The Eye Color Experiment: From Berlin to Auschwitz and Back

Richard H.C. Zegers MD PhD[1,2]

[1]Department of Ophthalmology, Diakonessenhuis, Utrecht/Zeist, The Netherlands
[2]Department of Ophthalmology, Amsterdam UMC, Location AMC, Amsterdam, The Netherlands

ABSTRACT: **Background:** In an effort to alter eye color during World War II, devout Nazi researcher Karin Magnussen had adrenaline eye drops administered to inmates at the concentration camp Auschwitz-Birkenau. A Sinti family, with a high prevalence of heterochromia iridis, was forced to participate in this study. Members of this family, as well as other victims, were later killed and had their eyes enucleated and sent to Magnussen for examination. Magnussen articulated the findings of these events in a manuscript that has never been published. The author is the first ophthalmologist to review this manuscript. The generation who experienced the atrocities of World War II will soon be gone and awareness of what happened during this tragic chapter of world history is fading.

Objectives: To describe these events to raise awareness among future generations.

Methods: A literature review and archival search was conducted.

Results: Magnussen's research was based on an animal study published in 1937. For Magnussen's study, adrenaline drops were administered to inmates, including a 12-year-old girl from the Sinti family. As there was a reported case of deaf-mutism within the family, Waardenburg syndrome seems to be the most plausible explanation for this family's heritable heterochromia.

Conclusions: The effort to change eye color was doomed to fail from the beginning because there was a probable diagnosis of Waardenburg syndrome. Extinction of humans for ophthalmological research is a horrible act beyond imagination. For the sake of these victims, and for the generations who still feel their pain, it is imperative to tell their stories.

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KEY WORDS: Auschwitz-Birkenau; eye color; Holocaust; Josef Mengele; Karin Magnussen; Mechau family, Oldenburg (Germany)

The year 2020 marks the 75th anniversary of the liberation of the concentration camp Auschwitz-Birkenau (CCAB). The generation who experienced the atrocities of World War II will soon be gone, and the awareness of what happened during this tragic chapter of world history is fading in the memories of younger generations. To the best of my knowledge, I am the first ophthalmologist to describe a medical experiment meant to change eye color. Considering the insanity of the eye color experiment and the horrible fate of the victims, I believe it is necessary to tell this story to raise awareness.

PATIENTS AND METHODS

The present study began in 2008. To identify relevant existing literature, I searched all major databases (PubMed, WorldCat, Google Scholar, and Google Books) in the German and English languages with (a combination of) the terms "(Karin) Magnussen," "Mechau," "Auschwitz," "(Josef) Mengele," "eye color (project)," "eye experiment," and "eyes Auschwitz." In addition, I visited the German General Archives in Berlin, the archives of the concentration camp Auschwitz-Birkenau (Poland), and the archives of Hadamar Memorial Museum (Germany).

RESULTS

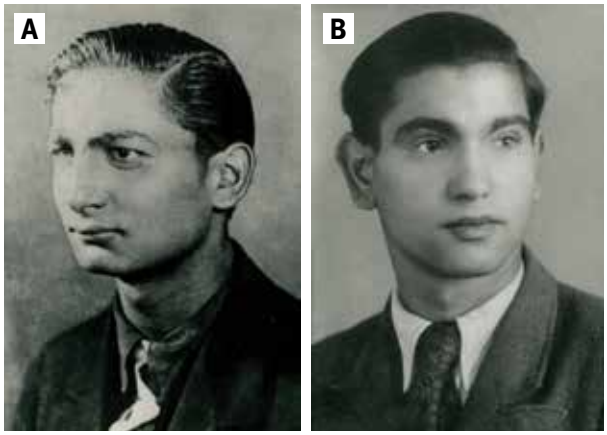
THE EYE COLOR PROJECT

Before and during WWII, the Nazi government (ab)used science to achieve several goals: to implement eugenics so that the Aryan race could be perfected; to identify the most efficient methods for the systematic annihilation of Jews, gypsies, and other minority groups considered inferior; and to invent improved remedies to treat disease and trauma, and thus strengthen the German army. To achieve their goals, Nazi physicians performed many (pseudo) scientific experiments on inmates in concentration camps without ethical consideration or informed consent. Horrors of the concentration camp experience have been described previously by former inmate and physician, Elie Aron Cohen [1].

In 1943 and 1944, Otmar Freiherr von Verschuer, the director of the Kaiser Wilhelm Institute (KWI) in Berlin, received orders from the German government to carry out scientific research on the phenogenetics of eye color (*Projekt Augenfarbe*) [2]. One of von Verschuer's employees, Karin Magnussen (1908–1997), a devout Nazi, was assigned to this project.

Among Magnussen's study population was the Sinti family, which showed a high prevalence of heterochromia iridis [Figure 1]. Magnussen photographed the eyes of the Mechau family in the winter of 1942/1943. Soon after, the entire family

Figure 1. Photographs of unknown date **[A]** Robert Mechau (1921–1945?) and **[B]** Baldwin Mechau (1925–1944)
Robert had a blue right eye and brown left eye. Baldwin was diagnosed with deaf-mutism and heterochromia iridis, the latter clearly visible. The brothers were incarcerated in different concentration camps. The exact circumstances of Robert's death are not known. Baldwin was liquidated by Mengele, having his eyes enucleated and sent to Berlin for examination by Magnussen [9]. There is no known photograph of their 12-year-old sister, Waltraud (1932–1944)



Photograph courtesy of Günter Heuzeroth (obtained from the private collection of Günter Heuzeroth, originals from Hugo Mechau)

Figure 2. Auschwitz-Birkenau conjunctival smear application form
Baldwin Mechau is mentioned in this application form, which is dated 13 December 1943 and signed by Mengele, to rule out conjunctival diphtheria. As more inmates were tested for conjunctival diphtheria by physicians other than Mengele, it remains unknown whether this examination is related to the eye color experiment. Baldwin tested positive, which could equate to a death sentence in the concentration camp. Nevertheless, he survived the illness, possibly because of his heterochromia, only to be murdered at a later time. Either way, this episode of conjunctival diphtheria is not mentioned in Magnussen's unpublished manuscript


Hyg.-bakt. Unters.-Stelle
der Waffen-SS, Südost

Auschwitz OS., am 13. Dezember 1943

233031/V/5759/43

Anliegend wird übersandt: Bl.20

Material: Augenabstrich entnommen am 13.12.1943

zu untersuchen auf Di-Bazillen

Name, Vorname: Z 2109/644 Mechau Baldwin

Dienstgrad, Einheit:

Klinische Diagnose: Conjunctivitis

Anschrift der einsendenden Dienststelle: H.-Krankenbau des Zig.Lagers Auschwitz Birkenau B II e

Bemerkungen:

(Stempel, Unterschrift)

Photograph courtesy of the archives of the Auschwitz-Birkenau State Museum

was deported to CCAB [2]. Magnussen's plan was to conduct a study to alter eye pigmentation in this family and others; however, as a civilian, she did not have access to CCAB. For this reason, she chose to collaborate with a colleague of the KWI who was stationed in CCAB [3], physician Josef Mengele (1911–1979), who became notorious after the war for many criminal medical experiments committed on inmates in CCAB [Figure 2] [4].

Subsequently, Mengele conducted eye color experiments on many inmates. He even had multiple heterochromic prisoners, including members of the Sinti family, killed by means of intracardial chloroform injections, only to have their eyes enucleated and sent to Magnussen in Berlin for examination [5]. No change of eye color ever took place during these experiments.

It is known that Mengele administered adrenaline eye drops to inmates, mainly children, that caused inflamed eyes that temporarily diminished sight, created lots of fear and distress, and even caused the death of a newborn baby [6]. In addition to adrenaline, it has been postulated that the eye drops could have also contained atropine, physostigmine, or carbachol [2]. Intraocular injections of methylene blue have also been described [7].

Twenty-two members of the Mechau family, including one person with combined heterochromia and deaf-mutism, died in CCAB [8]. After the war, Magnussen was prosecuted only for her opportunistic membership in the Nazi party and, in 1948, she received a fine of 490 Reichsmark [2].

Magnussen articulated her findings concerning the eyes of this family, as well as the results of the eye experiment, in a manuscript that she attempted in vain to publish, even after the war [2,6]. After her death, the manuscript was discovered by a relative, who handed it over to journalist Ernst Klee (1942–2013) [6]. Unfortunately, Klee kept the paper in his private collection for many years without making it accessible to others. In 2018, the Klee archive was given to the Hadamar Memorial Museum. The manuscript was then made available to researchers, finally enabling a reconstruction of the rationale behind this ophthalmological experiment.

MAGNUSSEN'S MANUSCRIPT

In her six-page manuscript, Magnussen stated that she was ordered to study familial heterochromia in a gypsy clan [9]. She explained that she was chosen for this project because she had conducted heterochromia-related research in rabbits and she was familiar with all relevant scientific literature concerning the topic. She claimed that an important first step in this type of research was to contact the family, explain to them the objective of the study, and try to make them cooperate. She wrote, "humans are partners in research, not objects!"

In her manuscript, Magnussen referred to a study published in 1937, which described how Horner syndrome was artificially generated in 2-week-old rabbits by unilateral surgical removal

of the superior cervical ganglion [10]. The subsequent induced heterochromia was treated for 10 weeks with adrenaline 0.1% eye drops two to three times per day, which eventually caused an increase in iris pigmentation. Physostigmine 0.1% and atropine 0.1% eye drops were also administered to a herd of rabbits; however, these medications generated no effect on iris pigmentation [10]. This study served as the model for Magnussen's later experiments on human beings [9].

In her manuscript, Magnussen reported anonymously on the Sinti family, and even included a genogram [Figure 3] [9]. She identified seven cases of total heterochromia in three generations of this family. She also noted her regret that the family was deported to the concentration camp soon after she had encountered them because it was "a major obstruction of the research."

Magnussen reported that "after a long time two heterochromia patients passed away," after which she received the enucleated eyes, together with records of their medical histories and autopsies [9]. She hypothesized that these patients had a "pathologic genetic predisposition; possibly a unilateral malfunctioning of the sympathetic nervous system." Furthermore, Magnussen suggested that tuberculosis could be the cause of death.

One of the family members, a 12-year-old heterochromic girl, was described as a subject of the eye color experiment [9]. Magnussen explained her motivation for choosing this girl by stating that, in her experience, a physiological increase in human iris pigmentation was possible only until the age of 15 years. Similar to the rabbit eye color experiment, adrenaline eye drops were administered to the girl and she died after 6 months, with an official cause of death registered as tuberculosis. After another unspecified period, three more heterochromic family members perished and, yet again, tuberculosis was claimed as the cause of the deaths. The eyes of all described victims underwent histologic examination by Magnussen, just like those of three homochromatic family members.

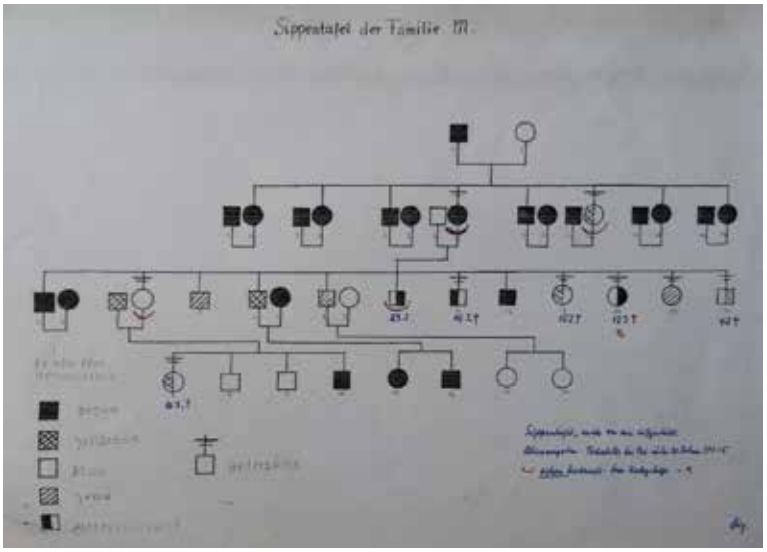
DISCUSSION

The extermination of a family to facilitate research on eye color represents a horrible medical experiment and unethical behavior beyond imagination. Since Nazi scientists often concealed all evidence of their medical experimentation, it is a very unique opportunity to have access to this written first-hand account of the researcher.

As the manuscript reveals, Magnussen exactly reproduced the 1937 study on rabbits in human beings. Moreover, she seemed to justify her research by labeling heterochromia as a "pathological condition" and by talking about "heterochromia patients."

In the Mechau family, the combination of heterochromia and deaf-mutism raises a strong suspicion for Waardenburg syndrome [Table 1]. Waardenburg syndrome is an auditory–pigmentary disorder with congenital sensorineural hearing loss and pigmentary disturbances of the iris, hair, and skin [11].

Figure 3. Mechau family genogram
The genogram depicting the Mechau family of Oldenburg (Germany) [8], found in Magnussen's unpublished manuscript [9]. Individuals depicted with two different colors represent those with heterochromia iridis. Two small horizontal dashes above a family member indicate eyeballs examined by Magnussen



Photograph courtesy of the archives of Hadamar Memorial Museum (Sippentafel der Familie M. [genogram of the M. family] in: Gedenkstätte Hadamar, Sammlung, N Klee, Hängeregister, Nachlass Magnussen)

Table 1. Comparison of clinical features in Waardenburg syndrome 1 (WS1) and Waardenburg syndrome 2 (WS2) [11]

	% of affected individuals	
Finding	**WS1**	**WS2**
Sensorineural hearing loss	47–58%	77–80%
Heterochromic irides	15–31%	42–54%
Hypoplastic blue irides	15–18%	3–23%
White forelock	43–48%	16–23%
Early graying	23–38%	14–30%
Leukoderma	22–36%	5–12%
High nasal root	52–100%	0–14%
Medial eyebrow flare	63–73%	7–12%

WS1 is distinguished from WS2 by the presence in WS1 of lateral displacement of the inner canthi [11]. Given the physical characteristics on the family photographs, which depict no signs of facial dysmorphia and total heterochromia in combination with deaf-mutism, WS2 seems the most likely diagnosis in the Mechau family. Inheritance of WS2 occurs most commonly in an autosomal dominant pattern [11]

Hearing loss in Waardenburg syndrome can be unilateral or bilateral and is present to a greater or lesser extent. Hence, it is possible that multiple Mechau family members presented with hearing loss without it being noticed by others or that they experienced hearing loss that may not have been considered worth mentioning to others. Dutchman Petrus Johannes Waardenburg first described the syndrome in 1951; therefore,

it was not identifiable at the time Magnussen conducted her study or wrote the manuscript [12].

Heterochromia can also be observed in familial congenital Horner syndrome [13]. This diagnosis seems less likely in the Mechau family, however, because ptosis and miosis–hallmarks of Horner syndrome–are not present in the family photographs. Heterochromia in Waardenburg syndrome is caused by hypopigmentation of the lighter iris due to a reduction in the number of stromal melanocytes, which contain fewer and smaller melanosomes (e.g., the location for synthesis, storage, and transport of melanin) compared to the brown iris [14]. In congenital Horner's syndrome, interruption of the sympathetic nerve supply to the eye inhibits synthesis of melanin pigment in the melanocytes, hence influencing iris color [15]. The difference in mechanisms between the two disorders can account for why Magnussen's experiment failed. Hypopigmentation in the victims' eyes was not caused by insufficient adrenergic stimulation of the melanocytes–like in the rabbits–but by a diminished number of melanocytes.

Considering that most inmates would have had normal adrenergic nervous systems, adrenaline was unlikely to have elicited the desired effect, presuming that no infants with congenital Horner syndrome had coincidentally been present among the test subjects. Moreover, adrenaline has now been in use for decades as a topical antiglaucoma therapy. Empirically, it is well known that it can cause hyperpigmentation of the conjunctivae and, in very rare cases, of the corneas, but not of the irides [16].

Nevertheless, the eye experiment did cause adverse effects. Witness accounts describe several ocular side effects, including blurred vision, irritation, inflammation, and lacrimation [6]. It is possible that these effects may have been attributable to ingredients other than adrenaline in the eye drops. Systemic effects of adrenaline eye drops are considered to be rare [17], although are more likely in children, especially when they are weakened due to poor living conditions and malnutrition. Systemic side effects of adrenaline can include headaches, benign ventricular extrasystoles, and even severe hypertensive reactions within minutes after instillation [18]. Complications of the latter condition can even lead to death, a plausible explanation for the reported death of a newborn baby in the experiment [6].

It remains unknown if other substances were used for the eye color experiment. Atropine and physostigmine [2] seem less likely because the 1937 study showed that these medications had no effect on iris pigmentation in rabbits [10]. Although suggested, there is no evidence that methylene blue was injected into eyes [7]. This substance would most likely have caused marked inflammation and corneal decompensation with severe visual loss as a consequence [19].

CONCLUSIONS

In the name of science, Magnussen and Mengele submitted innocent and defenseless people to malicious and meaningless medical experimentation, eventually murdering them for the purpose of additional research. The Nazi eye color experiment, and the fate of the Mechau family and others, is just one chapter of a tragic history. For the sake of these victims, and for the generations who still feel their pain, it is imperative to tell their stories.

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Correspondence
Dr. R.H.C. Zegers
Dept. of Ophthalmology, Diakonessenhuis, 3700 BA Zeist, The Netherlands
Phone: (31-88) 2509440
Fax: (31-88) 2509728
email: r.h.zegers@gmail.com

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Capsule

Cancer therapy in good order

Treatment of cancer patients with two or more drugs acting through different mechanisms is a strategy that has prolonged many lives. Whether the drugs within these combination therapies are delivered concurrently or sequentially can have a major impact on efficacy. A new study illustrates this principle for drugs that inhibit cell cycle kinases CDK4 and CDK6 (CDK4/6 inhibitors), which have attracted great interest because of their clinical efficacy in breast cancer. Studying mouse models of pancreatic cancer, **Salvador-Barbero** et al. found that sequential treatment with Taxol (which inhibits mitosis) followed by a CDK4/6 inhibitor (which prevents cell cycle entry) offered substantially more therapeutic benefit than concurrent treatment with the drugs. Mechanistically, this is because the CDK4/6 inhibitor prevents cancer cells from repairing the chromosomal damage caused by Taxol.

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Eitan Israeli

Capsule

Deadenylate or activate?

When cells are quiescent, they undergo reversible cell cycle arrest and evince low basal metabolism. Naïve T cells are normally quiescent until they recognize cognate antigens through T cell receptor-costimulatory molecule signaling. T cell quiescence appears to be an active process, but the mechanistic details are poorly understood. **Hwang** et al. reported that the transcription factors BTG1 and BTG2 are selectively expressed in quiescent T cells. In mice, T cells conditionally knocked out for both factors showed enhanced proliferation and a lowered threshold of activation both in vitro and in response to *Listeria monocytogenes* infection. Deficiency of BTG1 and BTG2 resulted in increases in global messenger RNA half-life, suggesting that messenger RNA deadenylation and degradation are important processes for maintaining T cell quiescence.

Science 2020; 367: 1255
Eitan Israeli

Capsule

Maternal obesity affects offspring

Alterations in cellular homeostasis can cause endoplasmic reticulum (ER) stress and activation of the stress pathway. Obesity in mice induces ER stress in tissues and the hypothalamus, a brain region that plays a role in many important functions, including controlling food intake and energy expenditure. **Park** et al. found that diet-induced obesity in pregnant mice resulted in postnatal ER stress in the pancreas and hypothalamus of offspring. These mice had increased food intake, adiposity, and body weight and showed disrupted development of specific hypothalamic neurons associated with energy homeostasis. Treatment of offspring with an ER stress-relieving drug reversed these effects. This finding suggests that in mice, maternal physiology has important nutritional programming effects on offspring.

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