to a defect: (1) the epidemiology must report a consistent teratological effect; (2) secular trend analysis; (3) animal studies; (4) the dose response curve; and (5) biological plausibility. Tr. at 140-50.

First, the epidemiology should define a group of malformations. Tr. at 140. One looks at the spectrum of malformations. *Id.* There are 45 causes of malformations, including infections, parasites, bacteria, and viruses. *Id.* Although Dr. Maertens inferred one would need hundreds of cases to posit a causative relationship, this is not true. Tr. at 141. In 1939, Dr. Greg described congenital rubella syndrome from just seven cases. The nature of teratogenic syndromes involves a narrow spectrum of malformation. Tr. at 141. With measles vaccine, we have meager data and no spectrum of malformations. *Id.* Birth defects occur in four to eight percent of births, or 40 to 80 births out of 1,000. Tr. at 142.

The Congenital Malformation Branch of the CDC monitors pregnancies and, therefore, we have a good idea of birth defects. Tr. at 145. Thirty out of every 1,000 have severe defects. Tr. at 146. Another 30 have less severe congenital malformations. *Id.* Animal studies are not helpful in studying viruses. Tr. at 147. Dr. Brent would not vaccinate a pregnant woman with measles vaccine, but he would not recommend abortion if she were vaccinated. *Id.*

Second, a secular trend analysis could be done if there was a large segment of a population of pregnant women exposed to something. Tr. at 148. But this is not possible with measles vaccine because not enough pregnant women are exposed to it, and so there is not enough data to find a secular trend of birth defects. Tr. at 149.

Third, animal studies are not helpful because measles virus is species specific. *Id.*

Fourth, a dose response curve is not useful with vaccine virus or live virus because virus replicates and there is no real dose. *Id.*

Fifth, although biological plausibility or common sense tells us measles is a serious illness and measles vaccine has made a tremendous difference in morbidity and mortality, we know that women with severe measles lose pregnancies, but it has nothing to do with the virus but with their being sick while pregnant. Tr. at 150. When pregnant mothers sick with measles do give birth prematurely, there is no growth retardation. Tr. at 151. This indicates that the virus has not come across the placenta and injured the fetus. *Id.* The prematurity was caused because the mother was very sick. *Id.*

No syndrome such as agenesis of the corpus callosum has been recorded in 300 adverse reaction reports. Tr. at 151-52. The vaccine does not produce a recognizable syndrome. *Id.* Teratogens have great specificity. Tr. at 152. Rubella (German measles) has a spectrum of malformations. *Id.* Wild measles virus does not have specificity in malformations nor is there any data on its crossing the placenta. *Id.*

Mrs. Rooks received measles vaccine on the 17th day of pregnancy, but cleft palate and hypospadias (which Tyler has) are late malformations. Tr. at 153. His cerebellum has heterotopias, which are a known teratogenic effect, but they are also late and have nothing to do with a 17th-day exposure. *Id.* If measles virus crossed the placenta on the 17th day of development, the mostly likely occurrence would be that the fetus would die. Tr. at 154. Organogenesis begins on the 18th to 20th day. *Id.* Since Mrs. Rooks had already been immunized with measles vaccine, she probably was immune to measles. *Id.* Her second measles vaccination was very unlikely to produce significant viremia (i.e., virus circulating in her blood), and, therefore, very unlikely to cross the placenta. Tr. at 154 & 56. We do not even have evidence that wild measles virus crosses the placenta. Tr. at 154.

We should not rule out a genetic cause for Tyler's defects because the cause of 50 percent of birth defects is unknown. Tr. 155. There are 17 hereditary diseases that cause agenesis of the corpus callosum. Tr. at 154-55.

Dr. Brent expected that if Tyler were exposed to measles in utero, nothing would have occurred. Tr. at 156. There is no evidence that wild measles caused Tyler's congenital defects. *Id.* The cerebellum and the cortex are not present on the 17th day. Tr. at 157. It is an all or none period when the fetus is most susceptible to lethal effects of a toxic agent. *Id.* If the virus were there, it would be there for weeks or months. *Id.*

Tyler's syndactyly (the fingers are not completely separated) is inherited. Tr. at 158. His cleft palate and hypospadias are genetic although they could be teratogenic. *Id.* His agenesis of the corpus callosum is genetic and not teratogenic. *Id.* Tyler's syndrome is very rare. *Id.*

Dr. Brent has taught medical students for 45 years. Tr. at 160. He can take two, three, and four features and describe the agent causing a birth defect by using the concept of a syndrome. Tr. at 161. Measles vaccine does not have a syndrome or constellation of features pointing to one etiology. *Id.* If virus is in the cell, there is no time window. Tr. at 162. It could be in the embryo for weeks or months. *Id.* Forty-seven new genes have been isolated this year. Tr at 163. Dr. Brent does not know the cause of Tyler's birth defects, but stated that it was not measles vaccine. Tr. at 166.

Mrs. Rooks' immune system would have destroyed the live virus after vaccination. *Id.* Moreover, there is no placenta or fetus at the 17th day. Tr. at 173. Tyler was an embryo. *Id.* An embryo has three germ layers: the beginning of an amnion, a yolk sac, and the embryo. *Id.* It is in direct contact with the mother. *Id.* The blood vessels are the maternal barrier, but there is no

umbilical cord. Tr. at 173-74. Measles virus would reach the embryo in 24 hours if it were replicating in the mother's blood stream. Tr. at 175.

At the day of conception, there are two cells. Tr. at 176. At four days, there are 16-32 cells. *Id.* At five and a half days, it is a blastocoel. *Id.* On the 17th day, there are three germ layers. *Id.* On the 28th day, the central nervous system is closed. *Id.* The fetus is insensitive to destructive effects on the brain up to the 28th day because it has great recuperative powers. Tr. at 177-78. The main time to produce mental retardation from a teratogen is from the 8th to the 15th week, when those recuperative powers no longer exist. Tr. at 178.

Measles virus cells are small. Tr. at 180. The virus cell in an adult lasts from eight to ten days. *Id.* In a child, measles virus vaccine is given late (in the second year) because before then the child does not have a good response. Tr. at 181. A newborn is immunologically immature. *Id.* Dr. Sever opined at this point that the measles vaccine virus in a child would disappear in a few days. Tr. at 183-84. Dr. Brent stated that the embryo's sensitive period for organogenesis is from the 18th to the 40th day. Tr. at 185. Fifteen percent of early pregnancies are spontaneously aborted, and 70 percent of them have chromosomal abnormalities. Tr. at 192.

Dr. Brent gives consultations on birth defects around the world, particularly in the context drug testing. Tr. at 187. Drug companies send him animal studies to determine if there is a risk for humans. *Id.*

Submitted Material

Petitioner submitted medical articles or chapters, filed June 30, 1997, the first of which (P. Ex. A) is ""Chickenpox, Measles and Mumps," by A.A. Gershon.[2] The author refers to measles

[2] In *Infectious Disease of the Fetus and Newborn Infant*, 4th ed., by J.S. Remington and J.O. Klein (1995) 599-602.

vaccine administered to seven pregnant women who were in their 2nd to 8th month of pregnancy. All delivered healthy babies at term. Id. at 601.

Petitioner's Ex. B is "Measles Vaccination in Adults With and Without Complicating Conditions" by M. Gudnadottir and F.L. Black.[3] They describe seven Icelandic pregnant women who were vaccinated and delivered healthy babies. Id. at 522.

Petitioner's Ex. C deals with German measles (rubella).[4] If a pregnant woman receives rubella vaccine, the Academy does not recommend abortion because none of the 226 pregnant women who received rubella vaccine during the first trimester delivered a baby with congenital defects. Id. at 410-11.

Petitioner's Ex. D is "An Epidemic of Measles in Southern Greenland, 1951" by P.E. Christensen, et al.[5] (This is also respondent's Ex. G.) Eighty-three pregnant women, most of whom had passed through the first half of their pregnancies, contracted measles. Six women had premature delivery, seven had abortions in the third to fifth months, thirteen had delivery at term. Of the children born at term, two died on the first day, and three premature children died. No congenital measles was observed. Four of the women died. The authors opined that measles contracted during the first three months of pregnancy did not seem to have a harmful influence on the fetus. Id. at 447. No congenital abnormalities were seen in the children born to mothers who contracted measles during pregnancy. Id. at 448.

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[3] *Arch Ges Virusfordsch* 16:521-23 (1965).

[4] American Academy of Pediatrics, Red Book, 406-12 (1995).

[5] *Acta Med Scand* 144:430-49 (1953).

Petitioner's Ex. E is "Low Birth Weight and Maternal Virus Diseases. A Prospective Study of Rubella, Measles, Mumps, Chickenpox, and Hepatitis" by M. Siegel and H.T. Fuerst.[6] The article focuses on low-birth weight when mothers contracted viruses. They found an increase in major congenital defects only in cases of rubella occurring in the first 16 weeks of gestation. In other virus groups, there was no increase above control levels. Id. at 682. In measles, the ill effects were limited mainly to prematurity, and not to any significant increase in fetal deaths. Id. at 683. Except for rubella, evidence of viral infection of the fetus has rarely been found in the other diseases. Id. In the absence of direct infection of the fetus, authors have implicated high fever, anoxia, metabolic changes, and toxic products produced in the course of the mother's disease. But their precise effects on gestation and the fetus have not been defined. Id.

Petitioner's Ex. F is "The Effect of Maternal Measles on the Fetus" by E. Gazala, et al.[7] (This is also respondent's Ex. K.) Five mothers with measles gave birth. One child was stillborn with severe malformations (hydrocephalus, single ventricle, cleft lip and palate). None of the babies showed any clinical signs of measles. Serum samples were taken from the infants at birth and 2 to 4 weeks later. No specific measles IgG antibodies were detected. All the infants had low birth weight and three had severe respiratory distress syndrome requiring assisted ventilation. The authors explain the lack of measles in the babies by stating that transplacental transfer of IgG occurs mainly in the last trimester, and premature infants have decreased IgG tiers compared with term babies. Id. at 203. They conclude that maternal measles may lead to premature birth.

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[6] *JAMA* 197:680-84 (1966).

[7] *Ped Infect Dis* 4:203-04 (1985)

Petitioner submitted additional exhibits marked A, B, and C, filed June 1, 1998, which are different than the above and are attached to Dr. Maertens' report. Petitioner's Ex. A is a chapter Dr. Maertens co-authored with Paul R. Dyken entitled, "Viral Infections."[8] The authors state that the "constellation of multiple organ system malformations (teratogenicity) depends on the virulence of the virus on fetal tissues." Id. at 425. They state in a Table (19-14) that malformation syndromes are rare in measles. Id.

Petitioner's Ex. B is "Measles as a Cause of Fetal Defects. A Retrospective Study of Ten Measles Epidemics in Greenland" by C.S. Jespersen, et al.[9] (This is also respondent's Ex. H.) In a study of pregnant women infected with measles in the first trimester, the risk of fetal death was found to be high. Twenty-eight women infected in the first two months of pregnancy had live children, but four had congenital malformations, three of extreme rarity and severity, leading to death. Id. at 367. Eight cases of gross congenital malformations were diagnosed among 300 liveborn infants. Id. at 369. Out of 58 babies whose mothers were infected with measles in the first trimester, five babies or nine percent had congenital malformations. Id. The authors considered measles to be the dominant etiologic factor in these cases of congenital malformations. Id. at 371.

Respondent's Ex. C is a summary of the VAERS reports of fetal distress or congenital anomalies after receipt of measles vaccine by pregnant women as of August 28, 1997. Of the five reported cases, one was the instant case. One turned out not to be pregnant. One had the vaccination two weeks after conception and gave birth to a healthy child with a small growth in front of the ear.

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[8] In *Pediatric Neuropathology*, chapter 19, 396-434.

[9] *Acta Pediatr Scand* 66:367-72 (1977).

Another had vaccination two weeks after conception and gave birth by Caesarean section because of fetal distress to a child without congenital abnormalities. The fifth, who received measles vaccine three months before conception, had a spontaneous abortion. The fetus had unspecified congenital abnormalities on autopsy.

Respondent's Ex. D is "The Relationship of German Measles During Pregnancy to Congenital Ocular Defects" by B. Rones.[10] Of the four cases the author discusses, the fourth concerns measles which resulted in a baby born with congenital buphthalmos.[11] The mother contracted a severe case of measles in the last week of her third month of pregnancy, broke out in a severe rash, and stayed in bed seven to eight days with a high temperature. Id. at 286.

Respondent's Ex. E is "Final Observations on Congenital Defects in Infants Following Infectious Diseases During Pregnancy, with Special Reference to Rubella" by C. Swan, et al.[12] In the fourth series of cases the authors studied, three, and possibly four, of the mothers had measles. Id. at 896, 897. The mother with either measles or rubella who had been pregnant for seven weeks when she became ill gave birth to a child with microcephaly. Another mother, who had measles at six weeks of gestation gave birth to a normal child as did a mother with measles at four to five months gestation. The fourth mother had measles at two and a half to three months gestation and gave birth to a baby with heart disease. Id. at 897, 900. There were 18 measles cases among pregnant women in South Australia, resulting in one infected mother at the first month (with a

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[10] *Med Ann D.C.* 13:285-87 (1944).

[11] Buphthalmos is "enlargement and distention of the fibrous coats of the eye; hydrophthalmos; infantile glaucoma." Dorland's Illustrated Medical Dictionary, 27th ed. (1988) at 244.

[12] *Med J Australia* Vol. II, No. 26:889-908 (1946).

normal baby), two infected mothers in the second month (with normal babies), four infected mothers in the third month (with two abnormal babies), three infected mothers in the fourth month (with two abnormal babies) and no abnormal babies for those mothers infected in the remaining months. Id. at 901. The authors state, "With regard to the effect on the fetus of other infectious diseases during pregnancy, there is little evidence as yet to suggest that morbilli [measles] leads to congenital abnormalities, though it is possible that it may be a cause of abortion." Id. at 907.

Respondent's Ex. F is "Measles Complicating Pregnancy. Report of Twenty-Four Cases with Three Instances of Congenital Measles" by I. Dyer.[13] None of the 24 babies was born with congenital abnormalities. Id. at 602.

Respondent's Ex. I is "The Influence of Maternal Measles (Morbilli) on the Unborn Child" by A.D. Packer.[14] The author looked at 11,000 cases of measles in South Australia, among which were 18 cases of pregnancy. The majority of the babies were unaffected by the maternal measles. Id. at 836. There were two congenitally abnormal babies, one with mongoloidism and the other with partial deafness, born to mothers who had measles in the 5th and 8th weeks of gestation, respectively. The author states that the organogenetic period in humans during which various organs are formed lasts from the 4th to the 10th week of pregnancy. Id. The author opines that measles is "in no way comparable in importance to rubella as a teratogenic agent in humans." Id. at 837.

Respondent's Ex. J is "Congenital Malformations Following Chickenpox, Measles, Mumps, and Hepatitis. Results of a Cohort Study" by M. Siegel.[15] Out of 60 pregnant women with measles

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[13] *South Med J* 33:601-04 (1940).

[14] *Med J Australia*1:835-38 (1950).

[15] *JAMA* 226:1521- 24 (1973).

and 62 pregnant women without measles, each group had one baby born with congenital malformations Id. at 1522. There was no apparent difference. The abnormal child born to the mother who had measles was bilaterally deaf; the abnormal child born to the control mother had mental retardation. Id. at 1523. In two large studies, the author states, a causal relationship could not be established. Id.

Respondent's Ex. P is "Measles in Pregnancy" by M. Grandien and G. Sterner.[16] The authors state that measles during early pregnancy has not been firmly associated with abnormalities of the newborn and should not be a reason for abortion. Referring to an analysis of ten epidemics of measles in Greenland, the authors refer to 28 women infected in the first two months of pregnancy, and the birth of four children with congenital malformations. However, there was no particular constellation of abnormalities found. Newborns of measles-immune women were themselves immune to measles. Id. at 45. When inadvertently administered to pregnant women, measles vaccine has not been reported to damage the fetus. Id. at 46.

Respondent's Ex. Q is "Risk of Pregnancy among Adolescent Schoolgirls Participating in a Measles Mass Immunization Program" by J.M. Mann, et al.[17] Of 1,913 female vaccinees, two were pregnant at the time of vaccination and four became pregnant within three months after vaccination. One of the two already pregnant spontaneously aborted, and the other five delivered healthy infants. Id. at 528. The authors state, "There is no evidence to support teratogenicity or other adverse fetal effects of measles vaccine." Id. at 529.

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[16] *Scan J Infect Dis*, Suppl. 71:45-48 (1990).

[17] *Amer J Pub Health* 73:527-29 (1983).

Respondent's Ex. R is "Rougeole et grossesses. A propos de 16 cas, au Burkina Faso" by B. Dao, et al.[18] The authors describe an epidemic of 16 cases of measles hospitalized during pregnancy. Two had abortions, three had stillbirths, one delivered prematurely, and two had full-term births. Eight ongoing pregnancies were unable to be followed up after discharge from the hospital. Id. at 606.

DISCUSSION

Petitioner is proceeding on a theory of causation in fact. To satisfy her burden of proving causation in fact, petitioner must offer "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury. A reputable medical or scientific explanation must support this logical sequence of cause and effect." Grant v. Secretary, HHS, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Agarwsal v. Secretary, HHS, 33 Fed. Cl. 482, 487 (1995); see also Knudsen v. Secretary, HHS, 35 F.3d 543, 548 (Fed. Cir. 1994); Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993).

Without more, "evidence showing an absence of other causes does not meet petitioners' affirmative duty to show actual or legal causation." Grant, supra, 956 F.2d at 1149. Petitioner must not only show that but for the vaccine Tyler would not have had the injury, but also that the vaccine was a substantial factor in bringing about his injury. Shyface v. Secretary, HHS, 165 F.3d 1344 (Fed. Cir. 1999).

At first blush, Tyler's congenital abnormalities seem to fit quite nicely in Dr. Maertens' theory that Mrs. Rooks' second measles vaccination caused his illness. But further examination of his theory, and evaluation of the testimony and credentials of respondent's experts Dr. Sever and Dr.

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[18] *J Gynecol Obstet Biol Reprod* 26:606-09 (1997).

Brent as well as the medical literature, cast doubt on that first impression. To determine which experts are more credible, an examination of their training and background is useful.

Dr. Maertens is a pediatric neurologist who also treated Tyler. His subspecialty is neurosonography. P. Ex. C, p. 7. He has written 25 articles and 10 book chapters on topics ranging from MRI of the brain to inborn errors of metabolism, and one chapter on viral infections in a text entitled The Pathology of the Developing Human Nervous System. Id. at 14.

Dr. Sever is a pediatrician who specializes in microbiology and immunology. R. Ex. O, p. 5. In 1996, he gave a lecture at Sibley Memorial Hospital on "Prenatal Evaluation of Viral Exposure." He has a 37-page list of articles, and has edited 11 books on topics such as: clinical diagnostic immunology, infectious disease immunology (including viruses), perinatal infections, infectious complications of pregnancy, retroviruses in the nervous system, and environmentally induced birth defect risks. He has investigated the use of live rubella as well as rubella vaccine. Id. at 7, 8. His research interests are pediatrics, virology, epidemiology, infectious diseases, perinatal infections, chronic neurological diseases, and environmental studies. Id. at 8. He has written articles on rubella, measles, viruses and pregnancy, teratogens, vaccine safety, viral causes of genetic defects, and viral infections and malformations, totaling 598 articles.

Dr. Sever has been President of the Teratology Society (1976-77), President of the Association of Medical Laboratory Immunologists (1993-94), President of the Infectious Diseases Society for Obstetrics and Gynecology (1992-94), and President of the Pan American Society for Clinical Virology (1992-94). He received an award for outstanding contributions to the field of infectious diseases from the Infectious Diseases Society for Obstetrics and Gynecologists in 1998. He is or was a member of the editorial boards of the following journals: *Clinical and Diagnostic*

Laboratory Immunology, Infectious Diseases in Obstetrics and Gynecology, Journal of Clinical and Diagnostic Virology, Pediatric AIDS and HIV Infection, Molecular and Cellular Probes, American Journal of Reproductive Immunology and Microbiology. He was an associate editor of *Diagnostic Immunology*, a contributing editor of *Clinical Immunology Newsletter*, an editor of "Teratogen Update," *Teratology Journal*, and on the editorial board of Perinatal Press.

Dr. Brent is the Distinguished Professor of Pediatrics, Radiology and Pathology at Jefferson Medical College. He also has a Ph.D. in radiation biology and embryology. He is the head of the Laboratory of Clinical and Environmental Teratology at the DuPont Hospital for Children. He has been a member of the Teratology Society since 1960, of which he was President in 1968. He has been a member of the Congenital Anomalies Research Association of Japan since 1966. He was a member of the Environmental Hazards Committee of the American Academy of Pediatrics from 1968-76. He was on the Scientific Program Committee of the Third International Conference on Congenital Malformations. He was the associate editor of *Teratology* from 1969-76. He was the Chairman of the Subcommittee on the Prevention of Fetal and Perinatal Disease, Fogarty International Center, National Institutes of Health, from 1972-73. He was a member of the NIH Study Section in Human Embryology from 1970-74. He has been a member of the European Teratology Society from 1971 to the present. He is a member of the Society for Developmental Biology, Inc. He was appointed to the Medical Advisory Board of the Pennsylvania Association for Retarded Citizens in 1976. He was editor in chief of *Teratology* from 1976-92. He was appointed to the National Science Foundation Panel on Evaluation of Environmental Factors in Reproductive Biology as well as the Second Task Force for Research Planning in Environmental Health Sciences, Subtask Force on Reproduction of the National Science Foundation. He was appointed to the Task

Force on Prenatal Diagnosis of the National Institute of Child Health and Human Development in 1978. He was appointed to the editorial board of *Fetal Medicine* in1982. He was appointed to the editorial board of *Fetal Therapy* in 1986. He has been a consultant to the Teratogen Information System since 1985. He was the keynote speaker at the International Perinatal Conference in Singapore, November 12, 1996, speaking on "The Cause and Prevention of Human Birth Defects." He has 318 papers published, 16 papers submitted for publication, and 7 books and monographs.

Of his books and monographs, Dr. Brent has written about prevention of fetal disease and environmentally induced birth defect risks. His articles concern congenital malformations, the teratogenicity of drugs, antibodies and malformations, the effect of immune reactions on fetal development, the transport of molecules across placental membranes, morphologic alterations in the parietal yolk sac of the rat from the 12^{th} to the 19^{th} day of gestation, methods of evaluating alleged human teratogens, mechanisms of teratogenesis, and drug use in pregnancy.

There could hardly be two people more knowledgeable about congenital malformations, teratogens, and drugs than Drs. Sever and Brent. Compared to their exhaustive command of the very field which concerns this case, Dr. Maertens pales into insignificance. Consequently, when the undersigned heard Drs. Sever and Brent deny quite firmly that measles vaccine caused Tyler's congenital malformations, contrary to Dr. Maertens' testimony, their impressive backgrounds added considerable weight to their opinions.

Not only do their professional qualifications make Drs. Sever and Brent more credible than Dr. Maertens, but also what they said makes more sense in light of the evidence. The measles vaccine Mrs. Rooks received when she was pregnant with Tyler was her second one. As respondent's experts stated, she probably already had good antibody response against measles. Dr.

Maertens agreed that a patient would have some immunity following the initial vaccine, but he did not know to what extent. Since Tyler was just an embryo when Mrs. Rooks was vaccinated, he was fed directly through Mrs. Rooks' blood vessels, and her immunologic resistance was his. There is no reason for the court to assume that the vaccine had any more effect on him than it had on her, and it had no effect on her.

Respondent's experts pointed out that if an embryo or a fetus suffers because its mother received measles vaccine, it would be so only in the case in which the mother actually contracted measles. Mrs. Rooks testified that she did not become ill from the vaccine. Since she did not become ill, there would be no reason to assume harm to her embryo. Those few articles submitted into evidence showing children born with defects after a mother contracted measles concerned mothers who actually were sick with high fevers and the other signs of measles.

Furthermore, measles vaccine is not measles. It is an attenuated or weakened form of the virus. The undersigned cannot assume that everything measles may cause, the vaccine may cause. This is most obvious in the area of subacute sclerosing panencephalitis (SSPE). Robles v. Secretary, HHS, No. 90-3001V, 2000 WL ___ (Fed. Cl. Spec. Mstr. May 19, 2000); Carrico v. Secretary, HHS, No. 90-3712V, 1996 WL 422146 (Fed. Cl. Spec. Mstr. July 12, 1996).

The medical literature, as Dr. Brent states, does not show a syndrome of teratogenic defects as a consequence of measles or measles vaccine, unlike rubella and rubella vaccine. Medical expectation is reasonably that if a drug reaction produces harm, that harm is consistent. Dr. Brent said teratogens have great specificity. But the articles which showed defects did not indicate any pattern or syndrome, and another article which compared pregnant women vaccinated with measles

vaccine against controls showed no higher incidence of birth defects among the vaccinated than among the unvaccinated.

Apparently, the sole basis for Dr. Maertens' testimony is that some of Tyler's defects have been linked to teratogenic exposure of some type. But this ignores the fact that Tyler's other defects are not teratogenic. For the undersigned to accept Dr. Maertens' testimony would be to ascribe Tyler's defects both to the vaccine and to some unknown genetic or metabolic problem. That would be a profound coincidence. Moreover, the 17th day of gestation is before organogenesis begins. To accept Dr. Maertens' theory of causation would be also to accept that the vaccine lingered in Tyler's system (while Mrs. Rooks' was quickly depleting it through the same blood vessels) in order to affect organs that formed later. This assertion is unsubstantiated by any submission of medical literature or experience to which Dr. Maertens referred.

Based on the vast superiority in knowledge and experience of respondent's experts as well as the medical literature and clear logic, the undersigned holds that petitioner has failed to prove a prima facie case of cause and effect of Mrs. Rooks' second measles vaccination and Tyler's congenital defects and current condition.

The court commends Mrs. Rooks for her honesty in testimony and commiserates with her difficult task in tending to Tyler's substantial needs.

CONCLUSION

This case is dismissed with prejudice. In the absence of a motion for review filed pursuant to RCFC Appendix J, the clerk of the court is directed to enter judgment in accordance herewith.

IT IS SO ORDERED.

_____ _____

DATE Laura D. Millman
Special Master