fairly constant. Specifically, *males*, in the United States[10] as well as in Britain[11], smoke consistently over the years *at least twice* as many cigarettes as females. This difference holds true for the general population for the period 1920–1958 in Britain and especially for the age group 25–45 for the years 1955 and 1960[12] in the United States. In that age group in 1955[10] 65% of the females and only 22% of the males are non-smokers. Moreover, in the same age group five years later[13] 50.1% of the females and again only 19.7% of the males are non-smokers. In addition in 1960 the percent of males in the age group 30–39 who inhale deeply when smoking cigarettes is 35.9 compared to 17.9 for females[14].

According to the report of the Royal College of Physicians[11], the chances of dying between 35 and 55 for heavy cigarette smokers are 3–4 times those of non-smokers. The excess of deaths are mostly from cancer of the lung, coronary heart-disease, and bronchitis, all of which are much commoner in men than women. Interestingly, when rates of cardio-vascular diseases are plotted against age on log-log scales, remarkably straight lines are obtained over the greater part of adult life[15], resembling the straight line of our Figure.

Independently, PLATT concludes that most, if not the whole, of the excess of male deaths could be accounted for by cigarette smoking[16]. We believe, therefore, that there is substantial evidence to favour an affirmative answer to the question: Could the log difference in our Figure be the result of excess male smoking, contributing to the excess male mortality. It appears that the significant sex difference in smoking habits results in a log difference in death rates which obscures the metabolic-rate-dependent difference in lifespan between the sexes.

Zusammenfassung. Es wird der Nachweis geführt, dass die durch höheren Stoffwechsel bedingte kürzere Lebensdauer der Männer durch Rauchen besonders in der Altersgruppe zwischen 25 und 55 zusätzlich verkürzt wird.

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The Effect of *o,p*-DDD in the Chicken

A single i.v. injection of 1,1-dichloro-2-(*o*-chlorophenyl)-2-(*p*-chlorophenyl) ethane (*o,p'*DDD) into a dog will inhibit adrenal corticosteroid production and glucose-6-phosphate dehydrogenase activity of the adrenal gland[1]. Attempts to inactivate the adrenal cortex by exposing embryos to *o,p'*DDD or by i.v. injection of chicks with this drug will be discussed in this paper. Also, the influence of *o,p'*DDD on antibody response of chicks will be presented.

*o,p'*DDD was dissolved in ethyl alcohol (EA) or a 1:2 dilution of EA and propylene glycol (PG). To the *o,p'*DDD-EA solution was added sufficient sesame oil to make final concentrations of 2.0, 8.0, 32.0, and 64.0 mg *o,p'*DDD/ml. A 5.3% stock solution of *o,p'*DDD was prepared for intravenous injection (i.v.) by adding the chemical to a 1:2 dilution of EA and PG[2]. The stock solution was diluted with lipomul, an oil-in-water emulsion, to yield 10 mg of *o,p'*DDD/ml.

At 12 h intervals, 12 3-week-old New Hampshire chickens received 3 i.v. injections of 10 mg *o,p'*DDD per injection while a different group of 12 birds received 3 i.v. injections of lipomul. 1 h after the last i.v. injection, 6 birds in each group received an i.m. injection of 8 IU ACTH per 100 g body weight and the remaining 6 received saline. A second group of birds received 2 i.v. injections of *o,p'*DDD or lipomul prior to an i.m. injection of ACTH. The right adrenals were removed 12 h after the i.m. injection of ACTH and analyzed for cholesterol[3]. The left adrenals were placed in Orth's fixative and then stained with Masson tri stain[4].

At 6 weeks of age normal birds received an i.v. injection of 10 mg *o,p'*DDD. Approximately 18 h after *o,p'*DDD administration, each bird received 40 mg of bovine serum albumin (BSA) per kg body weight. Birds were bled 7 days later and the serum antibody level to BSA determined[5].

Hatchability was significantly depressed by egg injections of *o,p'*DDD (Table I). Preliminary data suggested

Table I. Percentage hatchability of eggs injected with 0.1 ml of sesame oil containing varying amounts of *o,p*-DDD

No. eggs per group	No injection	Sesame oil	*o,p*-DDD, mg 0.2	0.8	3.2	6.4
50[a]	84	70	58[c]	54[c]	57[c]	
50[b]	78	63[c]				47[c]

[a] Injected in 1st day of incubation. [b] Injected on 8th day of incubation. [c] Significantly different ($P < 0.05$) from non-injected eggs.

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that dipping eggs in *o,p'*DDD solutions will significantly reduce hatchability[6]. At hatching there were no significant differences in body weight, or weights of the bursa, spleen, or adrenal glands. All birds hatched from eggs injected with *o,p'*DDD responded normally to later injections of ACTH by exhibiting significant increases in the number of absolute heterophils and a significant depletion of adrenal cholesterol. On the other hand, the adrenal cholesterol of birds injected i.m. with ACTH and previously injected 3 times with *o,p'*DDD was significantly higher than comparable controls (Table II). The adrenals of birds receiving ACTH and previously treated with lipomul exhibited extensive vacuolization of the fuchsinophilic cells of the adrenal cortex while the adrenals of birds pretreated with *o,p'*DDD before ACTH administration showed only occasional vacuoles in the fuchsinophilic cells of the adrenal cortex. Two i.v. injections of *o,p'*DDD did not significantly influence the chicken's response to ACTH as measured by adrenal cholesterol or adrenal histology. Intramuscular injections of 20 mg *o,p'*DDD for 16 days increased the level of steroids in the adrenal gland[7]. The adrenal cholesterol and histological data of this study demonstrate that adrenal cortical response to ACTH is significantly altered by 3 i.v. injections of *o,p'*DDD.

Table II. Micrograms of cholesterol per mg of adrenal tissue 12 h after an i.m. injection of 8 IU ACTH per 100 g body weight into birds pretreated with lipomul or *o,p*-DDD[a]

Saline		ACTH	
Lipomul + PG 1 ml-3X	*o,p*-DDD 10 mg-3X	*o,p*-DDD 10 mg-3X	Lipomul + PG 1 ml-3X
24.3	25.3	17.9	11.7
Lipomul + PG 1 ml-2X	*o,p*-DDD 10 mg-2X	*o,p*-DDD 10 mg-2X	Lipomul + PG 1 ml-2X
25.5	27.4	13.6	10.1

[a] 6 birds per mean.

All means not underlined by the same line are significantly different at the 5% level[8].

The serum antibody level to BSA of birds pretreated with 10 mg of *o,p'*DDD (58.8 μg antibody N/ml serum) was significantly less than birds pretreated with lipomul (100.6 μg antibody N/ml serum). A reduction in phagocytosis or interference with the immune response of immunologically competent cells might explain these results. All birds initially treated with an i.v. injection of *o,p'*DDD exhibited symptoms of anaphylactic shock that ranged from muscular weakness to convulsions. The lipomul birds did not exhibit these symptoms. The second and third i.v. injections of *o,p'*DDD were less effective in illiciting an anaphylactoid response. Apparently, *o,p'*DDD caused the release of histamine or some other pharmacological agent capable of producing anaphylactic shock[9,10].

Zusammenfassung. Die Ausbrütbarkeit von Hühnereiern wird durch die Injektion von *o,p*-DDD signifikant reduziert. Durch die Behandlung mit *o,p*-DDD wird die Nebennierenrindenfunktion der ausgeschlüpften Küken nicht beeinflusst. Verabreicht man 3 Wochen alten Küken 30 mg *o,p*-DDD, dann wird die Ansprechbarkeit der Nebennierenrinde auf ACTH signifikant verändert. Eine Vorbehandlung der Küken mit 10 mg *o,p*-DDD reduziert die Bildung der Antikörper im Serum gegenüber Rinderserumalbumin.

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A Contribution to the Temperature Sensitivity of Lorenzinian Ampullae of Elasmobranchs

Ampullae of Lorenzini of Elasmobranchs are bulb-like and jelly-filled sense organs which are located under the skin of the head. They are connected with the skin surface by thin, jelly-filled tubes, sometimes several centimetres long. The biological function of these sense organs remains uncertain. They are sensitive to thermal[1-3], mechanical[4-6], chemical[7,8], and electrical[8-10] stimulation. All these types of stimulation may be active under normal biological conditions. Behavioural studies[11] seem to indicate that the Ampullae of Lorenzini are used for the perception of electrical signals. The location of the Ampullae might also be of importance for their function[10].

Hitherto, in all investigations, the temperature sensitivity of the Ampullae of Lorenzini has been described as being that of the cold-receptors of homoiotherms: cooling

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