THE INFLUENCE OF LIGHT DURATION AND TYPE OF LIGHT ON THE INDUCTION OF CATARACTS IN BOBWHITE QUAIL

Thesis for the Degree of M. S.

MICHIGAN STATE UNIVERSITY
KENNETH LARS KLIPPEN

1975

ABSTRACT

THE INFLUENCE OF LIGHT DURATION
AND TYPE OF LIGHT ON THE INDUCTION OF
CATARACTS IN BOBWHITE QUAIL

By

Kenneth Lars Klippen

Three hundred day-old Bobwhite quail of mixed sexes were randomly divided into six groups and placed into cages with nine square feet of floor space.

During the next two weeks, all the quail were subjected to continuous light as a 250 watt infrared light (2060 lumens/watt) was used for brooding.

Light types and length of duration were changed after two weeks. One pen had three light types, 250 watt infrared (2060 lumens/watt), 40 watt fluorescent (2500 lumens/watt), and 60 watt incandescent (835 lumens/watt) lights operated continuously twenty-four hours a day. The other pen had the same light types operated cyclically twelve hours a day and absolute darkness the other twelve hours.

In the second year of the study, fluorescent lights and 60 watt incandescent lights were replaced with 200 watt incandescent (4010 lumens/watt) lights and 25 watt incandescent (235 lumens/watt) lights.

All the quail received the same ration at appropriate ages.

Kenneth Lars Klippen

Light duration significantly influenced the induction of cataracts in Bobwhite quail.

No significant relationship was observed between type of light and incidence of cataracts.

THE INFLUENCE OF LIGHT DURATION AND TYPE OF LIGHT ON THE INDUCTION OF CATARACTS IN BOBWHITE QUAIL

By

Kenneth Lars Klippen

A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

MASTER OF SCIENCE

Department of Poultry Science

1975

ACKNOWLEDGEMENTS

The author wishes to express his sincere appreciation to Dr. Theo H. Coleman, Department of Poultry Science and Dr. Janver D. Krehbiel, Department of Veterinary Pathology for their supererogating efforts in making this research a reality.

The author is also grateful to Dr. Lloyd R. Champion and Dr. Howard C. Zindel for their guidance and careful review of this manuscript.

Sincere thanks are also extended to Dr. John L. Gill, Department of Dairy Science and Mr. Terry L. Wing, Department of Poultry Science for their statistical review of the data.

To Dr. H. C. Zindel, Chairman of the Poultry Science Department, the author extends his appreciation for financial support provided.

The author is grateful to all individuals in the Poultry Science Department at Michigan State University for their moral support and to Mrs. Barbara Jacobs, Miss Nancy Creed and Miss Patricia Shanks for their technical assistance.

Finally, the author is indebted to his parents and his girl, Patricia, for their never-ending courage and faith that this manuscript will someday be finished.

TABLE OF CONTENTS

	Page
LIST OF TABLES	vi
INTRODUCTION	1
LITERATURE REVIEW	2
Disease-Related Cataracts	2
Drug-Related Cataracts	3
External Stimulation	5
Internal Stimulation	5
Nutrition Cataract	6
Radiation	7
Heat and Light Cataracts	9
PROCEDURE	13
Experiment I	13
Experiment II	15
RESULTS AND DISCUSSION	17
CONCLUSION	26
BIBLIOGRAPHY	27
APPENDICIES	
A. Light Influence on Cataract Induction for Experiment I	36
B. Light Influence on Cataract Induction for Experiment II	63
C. Light Influence on Mortality for Experiment I	72

Page

D. Light Influence on Mortality for Experiment II 75

E. Quail Diets 80

LIST OF TABLES

Table Page

1. The relationship of Bobwhite quail mortality to cataract incidence, by light treatment over an eleven month observation period 19

2. Proportion of cataracts in Bobwhite quail within treatment combinations 22

3. Analysis of variance for data summarized in Table 2 23

4. The influence of cataracts on the mortality of Bobwhite quail reared in cages to eleven months of age 25

Appendix A (Experiment I)

1. The influence of light treatment on the induction of cataracts in Bobwhite quail from three to eleven months of age 36

2. The influence of light duration on the induction of cataracts in Bobwhite quail from three to eleven months of age 45

3. The influence of light type on the induction of cataracts in Bobwhite quail from three to eleven months of age 54

Appendix B (Experiment II)

1. The influence of light treatment on the induction of cataracts in Bobwhite quail from four to six months of age 63

2. The influence of light duration on the induction of cataracts in Bobwhite quail from four to six months of age 66

3. The influence of light type on the induction of cataracts in Bobwhite quail from four to six months of age 69

Page

Appendix C (Experiment I)

1. The influence of light treatment on mortality of Bobwhite quail to eleven months of age 72

2. The influence of light duration on Bobwhite quail mortality to eleven months of age 73

3. The influence of light type on Bobwhite quail mortality to eleven months of age 74

Appendix D (Experiment II)

1. The influence of light treatment on mortality of Bobwhite quail to thirteen months of age 75

2. Proportion of dead Bobwhite quail within light treatment 76

3. Analysis of variance for data summarized in Table 45 77

4. Proportion of dead Bobwhite quail within treatment combinations 78

5. Analysis of variance for data summarized in Table 47 79

Appendix E

1. Ingredient analysis 80

2. Ingredients 81

INTRODUCTION

The crystalline lens is suspended in the visual axis of the eye by zonular ligaments. Aqueous humor bathes the anterior surface of the lens while the posterior surface abuts the more viscous vitreous humor. Light energy must pass through these refractive indices and focus on the macula densa, a specific point of the retina. Changes in the lens will cause fluctuations in its refractive index deflecting light transmittance (Philipson, 1973). Any alteration in the normal transparency of the lens may be termed cataractous (Bourne, 1937).

Attributable factors to cataract induction are innumerable, but the factors of greatest concern are those that involve ordinary, everyday living.

Cataracts in avian species have been noted following subjugation to extended exposures of light energy.

To supplement the pinning of this plausible postulate this research has been conducted investigating the influence of light duration and type of light on the induction of cataracts in Bobwhite quail.

LITERATURE REVIEW

The preventative element of cataracts is still unknown yet the contemporary treatment element of surgical extraction of the lens still utilize the basics as outlined by the French oculist, Jacques David in 1748 (Rucker, 1965).

The first reported cataract explanation was by Aurelius Cornelius Celsus, a Roman physician at the beginning of the Christian era. Celsus (Bellows, 1944) believed that a large space, Locus vacuus, existed between the cornea and the iris. A cataract, according to his conception, was a diseased humor, a suffusion that seeped from the brain into this space and solidified. Cataract, then meaning, "flowing down", was appropriate. The opaque lens conception was finally recognized by Francous Quarre and confirmed by Weiner Rolfinck in 1656.

Disease-Related Cataracts

Maternal rubella acting as a teratogenic agent in humans has focused interest on the possible role of viral infections in the production of congenital abnormalities.

Lens absence or defective development occurred in chick embryos inoculated with Newcastle Disease Virus (Blattner and Williamson, 1951), Influenza A Virus

(Williamson et al., 1956) and Enders Strain Mumps Virus (Robertson et al., 1964).

Cataracts were reported in adult hens exposed to Avian Encephalomyelitis (Peckham, 1957; Bridges and Flowers, 1958; Barber and Blow, 1963; Halpin, 1967) and Lymphomatosis (Rigdon, 1959).

Drug-Related Cataracts

During the summer of 1935, a sporadic outbreak of cataracts, mostly in young women, revealed a cataractogenic drug; dinitrophenol. Its prescribed use was as a metabolic stimulant. Reports of cataracts attributed to this drug flooded medical journals (Allen and Benson, 1935; Kniskern, 1935; Lazar, 1935; Horner et al., 1935; Shutes, 1935; Whalman, 1936; Spaeth, 1936; Mann, 1936; Hessing, 1937; Horner, 1942).

Dinitrophenol experimentation revealed an interference with the metabolism of the rabbit lens (Field et al., 1937). Bourne (1937) postulated that interference with lens metabolism may lead to cataract formation. The dinitrophenol cataract was induced in chickens (Buschke, 1947), chicks (Robbins, 1944; Bettman, 1946a; Rigdon et al., 1959), chick embryos (Feldman et al., 1958) and in mammals (Bettman, 1946b; Ogino and Yasukura, 1957).

1,4-Dimethanesulfonoxybutane, a radiomimetric chemotherapeutic drug used in treating chronic myeloid leukemia in humans, produced diffuse opacities in the posterior cortical layers of the rat lens during chronic toxicity tests by Solomon _et al_. (1955). He speculated that the mode of action of this aliphatic compound was due to an alkylation of cytoplasmic or nucleic proteins.

Methoxsalen (8-methoxypsoralen), an ingredient used in suntanning lotions, photosensitizes the mammalian lens to ultraviolet light (Cloud _et al_., 1960, 1961) and with prolonged use, cataracts developed. Visible light is transmitted by the cornea, the lens and reaches the retina. Ultraviolet light is transmitted by the cornea but is absorbed by the lens. The authors conceded that one omission made in planning these experiments was that no provision was made to determine whether methoxsalen alone was responsible.

Polymyxin B Sulfate, an antibiotic, was linked to cataract development in rabbits following intravitreal injections (Cotlier and Apple, 1973). The membranes of the lens fibers contain reactive acidic groups as a part of glycoproteins, phospholipids, polyphosphate nucleic acids and acid mucopolysaccharides. Polymyxin B Sulfate can bind to anionic sites in the lens fiber membranes and eliminate its selective permeability. Electrolyte and water imbalances result, eventually yielding cataractous changes. No lenticular alterations resulted from intravitreal injections of streptomycin sulfate, nystatin, bacitracin, penicillin, or chloramphenicol.

2,6-Dichloro-4-nitroaniline, a fungicide used to inhibit the growth of mold on soft fruits, helped to induce cataracts in beagle dogs exposed to both the fungicide and sunlight (Bernstein *et al.*, 1970). The authors indict a conjunction with light because the cataract was located in the visual axis.

External Stimulation

According to Duke-Elder (1926), Hess (1888) and Kiribuchi (1900) induced cataracts in animals following electrical stimulation via the use of a Leyden jar. Adam and Klein (1945) observed cataracts in a man resulting from an electric current he received in an industrial accident. Duke-Elder (1926) suggested that the electric effect might be complicated by concussion effects. A wedge-shaped opacification can be produced in the human lens by excessive radial mechanical stress (Fisher, 1973).

Internal Stimulation

The occurrence of lamellar cataracts in several generations of a family of humans is not very rare. Khan (1926) managed to pedigree this type of cataract but concedes that the pedigree does not fulfill all the requirements of the Mendelian theory. Khan justifies this by citing the difficulty involved in accumulating all the data on maternal conceptions. Anderson (1949) attempted a genetic explanation of the cortical cataract as a form of dominance with low penetrance; "...an individual possesses a dominant gene for cataract but does not exhibit the condition himself." Anderson conceded that the investigators of the genetic cataracts report such a diversity of findings that the results seem questionable.

Nutrition Cataract

Laboratory investigations following dietary deficiencies have revealed several cataractogenic determinates in several species.

Riboflavin deficiency cataracts have been reported in the rat (Day *et al.*, 1931, 1937; O'Brien, 1932; Yudkin, 1933; Day and Darby, 1938), mouse (Langston *et al.*, 1933), pig (Patek *et al.*, 1941; Wintrobe *et al.*, 1944) and in the cat (Gershoff *et al.*, 1959). Gyorgy (1935) was unable to induce riboflavin-deficiency cataracts in the rat, whereas, Day and Langston (1934) reported a 100% incidence. Day *et al.* (1938) also reported that small amounts of riboflavin would arrest further development of this nutrition cataract. Baum *et al.* (1942) reported that small amounts of riboflavin are needed for cataract development.

Ferguson *et al.* (1954) observed cloudiness in the central portion of the lens of turkey embryos produced by hens fed an all-vegetable protein diet without vitamin E. A second study by Ferguson *et al.* (1956) yielded a keratoconus condition in turkey embryos at 19 days of incubation, with liquefaction of part or essentially all of the lens protein.

Young rats develop cataracts when fed a diet deficient in tryptophan (Albanese and Buschke, 1942; Buschke, 1943; von Sallmann *et al.*, 1959), phenylalanine (Syndestricker et al., 1947; Bowles et al., 1947; Hall et al., 1948), or low protein (Rezende and deMoura Campos, 1942; Ferraro and Roizin, 1947). Nutritional-deficiency cataracts have been reported in guinea pigs with tryptophan deficiency (von Sallmann et al., 1959), the larvae of the tiger salamander with cystine deficiency (Patch, 1941), the pig on a low protein diet (McLaren, 1959) and young rabbits with calcium deficiency (Swann and Salit, 1941).

Mitchell and Dodge (1935) reversed the nutritional deficiency investigations by overfeeding lactose and observing cataractous development. Galactose-rich diets also induce cataracts according to some investigators (Krewson et al., 1939; Gifford and Bellows, 1939; Kinoshita and Merola, 1964; Sippel, 1967; Kuwabara et al., 1969). The clinical manifestations of the sugar cataract have been reported in individuals with galactosemia (Lerman, 1959; Kinoshita, 1965) or diabetes (Duke-Elder, 1926).

Lens opacities were noted in studies of thirst (Kudo, 1921) and anoxia (Bellows and Nelson, 1944).

Radiation

Roentgen Radiation. In 1895 Roentgen discovered the X-ray and two years later Chalupecky, as reported by Clapp, 1932, demonstrated lenticular opacities in irradiated rabbits, fifty days after exposure. Leinfelder and Kerr (1936) exposed the eyes of several groups of rabbits to various doses of roentgen rays, one eye exposed and the other eye shielded by 2 mm of lead serving as the control.

Lenticular changes occurred in all exposed eyes. Cogan and Donaldson (1951) utilized a wider range of roentgen dosage and rabbit age and reported that cataracts occurred with a latent period that was an inverse function of the dose. Rabbits, 4-10 weeks old, were exposed to a single dose of 2000 r (von Sallmann, 1951) and after the first week a spatial disarrangement of nuclei was observed in the bow and preequatorial zone of the lens epithelium. After two weeks, the spatial disarrangement erupted into gaps and disorganization of lens fibers.

Reducing the roentgen dosage to 1500 r revealed to von Sallmann (1952) the disappearance of dividing cells thirty minutes after irradiation. This inhibition lasted 3-4 days, then mitosis recurred at an accelerated rate. Pirie (1959) was unable to induce lens opacities in the chick embryo or the chicken, but did note lenticular damage in the rabbit lens following exposure to roentgen rays.

<u>Atomic Radiation</u>. Advances in molecular technology have yielded medical repercussions such as cataract development after atomic radiation exposure. Small vacuoles, thickening of posterior capsule, failure of cells at the equator to differentiate into lens fibers, early migration of cells beneath the posterior capsule toward the posterior pole are some pathological descriptions of the human lens after exposure to the Hiroshima and Nagasaki explosions (Schlaegel, 1947; Cogan <u>et</u> <u>al</u>., 1949; Kimura and Ikui, 1951; Cogan <u>et</u> <u>al</u>., 1952).

Experimentation with radiation induction of cataracts revealed a migration of lens epithelium (Reese, 1939) of the germinative zone toward the posterior pole (Hanna and O'Brien, 1963). Selective irradiation with thymidine-tritium on other areas of the lens required much larger doses to produce lenticular changes than were required to produce changes of the germinative zone (Hanna and O'Brien, 1961).

Cyclotron-induced cataracts have been reported by investigators (Abelson and Kruger, 1949; Cogan _et al._, 1952) to be similar in appearance to radiation cataracts.

Heat and Light Cataracts

The clinical manifestations of cataracts in occupations that subject people to extremes of heat and light, such as glass-blowers and iron-workers, enticed cataractogenic speculation of incident radiant energy on the lens deranging the lens' metabolism (Duke-Elder, 1926). This renders its proteins more prone to coagulation by changes in hydrogen ion concentration, salt content, osmotic changes, or metabolic disturbances.

Lenticular opacification due to heat rays yield posterior polar opacities in rabbits (Goldmann, 1933) when the rays strike the iris alone and not the whole lens. Goldmann postulated that environmental and metabolic temperatures act on the temperature of the anterior lens surface behind the iris.

DeVolt (1944) reported lamellar cataracts in chicks brooded in a cellar using electric light bulbs. Other procedures did not experience this phenomenon, so he reared a second brood of chicks, but with a shortened time period in the cellar. No cataracts developed in the second brood. The effects of continuous light on the avian eye were first reported to be an enlarged eyeball (Jensen and Matson, 1957). Lauber _et al_. (1961) confirmed and extended this research to include: accumulation of vitreous, thickening of retina, reduced thickness of nerve fiber layer and layer of rods and cones. In his studies on the effects of continuous light on the avian eye, he subjected chicks for 10 weeks to an intensity of 3 foot candles (32.28 lux). Lauber _et al_. (1970) prolonged the exposure of chicks to continuous light to 16 weeks and noted an increase in intraoccular pressure. An increase in intraoccular pressure disrupts lens metabolism (Brini and Flament, 1973). A disruption in lens metabolism may lead to cataractous development (Bourne, 1937).

Zigman and Vaughan (1974) exposed mice to near-ultraviolet (black) light for 12 hours a day over a period of 90 weeks. At 35 weeks they noted inhibition of lens epithelial cell differentiation into typical non-nucleated cortical fiber cells in the bow region. Lenticular opacities appeared after 50-60 weeks of exposure to the near-ultraviolet light. Kinsey (1948a) used a Beckman Spectrophotometer to measure the ultraviolet adsorption by the eye. His studies revealed that the lens is chiefly responsible for radiant energy absorption of wave lengths shorter than 300 millimicrons. He theorized that radiant energy from the sun would damage the cornea before it damaged the lens. Pirie (1971) and van Heyningin (1973) noted photo-oxidation of lens proteins following exposure to ordinary sunlight.

Exposure of chickens to continuous artificial light for several months has led to peripheral anterior synechia, rupture and/or detachment of the retina, metaplasia of the eye wall and cataracts (Lauber and McGinnis, 1966). Light-induced eye abnormalities in turkeys include progressive buphthalmus and loss of corneal convexity (Ashton *et al.*, 1973).

Other investigators have inculpated light for cataractogenic activity in humans (Milner, 1934), trout (Allison, 1962; Steucke *et al.*, 1968) and rabbits, when light was in conjunction with heat (Langley *et al.*, 1960).

Bercovitz *et al.* (1970) measured the thickness of lenses of White Leghorn chickens following light treatments and noted that greater intensities increased the thickness of the lens.

A thorough investigation by Krehbiel (1972) of lens opacities in quail revealed that metabolic disturbances, infectious microorganisms, congenital causes, population density, or nutrition did not significantly influence cataract formation but that a continuous light environment for quail as opposed to their natural diurnal experience, had a pronounced effect on cataract formation.

PROCEDURE

Experiment I

Three hundred Bobwhite quail (*Colinus virginianus*) of mixed sexes were hatched from the eggs of Michigan State University stock, wing banded and evenly distributed at random between six identically-constructed compartments. The compartments were three feet by three feet of one-half inch screened cages with four feet by four feet of three-eighths inch plywood intercompartmental dividers. Each compartment possessed one hanging light bulb fixture suspended thirty inches above the floor of the divider so direct light transmittance between compartments was inhibited. Two light-control pens (capable of complete darkness) had three compartments each. These pens were located in House #2 of the Michigan State University Poultry Science Research and Teaching Center.

Brooding infrared lamps were initially installed in the light bulb fixtures for the first two weeks while the quail were provided MSU Quail Starter Ration 72 and water *ad libitum*. After two weeks, the brooding lamps were replaced with the experimental lights: 250 watt infrared (2060 lumens/watt) bulbs in compartments 1 and 4, 40 watt Cool White flourescent (2500 lumens/watt) bulbs in compartments 2 and 5 and 60 watt incandescent (835 lumens/watt) bulbs in compartments 3 and 6. Compartments 1, 2 and 3 were located in pen G and the light system was set to operate continuously for one year. Compartments 4, 5 and 6 were located in pen F and operated on a cyclic light system; 12 hours of light, 12 hours of darkness each day.

Each pen had one main ceiling outlet into which all three light fixtures were plugged. Via the use of a time clock, this outlet produced either a continuous flow of electrical power (pen G) or the cyclic system (pen F).

Monthly eye examinations were performed on each bird. When the eye undergoes cataractous development, light will be reflected from the pupillary region, exhibiting an opaqueness of some form. Most generally, the afflicted quail exhibited centrally located petechial opacities which progressed with time into complete opacification of the lens. By simply shining a high intensity light onto this region, a positive identification of cataracts could be diagnosed.

When a cataract was detected, the compartment number, wing band number and particular eye (R,L) was recorded and the bird was returned to its compartment. A one-quarter inch thick plywood, portable intracompartmental divider prevented repeated handling of the same bird. All the birds in one compartment were persuaded to one side, then the divider was inserted. After each examination, the bird was placed on the other side of the divider. When the compartmental examination was finished, the intracompartmental divider was removed. The birds would then resume their routine activities until the next month's exam.

Quail Breeder Ration 72 replaced the starter ration after six months.

Experiment II

Experiment II began with a new hatch of three hundred and thirty-six Bobwhite quail of mixed sexes which were wing banded and randomly distributed among the six original compartments.

Newly-incorporated procedures included debeaking (two-thirds of the upper and lower beak), using a Lyons PDQ-2 Debeaker, replacement of the high intensity examination light with a Kowa SL Portable Slit Lamp Microscope and light-type changes. The fluorescent lights in compartments 2 and 5 were replaced with 200 watt incandescent (4010 lumens/watt) bulbs. The 60 watt incandescent bulbs in compartments 3 and 6 were replaced with 25 watt incandescent (235 lumens/watt) bulbs. Infrared lights (2060 lumens/watt) were retained in compartments 1 and 4.

Except for these few changes, brooding and operating procedures were identical to those described in Experiment I up to the six month stage, at which time two new light-control pens with three, new, identically-constructed compartments each, were incorporated. At that point, i.e., after six months, half of the number of remaining birds in each original compartment were transferred to new compartments utilizing the same type of light, but a new light system. For example: half the number of birds in the original compartment 1 (24 hours of infrared light) were transferred to a new compartment that operated on 12 hours of infrared light and 12 hours of darkness. The other half of the birds remained as controls in the original location.

RESULTS AND DISCUSSION

Etiologic investigations of cataracts have yielded two parameters for study concerning light: type and duration.

Three commercially-available light types were employed in two different light systems offering two different durations; six treatment combinations in all. With diet, ventilation, temperature, floor space and examination techniques identical for all the quail, the only varying factor was the type and duration of light. All of the diets used met the established protein requirements for optimum growth and maintenance as prescribed by Andrews *et al*. (1973).

Monthly data collections were analyzed using Chi-Square Analysis (Kempthorne, 1969) to determine if the six treatments had any cataract-inducing influence. In the entire two years of study, a significant cataract-inducing influence was attributable to the subjection of Bobwhite quail to the light treatments every month. Eighty-nine percent of the first year's data indicated a high degree of significance ($P<0.005$) in treatments influencing cataract induction.

The second year and a new brood of quail increased this highly significant percentage to 92% overall. Two more chi-square tests followed each monthly test of significance attributable to the treatments. Light duration was analyzed for its cataract-inducing influence and in the first year, produced a high level of significance in every month (78% at the 0.005 level and 22% at the 0.01 level of probability).

The second year reaffirmed the 100% cataract-inducing influence through light duration with 83% of the data at the 0.005 level of probability.

Light type was the third, monthly chi-square test. Only one month in the two year period showed any level of significance ($P<0.05$).

Tables 5 through 31 (see Appendix A) show the chi-square test statistics for the cataract-inducing influences of light in the first year of study. Tables 32 through 40 (see Appendix B) show light influences for the second year.

The data records begin at the first month that signs of cataract-afflicted quail are evident in any group.

The multi-dimensional contingency (Cochran, 1954), Table 1, reveal a highly significant ($P<0.005$) relationship of cataract incidence and light treatments to Bobwhite quail mortality.

Tables 41 through 43 (see Appendix C) are the chi-square tests used to single out the inculpatory factor.

Table 1. The relationship of Bobwhite quail mortality to cataract incidence, by light treatment over an eleven month observation period.

Light Duration	Treatment	OBSERVED Cataract Lived	Cataract Died	Cataract Total	Normal Lived	Normal Died	Normal Total	Total Population Lived	Total Population Died	Total Population Total	EXPECTED Cataract Lived	Cataract Died	Normal Lived	Normal Died
24 Hours	1	8	10	18	8	24	32	16	34	50	3	5	15	27
	2	3	6	9	19	22	41	22	28	50	3	5	15	27
	3	10	5	15	13	22	35	23	27	50	3	5	15	27
12 Hours	4	0	1	1	23	26	49	23	27	50	3	5	15	27
	5	0	1	1	5	44	49	5	45	50	3	5	15	27
	6	1	3	4	16	30	46	17	33	50	3	5	15	27
totals		22	26	48	84	168	252	106	194	300	18	30	90	162

$(\text{OBSERVED-EXPECTED})^2/\text{EXPECTED}$

Treatment	Cataract Lived	Cataract Died	Normal Lived	Normal Died
1	8.33	5.00	3.27	0.33
2	0	0.20	1.07	0.93
3	16.33	0	0.27	0.93
4	3.00	3.20	4.27	0.04
5	3.00	3.20	6.67	10.70
6	1.33	0.80	0.07	0.33

$\Sigma\left[(O-E)^2/E\right] = 73.27$***

***Significant, $p < 0.005$

Light treatments offer a high degree of significance toward influencing Bobwhite quail mortality, but no significant difference could be isolated in light duration and types analysis. Reinspecting Table 1, particularly treatment 5, an unusually high mortality is recorded. An uncontrollable case of cannibalism took its toll on this particular group, thus, influencing the test statistics. To counter such a reoccurrence in the second study, all the quail were debeaked at one day of age. The second trial (Table 44, Appendix D) shows no significant relationship between light treatments and Bobwhite quail mortality.

A radiant energy range of 200 to 400 nanometers (nm) is generally used to produce fluorescent rays. This range involves ultraviolet and the shorter wave lengths of visible light, the blue and violet spectral regions (White and Argauer, 1970). Kinsey in his spectrophotomic work (1948b) has demonstrated that the lens will absorb radiant energy of wave lengths shorter than 300 nm. No wave lengths shorter than 300 nm can be transmitted by glass. Kinsey speculated that solar energy would damage the cornea before damaging the lens.

The fluorescent lights used by the author were glassed, tubular encasements which produced the shorter wave lengths of visible light. The first experiment did not pinpoint a relationship between light type and cataract induction. The second experiment eliminated the fluorescent study and incorporated two new incandescent bulbs, 25 watt and 200 watt bulbs. These bulbs would offer a wider range of visible light wave length at two different intensities.

Can cataractous development be arrested by transferring cataract-free and cataract-afflicted quail from a continuous light system to the cyclic, or can cataractous development be stimulated by a transfer from cyclic to continuous? This question became an objective in the second experiment. Half the number of quail were transferred to a new compartment with the reciprocal light treatment. The remaining quail acted as controls. Table 2 indicates the proportion of cataracts within the treatment combinations. The numbers (12, 24) describe the number of hours of light initially (the first number) and for the last seven months (the second number). The analysis of variance (Table 3) reaffirms the significance of light duration (treatment combination) and exonerates light type.

Tukey's Test (Tukey, 1949) revealed that in this study of treatment combinations, the quail that began with 12 hours of light and were switched to 24 hours of light are not significantly different in cataract induction from those that began with 12 hours of light and remained at that light level.

<u>12--12, 12--24</u>   <u>24--12, 24--24</u>

There is a significant difference between those groups of quail that began with 24 hours of light and those that began with 12 hours of light.

Table 2. Proportion of cataracts in Bobwhite quail within treatment combinations.

Treatment combinations	Proportion of dead		Arcsin transformations	Σ
12-24*	No.	%		
Infrared	5/23	21.7	27.76	
200 watt incandescent	7/22	31.8	34.33	
25 watt incandescent	5/19	26.3	30.85	92.94
24-24				
Infrared	14/18	77.8	61.89	
200 watt incandescent	8/17	47.1	43.34	
25 watt incandescent	17/22	77.3	61.55	166.78
12-12				
Infrared	4/22	18.2	25.25	
200 watt incandescent	1/21	4.8	12.66	
25 watt incandescent	1/10	10.0	18.44	56.35
24-12				
Infrared	14/20	70.0	56.79	
200 watt incandescent	6/17	35.3	36.45	
25 watt incandescent	13/20	65.0	53.73	146.97

*The original treatment up to six months of age is indicated by the first number and the second number is the treatment for the following seven months in hours of light per day.

Table 3. Analysis of variance for data summarized in table 2.[1]

Source	Degrees of Freedom	Mean Square	F-Value
Treatment combination	3	847.49	18.45*
Light type	2	145.65	3.17
Error	6	45.93	

1. Calculated by arcsin transformation.

* Significant, $p < 0.05$

The symbols above are a convention for displaying the results of paired comparisons in order of magnitude. The underlined portion shows that these groups of means are not significantly different from one another.

The data collected consisted of the proportion of cataract-afflicted quail in the total population within compartments. The possibility that cataracts are lethal to Bobwhite quail would bias the results. Tests conducted on the effects of cataracts on Bobwhite quail mortality over an eleven month period (Table 4) revealed no significant relationship between the two. Light treatments and/or treatment combinations effects on Bobwhite quail mortality were also analyzed (Tables 45-48, Appendix D) resulting in a negative relationship between them.

Table 4. The influence of cataracts on the mortality of Bobwhite quail reared in cages to eleven months of age.

	Observed		Expected		$(Obs-Exp)^2/Exp$	
	C	N	C	N	C	N
Lived	22	84	17	89	1.47	0.28
Died	26	168	31	163	0.81	0.15
totals	48	252	48	252	$\Sigma[(O-E)^2/E] = 2.71$	

1. C = Number of cataract-afflicted quail.
2. N = Number of cataract-free quail.

CONCLUSION

The exposure of Bobwhite quail to a continuous light environment will lead to cataractous lenses. The type of light utilized will not significantly influence cataract induction.

Cataract-affliction in domestically-reared Bobwhite quail will not significantly influence mortality.

BIBLIOGRAPHY

BIBLIOGRAPHY

Abelson, P. H. and P. G. Kruger, 1949. Clyclotron-induced radiation cataracts. Science 110: 655-657.

Adam, A. L. and M. Klein, 1945. Electrical cataract: notes on a case and review of literature. Brit. J. Ophthal. 29: 169-175.

Albanese, A. A. and W. Buschke, 1942. On cataract and certain other manifestations of trytophan deficiency in rats. Science 95: 584-586.

Allen, T. D. and V. M. Benson, 1935. Late development of cataract following use of dinitrophenol about a year before. J.A.M.A. 105 pt. 1: 795.

Allison, L. M., 1962. Cataract among hatchery-reared lake trout. Prog. Fish Cult. 24: 155.

Anderson, C. L., 1949. Reduced penetrance in the inheritance of cortical cataract. J. Hered. 40: 157-161.

Andrews, T. L., R. H. Harms and H. R. Wilson, 1973. Protein requirement of the Bobwhite chick. Poultry Sci. 52: 2199.

Ashton, W. L. G., M. Pattison and K. C. Barnett, 1973. Light-induced eye abnormalities in turkeys and the turkey blindness syndrome. Res. Vet. Sci. 14: 42-46.

Barber, C. W. and W. L. Blow, 1963. A genetic influence on cataract formation among White Leghorn incrosses following an outbreak of avian encephalomyelitis. Avian Dis. 7: 495-500.

Baum, H. M., J. F. Michaelree and E. B. Brown, 1942. The quantitative relationship of riboflavin to cataract formation in rats. Science 95: 24-25.

Bellows, J. G., 1944. Cataract and Anomalies of the Lens. C. V. Mosby Company, St. Louis.

Bellows, J. and D. Nelson, 1944. Cataract produced by anoxia. Arch. Ophthal. 31: 250-252.

Bettman, J. W., 1946a. Production of cataracts in chicks with dinitrophenol. Arch. Ophthal. 36: 674-676.

Bettman, J. W., 1946b. Experimental dinitrophenol cataract, Amer. J. Ophthal. 29: 1388-1395.

Bercovitz, A. B., P. C. Harrison and G. A. Leary, 1970. Eye development of White Leghorns subjected to different intensity and spectrum light treatments. Poultry Sci. 49: 1367.

Bernstein, H. N., J. Curtis, F. L. Earl and T. Kuwabara, 1970. Phototoxic corneal and lens opacities in dogs receiving a fungicide, 2,6-dichloro-4-nitroaniline. Arch. Ophthal. 83: 336-348.

Blattner, R. J. and A. P. Williamson, 1951. Developmental abnormalities in the chick embryo following infection with Newcastle disease virus. Proc. Soc. Exptl. Biol. Med. 77: 619-621.

Bourne, M. C., 1937. Metabolic factors in cataract production. Physio. Rev. 17: 1-27.

Bowles, L. L., V. P. Syndenstricker, W. K. Hall and H. L. Schmidt, 1947. Cataracts resulting from a deficiency of phenylalanine in the rat. Proc. Soc. Exptl. Biol. Med. 66: 585-586.

Bridges, C. H. and A. I. Flowers, 1958. Iridocyclitis and cataracts associated with an encephalomyelitis in chickens. J.A.M.A. 132: 79-84.

Brini, A. and J. Flament, 1973. Cataracta glaucomatosa acuta. Exp. Eye Res. 16: 19-28.

Buschke, W., 1943. Classification of experimental cataracts in the rat: recent observations on cataract associated with tryptophan deficiency and with some other experimental conditions. A.M.A. Arch. Ophthal. 30: 735-750.

Buschke, W., 1947. Acute reversible cataract in chickens due to various nitrocompounds. Amer. J. Ophthal. 30: 1356-1358.

Clapp, C. A., 1932. The effects of x-ray and radium radiations upon the crystalline lens. Amer. J. Ophthal. 15: 1039-1044.

Cloud, T. M., R. Hankin and A. C. Griffin, 1960. Photosensitization of the eye with Methoxsalen I. Acute effects. A.M.A. Arch. Ophthal. 64: 346-351.

Cloud, T. M., R. Hankin and A. C. Griffin, 1961. Photosensitization of the eye with Methoxsalen II. Chronic effects. A.M.A. Arch. Ophthal. 66: 689-694.

Cochran, W. G., 1954. Some methods for strengthening the common X^2 tests. Biometrics 10: 417-451.

Cogan, D. G., S. F. Martin and S. J. Kimura, 1949. Atom bomb cataracts. Science 110: 654-655.

Cogan, D. G. and D. D. Donaldson, 1951. Experimental radiation cataracts I. Cataracts in the rabbit following single x-ray exposure. Arch. Ophthal. 45: 508-522.

Cogan, D. G., D. D. Donaldson and A. B. Reese, 1952. Clinical and pathological characteristics of radiation cataract. Arch. Ophthal. 47: 55-70.

Cotlier, E. and D. Apple, 1973. Cataracts induced by the polypeptide antibiotic Polymyxin B Sulfate. Exp. Eye Res. 16: 69-77.

Day, P. L., W. C. Langston and C. S. O'Brien, 1931. Cataract and other ocular changes in vitamin G deficiency. An experimental study on albino rats. Amer. J. Ophthal. 14: 1005-1009.

Day, P. L. and W. C. Langston, 1934. Further experiments with cataract in albino rats resulting from the withdrawal of vitamin G (B_2) from the diet. J. Nut. 7: 97.

Day, P. L., W. J. Darby and W. C. Langston, 1937. The identity of flavin with the cataract-preventive factor. J. Nut. 13: 389-399.

Day, P. L., W. J. Darby and K. W. Cosgrove, 1938. The arrest of nutritional cataract by the use of riboflavin. J. Nut. 15: 83-90.

Day, P. L. and W. J. Darby, 1938. The influence of different casein preparations in riboflavin-deficient diets upon the appearance of cataract. Biochem. J. 32 pt. 1: 1171.

DeVolt, H. M., 1944. Lamellar cataracts in chickens. Poultry Sci. 23: 346-348.

Duke-Elder, W. S., 1926. The pathological action of light upon the eye. The Lancet 1: 1250-1254.

Feldman, G. L., T. M. Ferguson, R. H. Rigdon, B. L. Reid, M. S. Cross and J. R. Couch, 1958. Effect of dinitrophenol on the lens of chick embryo. Proc. Soc. Exptl. Bio. Med. 98: 646-648.

Ferguson, T. M., R. L. Atkinson and J. R. Couch, 1954. Relationship of vitamin E to embryonic development of the avian eye. Proc. Soc. Exptl. Bio. Med. 86: 868-871.

Ferguson, T. M., R. H. Rigdon and J. R. Couch, 1956. Cataracts in vitamin E deficiency. An experimental study in the turkey embryo. A.M.A. Arch. Ophthal. 55: 346-355.

Ferraro, A. and L. Roizin, 1947. Ocular involvement in rats on diets deficient in amino acids. Arch. Ophthal. 38: 331.

Field II, J., E. G. Tainter, A. W. Martin and H. S. Belding, 1937. Studies on the oxygen consumption of the rabbit lens and the effect of 2-4 dinitrophenol thereon. Amer. J. Ophthal. 20: 779-794.

Fisher, R. F., 1973. Human lens fibre transparency and mechanical stress. Exp. Eye Res. 16: 41-49.

Flowers, A. I., L. C. Grumbles, R. T. Dubose and J. P. Delaplane, 1957. Cataracts: A new flock problem in chickens. Poultry Sci. 36: 1117.

Gershoff, S. N., S. B. Andrus and D. M. Hegsted, 1959. The effect of the carbohydrate and fat content of the diet upon the riboflavin requirement of the cat. J. Nut. 68: 75-88.

Gifford, S. R. and J. Bellows, 1939. Histologic changes in the lens produced by galactose. Arch. Ophthal. 21: 346-358.

Goldmann, H., 1933. Genesis of heat cataract. Arch. Ophthal. 9: 314.

Gyorgy, P., 1935. Investigations on the vitamin B2 complex. I. The differentiation of lactoflavin and the "rat antipellagra" factor. Biochem. J. 29 pt. 1: 741-759.

Hall, W. K., L. L. Bowles, V. P. Syndenstricker and H. L. Schmidt, Jr., 1948. Cataracts due to deficiencies of phenylalanine and of histidine in the rat. A comparison with other types of cataracts. J. Nut. 36: 277-296.

Halpin, F. B., 1967. Opacity of the lens in fowl associated with exposure to the virus of infectious avian encephalomyelitis--a case report. Avian Dis. 11: 146-148.

Hanna, C. and J. E. O'Brien, 1961. Cell production and migration in the epithelial layer of the lens. A.M.A. Arch. Ophthal. 66: 103-107.

Hanna, C. and J. E. O'Brien, 1963. Lens epithelial cell proliferation and migration in radiation cataracts. Rad. Res. 19: 1-11.

Hessing, E. E., 1937. Cataract due to DNP. Report of cases. Arch. Ophthal. 17: 513-515.

Horner, W. D., R. B. Jones and W. W. Boardman, 1935. Cataracts following the use of dinitrophenol. Preliminary report of 3 cases. A.M.A.J. 105 pt. 1: 108-110.

Horner, W. D., 1942. Dinitrophenol and its relation to formation of cataract. Arch. Ophthal. 27: 1097-1121.

Jensen, L. S. and W. E. Matson, 1957. Enlargement of avian eye by subjecting chicks to continuous incandescent illumination. Science 125: 741.

Kempthorne, O., 1969. An Introduction to Genetic Statistics. Iowa State Univ. Press, Ames, Iowa.

Khan, W. A., 1926. Pedigree of lamellar cataract. Brit. J. Ophthal. 10: 387-389.

Kimura, S. J. and H. Ikui, 1951. Atomic-bomb radiation cataract. Case report with histopathologic study. Amer. J. Ophthal. 34: 811-816.

Kinoshita, J. H. and L. O. Merola, 1964. Hydration of the lens during the development of galactose cataract. Invest. Ophthal. 4: 786-799.

Kinsey, V. E., 1948. Spectral transmission of the eye to ultraviolet radiations. A.M.A. Arch. Ophthal. 39: 508-513.

Kniskern, P. W., 1935. Cataracts following dinitrophenol. A.M.A.J. 105 pt. 1: 794-795.

Krehbiel, J. D., 1972. The pathology of spontaneous cataract in Bobwhite quail. Ph.D. dissertation at MSU.

Krewson, C. F., E. J. Schantz and C. A. Elvehjem, 1939. Relation of skim milk feeding to cataract production. Proc. Soc. Exp. Bio. 42: 573-576.

Kudo, T., 1921. Studies on the effects of thirst. I. Effects of thirst on the weights of the various organs and systems of adult albino rats. Amer. J. Ana. 28: 399-430.

Kuwabara, T., J. H. Kinoshita and D. G. Cogan, 1969. Electron microscope study of galactose-induced cataract. Invest. Ophthal. 8: 133.

Langley, R. K., C. B. Mortimer and C. McCullock, 1960. The experimental production of cataracts by exposure to heat and light. Arch. Ophthal. 63: 473-488.

Langston, W. C., P. L. Day and K. W. Cosgrove, 1933. Cataract in the albino mouse resulting from a deficiency of vitamin G (B2). Arch. Ophthal. 10: 508-514.

Lauber, J. K., J. V. Shutze and J. McGinnis, 1961. Effects of exposure to continuous light on the eye of the growing chick. Proc. Soc. Exptl. Bio. Med. 106: 871-872.

Lauber, J. K. and J. McGinnis, 1966. Eye lesions in domestic fowl reared under continuous light. Vis. Res. 6: 619-626.

Lauber, J. K., J. E. Boyd and T. A. S. Boyd, 1970. Intraoccular pressure and aqueous outflow facility in light-induced avian buphthalmos. Exp. Eye Res. 9: 181-187.

Lazar, N. K., 1935. Cataract following the use of dinitrophenol. A.M.A.J. 105 pt. 1: 794.

Leinfelder, P. J. and H. D. Kerr, 1936. Roentgen-ray cataract; an experimental, clinical and microscopic study. Amer. J. Ophthal. 19: 739-756.

Lerman, S., 1959. The lens in congenital galactosemia. Arch. Ophthal. 61: 88-92.

Mann, Jr., W. A., 1936. Cataract due to di-nitrophenol. Arch. Ophthal. 15: 116-117.

McLaren, D. S., 1959. The eye and related glands of the rat and pig in protein deficiency. Brit. J. Ophthal. 43: 78-87.

Milner, J. G., 1934. Irradiation cataract. Brit. J. Ophthal. 18: 497-511.

Mitchell, H. S. and W. M. Dodge, 1935. Cataract in rats fed on high lactose rations. J. Nut. 9: 37.

O'Brien, C. S., 1932. Experimental cataract in vitamin G deficiency. Arch. Ophthal. 8: 880-887.

Ogino, S. and K. Yasukura, 1957. Biochemical studies on cataract. VI. Production of cataracts in guinea pigs with dinitrophenol. Amer. J. Ophthal. 43: 936-946.

Patek, Jr., A. J., J. Post and J. Victor, 1941. Riboflavin deficiency in the pig. Amer. J. Physio. 133: 47-55.

Patch, E. M., 1941. Dietary production of cataracts in larval Amblystoma tigrinum. J. Nut. 22: 365-381.

Peckham, M. C., 1957. Case report--lens opacities in fowls possibly associated with epidemic tremors. Avian Dis. 1: 247-255.

Philipson, B., 1973. Changes in the lens related to the reduction of transparency. Exp. Eye Res. 16: 29-39.

Pirie, A., 1959. The effect of X-radiation on the lens of the embryo and the adult hen. Rad. Res. 11: 260-270.

Pirie, A., 1961. Difference in reaction to X-irradiation between chicken and rabbit lens. Rad. Res. 15: 211-219.

Pirie, A., 1971. Formation of N'-Formylkynurenine in proteins from lens and other sources by exposure to sunlight. Biochem. J. 125: 203-208.

Reese, A. B., 1939. Operative treatment of radiation cataract. Arch. Ophthal. 21: 476-485.

Rezende, C. and F. A. deMoura Campos, 1942. Cataract in rats fed a low protein diet. Arch. Ophthal. 28: 1038-1041.

Rigdon, R. H., 1959. Cataracts in chickens with lymphomatosis. Amer. J. Vet. Res. 20: 647-654.

Rigdon, R. H., G. L. Feldman, T. M. Ferguson, B. L. Reid and J. R. Couch. Cataracts produced by dinitrophenol. Arch. Ophthal. 61: 249-257.

Robbins, B. H., 1944. Dinitrophenol cataract: production in an experimental animal. J. Pharmacol. Exp. Ther. 80: 264-271.

Robertson, G. G., A. P. Williamson and R. J. Blattner, 1964. Origin and development of lens cataracts in mumps-infected chick embryos. Amer. J. Ana. 115: 473-486.

Rucker, C. W., 1965. Cataract: a historical perspective. Invest. Ophthal. 4: 377-383.

Salit, P. W., 1938. Water content and solids of cataractous and sclerosed human lenses. Amer. J. Ophthal. 21: 755.

Schlaegel, T. F., 1947. Occular histopathology of Nagasaki atomic bomb casualties. Amer. J. Ophthal. 30: 127-135.

Shutes, M. H., 1935. Notes, cases, instruments--dinitrophenol. Amer. J. Ophthal. 18: 752.

Sippel, T. O., 1967. Enzymes of carbohydrate metabolism in developing galactose cataracts of rats. Invest. Ophthal. 6: 59-63.

Solomon, C., A. E. Light and E. J. DeBeer, 1955. Cataracts produced in rats by 1,4-dimethanesulfonoxy-butane (Myleran®). Arch. Ophthal. 54: 850-852.

Spaeth, E. B., 1936. Dinitrophenol cataracts with signs of tetany. Amer. J. Ophthal. 19: 320-323.

Steucke, Jr., E. W., L. H. Allison, R. G. Piper, R. Robertson and J. T. Bowen, 1968. Effects of light and diet on the incidence of cataract in hatchery-reared lake trout. Prog. Fish Cult. 30: 220-226.

Swann, K. C. and P. W. Salit, 1941. Lens opacities associated with experimental calcium deficiency. Preliminary report. Amer. J. Ophthal. 24 pt. 1: 611-614.

Syndenstricker, V. P., H. L. Schmidt, Jr. and W. K. Hall, 1947. The corneal and lenticular changes resulting from amino acid deficiencies in the rat. Proc. Soc. Exptl. Bio. Med. 64: 59-61.

Tukey, J. W., 1949. Comparing individual means in the analysis of variance. Biometrics 5: 99-114.

van Heyningen, R., 1973. Photo-oxidation of lens proteins by sunlight in the presence of fluorescent derivatives of kynurenine, isolated from the human lens. Exp. Eye Res. 17: 137-147.

von Sallmann, L., 1951. Experimental studies on early lens changes after roentgen irradiation. I. Morphological and cytochemical changes. Arch. Ophthal. 45: 149-164.

von Sallmann, L., 1952. Experimental studies on early lens changes after roentgen irradiation. III. Effects of X-radiation on mitotic activity and nuclear fragmentation of lens epithelium in normal and cysteine-treated rabbits. Arch. Ophthal. 47: 305-320.

von Sallmann, L., 1957. The lens epithelium in the pathogenesis of cataract. Amer. J. Ophthal. 44: 159-170.

von Sallmann, L., M. E. Reid, P. A. Grimes and E. M. Collins, 1959. Tryptophan-deficiency cataract in guinea pigs. Arch. Ophthal. 62: 662-672.

Whalman, H. F., 1936. Dinitrophenol cataract. Amer. J. Ophthal. 19: 885-888.

White, C. E. and R. J. Argauer, 1970. Fluorescence Analysis. Marcel Dekker, Inc., New York.

Williamson, A. P., L. Simonsen and R. J. Blattner, 1956. Specific organ defects in early chick embryos following inoculation with influenza A virus. Proc. Soc. Exptl. Bio. Med. 92: 334-337.

Wintrobe, M. M., W. Buschke, R. H. Follis, Jr. and S. Humphreys, 1944. Riboflavin deficiency in swine. With special reference to the occurrence of cataracts. J. Hopkins Hosp. Bul. 75: 102-114.

Yudkin, A. M., 1933. Occular disturbances produced in experimental animals by dietary changes. J.A.M.A. 101 pt. 1: 921-926.

Zigman, S. and T. Vaughan, 1974. Near-ultraviolet light effects on the lenses and retinas of mice. Invest. Ophthal. 13: 462-465.

APPENDIX A

Table 5. The influence of light treatment on the induction of cataracts in Bobwhite quail to three months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	(Obs-Exp)^2/Exp C	(Obs-Exp)^2/Exp N
24 Hours	1	Infrared	6	43	2	47	8.00	0.34
	2	Fluorescent	1	41	2	40	0.50	0.02
	3	Incandescent	3	45	2	46	0.50	0.02
12 Hours	4	Infrared	0	37	2	35	2.00	0.11
	5	Fluorescent	0	30	1	29	1.00	0.03
	6	Incandescent	0	21	1	20	1.00	0.05
		totals	10	217	10	217	$\Sigma[(O-E)^2/E] = 13.57$*	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

* Significant, $p < 0.05$

Table 6. The influence of light treatment on the induction of cataracts in Bobwhite quail to four months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	$(Obs-Exp)^2/Exp$ C	$(Obs-Exp)^2/Exp$ N
24 Hours	1	Infrared	12	37	4	45	16.00	1.42
	2	Fluorescent	2	40	3	39	0.33	0.33
	3	Incandescent	3	42	4	41	0.23	0.02
12 Hours	4	Infrared	0	37	3	34	3.00	0.26
	5	Fluorescent	1	29	2	28	0.50	0.04
	6	Incandescent	0	21	2	19	2.00	0.21
		totals	18	206	18	206	$\Sigma[(O-E)^2/E] = 24.06$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 7. The influence of light treatment on the induction of cataracts in Bobwhite quail to five months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	$(Obs-Exp)^2/Exp$ C	$(Obs-Exp)^2/Exp$ N
24 Hours	1	Infrared	11	36	4	43	12.25	1.14
	2	Fluorescent	2	40	3	39	0.33	0.03
	3	Incandescent	3	40	3	40	0	0
12 Hours	4	Infrared	0	37	3	34	3.00	0.26
	5	Fluorescent	1	23	2	22	0.50	0.05
	6	Incandescent	0	21	2	19	2.00	0.21
		totals	17	197	17	197	$\Sigma[(O-E)^2/E] = 19.77$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 8. The influence of light treatment on the induction of cataracts in Bobwhite quail to six months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	$(Obs-Exp)^2/Exp$ C	$(Obs-Exp)^2/Exp$ N
24 Hours	1	Infrared	11	24	4	31	12.25	1.58
24 Hours	2	Fluorescent	3	36	5	34	0.80	0.12
24 Hours	3	Incandescent	7	32	5	34	0.80	0.12
12 Hours	4	Infrared	1	36	5	32	3.20	0.50
12 Hours	5	Fluorescent	1	11	2	10	0.50	0.10
12 Hours	6	Incandescent	0	21	3	18	3.00	0.50
		totals	23	160	24	159	$\Sigma[(O-E)^2/E] = 23.47$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 9. The influence of light treatment on the induction of cataracts in Bobwhite quail to seven months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	(Obs-Exp)^2/Exp C	(Obs-Exp)^2/Exp N
24 Hours	1	Infrared	11	24	4	31	12.25	1.58
	2	Fluorescent	1	34	4	31	2.25	0.29
	3	Incandescent	7	31	5	33	0.80	0.12
12 Hours	4	Infrared	1	35	4	32	2.25	0.28
	5	Fluorescent	1	10	1	10	0	0
	6	Incandescent	0	21	3	18	3.00	0.50
		totals	21	155	21	155	$\Sigma[(O-E)^2/E] = 23.32$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 10. The influence of light treatment on the induction of cataracts in Bobwhite quail to eight months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	$(Obs-Exp)^2/Exp$ C	$(Obs-Exp)^2/Exp$ N
24 Hours	1	Infrared	10	20	5	25	5.00	1.00
	2	Fluorescent	3	27	5	25	0.80	0.16
	3	Incandescent	8	29	6	31	0.67	0.13
12 Hours	4	Infrared	0	31	5	26	5.00	0.96
	5	Fluorescent	1	9	2	8	0.50	0.12
	6	Incandescent	2	19	3	18	0.33	0.06
		totals	24	135	26	133	$\Sigma\left[(O-E)^2/E\right] = 14.73$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 11. The influence of light treatment on the induction of cataracts in Bobwhite quail to nine months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	$(Obs-Exp)^2/Exp$ C	$(Obs-Exp)^2/Exp$ N
24 Hours	1	Infrared	11	19	5	25	7.20	1.44
	2	Fluorescent	2	24	4	22	1.00	0.18
	3	Incandescent	9	26	6	29	1.50	0.31
12 Hours	4	Infrared	0	29	5	24	5.00	1.04
	5	Fluorescent	1	6	1	6	0	0
	6	Incandescent	1	18	3	16	1.33	0.25
		totals	24	122	24	122	$\Sigma\left[(O-E)^2/E\right] = 19.25$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 19. The influence of light duration on the induction of cataracts in Bobwhite quail to eight months of age.+

	Observed		Expected		$(Obs-Exp)^2/Exp$	
Light Hours/Day	C	N	C	N	C	N
24	21	76	15	82	2.40	0.44
12	3	59	9	53	4.00	0.68
totals	24	135	24	135	$\Sigma\left[(O-E)^2/E\right] = 7.52$**	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

** Significant, $p < 0.01$

Table 33. The influence of light treatment on the induction of cataracts in Bobwhite quail to five months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	$(Obs-Exp)^2/Exp$ C	$(Obs-Exp)^2/Exp$ N
24 Hours	1	Infrared	11	40	5	46	7.20	0.78
	2	200 watt incandescent	7	49	5	51	0.80	0.08
	3	25 watt incandescent	9	43	5	47	3.20	0.34
12 Hours	4	Infrared	1	51	5	47	3.20	0.34
	5	200 watt incandescent	0	51	5	46	5.00	0.54
	6	25 watt incandescent	0	50	4	46	4.00	0.35
		totals	28	284	29	283	$\Sigma[(O-E)^2/E] = 25.83$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 13. The influence of light treatment on the induction of cataracts in Bobwhite quail to eleven months of age.+

Light Duration	Treatment	Type	Observed C	Observed N	Expected C	Expected N	$(Obs-Exp)^2/Exp$ C	$(Obs-Exp)^2/Exp$ N
24 Hours	1	Infrared	8	8	3	13	8.33	1.92
	2	Fluorescent	3	19	4	18	0.25	0.06
	3	Incandescent	10	13	5	18	5.00	1.39
12 Hours	4	Infrared	0	23	5	18	5.00	1.39
	5	Fluorescent	0	5	1	4	1.00	0.25
	6	Incandescent	0	17	3	14	3.00	0.64
		totals	21	85	21	85	$\Sigma[(O-E)^2/E] = 28.23$***	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

*** Significant, $p < 0.005$

Table 28. The influence of light type on the induction of cataracts in Bobwhite quail to eight months of age.+

	Observed		Expected		$(Obs-Exp)^2/Exp$	
Type	C	N	C	N	C	N
Infrared	10	51	9	52	0.11	0.02
Fluorescent	4	36	6	34	0.67	0.12
Incandescent	10	48	9	49	0.11	0.02
totals	24	135	24	135	$\Sigma\left[(O-E)^2/E\right] = 1.05$	

1. + = The results pertain to the number of surviving quail at this time.
2. C = Number of cataract-afflicted quail.
3. N = Number of cataract-free quail.

APPENDIX C

Table 41. The influence of light treatment on mortality of Bobwhite quail to eleven months of age.

Light Duration	Treatment	Type	Observed L	Observed D	Expected L	Expected D	(Obs-Exp)^2/Exp L	(Obs-Exp)^2/Exp D
24 Hours	1	Infrared	16	34	18	32	0.22	0.12
	2	Fluorescent	22	28	18	32	0.89	0.50
	3	Incandescent	23	27	18	32	0.39	0.78
12 Hours	4	Infrared	23	27	18	32	1.39	0.78
	5	Fluorescent	5	45	18	32	9.39	5.28
	6	Incandescent	17	33	18	32	0.06	0.03
		totals	106	194	108	192	$\Sigma[(O-E)^2/E] = 20.83$***	

1. L = Number of surviving quail.
2. D = Number of dead quail.

*** Significant, $p < 0.005$

Table 42. The influence of light duration on Bobwhite quail mortality to eleven months of age.

	Observed		Expected		$(Obs-Exp)^2/Exp$	
Light Hours/Day	L	D	L	D	L	D
24	61	89	53	97	1.21	0.66
12	45	105	53	97	1.21	0.66
totals	106	194	106	194	$\Sigma\left[(O-E)^2/E\right] = 3.74$	

1. L = Number of surviving quail.
2. D = Number of dead quail.

Table 43. The influence of light type on Bobwhite quail mortality to eleven months of age.

	Observed		Expected		$(Obs-Exp)^2/Exp$	
Type	L	D	L	D	L	D
Infrared	39	61	35	65	0.46	0.25
Fluorescent	27	73	35	65	1.83	0.98
Incandescent	40	60	35	65	0.71	0.38
totals	106	194	105	195	$\Sigma\left[(O-E)^2/E\right] = 4.61$	

1. L = Number of surviving quail.
2. D = Number of dead quail.

APPENDIX D

Table 44. The influence of light treatment on mortality of Bobwhite quail to thirteen months of age.

Light Duration	Treatment	Type	Observed L	Observed D	Expected L	Expected D	$(Obs-Exp)^2/Exp$ L	$(Obs-Exp)^2/Exp$ D
24 Hours	1	Infrared	51	2	52	1	0.02	1.00
	2	200 watt incandescent	54	1	54	1	0	0
	3	25 watt incandescent	50	1	50	1	0	0
12 Hours	4	Infrared	52	0	51	1	0.02	1.00
	5	200 watt incandescent	50	2	51	1	0.02	1.00
	6	25 watt incandescent	49	2	50	1	0.02	1.00
		totals	306	8	308	6	$\Sigma\left[(O-E)^2/E\right] = 4.08$	

1. L = Number of quail that survived.
2. D = Number of quail that died.

Table 45. Proportion of dead Bobwhite quail within light treatment.

Treatment	Proportion of dead		Arcsin transformation	Σ
24 Hours	No.	%		
Infrared	2/53	3.8	11.24	
Fluorescent	1/55	1.8	7.71	
Incandescent	1/51	2.0	8.13	27.08
12 Hours	No.	%		
Infrared	0	0	0	
Fluorescent	2/52	3.8	11.24	
Incandescent	2/51	3.9	11.39	22.63

Table 46. Analysis of variance for data summarized in table 45.[1]

Source	Degrees of Freedom	Mean Square	F-Value
Light duration	1	3.3	0.092
Light type	2	10.7	0.300
Error	2	35.71	

1. Calculated by arcsin transformation.

Table 47. Proportion of dead Bobwhite quail within treatment combinations.

Treatment combinations	Proportion of dead		Arcsin transformations	Σ
12-24*	No.	%		
Infrared	3/26	11.5	19.82	
200 watt incandescent	2/24	8.3	16.74	
25 watt incandescent	5/24	20.8	27.13	
				63.69
24-24				
Infrared	5/23	21.7	27.76	
200 watt incandescent	9/26	34.6	36.03	
25 watt incandescent	1/23	4.3	11.97	
				75.76
12-12				
Infrared	4/26	15.4	23.11	
200 watt incandescent	2/23	8.7	17.16	
25 watt incandescent	14/24	58.3	49.78	
				90.05
24-12				
Infrared	4/24	16.7	24.12	
200 watt incandescent	9/26	34.6	36.03	
25 watt incandescent	3/20	15.0	22.79	
				82.94

*The original treatment up to six months of age is indicated by the first number and the second number is the treatment for the following seven months in hours/day.

Table 48. Analysis of variance for data summarized in table 47.[1]

Source	Degrees of Freedom	Mean Square	F-Value
Treatment combination	3	42.15	0.246
Light type	2	18.38	0.107
Error	6	171.49	

1. Calculated by arcsin transformation.

APPENDIX E

Quail Diets

	MSU 72-15 Starter	MSU 72-16 Breeder
Protein--g/g diet	0.29	0.235
Metabolizable energy--kcal/g	3.02	2.88
(Prot/M.E.) x 100	9.60	8.15
Methionine--% of prot.	1.94	1.95
Ca--%	1.02	2.75
P, avail.--%	0.65	0.53
Fat--%	8.2	8.4
Fiber--%	3.2	3.1

Quail Diets

Ingredient	QS 72 Starter	QB 72 Breeder
	Pounds per ton	
Corn, #2 Yellow	767.0	900.4
Soybean Meal, 49%	846	654
Fish Meal	62	---
Meat Scrap, 50%	70	100
Alfalfa Meal, Dehy.	90	90
Animal Fat, Stabl.	112	114
Limestone	---	100
Dicalcium Phosphate	26	14
Choline Chloride, 50%	6	6
Methionine Hydroxy Analogue	2	2
Salt, Iodized	7	7.6
Mineral Mix A	6	6
Vitamin Mix A	6	6
Antioxidant (Ethoxyquin/ or BHT)	113.6 g	113.6 g