

“Congenital Cataract following German Measles in the Mother” (1941), by Norman McAlister Gregg

In Australia in the 1940s, Norman McAlister Gregg observed a connection between pregnant women who contracted the rubella virus, or German measles, and cataract formation in their children's eyes. Gregg published his findings in the 1941 article “Congenital Cataract following German Measles in the Mother” in Transactions of the Ophthalmological Society of Australia. In the article, Gregg analyzed seventy-eight cases of congenital cataracts and suggested that the mothers' environmental factors could cause birth defects, otherwise known as teratogenic effects. Gregg's paper on the teratogenic effects of an environmental agent, the rubella virus, changed the study of birth defects to include viruses as potential causes or teratogens.

Gregg, an eye surgeon at Royal Prince Alfred Hospital and at Royal Alexandra Hospital for Children, both in Sydney, Australia, noticed that abnormally high numbers of children were born with cataracts after a rubella virus outbreak swept through Australia in 1940. Gregg hypothesized that measles could alter fetuses as they developed, but he encountered initial opposition to that claim. Doctors characterized rubella as a mild infection consisting of a slight cough, fever, and rash that disappeared within two weeks. Few accepted that the mild rubella virus could be connected to severe birth defects, as many doctors claimed that congenital defects could only be inherited.

Gregg's article first notes the prevalence of congenital cataracts and then by reviews the physical symptoms of the disease. Gregg discusses some of the extraordinary symptoms that he and other doctors observed among seventy-eight cases of congenital cataracts. Then, based on anatomy and development of the cataracts, Gregg suggests that such a disease could be caused by an infection, such as measles. He then argues that in the cases he presents, congenital cataracts in newborns likely associated with German measles in the mothers when they were still pregnant. He concludes with a discussion of the potential management and treatment of cataracts.

The children included in the study, mainly patients at the Royal Alexandra Hospital for Children, had advanced cataracts that affected much of the newborn's lenses, with the exception of the lenses' superficial layers. The affected layers form early in embryonic development, thus Gregg concluded that the disease affects embryos early in the growth process. Many of the children also had, compared to normal children, stunted development and malnutrition, darting eyes (nystagmus), small and underdeveloped eyes (microphthalmia), heart defects, and severe reactions to the muscle relaxant atropine. Gregg focused on monocular or single-cataract cases because they allowed him and his colleagues to have a controlled comparison of normal eyes and eyes with cataracts. After detailing the physiological symptoms for the cases, Gregg discusses the potential causes of congenital cataracts based on available data.

Gregg states that three major factors indicated that the children had not inherited the disease. First, he noticed that the cataracts clouded similarly in all cases, suggesting a cause to all of the afflicted children. Though there was some variation across children in progression of the disease, he explained the variation as an effect of the developmental time of contraction and the severity of the infection. Second, most of the affected children showed signs of heart complications, such as trouble feeding and general listlessness. These problems often arise when the heart develops abnormally and does not pump enough blood and oxygen to the rest of the body. Such developmental problems suggested a more systemic factor. Third, the children were all roughly the same age, and they came from all over Australia. Together, these pieces of evidence suggested that the congenital cataracts were caused by an environmental agent that was infectious. Gregg further discovered that all the mothers were in their first few months of pregnancy during the 1940 rubella outbreak.

When questioned, most of the mothers said that they had contracted the German measles during that time, while women with mild infections may not have recalled any ill effects.

The next few sections of the article review some facts, as Gregg mentions the incidence of German measles among the pregnant mothers, the wide occurrence of cases across Australia, and the severe symptoms he observed in patients with German measles during the 1940 outbreak. In the last two sections of the article, he discusses the management of the disease and the prognosis of the afflicted children. Gregg says that surgical removal of the cataracts is the best way to help afflicted children and that operating as soon as possible would help the eyes develop. Children with congenital cataracts face a future health that depends on factors including the severity of any associated heart defect, the possibility of more problems appearing later in life, and whether doctors can complete the corrective eye surgery before the children develop darting eyes. As the prognosis for children with congenital cataracts was uncertain, Gregg advocates that doctors improve prevention techniques, inoculating people from the rubella virus and isolating pregnant women from those infected. He also calls for improved medical records and public education to help prevent the fetuses from developing the disease.

Gregg's findings received mixed reviews from his peers in scientific and medical fields. In Australia, The National Health and Research Council of Australia arranged for Charles Swan, a medical researcher in South Australia, to corroborate Gregg's conclusions. Swan studied forty-nine cases of women who contracted the German measles during pregnancy. Thirty-one of those women subsequently had babies with birth defects, and in twenty-nine of those cases, the women contracted measles during the first trimester of pregnancy. Swan's findings, published in 1943, confirmed Gregg's conclusion that a measles infection during pregnancy could cause birth defects. Swan found deafness to be another affliction among the children in these cases. He proposed that the various malformations associated with rubella could be caused when pregnant women contracted the infection at different stages of pregnancy.

Many in the US accepted Gregg's findings, while many in the UK were more cautious, even after Swan's corroboration. A 1944 *Lancet* editorial stated that Gregg's conclusions were weak, while a 1946 *British Medical Journal* editorial accepted Gregg's conclusion as possible. Medical professionals later classified the German measles as teratogenic and created a rubella vaccine. The vaccine effectively eliminated the German measles from the developed world.

Sources

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